Implantable cardiac defibrillator lead failure or myopotential oversensing? An approach to the diagnosis of noise on lead electrograms

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The appearance of noise on electrograms (EGMs) recorded from the lead of an implantable cardioverter-defibrillator (ICD) may be owing to oversensing of myopotentials, insulation breach, conductor coil fracture, loose set screw, or electromagnetic interference (EMI) from an external source. The extraneous noise may lead to inappropriate shocks or inhibition of pacing. We describe two cases of pectoral myopotentials oversensing in patients with ICD and an approach to distinguish among the various extraneous noises recorded on EGMs. A systematic approach to identify the cause of the noise is important to render an appropriate treatment, which might include simple device re-programming or require re-operation and lead revision or replacement.

**KEYWORDS**
Implantable cardioverter-defibrillator; Oversensing of myopotentials

Introduction

The appearance of noise on electrograms (EGMs) recorded from the lead of an implantable cardioverter-defibrillator (ICD) may be owing to oversensing of myopotentials, insulation breach, conductor coil fracture, loose set screw, or electromagnetic interference (EMI) from an external source. The extraneous noise may lead to inappropriate shocks or inhibition of pacing. Some of these problems can be overcome by simply re-programming the device, whereas others require re-operation and lead revision or replacement. Thus, the ability to distinguish among the various possibilities is of crucial importance. We report two cases which illustrate some of the subtleties in the differential diagnosis of lead noise.

Case 1

A 63-year-old man with a history of anterior myocardial infarction, severe left ventricular dysfunction, and sustained ventricular tachycardia underwent dual-chamber ICD implantation (Vitality model T180 Guidant, St Paul, MN, USA). An atrial bipolar lead (model 4480, Guidant) was implanted in the right atrial appendage and an Endotak™ Reliance SG dual-coil, integrated bipolar lead (model 0185, Guidant) was positioned at the apex of the right ventricle (RV). Eight months later, the patient returned from a routine device follow-up without any complaints. Interrogation of the device revealed sensed R-waves of 7.1 mV, an RV pacing threshold of 1.0 V at 0.5 ms, and pacing lead impedance of 420 Ω, values which were unchanged from prior follow-up. Interrogation of the device revealed multiple episodes of high ventricular rate falling in the ventricular fibrillation (VF) zone. Inspection of the EGMs stored from these episodes revealed high-frequency non-physiological signals on both the integrated RV tip-to-distal coil bipolar sensing EGM and the high-voltage (shock) EGM recorded between the distal coil and pulse generator (Figure 1A). The noise could also be reproduced in the clinic by isometric left arm exercise (Figure 1B). Therefore, there was high suspicion on lead or connector problems vs. myopotential oversensing. Revision of the ICD system was performed. During surgical exploration, no loose set screws or reversal of pins was noted but the high-frequency non-physiological signals were still present on the ventricular and shock EGMs (Figure 1C). Prior to lead removal, different sensing configurations were examined in order to determine the source of the extraneous noise. The non-physiological signals were still present on the shock EGM even after the distal or the proximal coil ports were plugged. However, there was no evidence of noise when the pulse generator was removed from the pocket and similarly when the pacing system analyser cables were connected directly to

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the proximal and the distal coils without involving the can (Figure 2). The lead was replaced with a new Endotak™ Reliance 5G single-coil integrated bipolar lead (model 0181, Guidant), which was positioned on the RV septum. High-frequency noise was no longer present on the sensing EGM but was still visible on the shock EGM (Figure 3), which did not affect sensing. Therefore, the source of the remaining noise was the pectoral muscle myopotentials near the pocket, as it was present on the shock EGM only when the can was part of the sensing circuit. Whereas the noise on the sensing EGM was related to diaphragmatic myopotentials as it resolved after lead reposition, and the fact that it was present only on the sensing EGM. Examination of the originally implanted lead by the manufacturer revealed intact insulation integrity and normal electrical performance.

Case 2

An 82-year-old man with chronic atrial fibrillation following remote mitral valve replacement subsequently developed a non-ischaemic dilated cardiomyopathy and class II symptoms of congestive heart failure, prompting referral for implantation of a single-chamber ICD. A dedicated bipolar dual-coil active fixation lead (model 1581, St Jude Medical, St Paul, MN, USA) was implanted and connected to St Jude (model V193, St Jude Medical) single-chamber ICD. At the time of implantation, the sensed R-waves were 8.2 mV, the pacing threshold 0.6 V at 0.5 ms, the pacing lead impedance 624 Ω, and the defibrillation threshold (DFT) < 14 J. Two tachycardia zones were programmed; a monitor-only zone for rates 150 b.p.m. and a VF zone for rates > 180 b.p.m. He did well post-operatively and received no therapies from his device over the next 12 months. However, during his most recent ICD interrogation, an episode falling in the monitor zone had been logged by the device. The bipolar EGM recorded between the RV tip and RV ring stored during this episode was free of noise (V sense/pace, Figure 4), but noise was present on the bipolar EGM recorded between the superior vena cava (SVC) coil and pulse generator (SVC to can, Figure 4). During inspection of the real-time EGM, faint evidence of the same noise could be seen during manipulation of the pocket. However, all other aspects of device function, including pacing lead impedance, sensed R-wave, and pacing threshold were unchanged. Because of concern about possible conductor coil fracture or insulation breach, a chest radiograph was obtained which revealed twisting of the lead in the pocket. This was not present the day after device implantation, again raising the suspicion of conductor coil fracture. Preparations were made to explore the pocket and, if necessary, replace the lead.

Prior to surgical exploration, the patient was brought to the hospital to undergo DFT testing. Inspection of the real-time SVC to can EGM and RV coil to can signals revealed noise that was reproducible by isometric left arm exercise (Figure 5), but when the configuration was programmed from RV coil to SVC coil or from RV tip to SVC coil, no noise was seen. Defibrillation threshold testing was carried out, yielding a DFT of < 15 J, with a high-voltage lead impedance of 37 Ω. As all aspects of the device and the lead function were normal and since noise was only seen when the pulse generator was part of the collection set-up, it was concluded that this noise represented myopotential signals originating in or near the pocket and preparations for re-operation were cancelled.

Discussion

Myopotentials are high-frequency, low-amplitude electrical transients generated by skeletal muscles, including
inter-costal muscles or the diaphragm. Myopotentials appear as noise on EGMs recorded from an implanted device and must be distinguished from other potential sources of noise (Table 1). When myopotentials are sensed by an implanted pacemaker or defibrillator, inhibition of pacing or inappropriate detection of VF may ensue. Inhibition of ventricular pacing by oversensing of myopotentials was relatively common with unipolar pacemakers which typically used the pacing lead tip electrode as the active cathode and the pulse generator itself as the indifferent electrode or ground.1,2 Such a configuration is referred to as a 'single-ended input'; if myopotentials are generated only by the skeletal muscle of the chest wall originating in the vicinity of the pulse generator, the signals will be amplified in such a system and will appear as noise.

Pacemakers and defibrillators utilize bipolar sensing and rely on differential amplification and ‘common mode rejection’ to avoid sensing pectoral myopotentials. Three inputs were utilized in differential amplifications: the tip electrode, the ring electrode, and the can which still functions as the indifferent electrode. With this configuration, the noise generated from the pectoral myopotentials was common to the two signals: tip-to-can and ring-to-can. The noise, therefore, was cancelled out yielding only the tip-to-ring signal. Implantable cardiac defibrillators typically use the distal RV coil as the indifferent electrode in the sensing circuit rather than the can. Thus, pectoral muscle myopotentials generated in or near the pocket should not affect the sensing circuit. However, ICDs may still detect myopotentials generated by inter-costal muscles or the diaphragm, particularly if the ICD lead is an integrated bipolar rather than true bipolar as was seen in case 1. Integrated bipolar sensing is more susceptible to diaphragmatic oversensing because the large area of the RV shocking coil is shared as the anode for sensing.3,4 If the device’s programming leads to obligate ventricular pacing and/or the device utilizes automatic gain control (exclusive to Boston Scientific defibrillators) rather than auto-adjusting

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**Table 1** Differential diagnosis of myopotentials and diagnostic clues

| External electromagnetic interference | • Noise seen on all sensing channels |
| Lead/connector problem (header, adapter, or set screw) | • History of encounter with an EMI source |
| • It occurs during a small fraction (<10%) of the cardiac cycle |
| • It often saturates the amplifier |
| • The pacing-lead impedance is abnormal and intermittent |
| • Chest X-ray may reveal loose setscrew in the header |
| Lead fracture/insulation breach | • Abnormally high or low impedance |
| • Evidence of fracture on a chest X-ray |
| • Noise during manipulation of the pocket |
| Defibrillation leads reversed in the header | • Large P-waves on the shock EGM |
| • Unusually high DFT |
| Diaphragmatic oversensing | • Noise seen only on the ventricular or shock EGM |
| • Active diaphragmatic manoeuvres including deep inspiration, expiration, Valsalva, and coughing |
| Skeletal muscle oversensing | • Provocative skeletal isometric exercise involving the upper extremities and abdominal muscles |
| • Absence of noise when can is removed from the sensing circuit |

EMI, electromagnetic interference; EGM, electrogram; DFT, defibrillation threshold.
sensitivity, it may predispose to myopotential oversensing, especially at long RR intervals. Such oversensing sometimes, but not always, can be corrected by manually adjusting the sensitivity without impairing the detection of VF.

In the first case, both diaphragmatic and pectoral myopotentials were oversensed by the device. The diaphragmatic myopotentials were sensed on the integrated bipolar sensing EGM and were brought out by provocative manoeuvres. Proximity of the lead to the diaphragm, resulting from the apical position of the lead, contributed to the oversensing. Sensing of this noise on the ventricular EGM led to inappropriate detection of VF. Once the position of the lead was changed to a more septal location, the noise disappeared from the sensing EGM and was present only on the shock EGM (distal coil-to-can; Figure 4), revealing the presence of pectoral myopotentials. The persistent presence of noise on the shock EGM after the repositioning of lead was due to the distance between the distal coil and the can.

In both the presented cases, pectoral myopotentials were not detected on the rate-sensing electrode recorded between the RV tip and ring electrodes or SVC coil and RV coil, but rather on the EGM recorded between the distal or proximal coils and ICD pulse generator. Once the generator is removed from the sensing circuit and is not in contact with the skeletal muscle near the pocket, the noise is no longer present. This phenomena was confirmed by connecting the pacing system analyser cables to the proximal and the distal coils. Although the noise did not lead to inappropriate detection of VF or inappropriate inhibition of ventricular pacing because they were found only on the shocking EGM, its appearance did raise concern about possible insulation breach or conductor coil fracture. If insulation failure or conductor coil fracture had been present, one should have seen the same noise when other EGM configurations were tested. The observations in both the described cases indicate that the noise was only seen when the pulse generator itself is a part of the circuit. The DFT and high-voltage lead impedances were unchanged from the implant values indicating that this noise represents interleaved noise between the RV tip and ring electrodes or SVC coil and RV coil.


