Imaging in Cardiology: Ventricular Tachycardia and Ventricular Fibrillation During Pacemaker Implantation

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Introduction

Implantation of a pacemaker can be accompanied by different types of arrhythmias. In most cases, it involves short-lived atrial or ventricular arrhythmias induced by the mechanical contact of the manipulated lead. In a minority of these cases atrial fibrillation may persist, and can hamper the measurements following proper positioning of the atrial lead. Ventricular extrasystoles and short runs of ventricular tachycardia are always present during implantation, but very few cases have reported ventricular arrhythmias persisting during pacemaker implantation [1]. In this case report we describe the occurrence of an unforeseen ventricular tachycardia and ventricular fibrillation during pacemaker implantation.

Case Report 1

A 79-year-old man was referred to the cardiology department for evaluation of his syncopal events. The history revealed a myocardial infarction in 1981. Twelve years later the patient underwent coronary bypass surgery. One year after surgery he was admitted to the hospital for treatment of ventricular tachycardia (VT). A poor left ventricular function was diagnosed and amiodarone was administered. Shortly before his most recent admission to the hospital the patient experienced circulatory collapse with a spontaneous recovery, and immediately after the event the referring physician diagnosed a pulse rate of 24 beats/min (bpm). Intravenous atropine did not accelerate the heart rate, but the heart rate increased to 80 bpm after 2 mg of intravenous adrenaline was administered. An ECG indicated atrial fibrillation (AF) with a wide complex escape rhythm (the QRS width was 240 ms with a right bundle branch configuration and a left axis).

The patient was conscious with a blood pressure of 117/67 mmHg, a pulse rate of 33 bpm; auscultation over the heart revealed soft heart sounds without any murmurs. There were no signs of heart failure. The ECG showed AF with total atrioventricular (AV) block and an escape rhythm of 33 bpm with wide QRS complexes. The echocardiogram showed a moderate mitral insufficiency poor left ventricular function with an anteroseptal aneurysm.

Amiodarone was continued and a permanent pacemaker was implanted. After introducing the ventricular lead into the right ventricle for positioning into the right ventricular apex, VT was initiated following a ventricular extrasystolic beat (Figure 1). The VT terminated spontaneously. A new episode of VT occurred and deteriorated into ventricular fibrillation (VF), which was terminated by defibrillation (Figure 2). The defibrillation terminated not only the VF, but also the AF. The implantation was without further arrhythmic events.

Case Report 2

A nine-year-old pacemaker had to be replaced in an 82-year-old man, who had experienced symptomatic bradycardias five years after his coronary bypass surgery. During surgery the lead was damaged and had to be replaced. The new ventricular lead (Merox MEX 60-BP, Biotronik, Germany) was inserted, but it was very difficult to pass through the tricuspid valve, and once the tip of the lead passed though the valve it jumped into the right ventricular outflow tract. Repositioning to a lower, more stable location resulted in a non-sustained monomorphic VT with an irregular rate (Figure 3).

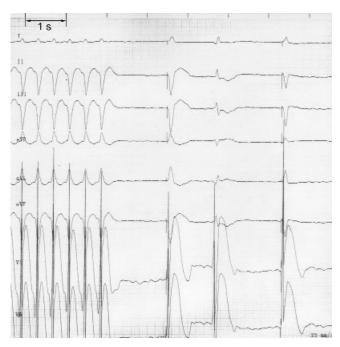


Figure 1. The ECG during the ventricular tachycardia shows a fast ventricular rhythm at a rate of 145 beats/min. Atrioventricular dissociation cannot be observed because of atrial fibrillation. After spontaneous termination of the ventricular tachycardia, atrial fibrillation is apparent. A temporary pacemaker had already been inserted. The two lower and identical ECG recordings (marked as VI and V6) display the intracardiac ECG, derived from the tip of the permanent pacemaker lead.

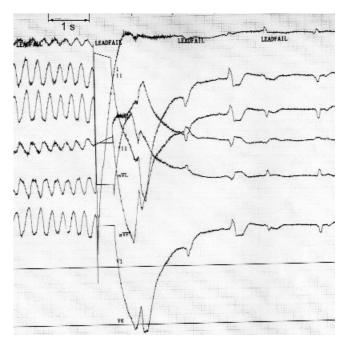


Figure 2. Ventricular fibrillation terminated by external defibrillation and restoration of the "normal rhythm."

Discussion

Many authors describe the occurrence of atrial and ventricular arrhythmias during pacemaker implantation, but specific case reports are rare [2-5]. Mechanical contact or irritation of the pacemaker lead may induce extrasystolic beats, especially when the stylet is partly withdrawn from the lead and the lead tip is freely movable in the cardiac cavity. The induced tachycardias may stop spontaneously after the lead is withdrawn into the proximal compartment of the heart; i.e., into the atrium when inducing arrhythmias in the ventricle, and into the vena cava when inducing arrhythmias in the right atrium. Manipulation of the lead in the right ventricular outflow tract may provoke polymorphous ventricular tachycardias.

Several risk factors can contribute to this condition: significant acid-base imbalance, electrolyte imbalance, physiologic imbalance, drug induced susceptibility enhanced by digitalis and catecholamine, impaired myocardial oxygenation and suboptimal regulated congestive heart failure [6,7]. Furthermore, the first patient was diagnosed with monomorphic VT's. Pre-operative preparation of the patient should include laboratory tests (for electrolytes and renal function), and assessment for the presence of myocardial ischemia and congestive heart failure. The heart rate should be stable and reliable during permanent pacemaker implantation, but the administration of isoproterenol may enhance the risk of ventricular arrhythmias [8]. On the other hand, complete heart block induces remodeling of the heart in the form of dilatation, which enhances the risk of ventricular arrhythmias [9].

The implanting cardiologist or surgeon should be prepared for the development of serious arrhythmias. A defibrillator must be fully charged and located near the patient, and in cases of high risk defibrillating electrodes should be strapped to the patient's chest. In cases of polymorphic ventricular arrhythmias the lead can be withdrawn, and when the stylet is pushed into the lead the stylet can be retracted to reduce the pressure of the lead tip on the myocardium.

It must be stressed that extrasystolic beats are not only or solely induced by mechanical movements, but also by the pressure of the lead tip itself (once it is anchored into the myocardial trabeculae). On the ECG the acute lesion si characterized by ST elevation on the intracardiac ECG recording (see figure 1: intracardiac ECG of spontaneous beat at end of repolarisation), which is possibly

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Figure 3. Pacing from the right ventricular apex is present at the beginning of the recording (three QRS complexes with negative configuration in limb leads II, III, aVF), and a monomorphic non-sustained VT can be observed. Note the different configuration, with more positive QRS complexes indicating that the origin is high on the interventricular septum.

caused by leakage of intracellular potassium caused by leakage of intracellular potassium ions; this ST elevation is very often accompanied by early depolarizations (Figure 1). Further investigations in this field are necessary to gain more insight into the origin of the extrasystolic beats during pacemaker implantation.

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