Combined Bipolar Dual Chamber Pacing and Automatic Implantable Cardioverter/Defibrillator

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A 67 year old man with recurrent hypotensive ventricular tachycardia, amiodarone-induced bradyarrhythmias and severe cardiac dysfunction underwent simultaneous implantation of an automatic cardioverter/defibrillator and bipolar atrioventricular (AV) pacemaker. The pacing electrodes were placed epicardially near the right atrial appendage and on the lateral right ventricular wall. The rate detector of the automatic defibrillator was placed epicardially on the posterobasal left ventricular wall. Effective bipolar AV pacing produced no false counting of the heart rate by the automatic cardioverter/defibrillator, and ventricular tachycardia properly inhibited the pacemaker. Long-term follow-up study confirmed the safety of this treatment. With proper precautions, bipolar AV pacing can be safely combined with an automatic cardioverter/defibrillator.

Case Report

Clinical features. A 67 year old man with a history of myocardial infarction (1963 and 1978) had an episode of out-of-hospital cardiac arrest in January 1984 for which he underwent serial electrophysiologic studies and was subsequently started on a regimen of amiodarone. His left ventricular ejection fraction, measured by radionuclide angiography, was 15%. In October 1984 he was readmitted to the hospital for another episode of syncpe due to ventricular tachycardia and required direct current cardioversion. While in sinus rhythm the patient was bradycardic with heart rates between 35 and 50 beats/min and episodes of sinus arrest and junctional escape rhythm, usually associated with diziness and chest tightness radiating to both arms. Programmed stimulation induced ventricular tachycardia at a rate of 200 beats/min, requiring cardioversion. He was referred to our center for implantation of an automatic defibrillator and a permanent pacemaker.

On examination, the patient looked chronically ill. The pulse rate was 48 beats/min and regular and the supine blood pressure was 128/60 mm Hg. Pertinent physical findings included a loud apical third heart sound and, on auscultation of the lungs, bilateral basilar rales. The 12 lead electrocardiogram revealed sinus bradycardia with periods of atrioventricular (AV) dissociation and junctional escape rhythm, first degree AV block, right bundle branch block, left anterior fascicular block, old inferior and anterior infarctions and a QT interval of 540 ms. Figure 1 is a representative rhythm strip recorded shortly after the patient's admission to the hospital.

Left ventricular and coronary angiography revealed severe left ventricular dysfunction, a large anteroapical aneurysm and severe inferolateral hypokinesia. The proximal...
left anterior descending coronary artery was occluded and there was 95 and 30% stenosis of the mid-right coronary artery and proximal circumflex artery, respectively.

**Surgical procedures.** The patient underwent resection of the anterior ventricular aneurysm, encircling endocardial ventriculotomy of the residual scar on the ventricular septum and subendocardial resection of additional scar on the lateral wall of the left ventricle, sequential bypass grafting of two branches of the right coronary artery and implantation of an automatic cardioverter/defibrillator and a bipolar dual chamber pulse generator.

**Electrode systems.** The electrode system for the cardioverter/defibrillator consisted of a large patch applied to the lateral wall of the left ventricle, two rate-counting screw-in electrodes (Medtronic 6917A) placed 10 mm apart on the posterobasal left ventricular wall and an intravascular spring electrode placed at the superior vena caval-right atrial junction, introduced through the innominate vein. The electrode system for the pacing device consisted of two screw-in electrodes (Medtronic 6917A) placed 10 mm apart on the lateral wall of the right ventricle for ventricular pacing, and two fish-hook electrodes (Medtronic 4951) implanted 10 mm apart near the right atrial appendage for atrial pacing. Figures 2A and 2B illustrate the electrode configuration viewed on a posteroanterior chest roentgenogram. Subcutaneous pockets were prepared in the supraumbilical area and in the abdominal right upper quadrant for housing of the cardioverter/defibrillator and the pacemaker, respectively, and the electrodes of each device were tunneled transthoracically to the appropriate pockets.

**Electrogram amplitude** measured from the epicardial rate-counting electrodes was 21 mV, and that measured from the transcardiac spring-patch bipole was 11 mV. Electrogram amplitude measured from the ventricular pacing bipole was 24 mV, and that measured from the atrial pacing bipole was 1.6 mV. The atrial pacing threshold was 2.3 V/4.9 mA, and the ventricular pacing threshold was 0.8 V/1.3 mA. The pacing leads were connected to the pulse generator (Medtronic Symbios, model 7006), which was temporarily programmed to an AOO mode at a rate of 70 beats/min, a pulse width of 0.5 ms and an output of 5 V. Further recordings obtained from the rate-sensing electrodes showed that atrial pacing produced no visible artifact. At the end of the procedure, cardioversion/defibrillation threshold measurements and successful testing of the unit (AID-B, rate cutoff 152.5 beats/min) were performed, and the chest was closed. The pulse generator was programmed to the AOO mode until the patient awakened, and was then reprogrammed to DDD pacing. Noninvasive, audible monitoring of the cardioverter/defibrillator function during AOO and AV sequential pacing confirmed that atrial pacing was not sensed by the unit and that ventricular activation was sensed as a single and only electrical event.

**Postoperative hemodynamic measurements** during dual chamber pacing showed a pulmonary capillary wedge pressure of less than 10 mm Hg and a cardiac index between 2.8 and 3.5 liters/min per m². Because of postoperative respiratory complications the patient remained for 7 weeks under continuous cardiac monitoring during which appropriate AV sequential pacing was consistently verified and no shock was recorded or reported by the patient.

**Electrophysiologic study.** Before his discharge from the hospital, the patient underwent another electrophysiologic study to measure the results of surgical treatment and to confirm the adequate function of the cardioverter/defibrillator. During the first part of the procedure the unit was activated but inhibited with a magnet, to avoid its triggering by the stimulation program. Programmed stimulation of the right ventricular apex, including triple extrastimulation after trains of ventricular paced rhythm at cycle lengths as short

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**Figure 1.** Rhythm strip obtained on the patient's admission to the hospital, showing intermittent atrioventricular dissociation due to sinus bradycardia and a junctional escape rhythm at a rate of 38 beats/min. Shown are (from top to bottom) leads II, V₁ and V₅. The recording speed is 25 mm/s.
as 400 ms, was unsuccessful in inducing sustained ventricular tachycardia. Instead, several episodes of nonsustained, monomorphic ventricular tachycardia with a cycle length of 370 ms were observed, the longest one lasting 27 seconds. During such episodes no inappropriate impulses originating from the AV sequential pacemaker were observed. Finally, the patient was sedated, the magnet was removed from over the cardioverter/defibrillator and nonsustained ventricular tachycardia with a cycle length of 360 ms was induced. This rhythm was properly sensed by the automatic device.

Figure 2. A, Posteroanterior chest roentgenogram showing configuration of the electrodes from the automatic cardioverter/defibrillator and from the dual chamber pacemaker. B, Artist’s representation and description of the electrode system as seen on chest roentgenogram.
which delivered a synchronized shock 13.5 seconds after the onset of programmed stimulation, 11.5 seconds after the onset of ventricular tachycardia and 5.5 seconds after its spontaneous termination. The amiodarone blood level on the day of this procedure, 50 days after discontinuation of therapy, was 0.6 μg/ml.

The patient was discharged from the hospital the next day on a drug regimen including amiodarone, 200 mg/day. After a follow-up of 12 months he has experienced neither recurrence of ventricular tachycardia nor discharge from the cardioverter/defibrillator.

Discussion

Potential interference of the cardioverter/defibrillator by an implanted pacemaker. The rapidly growing use of automatic cardioversion/defibrillation for the long-term treatment of life-threatening ventricular arrhythmias is presenting implanting physicians with several new technical problems that may have important clinical consequences. Reports already published (2) indicate that one such problem, namely, the coexisting need for long-term pacing and standby cardioversion/defibrillation, occurs in 5 to 10% of patients. Thus far, AV pacing could not be offered to those in need of automatic cardioversion/defibrillation because the unipolar pulses delivered by such pacemakers are known to interfere with the automatic device in two different fashions. The first and most common type of interference is an erroneous and excessive counting of the heart rate by the cardioverter/defibrillator, which may interpret atrial pacing or ventricular pacing, or both, as electrical events separate from ventricular activation, leading to a doubling or tripling of the sensed heart rate and inappropriate discharges of the device (3). The second form of interference occurs when pacing is not properly inhibited and continues during ventricular tachycardia or fibrillation. This may alter the detection capability of the cardioverter/defibrillator which, at any given time, bases its arrhythmia analysis on the largest intracardiac electrical signals, in this instance those originating from the pacing device. This results in failure of the automatic cardioverter/defibrillator to terminate the tachyarrhythmia.

Precautions to be observed with combined pacemaker and cardioverter/defibrillator implantation. From our observations it appears that the recent introduction of bipolar AV sequential pacemakers can offer a solution to the interference problem. However, precautions must be taken at the time of implantation to verify that even bipolar artifacts are not inappropriately sensed by the cardioverter/defibrillator. The electrodes should preferably be placed epicardially and as distant from each other as possible. The interelectrode spacing of the rate detector should be narrow, though one should keep in mind that the electrogram recorded from it has to be at least 5 mV in amplitude to provide an adequate rate-counting signal. The rate detector signals should then be recorded and examined during atrial pacing to verify that no pacemaker artifact is present or large enough to confuse the rate counting by the cardioverter/defibrillator. In addition, proper counting of the heart rate by the automatic cardioverter/defibrillator can be verified noninvasively by magnet application, which should produce a single audible beep synchronized with each ventricular cycle. Absence of inappropriate pacing should be established during an induced episode of ventricular tachycardia. Alternatively, it has been suggested that the proper detection function of the cardioverter/defibrillator be verified by the induction of ventricular tachycardia and fibrillation with the unit activated and the pacemaker placed in asynchronous mode at maximal output (4). Because of the different characteristics of the intracardiac signals generated by these two rhythms, it is important that proper sensing be verified during induction of both rhythms. Furthermore, if the patient is known to experience ventricular tachycardia of variable configuration, attempts should be made to verify proper detection of each configuration. In our patient, we decided against the induction of ventricular fibrillation because of his precarious cardiopulmonary reserve, and because all previously observed episodes of tachyarrhythmia, spontaneous or induced, were due to ventricular tachycardia and not fibrillation. We recognize, however, that this was an incomplete evaluation.

During testing of the cardioverter/defibrillator before release of the patient from the hospital, we observed its activation by a brief run of nonsustained ventricular tachycardia, with subsequent discharge during sinus rhythm. This sequence of events has been documented by long-term cardiac monitoring during treatment with the cardioverter/defibrillator (5), and is a known cause of unexpected internal shocks in the conscious, asymptomatic state.

A new generation of automatic cardioverter/defibrillators that will have pacing capability is in the making. To the best of our knowledge, however, these new devices will provide only single chamber, ventricular pacing. Many candidates for automatic cardioversion/defibrillation have severely impaired cardiac function and receive drugs that may produce chronic bradyarrhythmias; thus they are also in need of AV sequential pacing. Such patients will continue to pose the problem of pacemaker interference with the automatic cardioverter/defibrillator.

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References

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