

Cardioverter-Defibrillator Implantation to Safeguard Against Fatal Arrhythmias in Cardiomyoplasty Patients

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Abstract

A potentially low-risk surgery to improve hemodynamics and functional class in heart failure patients and others with advanced ventricular dysfunction, dynamic cardiomyoplasty (CMP) has been performed over 1,000 times worldwide. During long-term follow-up, arrhythmic sudden cardiac death remains a major cause of death with any advanced medical management (i.e., a 15%–50% risk of recurrent cardiac arrest despite drug suppression of inducible arrhythmia, including amiodarone).

Combining our research expertise and clinical capabilities in electrophysiology with the technological expertise of Medtronic, Inc., a newly devised protocol for combined cardioverter-defibrillator implantation (ICD) and CMP was created from Medtronic protocols for cardiomyoplasty, ICD implantation, and prevention of “cross-talk” (i.e., adverse interaction due to over-sensing signals emitted by another implanted device).

In this largest published series of patients who have undergone CMP with concomitant ICD implantation (5 patients at the Milwaukee Heart Institute), we describe measures to prevent cross-talk and categorize cases according to timing of ICD implantation: 1) soon after CMP, 2) several months after CMP (in patients who develop ventricular tachycardia), or 3) considerably before CMP. We have not yet experienced a fourth scenario, i.e., simultaneous ICD implantation and CMP; however, we believe that we have established the groundwork for this eventuality.

Key words: aged skeletal muscle, cardiomyoplasty, contractile force, electrical stimulation.

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In the United States alone, approximately 2-3 million patients suffer from heart failure - a number likely to increase [35, 18]. Most of these patients manifest frequent and complex ventricular ectopy, and the frequency and severity of the ventricular arrhythmia worsens with advancing ventricular dysfunction [18]. Their chances for survival are limited not only by progressive pump dysfunction, but also by the risk of sudden cardiac death - in those with class IV symptoms, the rate of one-year mortality exceeds 50%.

Recent advances in the medical management of heart failure have significantly ameliorated symptoms, improved functional class, and prolonged survival [10, 11, 13, 15, 38]; however, the risk of sudden death remains considerable - 28%-68% [15, 18, 20]. Dynamic cardiomyoplasty as surgical therapy for patients with advanced ventricular dysfunction has been performed worldwide in over 1,000 patients with generally rewarding results [22].

While dynamic cardiomyoplasty has potential as low-risk surgery to improve hemodynamics and functional class, it is limited by a progressive decrease in latissimus dorsi muscle power and by arrhythmic sudden cardiac death [25]. Sudden cardiac death remains a major cause of attrition in the long-term survival of patients who have undergone cardiomyoplasty [27]. Of the various available therapeutic strategies to safeguard patients against sudden death, the implantable cardioverter defibrillator (ICD) has been shown to be the most effective [3]. Adjunctive use of the ICD may, therefore, provide an optimal outcome for patients who undergo cardiomyoplasty [39].

Materials and Methods

The first clinical CMP in Milwaukee was performed on November 7, 1995. To date, 7 patients have undergone the procedure; in our report, the study group consists of the 5 patients who had combined CMP and ICD implantation. All patients were chosen for CMP according to

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the indications described by Carpentier and Chachques [6].

All patients deemed to be in need of ICD implantation - either before or after CMP - were in the pre-end stage of congestive heart failure: average left ventricular ejection fraction (LVEF) $16\pm3\%$; right ventricular ejection fraction (RVEF) $29\pm4\%$; left ventricular end-diastolic volume (LVEDV) 381 ± 63 ml; left ventricular end-systolic volume (LVESV) 309 ± 57 ml; and peak VO_2 15.1 ± 4 ml/min/m².

Medtronic Inc., protocols for cardiomyoplasty, for ICD implantation, and for evaluation to insure against adverse interaction between devices were followed in each case reported here. (This interaction due to over-sensing signals emitted by another implanted device is termed "cross-talk;" measures to prevent it are described in some detail in Case 1, below.)

The usual operative technique and protocol for CMP described previously were used [6, 26, 29]. In all cases, the left latissimus dorsi muscle was mobilized and wrapped posteriorly around the heart. A protocol for progressive muscle conditioning is then followed that uses an implanted Model 4710 cardiomyostimulator (Medtronic Inc., Minneapolis, MN) device for a 1:2 stimulation mode. Skeletal muscle flap stimulation starts 2 weeks post-operatively.

All 5 patients were evaluated 6 months after CMP and one year after CMP; 4 patients were evaluated at 1.5 years after CMP. NYHA functional class in 4 patients improved considerably (from 3.5 to 2.2) and remained the same in one. Hemodynamic indices showed an increase in LVEF from $16\pm3\%$ to $18\pm3\%$ one year post CMP and to $19\pm4\%$ a year and half after CMP. RVEF increased from $29\pm4\%$ to $33\pm9\%$ one year post CMP and to $32\pm7\%$ a year and half after CMP. LVEDV and LVESV both decreased: LVEDV from 381 ± 63 ml to 342 ± 51 ml one year post CMP and to 320 ± 56 ml a year and half after CMP, and LVESV from 309 ± 51 ml to 279 ± 42 ml one year post CMP and to 257 ± 37 ml a year and half after CMP. Despite this tendency for improvement in hemodynamics, the differences between the pre- and post-operative data

In short, the hemodynamic status of these patients before CMP was comparable to that considered "borderline" when indications and contraindications for CMP are weighed before the operation, and these results made the prospect of any tangible hemodynamic benefit from the operation very doubtful. On the plus side, the operation served to keep their hemodynamic status from deteriorating at one year (and one and a half years) post-operatively, even though it only remained stable. With the added safeguard against fatal arrhythmia provided by the ICD, in addition to stable hemodynamics, the benefits of the combined CMP-ICD were enough to justify the risk, especially considering that with this protection against sudden cardiac death comes the assurance of having the time needed for progressive latissimus dorsi muscle re-training.

Results

Scenario One: ICD implantation shortly after cardiomyoplasty

Case 1

A 65-year-old retired physician with advanced ventricular dysfunction related to coronary artery disease (CAD) was evaluated for cardiomyoplasty. The patient was NYHA functional class III and on optimal medical therapy. In 1991, he had undergone percutaneous transluminal coronary angioplasty (PTCA) for relief of his angina. In the months just prior to evaluation, he had experienced two episodes of unexplained syncope and suffered an out-of-hospital cardiac arrest, from which he had been successfully resuscitated. Monomorphic ventricular tachycardia (VT) was documented as the initial rhythm. After complete neurological recovery, he underwent further evaluation at the Milwaukee Heart Institute.

Cardiac hemodynamic evaluation revealed evidence of pulmonary hypertension: increased right pulmonary arterial pressure (58/25 mm Hg, mean 36 mm Hg); elevated pulmonary capillary wedge pressure, indicated by both

Table 1. Hemodynamic Changes After Combined CMP-ICD.

Hemodynamic Index	Before CMP n = 5	6 months later n = 5	1 year later n = 5	1.5 years later n = 4
LVEDV (ml)	381±63	361±40	342±51	320±56
LVESV (ml)	309±51	323±61	279±42	257±37
LVEF (%)	16±3	18±5	18±3	19±4
RVEF (%)	29±4	33±9	35±7	32±7
Peak VO_2 (ml/min/m ²)	15.1±4	16.3±5	16.1±4	17.2±3

LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; RVEF, right ventricular ejection fraction; Peak VO_2 , peak oxygen volume.

were statistically insignificant ($p > 0.05$) (Table 1).

A-wave (28 mm Hg) and V-wave (42 mm Hg) measure-

ments (mean 32 mm Hg); and a reduced cardiac index (2.12 L/min/m²). Coronary angiography showed severe native three-vessel CAD. The left anterior descending artery had moderate-to-severe proximal and mid-level disease; the circumflex had severe proximal disease; and the right coronary artery had diffuse moderate disease in its entirety and was occluded. The left ventricle was severely hypocontractile with an ejection fraction of 10%, LVEDV of 473 ml and LVESV of 392 ml. During electrophysiologic evaluation, there was easily inducible, sustained monomorphic VT of at least three morphologies, which was terminated by cardioversion. The patient was started on oral amiodarone loading to suppress VT.

Because this patient was free of end-stage heart failure, cardiomyoplasty was chosen over heart transplantation as a treatment option and a posterior wrap was performed through a medial sternotomy. A Medtronic cardiomyostimulator was implanted, and myocardial and skeletal muscle electrodes were placed in the conventional manner. The operation was completed without complications. Following satisfactory post-operative recovery, a repeat electrophysiologic study was performed to evaluate the efficacy of amiodarone therapy in which it was found that monomorphic VT of two morphologies, both hemodynamically compromising, remained persistently inducible.

The patient's history of advanced ventricular dysfunction, prior cardiac arrest, and electrophysiologic results indicating failed suppressive drug therapy, all contributed to the decision to proceed with implantation of an ICD for VT management and sudden cardiac death prophylaxis.

ICD implantation was performed 2 weeks after cardiomyoplasty and utilized a lead system consisting of a Medtronic Transvene® right ventricular transvenous defibrillation/rate sensing electrode and a second defibrillation electrode in the SVC/innominate junction, along with a subcutaneous patch placed along the left chest wall. A Medtronic pulse generator (Model 7219D) was used in conjunction with this lead system. Defibrillation testing was performed using step-down energies until a defibrillation threshold of 24 joules with a biphasic waveform was obtained. During defibrillation testing, the cardiomyostimulator was temporarily programmed to deliver 6 pulses at maximum amplitude to detect any interaction, or "cross-talk," between the cardiomyostimulator device and the ICD. No interaction was noted and the ICD appropriately detected all VF episodes, and the cardiomyostimulator was then reprogrammed to original settings. The patient had an excellent postoperative recovery.

Three days later, a follow-up electrophysiologic evaluation of the ICD and lead system was performed, specifically: (1) thorough re-evaluation of the potential for cross-talk between the cardiomyostimulator and the ICD and (2) defibrillation threshold testing to ensure an

adequate safety margin. The cardiomyostimulator was again programmed to deliver 6 pulses at maximum amplitude and pulse width during this testing; the ICD was programmed for maximum sensitivity to determine whether the ICD sensed signals from the cardiomyostimulator. No inappropriate sensing was observed, and satisfactory defibrillation was obtained at 24 joules in a biphasic waveform. The cardiomyostimulator was then reprogrammed to original settings, and the ICD was left active. Amiodarone was continued at a lower maintenance dose of 200 mg qd. At this point, the standard Medtronic cardiomyoplasty electrical stimulation protocol was started.

The final phase of training was reached 70 days after the initial cardiomyoplasty operation. The number of pulses was incrementally advanced to 6 and the pulse interval was set at 31 msec. During each follow-up visit, cross-talk between the cardiomyostimulator and ICD was evaluated by programming the cardiomyostimulator at maximal output and pulse width and setting the ICD sensitivity at maximal gain. No interaction was observed with these maneuvers.

Over the next 42 months while on amiodarone, the patient had 65 episodes of rapid VT, which required 11 shocks to save him from cardiac death. Interrogation and analysis of the stored electrograms revealed no cross-talk between the cardiomyostimulator and the ICD during these clinical events; VT was appropriately detected in each case by the ICD without over-sensing signals emitted by the cardiomyostimulator. The patient has reported continued subjective improvement in functional class. His hemodynamics have not improved significantly with the most recent levels as follows: LVEF 18%, RVEF 35%, LVEDV 411 ml, and LVESV 368 ml. However, it has been encouraging to find no deterioration in hemodynamic status 42 months after CMP.

Case 2

A 75-year-old patient with a history of coronary artery bypass grafting (1990) and percutaneous transluminal angioplasty (1996) developed advanced ischemic cardiomyopathy and was NYHA functional class III. Cardiac hemodynamic evaluation at our center in 1997 revealed evidence of severe left ventricular dysfunction: LVEF 15%, LVEDV 320 ml, LVESV 260 ml, and a peak VO₂ of 12 ml/min/m². Twenty-four hour Holter monitoring showed 146 premature supraventricular beats per min and 6 runs of supraventricular tachycardia.

Cardiomyoplasty was performed using a posterior muscle wrap through a medial sternotomy. A Medtronic cardiomyostimulator was implanted, and myocardial and skeletal muscle electrodes were placed in the conventional manner. The operation was completed without complications, but two weeks postoperatively, several runs of nonsustained VT were noted. During electrophysiologic evaluation, there was easily inducible, sus-

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tained monomorphic VT which was terminated by cardioversion.

ICD implantation was performed 3 weeks after cardiomyoplasty and utilized a non-thoracotomy lead system. The patient had 3 episodes of rapid VT in the 14 months after cardiomyoplasty, which required 2 shocks. Interrogation and analysis of the stored electrograms revealed no cross-talk between the cardiomyostimulator and the ICD during these clinical events. The patient has subjective improvement in functional class. His LVEF increased to 19%, LVEDV decreased to 287 ml, and LVESV decreased to 215 ml.

Scenario Two: ICD implantation several months after cardiomyoplasty

Case 3

A 44-year-old patient with CAD, prior PTCA, ischemic cardiomyopathy, and severe left ventricular dysfunction was evaluated for cardiomyoplasty at our center. The patient denied any history of syncope, but hemodynamic evaluation revealed significant left ventricular failure: LVEDV 527 ml, LVESV 430 ml, but LVEF 25% and peak VO_2 of 22 L/min/m². Cardiomyoplasty was performed without complications. Three months postoperatively, Holter monitoring showed nonsustained monomorphic VT and appropriate cardiomyostimulator activity.

Although this patient continued to be asymptomatic for ventricular ectopy (i.e., no arrhythmic episodes in the 3 months prior to the decision to implant an ICD), electrophysiologic studies were performed for risk stratification, during which procainamide infusion induced sustained monomorphic VT that was severely hemodynamically compromising, indicating that this patient was at high risk for VT and subsequent sudden cardiac death.

ICD implantation was performed 3 months after cardiomyoplasty. One year after cardiomyoplasty, LVEF had fallen to 15% and clinical events related to CAD continued unabated. Fifteen months after cardiomyoplasty, the patient underwent 4 successful PTCA's and placement of two stents. Two and a half years after CMP, his LVEF remains the same as before CMP, but his RVEF has increased to 34%; his LVEDV has decreased to 440 ml, and his LVESV has decreased to 380ml. During the first 15 months postoperatively, he had no episodes of VT; however, during the next 9 months, he had 6 episodes, which required 5 shocks, confirming the soundness of our strategy of implanting an ICD in this patient who was at high risk for sudden cardiac death.

Follow-up interrogation and analysis of the stored electrograms revealed no cross-talk between the cardiomyostimulator and the ICD.

Case 4

A 72-year-old patient with coronary artery disease and congestive heart failure had a pacemaker implanted for complete heart block. His hemodynamics prior to the decision to perform cardiomyoplasty were very poor: LVEF 10%, RVEF 40%, LVEDV 290 ml, LVESV 238 ml, and peak VO_2 19.0 L/min/m². In addition, he had one episode of nonsustained supraventricular tachycardia.

Cardiomyoplasty was performed 5 years after pacemaker implantation. During postoperative recovery, he was free of complications, but one and a half years later (6.5 years after pacemaker implantation), the patient was admitted to the hospital for generator explantation and removal of the lead system when erosion was discovered in the pacemaker pocket. During evaluation in the electrophysiology laboratory for pacemaker reimplantation in the other side of the chest, the patient developed spontaneously polymorphic ventricular tachycardia which degenerated to ventricular fibrillation and needed a 360 joule shock for cardioversion.

A new permanent dual chamber pacemaker and an ICD were implanted; this patient had one episode of ventricular tachycardia during the next 5 months (converted to sinus rhythm without the need for any shock). No cross-talk was noted between any of these three devices, i.e., the cardiomyostimulator, the pacemaker, and the ICD. The patient has reported continuing subjective improvement in functional class. Hemodynamic improvement has been encouraging: LVEF has improved from 10% to 18% although RVEF has remained the same at 40%; LVEDV has decreased from 290 ml to 250 ml and LVESV from 238 ml to 194 ml.

Scenario Three: cardiomyoplasty after ICD implantation

Case 5

A 38-year-old patient with advanced dilated cardiomyopathy and history of VT underwent electrophysiology studies. A variety of medications was used (Norpace, mexiletine, propafenone, sotalol) and each study was persistently positive for inducible sustained monomorphic VT. An ICD was implanted using the non-thoracotomy lead system.

Two years later, the patient had an episode of atrial fibrillation with a controlled ventricular response and occasional ventricular pacing (40 beats per min) that was successfully treated with cardioversion; cardioversion was also required one week later. A permanent dual chamber pacemaker was therefore implanted to treat sinus bradyarrhythmia associated with symptomatic AV block. No interaction was noted between the ICD and the pacemaker, and during the next 5 years, there was no evidence of any spontaneous ICD therapy. Noninvasive evaluation of the ICD revealed normal functioning with minimum defibrillation thresholds (28 joules with reverse

lead polarity) along with demonstration of normal bradycardiac pacing function.

Prior to the decision to perform cardiomyoplasty, the patient developed severe congestive heart failure: LVEF 20%, RVEF 20%, LVED 310 ml, LVESV 257 ml, and a peak VO_2 of 17 L/min/m².

Cardiomyoplasty was performed 5 years after ICD implantation. During in-hospital recovery, 4 ICD discharges were recorded as therapy for one episode of VT. Testing showed appropriate functioning of the pacemaker, the ICD, and the cardiomyostimulator. During recovery, this patient has had 2 additional ICD discharges, but again, there has been no adverse interaction between the 3 devices.

Unfortunately, one year after CMP, the patient's condition had not improved: LVEF (22%) and RVEF (20%) were the same as before CMP, and peak VO_2 had decreased considerably (from 16.9 ml/min/m² to 14.2 ml/min/m²). During this period, there were 14 episodes of VT, which required 14 shocks. Fourteen months after CMP, successful heart transplantation was performed.

Discussion

As noted earlier, the principal mechanism of sudden cardiac death in patients with heart failure is arrhythmia - most commonly, VT degenerating into VF [24, 41, 42]. Bradyarrhythmic cardiac arrest and electromechanical dissociation are also well described and may be more common in idiopathic dilated cardiomyopathy [24]. VT usually develops due to reentry and may be modulated by a variety of triggers (e.g., changes in myocardial wall stress from adjustments to preload or afterload, myocardial ischemia, fluctuating neurohormonal tone, electrolyte abnormalities, proarrhythmic effect of antiarrhythmic drugs, and others) [8, 12, 17, 32, 33].

It has also been postulated that placing the paced skeletal muscle onto a diseased cardiac muscle can create substrates for reentry due to scar and fibrous tissue development and that mechanical compression and stress may further contribute to proarrhythmia in cardiomyoplasty patients.

Worldwide, 4 groups have published long-term results of clinical dynamic cardiomyoplasty, each reporting arrhythmic sudden cardiac death as the major cause for late mortality. In the series at Allegheny General Hospital in Pittsburgh, 9 of 38 patients died of arrhythmia [25]; at the Heart Institute in São Paulo, Brazil, 6 of 33 died suddenly [29]. All patients who died suddenly were on antiarrhythmic therapy for ventricular arrhythmias or atrial fibrillation. Data from Hospital Broussais in Paris confirmed sudden death as the cause of mortality in 3 of 52 patients [6]. The Bakulev Institute for Cardiovascular Surgery in Moscow reported one death from VT and one from sudden death out of 25 total patients [9]. Combining these reports therefore yields an overall incidence of

20/148 (13.5%) for sudden death related to VT following dynamic cardiomyoplasty.

In 1996, Magovern [38] combined data from the United States and Canada and reported that 19 of 57 patients died in the early and late stages following dynamic cardiomyoplasty; in 12 of these 19 (63%), the cause was suspected arrhythmia. In the same year, Moreira [30], reporting on combined data from South America (112 cardiomyoplasties), found that 62% of late deaths after cardiomyoplasty were related to progression of heart failure; sudden cardiac death accounted for 38% of these deaths. Obviously these reports are only roughly comparable, given differences in patient populations, surgical techniques, and concomitant therapies. Van den Berg et al. [40] reported that 3 of 4 patients died suddenly without one year after cardiomyoplasty, probably due to ventricular arrhythmias.

Still, it is clear that patients with advanced ventricular dysfunction who undergo dynamic cardiomyoplasty are at high risk for sudden cardiac death. Since improvements in hemodynamic status and functional class by any intervention do nothing to ameliorate this risk, both primary and secondary prevention of sudden cardiac death need to be addressed.

Used either in empirical therapy or in therapy guided by risk stratification, beta blockers, angiotensin inhibitors, hydralazine-nitrates, and amiodarone have all shown benefit for primary prevention of sudden cardiac death [11, 13, 15, 20, 38]. Unfortunately, their efficacy in advanced left ventricular dysfunction is so variable that they cannot be relied on for this population.

For example, a large randomized controlled study in the United States showed that amiodarone did not help to reduce sudden death mortality in patients with heart failure, although it was successful in the subset of patients with *nonischemic* cardiomyopathy [37]. In contrast, the GESICA trial showed that amiodarone extended survival, but the study did not separately analyze patients with and without ischemic heart disease [14].

Since it is known that at least 80% of cardiac arrest victims do not survive to hospital discharge [34], it is not surprising that, in patients with advanced left ventricular dysfunction (as in Case 1), sudden cardiac death is not greatly reduced by preventing recurrent cardiac arrest. In addition, despite the availability of resources for cardiopulmonary resuscitation in many communities, there is still significant out-of-hospital attrition due to cardiac arrest.

Small and large ongoing and completed trials have demonstrated important limitations to anti-arrhythmic drug therapy, including amiodarone, when prescribed empirically and guided by electrophysiologic testing [7, 28, 36]. Advanced left ventricular dysfunction (i.e., high risk for sudden cardiac death) is identified by risk factor assessment and the following indices for risk stratification: left ventricular systolic function, signal-averaged

electrocardiography, heart rate variability, ambulatory Holter monitoring, and programmed ventricular stimulation [2]. No single factor is predictive, but combining these data usually provides a good clinical perspective [2].

Unfortunately, due to limitations discussed elsewhere, these screening techniques are flawed and must be applied with caution. In patients with idiopathic cardiomyopathy, there is no clear association between functional class or age and a patient's risk of dying suddenly [19]. Standard risk factors do little to help discriminate between those who are and are not at risk for arrhythmic or nonarrhythmic death.

The ICD stands out by offering the best chance for sudden death risk reduction in this population [3], and it has been used successfully as a bridge to cardiac transplantation [5, 21]. The ICD has been reported to cut sudden cardiac death rates at five-year follow-up to about 4.5% [1, 31, 43]. It appears to be the best safeguard against arrhythmic death in dynamic cardiomyoplasty patients.

As the above case reports illustrate, the timing of ICD implantation in relationship to dynamic cardiomyoplasty cannot be standardized. Cardiomyoplasty may be performed shortly after or long after patients have had ICD implantation; it may be performed concomitantly with ICD implantation [37], or ICD implantation may be performed at some time after cardiomyoplasty surgery. In some patients, neither VT nor cardiac arrest has recurred following cardiomyoplasty - perhaps as a result of improved hemodynamics, ventricular stretch, or corrected autonomic imbalance. In these patients, there is no need to defer ICD implantation, since the "long-term anti-arrhythmic effect" of cardiomyoplasty cannot be predicted.

At present, information is limited about the potential interactions between the ICD and the cardiomyoplasty system [16], but care taken to insure against such interaction during implantation of a second device should yield long-term follow-up results similar to those when an ICD and pacemaker implantation are combined [18].

In conclusion, while dynamic cardiomyoplasty may help improve hemodynamic status and functional class in patients with advanced ventricular dysfunction, the risk of sudden death remains a vexing problem. For reducing their risk of arrhythmic death, anti-arrhythmic drug therapy alone has several limitations, but a favorable impact on the survival of these patients can be anticipated when the ICD is utilized in conjunction with dynamic cardiomyoplasty to buy them the needed time to complete skeletal muscle training. In our 5 patients, there have been 87 episodes of ventricular tachycardia, which have required 33 DC shocks for cardioversion.

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References

- [1] Akhtar M, Avitall B, Jazayeri M, *et al*: Role of implantable cardioverter defibrillator therapy in the management of high-risk patients. *Circulation* 1992; 85 (suppl I): I-131.
- [2] Akhtar M: Sudden cardiac death: Management of high-risk patients. *Ann Intern Med* 1991; 114: 499-512.
- [3] Axtell K, Tchou P, Akhtar M: Survival in patients with depressed left ventricular function treated by implantable cardioverter-defibrillator. *PACE* 1991; 14: 291-296.
- [4] Blanck Z, Niazi I, Axtell K, *et al*: Feasibility of concomitant implantation of permanent transvenous pacemaker and defibrillator systems. *Am J Cardiol* 1994; 74: 1249-1253.
- [5] Bolling SF, Deeb GM, Morady F, *et al*: Automatic internal cardioverter defibrillator: A bridge to heart transplantation. *J Heart Lung Transplant* 1991; 10: 562-566.
- [6] Carpentier A, Chachques JC, Acar C: Dynamic cardiomyoplasty at seven years. *J Thorac Cardiovasc Surg* 1993; 106: 42-54.
- [7] CASCADE Investigators: Randomized antiarrhythmic drug therapy in survivors of cardiac arrest (The CASCADE Study). *Am J Cardiol* 1993; 72: 280-287.
- [8] Castaigne A: Editorial overview. *Am J Cardiol* 1990; 65: 39K-40K.
- [9] Chekanov VS, Krakovsky AA, Buslenko NS, *et al*: Cardiomyoplasty: Review of early and late results. *Vasc Surg* 1994; 28 (7): 481-489.
- [10] Cohn JN, Archibald DG, Ziesche S, *et al*: Effect of vasodilator therapy on mortality in chronic congestive heart failure: Results of a Veterans Administration Cooperative Study. *N Engl J Med* 1986; 314: 1547-1552.
- [11] Cohn JN, Johnson G, Ziesche S, *et al*: A comparison of enalapril on mortality and the development of heart failure in symptomatic patients with reduced left ventricular ejection fraction. *N Engl J Med* 1992; 327: 685-691.

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- [12] Cohn JN, Levine TB, Olivari MT: Plasma norepinephrine as a guide to prognosis in patients with congestive heart failure. *N Engl J Med* 1984; 311: 819-823.
- [13] CONSENSUS Trial Study Group: Effects of enalapril on mortality in severe congestive heart failure: Results of the Cooperative North Scandinavian Enalapril Study (CONSENSUS). *N Engl J Med* 1987; 316: 1429-1435.
- [14] Doval HC, Nul DR, Grancelli HO, *et al*: Randomized trial of low-dose amiodarone in severe congestive heart failure. *Lancet* 1994; 344: 4933-4998.
- [15] Fonorow GC, Chelimsky-Fallick C, Stevenson LW, *et al*: Effect of direct vasodilation vs. angiotensin-converting enzyme inhibition on mortality in advanced heart failure: The Hy-C trial. *J Am Coll Cardiol* 1992; 19: 842-850.
- [16] Francischelli D, Peterson D, Stein PM, Gealow K, Steuble CD, Grandjean P: Combined cardiomyoplasty and antitachyarrhythmia systems. *PACE* 1995; 18 (4): 11;800 [abstract].
- [17] Garan H, McGovern BA, Canzanella VJ, *et al*: The effect of potassium ion depletion on postinfarction canine cardiac arrhythmias. *Circulation* 1988; 77: 696-704.
- [18] Greene HL: Clinical significance and management of arrhythmias in heart failure patients. *Clin Cardiol* 1992; 15 (suppl I): I-13-I-21.
- [19] Hofmann T, Melnert T, Kasper W, *et al*: Mode of death in idiopathic dilated cardiomyopathy: A multi-variate analysis of prognostic determinants. *Am Heart J* 1988; 116: 1455-1469.
- [20] Keogh AM, Baron DW, Hickie JB: Prognostic guides in patients with idiopathic or ischemic dilated cardiomyopathy assessed for cardiac transplantation. *Am J Cardiol* 1990; 65: 903-908.
- [21] Klein H, Troster J, Haverich A: AICD as a bridge to transplant. *Rev Eur Technol Biomed* 1990; 12: 110A [abstract].
- [22] Lerner AA: Cardiomyoplasty: a summing up. *Artif Organs* 1995; 19 (3): 199-203.
- [23] Lorusso R, Marchini A, Bianchetti F, *et al*: Cardiomyoplasty and implantable cardioverter defibrillator: Efficacy and safety of concomitant device implantation: Sudden death and cardiomyoplasty. *J Card Surg* 1998; 13 (2): 150-155.
- [24] Luu M, Stevenson LW, Brunken RC, *et al*: Diverse mechanisms of unexpected cardiac arrest in advanced heart failure. *Circulation* 1989; 80: 1675-1680.
- [25] Magovern GJ, Sr: Paced skeletal muscle for dynamic cardiomyoplasty. *Ann Thorac Surg* 1995; 60: 1153-1154.
- [26] Magovern GJ, Simpson KA: Clinical cardiomyoplasty: Review of the ten-year United States experience. *Ann Thorac Surg* 1996; 61: 413-419.
- [27] Magovern J, Magovern GJ Sr, Maher TD, *et al*: Operation for congestive heart failure: transplantation, coronary artery bypass, and cardiomyoplasty. *Ann Thorac Surg* 1993; 56: 418-425.
- [28] Moosvi AR, Goldstein S, Vandenburg MS, *et al*: Effect of empiric antiarrhythmic therapy in resuscitated out-of-hospital cardiac arrest victims with coronary artery disease. *Am J Cardiol* 1990; 65: 1192-1197.
- [29] Moriera LFP, Stolf NAG, Bocchi EA, *et al*: Clinical and left ventricular function outcomes up to five years after dynamic cardiomyoplasty. *J Thorac Cardiovasc Surg* 1995; 109: 353-463.
- [30] Moriera LFP, Stolf NAG, Brile DM, *et al*: Dynamic cardiomyoplasty in South America. *Ann Thorac Surg* 1996; 61: 408-412.
- [31] Nisam S, Mower M, Moser S: ICD clinical update: first decade, initial 10,000 patients. *PACE* 1991; 14: 255-262.
- [32] Podrid PJ, Fogel RI, Fuchs TT: Ventricular arrhythmia in congestive heart failure. *Am J Cardiol* 1992; 69: 82G-96G.
- [33] Roden DM: Drug therapy: risks and benefits of antiarrhythmic therapy. *N Engl J Med* 1988; 116: 1455-1469.
- [34] Schaffer WA, Cobb LA: Recurrent ventricular fibrillation and mode of death in survivors of out-of-hospital ventricular fibrillation. *N Engl J Med* 1975; 293: 259-262.
- [35] Schocken DD, Arrieta MI, Leaverton PE, *et al*: Prevalence and mortality rate of congestive heart failure in the United States. *J Am Coll Cardiol* 1991; 20: 301-306.
- [36] Siebels J, Kuck KH, CASH Investigators: Implantable cardioverter defibrillator compared with anti-arrhythmic drug treatment in cardiac arrest survivors (the Cardiac Arrest Study Hamburg). *Am Heart J* 1994; 127: 1139.
- [37] Singh SN, Fletcher RD, Fisher SG, *et al*: Amiodarone in patients with congestive heart failure and asymptomatic ventricular arrhythmia. *N Engl J Med* 1995; 333: 77-82.
- [38] SOLVD Investigators: Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure. *N Engl J Med* 1991; 325: 293-302.

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- [39] Thakur RK, Chow LH, Guiraudon GM, *et al*: Latis-simus dorsi dynamic cardiomyoplasty: role of combined ICD implantation. *J Card Surg* 1995; 10: 295-297.
- [40] van den Berg MP, Brouwer MH, Brugemann J, *et al*: Initial Groningen experiences with dynamic cardiomyoplasty. [Dutch] *Ned Tijdschr Geneesk* 1997; 141 (30): 1480-1484.
- [41] Weinberg BA, Miles WM, Klein LS, *et al*: Five-year follow-up of 589 patients treated with amiodarone. *Am Heart J* 1993; 125: 109-120.
- [42] Wilber DJ, Garan H, Finkelstein D, *et al*: Out-of-hospital cardiac arrest: use of electrophysiologic testing in the prediction of long-term outcome. *N Engl J Med* 1988; 318: 19-24.
- [43] Winkle RA, Mead RH, Ruder MA, *et al*: Long-term outcome with the automatic implantable cardioverter-defibrillator. *J Am Coll Cardiol* 1989; 13: 1353-1361.