EDITORIAL COMMENT

Should ICDs Be Implanted in All Patients With Dilated Cardiomyopathy and Unexplained Syncope?*

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Syncope is a frequent symptom, but when it occurs in the presence of organic heart disease and there is no obvious explanation, it carries with it a high incidence of subsequent sudden death. Several investigators have demonstrated that unexplained syncope in patients with dilated cardiomyopathy, particularly with clinical congestive heart failure (CHF), is a predictor of overall mortality and sudden death (1–3). The risk of sudden death maybe as high as 45% regardless of the cause of syncope. Although electrophysiologic testing has been useful to risk stratify patients with “ischemic” cardiomyopathy (20), it has been less useful in patients with asymptomatic dilated idiopathic cardiomyopathy (3–6,11). Only two prior studies have evaluated the outcome of patients with dilated cardiomyopathies and syncope treated with implantable defibrillators (ICDs). In the study of Fazio et al. (21), syncope had already been documented to be ventricular tachycardia, and most patients had inducible ventricular arrhythmias at the electrophysiologic study (EPS). In the study of Grimm et al. (22) only six ICD patients had dilated cardiomyopathies, and these patients had spontaneous nonsustained ventricular tachycardia.

In this issue of the Journal of the American College of Cardiology, Knight (23) presents a prospective study comparing patients with unexplained syncope and idiopathic dilated cardiomyopathy treated with devices versus a comparable group of 19 patients with prior cardiac arrests in whom devices had been implanted. The patients were comparably matched, although the arrest group had a slightly increased use of beta-adrenergic blocking agents and amiodarone therapy than the syncope group, neither of which were statistically significant. The basic message of this article is that patients with syncope had a comparable number of appropriate shocks than those patients who presented with cardiac arrest, and in those patients who had appropriate shocks, the time for the first shock was shorter (10 ± 14) in the syncope group than in the arrest group (48 ± 47 months; p = 0.06). This borderline significance may be due to either the low number of patients included in this study or the slightly increased use of amiodarone and beta-blockers in the arrest group. Of note is that the rate of the arrhythmia for which the first shock was delivered was comparable. It is significant that no patients in the syncope group with an ejection fraction greater than 35% received an inappropriate shock; however, only 3 of the 14 patients had ejection fractions of greater than 35%. All patients who had class III functional New York Heart Association status experienced appropriate shocks, but one patient with class I symptoms also received an appropriate shock. Thus, although it appears that people with more severe clinical CHF are likely to receive appropriate shocks, the small number of patients in this study (with the overlap noted) limit the usefulness of clinical characteristics as predictors of those patients who are likely to receive appropriate shocks.

These data might suggest that implantation of ICDs is appropriate for those patients who have syncope of indeterminate cause and in patients with idiopathic dilated cardiomyopathy. This seems rational because most of the patients with syncopal episodes had documented ventricular arrhythmias that the device appropriately and successfully treated. Moreover, on the basis of the results of the AVID trial (24), devices appear to have a greater benefit and overall survival than drugs in patients with arrhythmias. However, several words of caution are applicable before widespread adoption of this philosophy. First is that this study was small, including only 14 patients with unexplained syncope. Second and most important is that this study was not conducted to see whether the device improved overall mortality. Although it is true that the ICD delivered appropriate shocks and probably saved the lives of those people in whom it delivered appropriate shocks, there was still a 28% mortality in an average two year follow-up. To recommend ICDs as initial therapy for unexplained syncope in dilated cardiomyopathy, one would need to demonstrate that it was more cost effective than antiarrhythmic drugs with or without pacemakers in this group of people. Furthermore, it should be noted that ICDs are not necessarily associated with improved quality of life. Fifty percent of the patients in the syncope group received inappropriate shocks, many of which were due to atrial fibrillation. In view of the fact that amiodarone is now being widely used in patients with idiopathic dilated cardiomyopathy as a means to improve survival, particularly those with atrial fibrillation, a prospective study will be necessary to assess whether or not ICD implantation is a preferred method of treatment. In this cost-conscious environment, this will be mandatory.

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Other issues should be addressed. First is the definition of unexplained syncope. What may be unexplained in some physicians’ minds may be obvious in other physicians’ minds. This depends on how careful a history is taken. Potential abuses of ICDs will occur if reversible or treatable causes are not sought. For example, drugs are a very frequent cause of syncope. Use of angiotensin-converting enzyme inhibitors and diuretics are frequently used in patients with cardiomyopathy, and they may be important causes of syncope. Neurocardiac syncope can also occur in patients with cardiomyopathies, particularly if they are dehydrated. The classic symptoms of nausea, flushing and weakness are often not present. Bradycardic events must also be considered in those patients who have conduction disturbances. Although conduction disturbances predispose to malignant ventricular arrhythmias, they also may result in high-degree atrioventricular (AV) block particularly when antiarrhythmic drugs are used. The same is true for sinus node dysfunction. Amiodarone is certainly a drug that can produce both AV block and sinus node dysfunction. Although I am sure the authors excluded all of these causes, the detail with which such causes are sought may not be comparable in other settings and in studies conducted by other physicians.

Secondly, newer ICDs with dual chamber pacing (some with defibrillation capabilities) are being evaluated, even dual chamber defibrillation. These devices can provide important physiologic anti-bradycardia pacing and decreasing incidents of inappropriate shocks. In addition, there is a great interest in the use of pacing to synchronize ventricular activation to improve heart failure. Several companies are evaluating left ventricular pacing in patients with interventricular conduction defects as a means to improve left ventricular function. If these devices can be coupled with the dual chamber defibrillator concept, one would have a device that can improve ventricular function, convert ventricular fibrillation or atrial fibrillation when present, provide physiologic rate responsive pacing and allow for the safe use of antiarrhythmic agents. I am sure trials will soon be underway evaluating the advances in technologies in patients with heart failure, particularly those with dilated cardiomyopathy. Although much enthusiasm abounds in developing these technologies, this should all be taken in context of the recognition that once the heart reaches a certain level of dysfunction, the only alternative to improve survival is cardiac transplantation.

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REFERENCES


