

Effect of Electrical Cardioversion on Stented Coronary Artery

Research Article

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Received 22 July 2009; Accepted 5 October 2009

Abstract: Direct current cardioversion, which produces electrical energy, is highly effective for the termination of cardiac arrhythmia and sometimes is indicated in patients with coronary artery stents due to arrhythmias. Only a few reports have been published describing the potential adverse interactions between foreign bodies and electrical cardioversion. The aim of this animal study was to investigate the acute effect of repeated external defibrillation on coronary artery tissue and adjacent myocardium at the implantation site of coronary stents. Custom-made stainless steel stents were implanted in the coronary arteries of 7 dogs. Rapid ventricular pacing was performed to induce ventricular fibrillation. Defibrillation was achieved [5 J/kg; n=2 and 8 J/kg; n=3]. In 2 animals, coronary stent was implanted but defibrillation was not performed [control group]. The animal's heart were excised and sent for microscopic examination. The light and electron micrographs of heart muscles showed no histological and ultrastructural changes in defibrillated and control dogs. It is concluded that nickel coating provides good resistance to heat in coronary stents and repeated defibrillation does not cause histopathological changes typical of thermal injury at the implantation site of coronary stent.

Keywords: Cardioversion • Coronary • Injury • Myocardium • Stent

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1. Introduction

Direct current cardioversion, which can produce electrical energy, is highly effective for the termination of cardiac arrhythmia [1-4] and sometimes is indicated in patients with coronary artery stents due to malignant rhythm disturbances. Electrical energy can be converted into thermal energy in metal materials such as coronary stents, which are stainless steel mesh tube. It is hypothesized that electrical cardioversion may heat the coronary stents and subsequently produce injury in the coronary artery and myocardium at the implantation site of coronary stent. To date, there is no published report regarding the potential adverse interactions between coronary artery stents and electrical cardioversion. The aim of this experimental study was to investigate

the acute effect of repeated external defibrillation on coronary artery tissue and adjacent myocardium at the implantation site of the coronary stent.

2. Material nad Methods

This animal study was approved by the local Institutional Animal Care and Use Committee and conforms to the guidelines established in the "Position of the American Heart Association on Research Animal Use" adopted by the American Heart Association on November 11, 1984.

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2.1. Animal Preparation and Instrumentation

Seven Mongrel dogs of either sex (mean weight 28 kg) were pre-anesthetized with 20 mg/kg IM ketamine hydrochloride. Anesthesia was induced with sodium pentobarbital [10 mg/kg IV bolus] and maintained by halothane inhalation. Once anesthesia was induced, the animals were endotracheally intubated and ventilated with a respirator with supplemental oxygen. Blood pressure and surface ECG leads were continuously displayed on a physiological monitor.

2.2. Stent Implantation

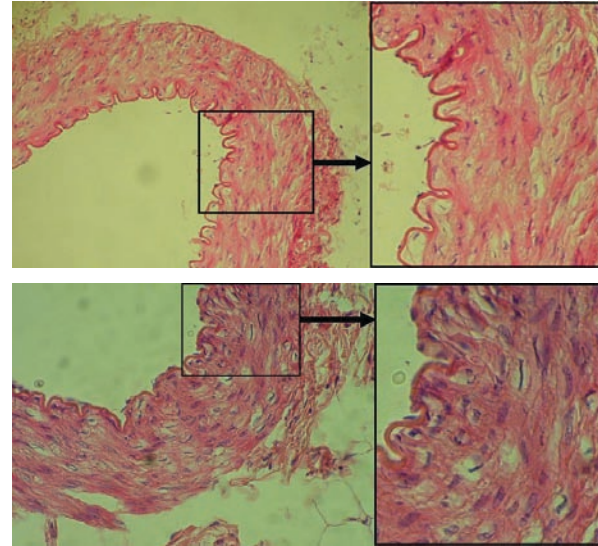
A custom-made, stainless steel, slotted-tube stents (BeStent, Medtronic, Inc) of 15-mm length and 2.5 mm expanded diameter were used in the present study. Arterial access was established by cut-down of the right carotid artery and insertion of a 7F arterial sheath with hemostatic valve under sterile conditions. Heparin sodium (100 IU/kg IV) was administered. Coronary artery stenting was performed by positioning a 0.014-in guidewire in the distal coronary artery and advancing the manually crimped stent on a 3.0-mm balloon catheter. The stent was implanted in a selected coronary artery segment.

Inflation pressures were chosen according to the manufacturer-derived compliance curves. The animals received 300 mg acetylsalicylic acid; from this group, a total of 7 dogs were instrumented. Rapid ventricular pacing was performed to induce ventricular fibrillation. Cardioversion was achieved with repeated defibrillation, as is usually done in patients to successfully terminate cardiac fibrillation. For successful defibrillation, 5 J/kg was given for 3 times in 2 animals while and 8 J/kg was administered for 2 times in 3 animals. Control group consisted of 2 animals in which coronary stents were implanted, but defibrillation was not performed.

2.3. Light Microscopic Examination

The animals were euthanized 2 hours after stent implantation. The heart was arrested by injection of 20 mL KCl into the aortic root. The heart was excised and fixed in 300 ml buffered 4% formaldehyde. A few days later, the coronary arteries were carefully dissected free, and the stented coronary artery with adjacent myocardium was embedded in polymethylmethacrylate. The fixed coronary artery segments and adjacent myocardium were sectioned transversely, cut into 800- μ m sections, and polished to a thickness of 100 μ m. Histological evaluation was performed by an observer unaware of the cases and controls. Within the stented coronary segments, 3 sections corresponding to the proximal, middle, and distal parts of the stent were examined.

Figure 1, 2. Light microscopic examination of the coronary arteries of defibrillated dogs. No acute histo-pathological change typical of tissue injury was detected in the defibrillated dogs [Right panel is the magnified view of the left panel].



2.4. Electron Microscopic Examination

The animals were euthanized 2 hours after stent implantation. The heart was arrested by injection of 20 ml KCl into the aortic root and excised. Tissues were fixed in 3% glutaraldehyde in 200 mM sodium phosphate buffer, pH 7.4, for 3 h at 4°C for electron microscopic examination. Materials were washed with the same buffer and post-fixed in 1% osmium tetroxide and in sodium phosphate buffer, pH 7.4, for 1 h at 4°C. Tissue samples were washed with the same buffer for 3 h at 4°C, dehydrated in a graded ethanol series and embedded in Araldite. Thin sections were cut with a Reichert OM U3 ultramicrotome. The sections were viewed and photographed under a Jeol 100 CX II transmission electron microscope at 80 kV.

3. Results

3.1. Light Microscopy

Histological examination of the coronary tissue, as performed by a blinded pathologist, revealed no evidence of tissue injury at the site of stent implantation in cases of animals in whom stent was implanted and defibrillation was done [$n=5$] when compared to the controls [$n=2$] (Figure 1, 2). Histological examination of the myocardial tissue adjacent to the implantation site of stent, revealed no evidence of tissue injury in cases compared to the controls (Figure 3). No evidence of stent thrombosis was reported in cases and controls.

Figure 3. Light microscopic examination of the myocardium of defibrillated dogs. No acute histo-pathological change typical of tissue injury was detected in the defibrillated dogs [Right panel is the magnified view of the left panel].

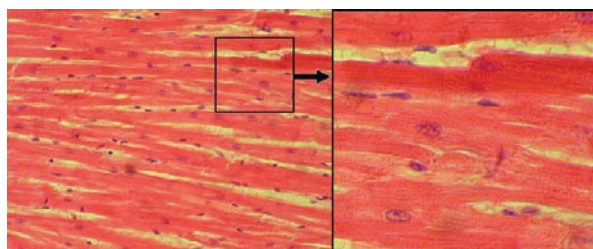
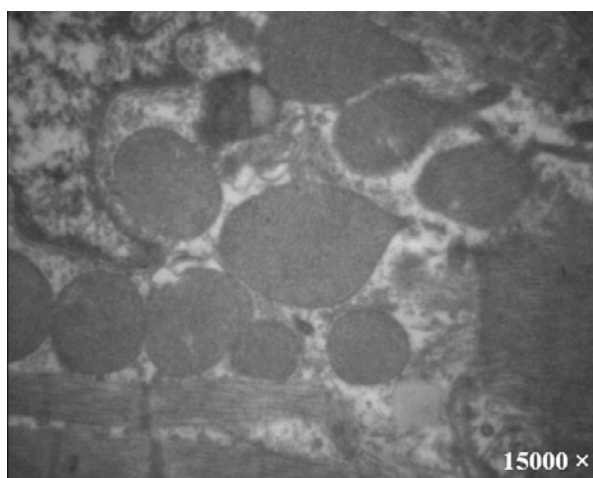


Figure 4. Electron micrographs of heart muscles of defibrillated dogs. Note that the mitochondria is intact.



3.2. Electron Microscopy

The electron micrographs of heart muscles of defibrillated dogs and control dogs showed no evidence of ultrastructural changes. Sub-cellular structures including mitochondria (Figure 4), cell membrane (Figure 5), nucleus (Figure 6), intercalated disc (Figure 7), sarcomere (Figure 8) and Golgi complex (Figure 9) showed no difference between defibrillated and control dogs.

Figure 5. Electron micrographs of heart muscles of defibrillated dogs. Note that the cell membrane is intact [white arrow].

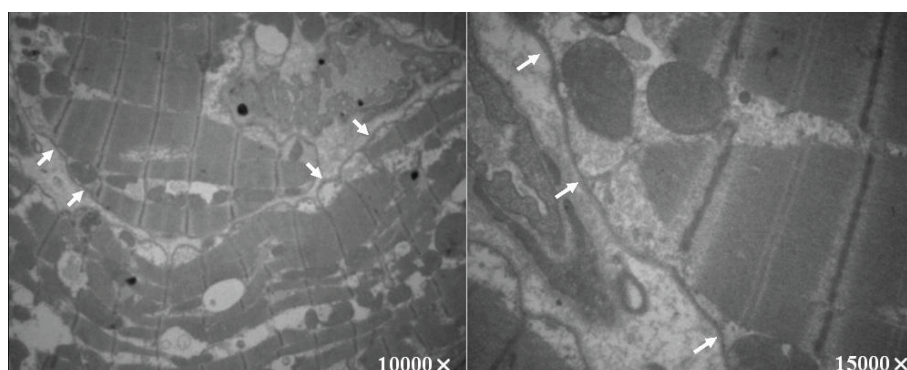
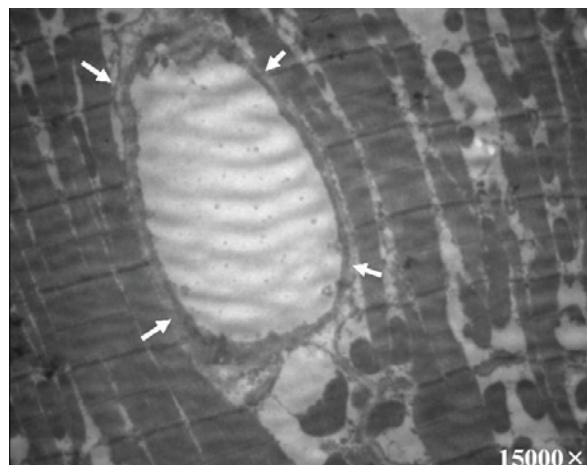


Figure 6. Electron micrographs of heart muscles of defibrillated dogs. Note that the nucleus is intact [white arrow].



4. Discussion

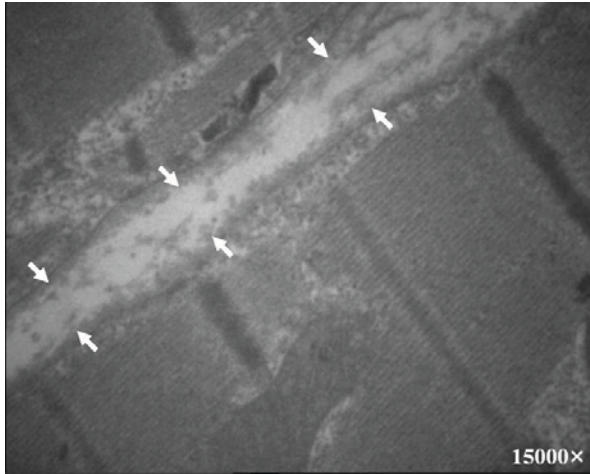
4.1. *In situ* foreign bodies and electrical cardioversion

External cardioversion may result in pacemaker dysfunction even in modern devices [5] and can induce thermal injury at the implantation site of atrial and ventricular pacemaker leads [6,7]. One study has found that repeated defibrillation did not cause acute histo-pathological changes typical of thermal injury at the implantation site of cerebral electrodes in pigs [8]. Another study described a patient with a descending thoracic aortic aneurysm that was successfully treated with endograft repair yet developed graft failure (endoleak) immediately after a single cardioversion [9].

4.2. Coronary stent and electrical cardioversion

A coronary stent is a stainless steel mesh tube with slot. Nickel constitutes an important component of stainless steel stents. Specific heat capacity is the heat required

Figure 7. Electron micrographs of heart muscles of defibrillated dogs. Note that the intercalated disc is intact [white arrow].



to raise the temperature of 1 kg of a substance by 1°C (or one Kelvin). The thermal conductivity of a material is the quantity of heat that passes in unit time through the unit area of a plate. Nickel has a specific heat capacity of 440 Joule per kilogram-Kelvin ($\text{J} \cdot \text{kg}^{-1} \cdot \text{K}^{-1}$) and a thermal conductivity of 90 watts per meter-Kelvin ($\text{W} \cdot \text{m}^{-1} \cdot \text{K}^{-1}$). With comparison between thermal characteristics of nickel and other metals, it is concluded that nickel-based materials are poor heat conductors. Therefore, nickel coating provides good resistance to heat in coronary

stents. In the present study, we evaluate the possible interaction between coronary artery stents and electrical cardioversion. We found that repeated defibrillation does not cause thermal injury at the implantation site of coronary stents and adjacent myocardium in a canine heart model. To the best of our knowledge, there is no published report describing the interaction between coronary artery stents and electrical cardioversion.

4.3. Electrical cardioversion and drug eluting stents

The Cypher drug-eluting stents are constructed out of stainless steel and are coated with *PARYLENE C* which is a polymer. *PARYLENE C* has a thermal conductivity of 82 watts per meter-Kelvin ($\text{W} \cdot \text{m}^{-1} \cdot \text{K}^{-1}$) [in comparison to 90 watts per meter-Kelvin for Nickel in bare metal stents]. Therefore, *PARYLENE C* is a poor heat conductor. Although polymer-coated, drug-eluting stents were not tested in our study, but with comparison between the nickel and polymer heat conductivity, one would expect that polymer coating would provide good heat resistance in drug-eluting stents if polymer and nickel heat conductivity were to be compared.

Figure 8. Electron micrographs of heart muscles of defibrillated dogs. Note that the sarcomeres and z-lines are intact.

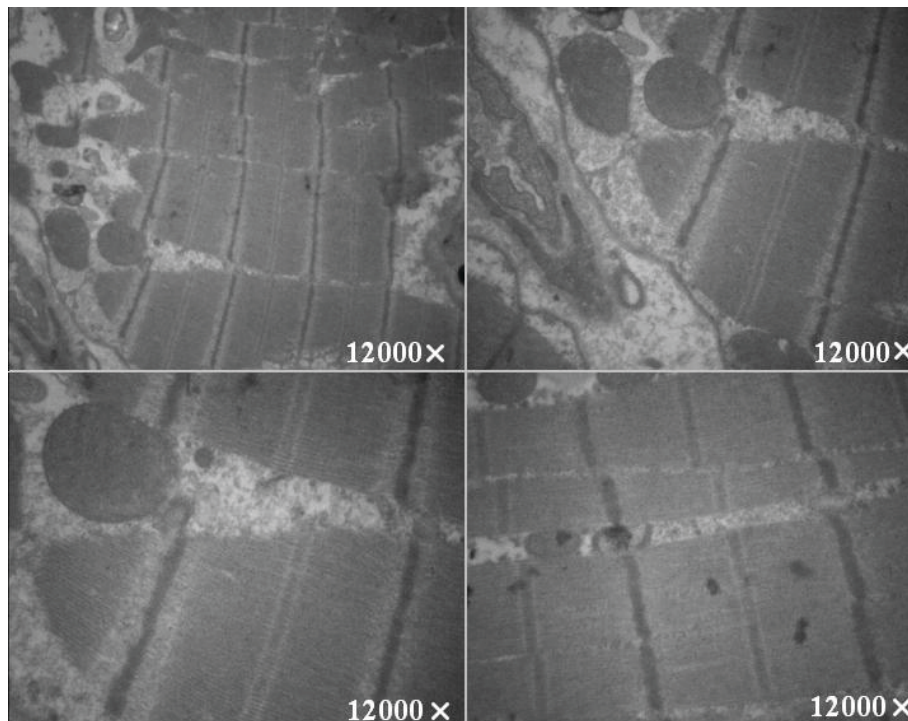
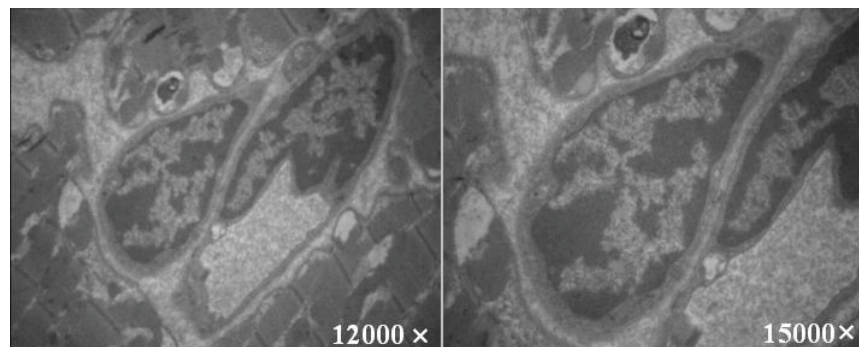


Figure 9. Electron micrographs of heart muscles of defibrillated dogs. Note that the digestive system [Golgi complex] is intact.



5. Conclusion

In conclusion, nickel coating provides good resistance to heat in coronary stents and repeated defibrillation does not cause thermal injury at the implantation site of coronary stent or adjacent myocardium.

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