Temporary Overdriving Pacing as an Adjunct to Antiarrhythmic Drug Therapy for Electrical Storm in Acute Myocardial Infarction

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A 55-year-old man with diabetes mellitus was admitted to hospital because of chest pain. He was diagnosed as anterior acute myocardial infarction and treated with stent placement. After 7 days, ventricular fibrillation occurred because of a subacute reocclusion and balloon angioplasty was performed. Despite reperfusion therapy, intraaurtic balloon pumping, antiarrhythmic drugs and β-blocker, ventricular tachycardia or fibrillation relapsed and cardioversion was performed 29 times during 32 h. Temporary overdrive atrioventricular sequential pacing was initiated and the malignant arrhythmia finally disappeared. Even after stoppage of 25 h overdrive pacing, it never recurred. Temporary overdrive pacing is an easy and feasible therapy for a drug-resistant electrical storm associated with AMI and should be performed in the early stage. (Circ J 2005; 69: 613–616)

Key Words: Electrical storm; Myocardial infarction; Pacing

The phenomenon of rapidly clustering episodes of ventricular fibrillation requiring multiple cardioversions is called an 'electrical storm' and in acute myocardial infarction (AMI) important contributory factors include heightened adrenergic nervous system tone, hypokalemia, hypomagnesemia, intracellular hypercalcemia, acidosis, free fatty acid production from lipolysis and free radical production from reperfusion of the ischemic myocardium.3–4 The current recommendations for antiarrhythmic drug therapy include class I antiarrhythmic drugs (β-blocker,2 and in Japan nifekalant, a novel class III drug, is also used).6,7 Although overdrive suppression of refractory ventricular arrhythmia by rapid pacing has become widespread in its application since 1964, there have been few reports about its effectiveness for drug-resistant electrical storm in AMI.

We present a case of a patient who developed a drug-resistant electrical storm even after reperfusion, which was successfully overcome with temporary overdrive pacing.

Case Report

A 55-year-old man with diabetes mellitus was admitted to hospital because of chest pain on June 7 2003. On physical examination, his level of consciousness was clear, temperature 36.6°C, pulse 76 beats/min, blood pressure 120/76 mmHg and oxygen saturation 96% in room air. Auscultation revealed neither cardiac murmurs nor rales, and there were no neurological abnormalities. The admission chest X-ray did not show pulmonary congestion, but the electrocardiogram (ECG) showed ST-segment elevation in leads V1–4 and abnormal Q wave in leads V1–3. Transthoracic echocardiography showed severe hypokinesis or akinesis of the anterior and apical walls and a left ventricular ejection fraction of 40%. He was diagnosed as anterior AMI and underwent emergency angiography, which revealed occlusion of the proximal left anterior descending artery (LAD) that was treated with stent placement. He had no symptoms after the treatment.

On June 14, ventricular fibrillation occurred after frequent ventricular premature beats and cardioversion was performed (Fig 1). Because the ECG after cardioversion showed re-elevation of the ST-segment in leads V1–4, he underwent emergency angiography. Left coronary angiography showed reocclusion of the proximal LAD, which was treated with balloon angioplasty. After placement of an intra-aortic balloon pump to preserve favorable hemodynamics and prevent reocclusion, the patient returned to the coronary care unit. Ventricular premature beats appeared frequently even after the successful reperfusion and he was treated with intravenous lidocaine and magnesium sulphate.

On June 15, ventricular fibrillation occurred and cardioversion was performed. Despite drug therapy (intravenous propranolol, procainamide, nifekalant (0.25–0.40 mg·kg⁻¹·h⁻¹) and oral amiodarone (800 mg)) and mechanical ventilation performed under general anesthesia with propofol, ventricular tachycardia or fibrillation relapsed. Cardioversion was performed 29 times during 32 h. We placed a pacing lead in the right ventricular apex through a right femoral approach and initiated overdrive ventricular pacing at a rate of 110 beats/min. The malignant arrhythmia finally disappeared. We placed the other pacing lead in the high right atrium and performed overdrive atrioventricular sequential pacing at an atrioventricular delay of 100 ms (Fig 2). We ceased the overdriving pacing 25 h after its initiation and the malignant arrhythmia did not recur (Fig 3). Thus, the electrical storm passed while overdrive...
pacing was being performed. All antiarrhythmic drugs, including nifekalant and amiodarone, were reduced gradually and ceased before discharge without recurrence of the malignant arrhythmia. Predischarge coronary angiography showed that the proximal LAD had been restored and he was discharged with aspirin, carvedilol and enalapril. No major adverse cardiac events occurred during the following 14 months.

**Discussion**

We have described a patient with incessant ventricular tachycardia or fibrillation that could not be controlled with reperfusion therapy, intraaortic balloon pumping, antiarrhythmic drugs or ß-blocker. Overdrive pacing was effective in suppressing the electrical storm in AMI.

Persistent electrical storm is rare, but fatal in AMI. Conventional antiarrhythmic drug therapy often fails to maintain sinus rhythm and increased sympathetic activity contributes to the occurrence of an electrical storm. In animal models sympathetic blockade is known to prevent ventricular arrhythmia. Zipes reported that myocardial ischemia and infarction affected the denervation of sympathetic–parasympathetic fibers, which enhanced sympathetic activity, thereby increasing the propensity for ventricular arrhythmia. In the clinical setting, Nademanee et al reported that the 1-week mortality rate was lower in AMI patients treated with sympathetic blockade treatment than in those treated with conventional antiarrhythmic drugs (22% vs 82%, p<0.01). Currently, ß-blocker are favored over...
conventional antiarrhythmic drugs for treating an electrical storm and therefore we administered intravenous propranolol, but the malignant arrhythmia relapsed. It has been recently reported that massive doses of oral amiodarone or intravenous nifekalant prevents malignant arrhythmia in AMI, but neither of these was effective in the present case. Because intraaortic balloon pumping preserved the hemodynamics during sinus rhythm, it was unclear whether additional percutaneous cardiopulmonary support would prevent the malignant arrhythmia. Under these conditions, overdrive pacing was remarkably effective in preventing relapse of the malignant arrhythmia.

When the pacing rate was reduced, ventricular premature beats recurred immediately. There are several mechanisms by which ventricular ectopic activity is related to heart rate, involving both reentry and automaticity. Decreasing heart rate results in increasing heterogeneity of ventricular repolarization, setting the stage for reentry extrasystoles of the focal reexcitation type. Furthermore, slower heart rates mean longer periods of diastole and hence a greater opportunity for an ectopic pacemaker. Because we started overdriving pacing as an adjunct to antiarrhythmic drug therapy, it is unclear whether the malignant arrhythmia was prevented only by the overdriving pacing, but we have demonstrated that it is an easy and feasible bailout therapy for an electrical storm, even in AMI.

Very recently, Bansch et al reported that radiofrequency ablation of the ventricular premature beats triggering incessant ventricular tachyarrhythmias after AMI was feasible and could prevent drug-resistant electrical storm. However, it requires an advanced electrophysiological technique and is very difficult in an emergency. They also reported that temporary overdrive pacing suppressed the ventricular premature beats and allowed for acute stabilization until radiofrequency ablation in 3 of the 4 patients, although the duration of the pacing was not described. Because an electrical storm is likely to be transient in reperfused AMI, we should only bear rather than treat it. From this point of view, temporary overdrive pacing is an easy and feasible bailout therapy in an emergency.

Nielsen et al reported that atrioventricular pacing reduced inferior, septal and global mean myocardial blood flow as well as the left ventricular ejection fraction, as compared with atrial pacing in patients with sick sinus syndrome. Therefore, overdrive ventricular pacing may cause left ventricular desynchronization, resulting in worsening hemodynamics especially in patients with depressed left ventricular function. However, in the present patient, overdrive ventricular pacing was effective in suppressing the electrical storm even at the sacrifice of worsening hemodynamics. In patients with depressed left ventricular function it may be essential that overdrive ventricular pacing is stopped immediately after the electrical storm passes. Overdrive atrial pacing is not essential, but it can suppress atrial fibrillation and lead to favorable hemodynamics through an atrioventricular sequence and thus may indirectly suppress an electrical storm.

In the present patient, once the electrical storm had passed, the malignant arrhythmia did not recur even without antiarrhythmic drug therapy, which suggests that an electrical storm during the early phase of AMI is not always associated with an increased risk of long-term mortality. If ventricular tachycardia is documented in the future, invasive electrophysiological testing should be considered for risk stratification.

In conclusion, temporary overdrive atrioventricular sequential pacing is an easy and feasible bailout therapy for an electrical storm in AMI and should be performed in the early stage.

References


