Lesson of the week

Pacemaker induced ventricular fibrillation in coronary care units

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Temporary transvenous pacing is an established treatment for many bradyarrhythmias associated with haemodynamic compromise in patients who have had acute myocardial infarction. Procedural complications associated with insertion of temporary pacing electrodes are well recognised and documented, as is subsequent loss of capture due to rising stimulation threshold or electrode displacement.¹ Temporary transvenous pacing, if not applied correctly, may also directly provoke ventricular arrhythmias. Here we report two cases of ventricular fibrillation that were caused by a temporary pacemaker in a coronary care unit.

Case reports

Case 1

A 64 year old man was admitted to the coronary care unit with acute inferior myocardial infarction. He was noted to be hypotensive and have a nodal rhythm with no atrial activity at 30 beats/min. There was no response to atropine. A temporary pacing electrode was inserted and connected to an external ventricular demand pacemaker. During the following two days, at least 15 episodes of ventricular fibrillation and many sustained episodes of ventricular tachycardia occurred. Review of the electrocardiograms showed occasional failure of pacing and sensing and of pacing impulses delivered into the ST segment and the T wave of spontaneous beats causing ventricular fibrillation (fig 1). The pacemaker was turned off. A bradycardia with first degree atrioventricular block was noted, but the episodes of ventricular fibrillation stopped immediately and did not recur.

Case 2

A 73 year old man was admitted with breathlessness and lethargy. He had experienced pain typical of myocardial infarction two days previously. He was taking an angiotensin converting enzyme inhibitor, a calcium antagonist, and a β blocker for hypertension. His pulse was 32 beats/min, and an electrocardiogram showed complete heart block with left bundle branch block. His blood pressure was 70/30 mm Hg even after intravenous fluid administration. A temporary pacing electrode was inserted and ventricular demand pacing started. Troponin T was 4.58 $\mu\text{g/l}$ (normal <0.03 $\mu\text{g/l}$); creatine kinase was 1025 units/l (normal <190 units/l). There was evidence of undiagnosed diabetes and renal impairment (glucose 20.8 mmol/l; urea 16.5 mmol/l; creatinine 311 $\mu\text{mol/l}$; HbA_{1c} 0.092%). An echocardiogram showed inferior wall akinesis, moderate mitral regurgitation, and raised right ventricular systolic pressure.

He was treated with insulin, and β blockade was stopped. Aspirin and simvastatin were also given.

His condition improved, but four days after admission he suddenly developed ventricular fibrillation.

Defibrillation was achieved with a 360 J shock, after two shocks at 200 J were ineffective. Episodes of ventricular tachycardia also occurred. He was given an intravenous loading dose of amiodarone followed by a continuous infusion.

Three days later ventricular fibrillation recurred. Defibrillation with a single 200 J shock was successful. A magnesium infusion was started. Two further episodes occurred that night, each requiring defibrillation. Plans were made for referral to a tertiary cardiac centre for consideration of angiography, electrophysiological study, and a possible cardioverter or defibrillator implant.

Two days later he developed a temperature of 37.7°C. Intravenous benzylpenicillin and flucloxacillin were given. The following day the temporary pacing electrode was removed. The underlying rhythm was atrial fibrillation with a satisfactory ventricular response rate.

On day 10 a consultant cardiologist reviewed the monitor tracings of all the available episodes of ventricular fibrillation. All episodes seemed to be triggered by inadvertent pacing during the repolarisation phase of a non-sensed ventricular extrasystole. This was particularly noticeable when the extrasystole developed after a long escape cycle (fig 2). The ventricular stimulation threshold of the pacemaker was 1.0 V on day 1 and 0.6 V on day 2 but had risen to 1.8 V on day 4 and 2.1 V thereafter. β blockers were subsequently reintroduced without further conduction problems, and warfarin treatment was

Ventricular fibrillation may be caused by "R on T" stimulation from a temporary pacemaker

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Fig 1 Case 1. Top: Apparently satisfactory pacing (first QRS complex) is followed by fusion beats, but failure to sense becomes apparent when the pacemaker spike (narrow artefact) discharges into the ST segment. The first captured premature complex leads to ventricular tachycardia, which later degenerates into ventricular fibrillation. Further pacing spikes can be seen during the ventricular tachycardia. Bottom: Another ventricular fibrillation episode. The first paced beat is premature. The pacemaker has not sensed the first QRS complex. After two further beats, with the pacemaker discharging harmlessly during the QRS complex, another discharge in the vulnerable recovery phase of repolarisation leads to repetitive activity and ventricular fibrillation

Fig 2 Case 2: The underlying cardiac rhythm is atrial fibrillation with a broad complex idioventricular rhythm at 55 beats/min. A ventricular extrasystolic escape beat is followed by pacemaker discharge (sharp biphasic spike artefact) with immediate ventricular fibrillation

started. Prolonged cardiac monitoring, including ambulatory monitoring, showed no recurrence of ventricular arrhythmias.

Discussion

Although the phenomenon described in these two cases is uncommon, it is possible that some episodes of pacemaker mediated ventricular fibrillation after temporary pacing for acute myocardial infarction are not recognised despite constant electrocardiographic monitoring. The phenomenon is vital to recognise—the arrhythmia may be fatal—and it is easy to treat. Most cases of ventricular fibrillation after cardiac pacing in acute myocardial infarction occur during manipulation of the temporary pacing electrode into the right ventricle, particularly after inferior or right ventricular myocardial infarction.^{2 3} When there is poor electrode contact with the endocardium, failure to sense the underlying local action potential is usually also accompanied by poor pacing performance. The failure is usually noticed, and the electrode is then repositioned. Case 1 illustrates both poor pacing and poor sensing. In the event of poor sensing, stimulation (even at a relatively low voltage) in the repolarisation phase of the cardiac cycle is well recognised as a potential cause of ventricular fibrillation (the “R on T” phenomenon) and has been noted to occur in patients having treatment with a pacemaker.^{4–10}

Repetitive ventricular activity is particularly common when a premature beat falls on the T wave of a beat with a preceding long cycle length (case 2).^{4 11} Other conditions making repetitive ventricular activity more likely are hypoxia, cardiac ischaemia, myocardial disease, hypokalaemia, hypothermia, hypothyroidism, digoxin treatment, and drug induced prolongation of repolarisation.^{5 6 12–14} Direct mechanical stimulation of an ischaemic heart may also induce ventricular fibrillation. The need for temporary pacing after myocardial infarction should be carefully evaluated daily.

Temporary endocardial pacing systems use bipolar electrodes. The tip of the electrode is negative (cathode). Cathodal stimulation is less likely than anodal stimulation to cause ventricular fibrillation.^{6 8} When connecting a temporary pacemaker, operators should ensure that the tip is connected to the negative (black) pole of the pulse generator.

Unipolar pacing has occasionally been applied in acute myocardial infarction but is impractical and is associated with an increased risk of oversensing and inappropriate pacemaker inhibition. Bipolar pacing

systems offer advantages in terms of reduced sensing of artefact and myopotentials, which may cause inappropriate pacemaker inhibition.

Many external temporary pacing systems have relatively simple controls, so that those not very familiar with cardiac pacing do not have to make adjustments other than for stimulation rate and output voltage. The sensitivity of the detection of the underlying cardiac rhythm is usually set to about 2 mV, which is adequate to detect most normal or extrasystolic ventricular depolarisations but not so sensitive as to detect extraneous artefact with consequent inhibition of the pacemaker. Occasionally the sensitivity may be adjustable, which may prevent the problems described in these cases. Poor sensing, however, is usually accompanied by poor pacing. The appropriate strategy is usually to reposition the electrode in the right ventricle. In patients dependent on a pacemaker it may be safer to introduce another electrode. As seen here, however, often the best strategy is to turn the pacemaker off or to remove it altogether.

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Endpiece

Time

Time goes, you say?
Alas, Time stays, we go.

Henry Austin Dobson (1840-1921)

Nicholas Hayes, neonatal senior house officer,
London