

CASE REPORT

Ventricular tachycardia: an unusual pacemaker-mediated tachycardia

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Abstract

A 74 year old man had recurrent ventricular tachycardia, which was well controlled with amiodarone, and complete heart block for which a VVI permanent pacing system had previously been implanted. After an elective increase in the programmed pacemaker rate from 70 to 82 beats/min, there was recurrence of frequent episodes of ventricular tachycardia. Each episode of tachycardia was initiated by a fusion beat consisting of a ventricular extrasystole and a paced beat. When the pacemaker rate was reprogrammed to 70 beats/min the episodes of tachycardia ceased abruptly. It is proposed that the fusion of a ventricular extrasystole with a pacemaker beat may have induced ventricular tachycardia, even though neither of these beats occurring separately was sufficient to cause this.

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Pacemaker-mediated tachycardias are uncommon in patients with chronically implanted and correctly functioning VVI pacing systems, in the absence of acute ischaemia or metabolic disturbance. This is due to the sensing functions that prevent the delivery of a pacemaker impulse during the vulnerable part of the cardiac cycle. We describe a patient whose pacemaker was implicated in the occurrence of ventricular tachycardia despite normal pacemaker function and appropriate settings of the sensing values.

Case report

A 74 year old man was admitted to hospital after an episode of dizziness that was associated with rapid palpitation then collapse but not loss of consciousness.

Eleven years previously, he had had an anterior myocardial infarction. This was subsequently complicated by left ventricular failure, which was treated with bumetanide and enalapril, and non-sustained ventricular tachycardia associated with presyncope which was controlled with amiodarone. Two years previously, he had developed complete heart block for which a permanent VVI pacing system was implanted. Four weeks before admission, the programmed pacing rate was electively increased from 70 to 82 beats/min in an attempt to improve symptoms of

fatigue. From that time he noticed a recurrence of the palpitation from which he had been entirely free for the previous nine years. At the time of his admission his medication consisted of 200 mg amiodarone daily, 20 mg enalapril daily, and 2 mg bumetanide twice daily. He had no other medical history and smoked 10 cigarettes daily.

The initial electrocardiogram showed a uniform ventricular tachycardia (fig 1A) and the chest x ray film showed pulmonary oedema. The serum electrolytes, magnesium, creatine kinase, and thyroxine were normal. The tachycardia spontaneously reverted to a ventricular paced rhythm (fig 1B), with resolution of his symptoms.

During the next 12 hours, there were frequent, non-sustained episodes of ventricular tachycardia (5-30 beats) that occurred every one to three minutes (fig 2). All the recorded episodes of ventricular tachycardia were of the same configuration and each was initiated by a ventricular extrasystole on which a pacemaker impulse was superimposed (fig 2). The ventricular extrasystoles that initiated the ventricular tachycardia occurred at a mean (SD) coupling interval of 710(14) ms. On several occasions ventricular extrasystoles inhibited the pacemaker output. These beats never initiated ventricular tachycardia (fig 2B), and they occurred at a shorter coupling interval after the preceding pacing spike than the ventricular extrasystoles that initiated the episodes of ventricular tachycardia (653(41) ms *v* 710(14) ms, $p < 0.01$). Only two ventricular extrasystoles with superimposed pacing impulses were recorded that did not trigger ventricular tachycardia and these extrasystoles occurred at coupling intervals of 680 and 700 ms after the preceding pacing impulses.

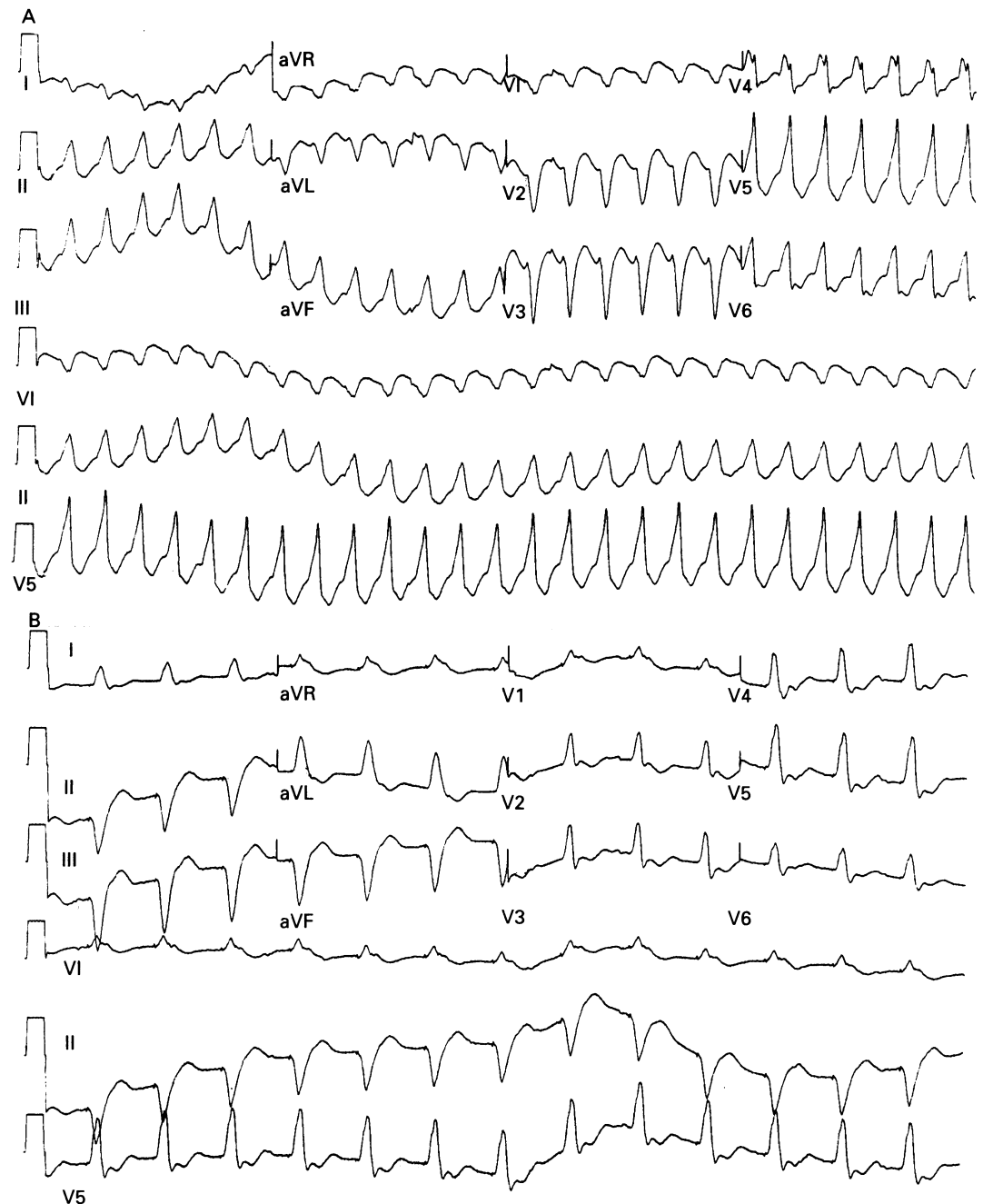
Twelve hours after admission to hospital, the patient's pacemaker was reprogrammed to 70 beats/min and this resulted in the immediate and complete end of the episodes of ventricular tachycardia. Subsequent pacemaker checks showed that the pacing system was functioning correctly with an appropriate sensing threshold. Continuous electrocardiographic monitoring in hospital for a further three days showed no further episodes of ventricular tachycardia, and there was a considerable reduction in the frequency of ventricular extrasystoles. At follow up nine months later, he remained free of palpitation and 24 hour ambulatory electrocardiographic monitoring showed no ventricular tachycardia.

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Figure 1(A) Twelve lead electrocardiogram recorded at admission showing a broad QRS complex tachycardia at 172 beats/min. (B) Twelve lead electrocardiogram recorded shortly after spontaneous resolution of the tachycardia.



Discussion

Several features of this case suggest that the episodes of ventricular tachycardia were related to pacemaker function. Firstly, there was a striking temporal relation between the increase in the pacing rate and the recrudescence of tachycardia. Secondly, there was immediate resolution of the episodes of tachycardia when the pacing rate was restored to the original frequency. Thirdly, there was no other abnormality such as a recent myocardial infarction or an electrolyte disturbance to explain the onset of the tachycardias. Also, the presence of a pacing impulse superimposed on the initiating ventricular extrasystole of each episode of ventricular tachycardia (fig 2) provided evidence that the tachycardias were triggered by the pacemaker. These initiating beats were of a different configuration from the usual paced beats and therefore represented fusion of separate impulses arising from the pacemaker and a

ventricular ectopic focus. Furthermore, the ventricular extrasystoles that inhibited the pacemaker output did not cause ventricular tachycardia (fig 2B).

The analysis of the recorded electrocardiograms showed that the ventricular extrasystoles occurred at a variable coupling interval of 640–720 ms, and that they were all of similar configuration. Those extrasystoles with a short coupling interval (653(41) ms) inhibited the output of the pacemaker and did not cause ventricular tachycardia. The ventricular extrasystoles with a long coupling interval that was close to the programmed cycle length of the pacemaker (710(14) *v* 732 ms) failed to inhibit the pacemaker output, presumably because depolarisation from the extrasystole had not spread to the pacemaker electrode site in time to inhibit the pacemaker. The resulting fusion beat usually triggered ventricular tachycardia.

Two mechanisms may have contributed to

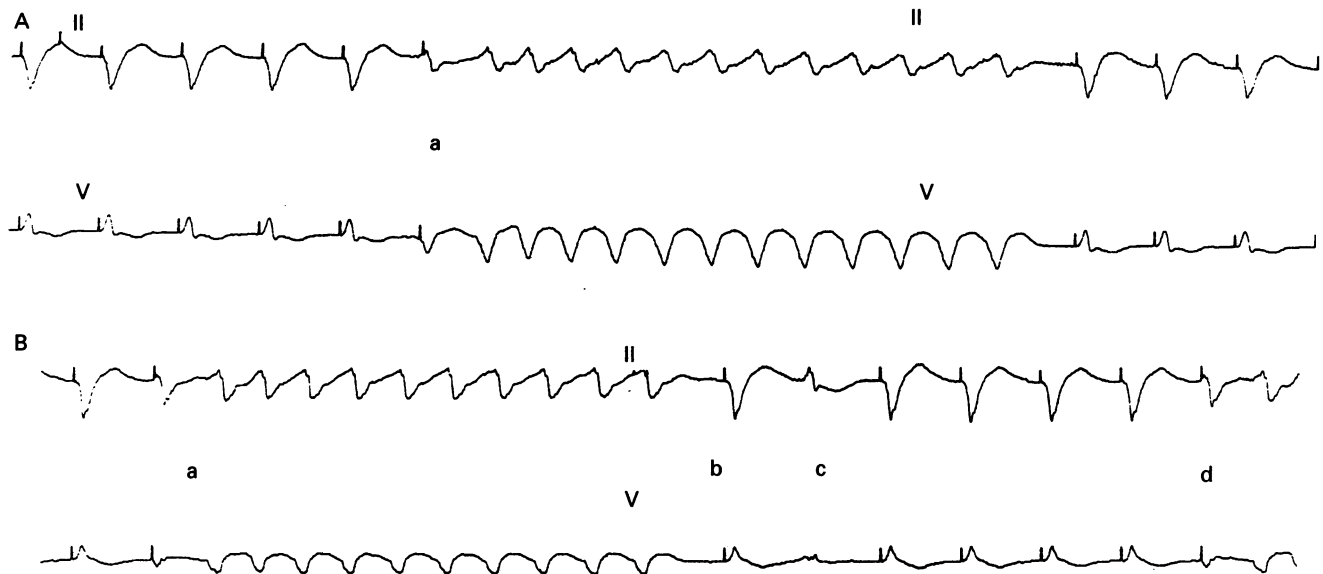


Figure 2(A) Electrocardiogram rhythm strip showing leads II and V1 of a representative episode of ventricular tachycardia that was initiated by a fusion beat (a). (B) Electrocardiogram rhythm strip showing leads II and V1 of an episode of ventricular tachycardia triggered by a fusion beat (a). The tachycardia stops and is followed by a single normal paced beat (b). A subsequent ventricular extrasystole (c) was sensed by the pacemaker and did not cause ventricular tachycardia. The beginning of a second episode of tachycardia triggered by a second fusion beat (d) is also seen.

the association of the tachycardias with the change in the rate of the pacemaker. Firstly, the presence and frequency of ventricular extrasystoles may be closely related to the underlying heart rate,¹ and in our patient ventricular extrasystoles were much more frequent when the pacing rate was 82 beats/min compared with 70 beats/min. Secondly, the relation between the coupling interval of the ventricular extrasystoles and the pacemaker cycle length was changed when the pacemaker rate was increased. At the increased rate of 82 beats/min, there was near synchrony between the two sources of depolarisation, and this caused the fusion beats that were critical in triggering the episodes of ventricular tachycardia.

In this patient, the combined activation of the ventricular ectopic focus and the pacemaker was necessary to cause ventricular tachycardia, whereas the activation of either source alone was insufficient. It is possible that the near synchronous activation of two separate ventricular ectopic foci may be an important mechanism for the occurrence of ventricular tachycardia. This would not normally be detectable on the surface electrocardiogram as the resulting fusion beat would be indistinguishable from an extrasystole arising from a single focus. In our patient, however, the impulses of the pacemaker acted as markers that allowed the separate origins of the fused beats to be distinguished.

Pacemaker mediated tachycardias may complicate the function of dual chamber pacing systems and sensor driven single chamber rate adaptive pacemakers.² Rarely runaway pacemakers cause potentially lethal tachycardias.³ In all these cases the tachycardia is both initiated and sustained by the pacemaker. By contrast, the tachycardia in this case was triggered by an interaction between the pacemaker and the ventricular extrasystoles, but was self-sustaining. Tachycardias mediated

by pacemakers are rare in patients with chronically implanted, normally functioning VVI pacemakers in the absence of acute ischaemia or metabolic disturbance, but when they do occur it is most often due to the occurrence of a pacing impulse during the T wave. This is normally prevented by appropriate sensing functions. Four cases have been described⁴ in which ventricular tachycardia was initiated by correctly timed impulses from normally functioning VVI pacemakers. These cases differed from our report in that the QRS configuration of the paced beats was similar to that during the tachycardia,⁴ and the mechanism of initiation of tachycardia depended on the close proximity of the tachycardia circuit and the pacemaker electrode. In the three patients studied with ventricular mapping⁴ the re-entry circuit was indeed found to be close to the right ventricular apex and the pacing electrode tip. In none of the four cases described⁴ was tachycardia initiated by a fusion beat, as was the case in our patient.

In summary, this is an unusual complication of reprogramming a pacemaker. As far as we know, it is the first reported case of ventricular tachycardia induced by the fusion of a pacemaker impulse and a ventricular extrasystole, and it indicates that fusion beats arising from two separate foci within the ventricular myocardium may trigger ventricular tachycardia when activation from either focus individually is insufficient to initiate tachycardia.

- 1 Coumel P. Rate dependence and adrenergic dependence of arrhythmias. *Am J Cardiol* 1989;64:41J-45J.
- 2 Lau C-P. Sensors and pacemaker mediated tachycardias. *PACE* 1991;14:495-8.
- 3 Mickley H, Andersen C, Nielsen LH. Runaway pacemaker: a still existing complication and therapeutic guidelines. *Clin Cardiol* 1989;12:412-4.
- 4 Karbenn U, Borggreffe M, Breithardt G. Pacemaker-induced ventricular tachycardia in normally functioning ventricular demand pacemakers. *Am J Cardiol* 1989;63:120-2.