We experienced 2 cases in which oversensing of a particular noise after the implantation of an implantable cardiac device was observed in the acute phase. These were unusual cases in which the noise exhibited a low frequency pattern and appeared several hours after the implantation, but disappeared within 1 week. Here we present these cases and the details of an experiment investigating the origin of the noise and the methods for its prevention.

The noise in these cases led to pacing inhibition and could have induced an inappropriate shock due to oversensing, but its morphology and electromagnetic interference were atypical for a lead failure or myopotentials. The noise spontaneously disappeared from the analysis of the data stored in the device. In an experiment based on the Irnich model, in which it was assumed that blood invaded a damaged grommet, low frequency noise occurred which was similar to the noise in the two cases. We concluded that care must be exercised when handling grommets.

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Introduction

In patients with a pacemaker or implantable cardioverter defibrillator (ICD), noise may cause oversensing and lead to inappropriate pacing inhibition as well as inappropriate ICD shocks. Factors involved in the oversensing include physiological noise such as T waves and myopotentials, and non-physiological noise such as electromagnetic interference (EMI) and lead fractures. However, we can distinguish between these types of noise by examining the relationship of the noise to the cardiac cycle. Extracardiac noise does not exhibit a constant relationship, but noise from physiologic signals other than myopotentials originates from intracardiac sources. Extracardiac noise has many possible causes, such as EMI, myopotentials, lead fractures, and connector (header, adapter, or set screw) prob-
lems, and it is difficult to identify the origin of the noise in individual cases.\textsuperscript{3,4)}

We experienced cases in which failure of the ventricular pacing occurred with the pacemaker. We also observed an episode in a patient with an ICD in whom oversensing occurred after transient noise in the acute phase after the implantation. Though these cases were at-risk patients, the noise was atypical for nonphysiological noise. We did not apply any intervention, and during our follow-up of the device data, the noise spontaneously disappeared. Here we describe 2 cases and the results of an in vitro experiment investigating the origin of this noise and the methods for its prevention.

**Case reports**

**Case 1**

An 80-year-old woman who had initially undergone a dual-chamber pacemaker implantation in 1996 for complete AV block underwent a third replacement of her pacemaker (Adapta\textsuperscript{TM}, Medtronic Inc., MN, USA). The atrial bipolar lead (model 4568; Medtronic Inc.) was screwed into the atrial appendage and the ventricle bipolar lead (model 4068; Medtronic Inc.) was screwed into the apex of the right ventricle (RV). The pacemaker was programmed as follows: DDD at a rate of 60–105 ppm, V amplitude 3.5 V at 0.4 ms, V sensitivity 2.8 mV, and a bipolar V sensing polarity.

We detected ventricular pacing failure on the electrocardiogram 4 hours after the implantation and then interrogated the data of the pacemaker (Figure 1). The interrogation of the pacemaker revealed that we could not sense an R-wave because of the absence of any intrinsic beats. The other parameters consisted of a V pacing threshold of 1.75 V at 0.4 ms and V pacing lead impedance of 1125 $\Omega$. These values were unchanged from those immediately after the implantation. No lead failure, such as a dislodgement, fracture, or insulation breach, was observed in the chest radiograph. Low frequency noise that was in synchrony with the exercise of the patient’s left upper extremity appeared during an inspection of the real-time EGM, and we considered that this resulted from oversensing (Figure 2A). The noise wave was about 10 mV, but it was difficult to adjust the R wave sensitivity because the R wave of the noise was higher in comparison than that of her escape beats. However, an adjustment from a bipolar V sensing polarity to a unipolar V sensing polarity led to the disappearance of the noise (Figure 2B).

One week later, the noise did not reappear when the sensitivity was temporarily adjusted to a bipolar V sensing polarity, even when the patient was prompted to move her left upper extremity. Furthermore, Holter monitoring did not produce any evidence of ventricular pacing failure. The measurement values such as the sensed waves, pacing threshold, and lead impedance remained unchanged during a follow-up of 1-year.

**Case 2**

This 53-year-old man had a history of cerebral infantile paralysis and had a sustained left upper extremity amputation in an accident. He underwent an ICD (Secura\textsuperscript{TM}; Medtronic Inc.) implantation for primary prevention of asymptomatic Brugada syndrome. An atrial bipolar lead (model 5076, Medtronic Inc.) was screwed into the right atrial appendage and an RV defibrillator lead (model 6947; Medtronic Inc.) was screwed into the septum of the RV. The ICD was programmed to AAI+ at 60–130 ppm. The tachycardia detection programmed shock therapy only for a VF zone; the V interval was 270 ms, and the lead integrity alert (LIA) was set to “OFF.”

We noticed one VT event recorded 3 hours after the implantation when we performed a scheduled measurement 1 week later. We observed noise with a low frequency noise pattern in the recorded EGM (RV tip to RV ring) and considered it to have been caused by an attempted inappropriate adjustment after oversensing (Figure 3). The sensing integrity counter (SIC), which cumulatively counts short (120–130 ms) V-V intervals and thereby typically indicates any intermittent oversensing of a noise, increased to 43 during a period of 1 week after the implantation. An interrogation of the ICD revealed a
sensed R wave of 10.4 mV, an RV pacing threshold of 0.625 V at 0.4 ms, and V pacing lead impedance of 627 Ω. These values were unchanged from those recorded immediately after the implantation, and the chest radiograph revealed no problems. In addition, we attempted provocative maneuvers such as an arm exercise, deep inspiration, and the Valsalva maneuver to evaluate the condition of the lead, but no noise appeared. Because the SIC decreased to 1 on the interrogation the next day, we did not change the setting of the ICD. Thereafter, the SIC remained at 0 during a follow-up of 1-year.

In vitro experiment

Materials and Methods

We designed an experiment to find out whether the grommet of an implantable cardiac device could be damaged and invaded by blood. In addition, it was necessary to simulate not only the blood invasion but also an environment in the pocket in which electrical differences would occur due to contact resistance variations, because the appearance of noise was sometimes transient after the implantation. A grommet is composed of silicone and forms the plug part for a torque wrench (Figure 4). We made a hole using an 18G injection needle and destroyed the grommet. The equipment used for the experiment, including a pacemaker (with a damaged grommet), lead, programmer, and pulse generator, were made by Medtronic Inc. (Figure 5). We filled a container with saline to simulate an environment of the device pocket, as that biomodeled by Werner Irnich; saline (0.18% w/t) was poured into a rectangular container to provide an electromagnetic simulation of the body tissue.5) We placed a circular electrode directly on top of the lead tip and applied a regular pulse of 60 ppm using a pulse generator. We fixed the telemetry wand of the programmer outside the container to record the real-time EGM under the following conditions.

(a) The pacemaker remained motionless in the container; we simulated a condition in which the patient was at rest after the invasion of blood into a pacemaker, but it was not enough to raise the contact resistance variation.

(b) We repeated movements of the pacemaker into and out of the container; we simulated a
condition in which the patient would move after the invasion of blood into a pacemaker, and electrical differences due to contact resistance variation occurred.

Results

The EGM recorded in the experiment showed (a) no oversensing of noise or (b) oversensing of a
low frequency noise pattern. When the pacemaker remained motionless in the container, the saline invaded the interior of the grommet and the baseline EGM was noisy (indicated by the arrow), but there was no oversensing (Figure 6a). On the other hand, when we repeated moving the pacemaker in and out of the container low frequency waves appeared on the EGM in synchrony with the maneuver (Figure 6b).

Discussion

Fortunately, in our 2 cases the noise disappeared spontaneously during the acute phase. We could get rid of the trouble only by fine adjustment of the devices. As for case 1, the noise disappeared by changing the sensing polarity from bipolar to unipolar. We suspected that a pin hole had been created only on the ring side of the grommet by the screw driver. Thus, the noise originated from the ring section and did not occur with unipolar pacing (tip-can).

However, such noise poses a serious risk of pacing failure or inappropriate ICD shocks, and if it had not disappeared, we would have had to consider a reoperation in the two cases. We therefore investigated the origin of the noise and possible methods for its prevention. Importantly, in both cases the noise appeared as a low frequency noise pattern on only 1 channel several hours after the device implantation. The pacing lead impedance and chest radiograph showed no problems and the noise disappeared over a period of 1 week.

Reported causes of noise include EMI, lead failures, connector problems, and myopotentials. However, the noise that we found was inconsistent in that it was a transient nonphysiological noise. EMI is reported to have several types of mechanisms, but usually differs from a low frequency noise pattern because it appears in all sensing channels and the noise pattern has an abrupt onset and is continuous. Lead failures could be diagnosed by the chest radiograph, or by variations in the lead impedance. Furthermore, lead fractures may be a factor with long-term complications. However, there were no abnormalities in either of our cases. Myopotentials are generated from skeletal muscle, including intercostal muscles and the diaphragm, but they have a high frequency and low amplitude. However, there was no abnormality of the chest radiograph or lead impedance, even at the point when the transient noise appeared. But we considered that the noise had a different origin, because the noise did not appear after the provocative maneuvers, including deep inspiration or the Valsalva maneuver, in the distant phase. Connector problems including the header, adapter and set screw are considered to have the same definition, and the symptoms resulting from those problems were similar to that in these cases. The cause and diagnosis were vague in these cases, and it was difficult to obtain conclusive evidence. Furthermore, unlike the situation with other noise, we could not rule out the possibility of an unknown cause. We therefore focused our attention on the possibility of damage to a grommet resulting from excessive

Figure 4 Grommet of the device.

Figure 5 Experimental use equipment; (a) programmer, (b) pulse generator, (c) Imrich model with a pacemaker and leads.
turning of the screw at the connection of the lead as the cause of this noise.

In an experiment, we confirmed that not only internal and external conduction, but also a potential difference was necessary for the appearance of the noise. The conduction was caused by the invasion of blood through a pinhole in a damaged part of the grommet, and the potential difference was caused by the body movement of the patient, and we speculated that the noise appeared under these conditions. Therefore, we considered that, over time, a condition developed that supported the conduction and potential difference, resulting in the appearance of noise after several hours. We surmise that slow body movements, such as exercising the left upper extremity, which was present in case 1, led to the development of a low frequency noise pattern. The disappearance of the noise within 1 week may be explained by the loss of a condition able to support the conduction as a result of insulation, such as blood clotting, absorbed effusion and the appearance of a dry pocket. We were unable to examine whether the invasion of blood resulted from damage to the grommet, how much bleeding there was, or the period during which the bleeding occurred. However, the conditions in the pocket have been reported to remain unstable during a period of about 1 month after the implantation due to pocket edema from an effusion including bleeding. Blood invasion might occur through a pinhole in the grommet, which may be affected by a wet pocket in the acute phase, but the blood is absorbed naturally over a few days in many cases. However, the actual cause is not clear because we did not resect the device. In addition, this

![Real time EGM during an experiment](image)
occurrence may be limited to devices manufactured by Medtronic Inc. because those used in both cases as well as the device we used in the vitro experiment were all made by the same manufacturer.

Assuming that oversensing of noise occurs as a result of damage to a grommet, we now will discuss the possible prophylactic measures. It is important to prevent damage to the grommet; however, the damage might not be noticed in many cases. Actually, this was consistent with our experience, as we had exercised care in the 2 cases. Therefore, we have to be more careful when handling a grommet. The grommet may be damaged by excessive turning or reverse turning of the screw with the torque wrench during a positional revision of a lead. If damage to the grommet is anticipated during a procedure, we recommend that a material such as a mounting medium should be preventively used. The use of an LIA was reported to be effective for reducing inappropriate shocks associated with noise and lead fractures in patients with an ICD.21) We feel that the LIA may provide protective benefits from inappropriate shocks in cases such as those with the noise experienced in the acute phase as in our cases. Of course, care should be taken when the LIA is triggered just after the implantation because the LIA might extend the interval for an appropriate shock. If a low frequency noise pattern regretfully appears after the implantation and no adequate diagnostic clue is obtained, we might avoid this situation by adjusting the polarity. In any case, observation using a suitable monitor and adequate measurements are necessary.

Conclusion

We experienced two cases of oversensing of a low frequency noise pattern, but the cause of the noise was difficult to ascertain. An experiment suggested that a low frequency noise pattern developed due to body movement in addition to damage to the grommet. Care in handling the grommets may be important for preventing inappropriate pacing inhibition and inappropriate ICD shocks.

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