Abrupt increase in impedance measurements as detected via remote monitoring: What is the cause?



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Introduction

A 57-year-old man with nonischemic dilated cardiomyopathy, New York Heart Association class III heart failure, and a narrow QRS complex was enrolled in an ongoing research study to assess the impact of cardiac resynchronization therapy (CRT) in patients with echocardiographically detected dyssynchrony and a narrow QRS complex (the EchoCRT study; http://clinicaltrials.gov/show/NCT00683696.). A Biotronik Linox Smart 65-18 implantable cardioverter-defibrillator (ICD) lead connected to a Biotronik Lumax 540 HF-T CRT-D device (Biotronik, Berlin, Germany) was implanted, and remote monitoring was enabled. According to study randomization, the pacemaker function was programmed to the VVI mode at 40 beats/min. In the absence of a landline phone, cellular monitoring was initiated.

On January 30, remote monitoring first conveyed an alert related to an abrupt increase in ICD pacing lead impedance. Multiple attempts to contact the patient were unsuccessful. The trends as reported by Home Monitoring are shown in Figure 1. What conclusions can be drawn about the cause of these abrupt changes in impedance?

Discussion

Measurement of lead impedance is one of the standard ways to measure lead integrity and function over time. The commonest causes of abrupt, large increases in lead impedance are conductor fracture or a connector problem, while abrupt decreases are indicative of insulation breach (current leak). Smaller changes in impedance can be seen owing to changes in a patient's physiologic state. Unipolar impedance changes occurring throughout the respiratory cycle or with changes in myocardial contractility have been incorporated

KEYWORDS Implantable cardioverter-defibrillator; Impedance; Remote monitoring; Death

ABBREVIATIONS CRT = cardiac resynchronization therapy; ICD = implantable cardioverter-defibrillator (Heart Rhythm Case Reports 2015;1:51–53)

into various rate-adaptive sensor technologies (minute ventilation, closed loop stimulation, etc), and transthoracic impedance trends are potentially useful in heart failure monitoring (OptiVol, CorVue, etc)

Remote monitoring has become a commonplace strategy in following patients with implanted pacemakers and defibrillators, and it facilitates early detection of device or lead malfunction. In this era of frequent ICD lead advisories (Fidelis, Riata, Riata ST, Quick-Flex, etc), remote monitoring provides an opportunity for the early detection of a lead problem before overt adverse clinical events. In this context, abrupt large increases in lead impedance usually suggest conductor fracture and prompt urgent intervention by health care providers.

In our patient, closer scrutiny of the Home Monitoring report indicated the following near-simultaneous changes (Figures 1 and 2):

- 1. a rapid increase in right ventricular pacing impedance up to 1500 Ω ;
- 2. a corresponding increase in shock impedance to $> 150 \Omega$;
- 3. a decrease in day-to-day heart rate from about 60–90 beats/min to a constant paced heart rate of 40 beats/min;
- 4. an abrupt increase in percentage of pacing from 0% to 100%;
- 5. a complete loss of activity (as detected by the device accelerometer); and
- 6. loss of detectable R waves (not shown in the figure but reported in the printout of the remote transmission).

A review of these findings led us to conclude that the patient had suffered electromechanical dissociation because no tachyarrhythmia was recorded and that the patient was deceased. The vertical line in Figure 1 coincides with January 31, the presumed date of death. A police search was initiated, and his body was subsequently discovered 2 days later. The postmortem interrogation of the device (Figure 2) confirmed all the findings of the initial remote monitoring alert. Device and lead analysis confirmed normal functioning of the ICD system.

We performed an exhaustive literature search regarding impedance changes immediately before death and found

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KEY TEACHING POINTS

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- This case demonstrates that data provided by remote monitoring must be interpreted cautiously.
- An abrupt increase in pacing impedance, although most often related to conductor fracture, can also be related to patient demise.
- In our case, multiple variables (such as sensed signal amplitudes, percentage of pacing, activity levels, and heart rate) had to be reviewed in conjunction with the impedance data to discern the true cause for an impedance increase—patient demise.

only 1 other such report,¹ although there is a modest body of work describing postmortem impedance changes in animal models. We found 1 reference to pacing impedance increase during dehydration in a living patient,² and whole body bioimpedance spectroscopy has been reported to be a reliable method of assessing hydration status in children.³ We also found data in a dog model that revealed increases in transthoracic impedance (measured between an esophageal

electrode and a skin patch) after death.⁴ Similarly, transabdominal impedance was noted to increase within the first 5 to 24 hours of death in a rat model.⁵ It may be postulated that progressive clotting of intravascular blood, coupled with insensible water loss after demise, may result in impedance increase, although the rapidity of the impedance increase seen in this case is difficult to explain.

For this model of Biotronik ICD, the daily average of the heart rate is a mathematical average of every heart beat over the preceding 24 hours. The daily average of pacing and shocking impedance measurements is a mathematical average of measurements made every 30 seconds using subthreshold stimuli. The daily average of activity is a mathematical average of accelerometer readings made every 10 ± 2 minutes. When a daily transmission is not possible (owing to nonproximity to the cellular transmitter), the next measurement is made a day later and reflects values only from the preceding 24 hours. As seen in Figure 2, there is a missing transmission on January 30. The plot simply "connects the dots" and provides an appearance of a "gradual" increase in impedance. The fact that the average heart rate and activity level at the end of January 31 show no overt change than those on prior dates, coupled with the fact that these values change to 40 beats/min (paced rate) and

Mean Ventricular Rate (bpm)

Mean ventricular rate (bpm)

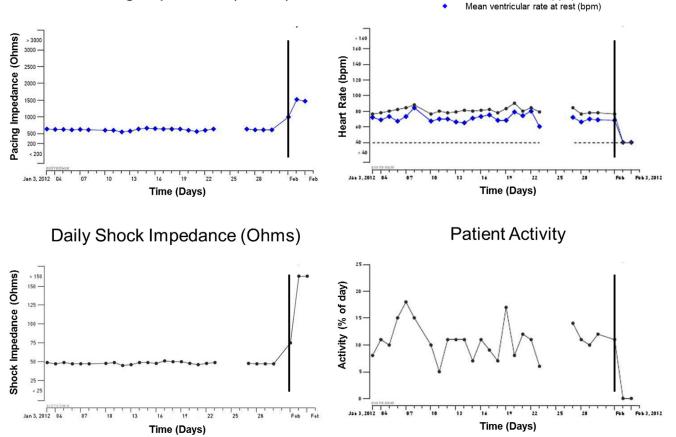


Figure 1 Detailed information from remote monitoring. Note that there is a missing data point on January 30 on all graphs, likely owing to nonproximity to the cellular transmitter (see text for details). This missing value is responsible for the appearance of a "gradual" increase in impedance values from January 29 to January 31 because the individual daily data points are simply connected by a straight line. bpm = beats/min; RV = right ventricular.

RV Pacing Impedance (Ohms)

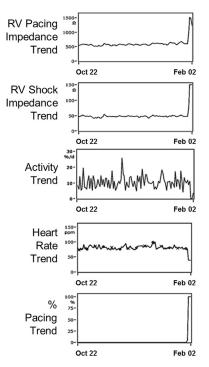


Figure 2 Device trends obtained at postmortem interrogation indicate near-simultaneous changes in pacing and shocking impedance measurements, heart rate and percentage of RV pacing, and patient activity and sensed P and R waves (see text for details). The terminal increase in the activity graph corresponds to the transportation of the patient's body to the morgue after demise. RV = right ventricular.

zero activity by the end of February 1, suggests that the time of death was close to the time of the transmission on January 31. The exact time course of impedance increase is not precisely seen, but the available heart rate and activity data suggest that the impedance increase may have *preceded* the actual demise by about 12–24 hours. If this can be validated, impedance changes may be a potentially useful marker of impending demise due to electromechanical dissociation.

No information was available on impedance changes from the atrial and left ventricular leads as the device was programmed to the VVI mode according to EchoCRT study randomization.

Conclusion

These findings suggest that increases in pacing and shocking impedance measurements occur around the time of death, possibly preceding death by several hours. If validated, these observations may play a useful clinical role in monitoring patients with implanted devices. Although our initial concern was about lead integrity, the combination of the abrupt increase in impedance and the loss of activity, loss of measured native signals, and 100% pacing at the base rate allowed us to come to the correct conclusion about this patient's demise. We were able to surmise the time of death from careful analysis of the trends. A sudden increase in multiple lead impedance parameters, coupled with the other changes described herein, should prompt consideration of patient demise rather than lead integrity issues.

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