

CASE REPORT

Ventricular tachycardia initiated by high energy cardioversion in a patient with an implantable cardioverter defibrillator

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Abstract

A transvenous implantable cardioverter defibrillator (ICD) was implanted into a 58 year old woman with idiopathic dilated cardiomyopathy who had drug refractory monomorphic ventricular tachycardia (VT). Antitachycardia pacing failed to terminate the VT; termination was attempted at 24 J, which was above the defibrillation threshold. When cardioversion at 24 J was delivered, VT with a different morphology and slower rate was reproducibly initiated. At 3 J, however, the original VT was successfully terminated without initiation of the slower VT. A new VT may be induced by high energy cardioversion. This may be a manifestation of the proarrhythmic potential of ICDs.

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Keywords: cardioversion; ventricular tachycardia; implantable cardioverter defibrillator

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For intractable ventricular tachycardia/ventricular fibrillation (VT/VF), transvenous implantable cardioverter defibrillators (ICD) have proved to be effective.^{1,2} However, degeneration of VT into VF may be observed in some patients especially when low energy shocks are delivered.³⁻⁵

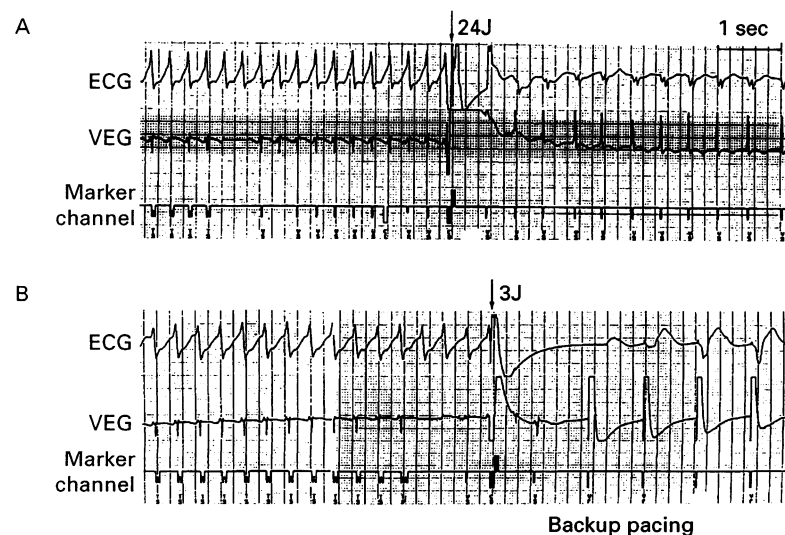
In the present case, VT with a different QRS morphology and a slower rate was reproducibly initiated when a high energy shock (24 J) was delivered. Low energy cardioversion (3 J) successfully terminated the VT without initiation of a new VT.

Case report

A 58 year old woman was referred to our hospital for treatment of recurrent sustained VT. Since 1990, she has been treated with diuretics and an angiotensin converting enzyme inhibitor for idiopathic dilated cardiomyopathy. In December 1991, she complained of palpitation and fainting. Monomorphic sustained VT with four different QRS morphologies was recorded on 12-lead electrocardiograms.

On admission, chest radiography revealed mild cardiomegaly but no pulmonary congestion. A cross sectional echocardiogram showed dilatation of both ventricles and the left atrium; the wall of the left ventricle was diffusely thin and hypokinetic. Left ventriculography revealed moderate mitral regurgitation, and the ejection fraction was 36%. The 12-lead ECG during sinus rhythm showed a complete right bundle branch block pattern with a superior axis.

After obtaining informed consent, electrophysiological study was performed using the standard technique. Four monomorphic VTs (two with left bundle branch block pattern and two with right bundle branch block pattern) were reproducibly induced by programmed stimulation. The site of each VT origin was mapped on either side of the interventricular septum (three sites) and in the free wall of the left ventricle (one site). During electrophysiological study, criteria for transient entrainment were confirmed by rapid ventricular pacing⁶; however, pacing at shorter cycle lengths was unable to terminate, and usually accelerated, the VT. External cardioversion had to be used to restore sinus rhythm.



Cardioversion at 24 J was delivered to terminate VT; the original VT was terminated but VT with a new QRS morphology and a slower rate was observed immediately after the shock (A). The cycle length of the new VT was longer than the tachycardia detection interval (less than 400 ms), and the dysrhythmia terminated spontaneously. The initiation of new VT was avoided by cardioversion at 3 J (B) and back-up pacings initiated after the low energy shock. ECG, lead II of surface electrocardiogram. VEG, intraventricular electrogram.

Antiarrhythmic drugs including DL-sotalol failed to prevent the induction or recurrence of VT. Radiofrequency catheter ablation was effective in eliminating only one VT morphology. The patient was considered as a candidate for ICD treatment.

In September 1995, a transvenous ICD system (Medtronic Jewel-Plus, 7220C, Active Can, Minneapolis, Minnesota, USA) was implanted. During the operation, a shock of 24 J was twice confirmed capable of terminating VF. A shock of 24 J was set as the first treatment for VF and VT.

Two days after the operation, VT recurred and cardioversion at 24 J was appropriately delivered between the body of ICD generator and the common lead in the right ventricle. However, VT with a non-clinical QRS morphology and longer cycle length was initiated immediately by the shock (fig). The VT showed a gradual prolongation of the R-R interval and terminated spontaneously within 10 seconds. The same phenomenon was reproduced after administration of antiarrhythmic drugs (aprimidine and bepridil hydrochloride). The energy of the first shock was then set at 3 J which successfully terminated VT without initiation of the non-clinical VT (fig).

Discussion

In the present case, the mechanism of the non-ischaemic VT was shown, by confirming the criteria for transient entrainment, to be reentry with an excitable gap.^{6,7} However, VT could not be terminated with rapid pacing⁸ and termination of VT/VF was attempted by the delivery of shock at 24 J.

Immediately after the shock, VT with different morphology was reproducibly induced. The non-clinical VT was characterised by a gradual slowing and spontaneous termination; automaticity seems to be the likely mecha-

nism. VT might be induced by shock delivery³⁻⁵ but characteristics of the shock induced VT has not been fully described. Interestingly, the induction of VT was prevented by a shock of 3 J which successfully terminated the original VT.

Shocking with high energy may enhance automaticity in some diseased myocardium,^{9,10} and may have been the mechanism of the phenomenon seen in the present case. Mechanical stimulation by an implanted lead could be excluded because the QRS morphology of the non-clinical VT was different from that of the paced one at the site (fig).

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