Case Report

ELECTROSURGERY-INDUCED VENTRICULAR FIBRILLATION DURING PACEMAKER REPLACEMENT – A UNIQUE MECHANISM

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ABSTRACT. Arrhythmias and pacemaker malfunction are known to occur from the use of an electrosurgical device. The present case report describes a patient with sick sinus syndrome who experienced ventricular fibrillation while undergoing surgery. During replacement of his non-functioning cardiac pacemaker under general anesthesia, electrosurgery was used to ensure hemostasis. Electric current may have stimulated myocardial leads present in the surrounding tissue, leading to ventricular fibrillation. The patient was resuscitated from the episode without any residual sequelae. Microshock and possible mechanisms that can lead to ventricular arrhythmias in patients with pacemakers during electrosurgery are discussed.

KEY WORDS. Cardiac pacemakers; transposition of the great arteries; Mustard procedure; ventricular fibrillation; electrosurgery; microshock.

INTRODUCTION

Following corrective surgical repair for transposition of the great arteries (the Mustard procedure), patients have a high incidence of atrial arrhythmias, including sick sinus syndrome [1]. Cardiac pacemakers have been utilized as a treatment for arrhythmias in such patients. Arrhythmias and pacemaker malfunction have been reported during the use of electrosurgery in patients with cardiac pacemakers [2–5]. We describe a case in which a patient with sick sinus syndrome experienced ventricular fibrillation intraoperatively while undergoing replacement of his cardiac pacemaker.

CASE REPORT

A 22-year-old man was scheduled for replacement of his cardiac pacemaker under general anesthesia. He was admitted to the hospital with the chief complaint of pain in the left upper quadrant of his abdomen in proximity to the generator insertion site. The cardiac pacemaker was ascertained to be non-functioning (neither pacing nor sensing either chamber). The patient was on no medications and his serum electrolytes were normal.

The patient was diagnosed with transposition of the great arteries with a ventricular septal defect in early childhood and underwent a Mustard procedure at the age of two. He began to experience syncope at the age of twenty. A diagnosis of sick sinus syndrome was made, and a transthoracic dual chamber cardiac pacemaker (DDD) was subsequently implanted. The pacemaker had

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stopped sensing and pacing the atria and was therefore reprogrammed to the VVI mode during a subsequent hospitalization. Full details of his earlier hospitalizations were unknown. Physical examination revealed a nonradiating, III/VI systolic ejection murmur, which was best heard along the left lower sternal border. The remainder of the physical examination was unremarkable. The electrocardiogram demonstrated a normal sinus rhythm with a rate of 72 beats/min and nonsignificant Q waves in leads II, III, and aVF. Discontinuity of both ventricular pacemaker leads was observed on the chest radiograph; however, the atrial lead appeared to be intact. After institution of standard monitoring, including electrocardiogram (leads II, V5), pulse oximetry, and automated, noninvasive blood pressure, general anesthesia was induced with fentanyl (3 μg/kg), sodium thiopental (3.5 mg/kg), and succinylcholine (1.5 mg/kg). After endotracheal intubation, anesthesia was maintained with isoflurane (0.5 to 0.75%, end-tidal) in 50% oxygen and 50% nitrous oxide. An additional dose of fentanyl (2 μg/kg) was administered during the procedure, and pancuronium (0.05 mg/kg) was given for muscle relaxation.

The initial intraoperative course was stable. During surgical exploration of the pacemaker generator site, the ventricular leads were found to be completely severed and lying free in the surrounding tissue. The sections of these leads from the pacemaker generator to the wall of the pocket were removed. However, the internal sections of these leads from the wall of the pocket to the myocardium could not be located and removed because of adhesions secondary to earlier operations. Upon interrogation, the generator was found to be nonfunctional and was removed. The atrial lead was able to pace the ventricle with a threshold of 2.8 mA. An R-wave of greater than 20 mV with an impedance of 670 ohms was observed in the atrial lead; however, this lead was abandoned since the insulating layer was broken on one side. This lead was capped and patched. Two epicardial screw-in leads (Model 6917–53T, Medtronic) were placed on the inferior surface of the right ventricle via a subcostal approach. Threshold data for the replacement leads was 1.6 V, 3.1 mA with an R-wave of 7–8 mV and an impedance of 930 ohms. These leads were connected to a new VVI generator (Model 1204 META11, Teletronics). The pacemaker settings consisted of a pulse amplitude of 3.7 V, a pulsewidth of 375 msec, a rate of 50 beats/min, a sensitivity of 2 mV, and a refractory period of 300 msec. The generator was replaced in the abdominal pocket of the old generator, and unipolar electrosurgery (Force 2, Valleylab) was used to provide hemostasis. A disposable, pre-gelled dispersal pad was placed on the patient's left anterior thigh. The electrosurgery unit (ESU) was used in a blend mode with cutting set at 20 and coagulation set at 30. The ESU had a return electrical monitoring system with an audible alarm for fault.

During the use of the ESU, cardiac rhythm suddenly changed to ventricular fibrillation. Nitrous oxide and isoflurane were discontinued to provide 100% oxygen. Defibrillation was accomplished with external paddles at 300 joules, and a rhythm of ventricular tachycardia was then established. Ventricular tachycardia subsequently converted to sinus tachycardia following intravenous administration of a bolus of lidocaine (1.5 mg/kg). Heart rate subsequently decreased to 85 beats/min. The entire episode lasted approximately 3 minutes. Dopamine was started at 5 μg/kg/min to treat mild hypotension (blood pressure 90/50 mmHg). The pacemaker generator was re-evaluated and was noted to be functioning properly. The ESU was examined for faults postoperatively and found to be functioning normally. No other instruments that could have provided conduction for electrical current were in the field. The generator was repositioned in the pocket, and the site was closed. For the remainder of the intraoperative period, the patient was hemodynamically stable. The patient was left intubated and transferred to the coronary care unit for recovery and further observation. Examination of the patient revealed no areas of skin burns. Dopamine delivery was slowly decreased and the patient was successfully extubated the same evening. Myocardial infarction was ruled out by serial cardiac enzymes and electrocardiograms, and the patient was discharged home 4 days later without any further complications.

DISCUSSION

Ventricular fibrillation is a rare complication of electrosurgery in patients with cardiac pacemakers [6, 7]. In the present case, we suggest that ventricular fibrillation occurred because of the direct conduction of current from the tip of the electrosurgery applicator to the surrounding tissues, and then via the exposed ends of the inner portion of severed pacing wires to the myocardium. This event can be classified as microshock. Figure 1 schematically depicts the proposed pathway for conduction of electrosurgery current, in this case resulting in ventricular fibrillation.

As opposed to macroshock, which results from large amounts of current coming into contact with the surface of the body, microshock is due to very small amounts of current directly applied to the heart, producing a large current density. Various factors that determine whether a particular episode of electric shock will result in ventricular arrhythmias include duration of contact, intensity of

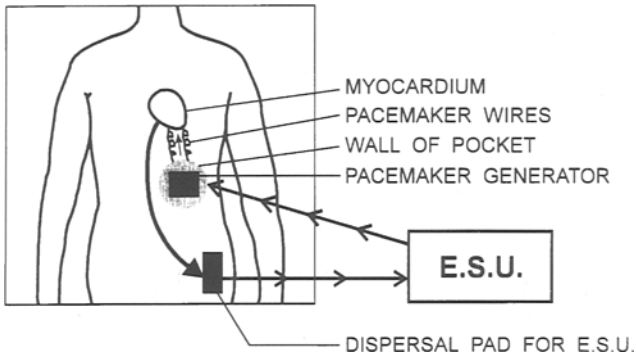

Fig 1. Schematic pathway for conduction of electric current from electrosurgery unit (ESU) to the myocardium. The electric current was conducted from the tip of the electrosurgery applicator to the tissues surrounding the pacing wires and then via the pacing wires to the myocardium. The return pathway was completed from the myocardium through the body tissues to the ESU dispersal pad and then to the ESU.

the current and type of electricity [7–10]. Ventricular fibrillation occurs only if the duration of the shock is 10 msec or greater [8]. Direct current requires several times more energy to cause ventricular fibrillation than does alternating current. One hundred to 300 mA of 60 Hz alternating current is required for macroshock, while only 100 uA of current applied directly to the heart can lead to ventricular arrhythmias [9]. Very large currents, in the range of amperes, are useful for defibrillation. Also, there is a wide individual variation in the fibrillation threshold, depending on body habitus. Electrolyte imbalances such as hypokalemia and hypomagnesemia, as well as previous myocardial infarction, can perhaps lower the fibrillation threshold. Raftery and colleagues [10] were able to produce ventricular fibrillation in some of their patients with a current of only 80 uA applied directly to the myocardium. This amount of current is far below the threshold of human perception and cannot be detected by line isolation monitors, which alarm at much higher current levels (2 to 3 mA).

ESUs generate very high frequency current (500,000 to 1,000,000 Hz). When this high-frequency current is used to cut or coagulate tissues, it is usually harmless to the heart as it has low tissue penetration and passes across the pericardium without causing ventricular arrhythmias. However, when an electrosurgery applicator is used, an arc may occur between the tissue and the tip of the applicator (the active electrode). Foster [11] noted that Pierce had shown that the presence of an arc in an electrosurgical current caused the energy spectrum to widen, resulting in enhanced lower frequency components, including frequencies below 10 kHz. This modified current may produce malignant ventricular arrhythmias if conducted directly to the heart. In this case, 500-kHz bursts of sinusoid current at a duty cycle of 25 to 50% recurring at 31 kHz are thought to have traveled through tissue into the ends of fractured leads.

Microshock can lead to both ventricular tachycardia and/or fibrillation. Central arterial or venous catheters and myocardial or esophageal pacing and sensing electrodes represent potential hazards for microshock. In patients with such instrumentation, microshock should be suspected as the etiology for sudden and unexplained ventricular arrhythmias. Proper precautions must be taken when handling such devices. These include wearing latex or rubber gloves, using monitors with electrically isolated inputs and outputs, and covering contact points of external leads with a nonconducting material such as a surgical glove to prevent conduction of leakage current. Precautions necessary in patients with pacemakers are described in detail elsewhere [12].

Another possible mechanism that can lead to ventricular fibrillation is competing rhythm. Demand pacemakers in the presence of prolonged electromagnetic interference from the continuous use of electrosurgery are programmed to change to an asynchronous mode (pacemaker fires at a fixed rate irrespective of the spontaneous cardiac activity). Hayes et al. [13] have shown that pacemakers revert to this alternate mode in 21% of procedures in which electrosurgery is used. Competing rhythms may occur in the patient with spontaneous rhythm when the pacemaker is in an asynchronous mode. Ventricular tachycardia or ventricular fibrillation can result if the cardiac pacemaker stimulus occurs during the vulnerable period of the cardiac cycle and the patient has concomitant underlying susceptibility to arrhythmias. This mechanism was thought to be unlikely in the present case, as the electrosurgery was used for only a very brief period and no pacer spikes were observed on the electrocardiogram. The cardiac pacemaker should not be routinely reprogrammed to the asynchronous mode by placing a magnet on the pulse generator. The ESU should be used only in short bursts so as not to produce pacemaker inhibition, and to prevent potential occurrence of malignant ventricular arrhythmias.

In conclusion, ventricular arrhythmias can occasionally develop in patients with cardiac pacemakers if an ESU is used without proper precautions. Careful, continuous monitoring of cardiac rhythm and pulse rate are essential for the safe conduct of an anesthetic in these patients. The present case report describes a unique potential mechanism, stimulation of myocardial leads by electrosurgery, whereby malignant ventricular arrhythmias can be induced.

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