

Shock-induced right ventricular pacing failure caused by a short circuit: Uncommon but life-threatening complication of the Riata lead



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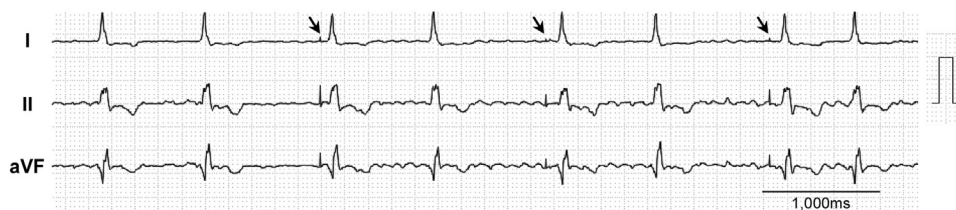


Figure 1 Surface electrocardiogram showing right ventricular pacing failure. The right ventricular pacing (arrows) at the programmed lower rate of 60 beats/min due to appropriate mode switching during atrial fibrillation did not capture the myocardium.

Introduction

The Riata and Riata ST implantable cardioverter-defibrillator (ICD) leads, a narrow caliber silicone lead without an outer jacket, have been shown to be prone to structural^{1–4} and electrical failure.^{1–3,5–8} While much interest has been shown in potentially lethal high-voltage (HV) failures^{5,9,10} caused by electrical short circuits due to a breach in the ethylene tetrafluoroethylene (ETFE) insulation of the defibrillation cables,⁸ the clinical manifestation of Riata/ST lead failures has not been fully elucidated. In this report, we present a case of shock-induced Riata lead pacing failure and discuss clinical approach to the management of Riata/ST leads.

Case report

A 59-year-old man with dilated cardiomyopathy who had a dual-chamber ICD (ATRAS DR, St Jude Medical, Inc, Sylmar, CA; right ventricular [RV] lead, Riata 1570-65, St

Jude Medical) implanted in 2005 visited the emergency department after experiencing 4 successive sets of shocks (1, 6, 6, and 6 shocks, respectively) within an hour in 2013. The surface electrocardiogram showed atrial fibrillation (AF) with RV pacing failure (Figure 1). Externalized conductors (ECs) were not apparent using multidirectional fluoroscopy. Measurement values related to the lead since the implantation had been within the normal ranges and stable up to and including the last evaluation conducted 5 weeks before the event, with the normal ranges as follows: an RV pacing impedance of 400–450 Ω , an RV pacing threshold of 0.25–0.5 V/0.5 ms, an RV sensing threshold of 4.0–6.0 mV, and an HV lead impedance of 52–57 Ω . Device interrogation revealed that all 19 shocks of 36 J were delivered inappropriately owing to AF. The HV lead impedance decreased to 24 Ω for the first shock and was 48, 48, and 45 Ω for the subsequent sets of shocks, respectively. The RV sensing threshold decreased to 2.8 mV. The RV pacing threshold markedly increased to 6.0 V/0.5 ms with an increased impedance of 1500 Ω (Figure 2A). The stored intracardiac electrograms of the first shock episode were unavailable because of the accumulation of data from the subsequent episodes. The second shock episode is shown in Figure 2B. The shock was delivered inappropriately owing to the rapid ventricular response during AF. Noise became apparent on the RV lead after shock delivery. The noises were transiently seen afterward on the intracardiac electrograms of the subsequent episodes.

KEYWORDS Electrical failure; ICD; Pacing failure; Shock; Short circuit; Riata lead

ABBREVIATIONS AF = atrial fibrillation; EC = externalized conductor; ETFE = ethylene tetrafluoroethylene; HV = high voltage; ICD = implantable cardioverter-defibrillator; PTFE = polytetrafluoroethylene; RV = right ventricular; SVC = superior vena cava (Heart Rhythm Case Reports 2015;1:46–50)

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KEY TEACHING POINTS

- Electrical defects of the Riata lead may become evident only after a maximum shock delivery in the setting of previously unsuspected lead problem based on the routine device checkups.
- Externalized conductors may likely indicate a future risk of electrical failure, but may not necessarily precede the electrical defect.
- A high-output defibrillation testing is strongly recommended to assess the integrity of the Riata lead at the time of a generator exchange or when electrical defects are highly suspicious.

The patient underwent the implantation of a new defibrillator lead. The affected lead was intact as far as could be visually inspected in the pocket. The RV ring and tip electrode conductors were evaluated individually. The unipolar measurements of the RV ring electrodes were within the normal range, with a pacing impedance of 299 Ω and a pacing threshold of 1.9 V/0.5 ms, whereas those of the RV tip electrodes were abnormal, with a pacing impedance of 1953 Ω and a pacing threshold of 6.5 V/0.5 ms. All connectors of the damaged lead were capped, and the lead was not extracted. A new ICD lead was implanted, and a generator was exchanged. The explanted ICD was bench tested by the manufacturer, and no abnormal findings were obtained.

Discussion

This case demonstrates a shock-induced RV pacing failure, an uncommon manifestation of electrical short circuiting of the ICD lead.

It is quite evident that an electrical short circuit occurred during the first shock. However, a short circuit between the HV conductors was unlikely in this case, because the bench test found no abnormality in the internal circuitry of the explanted pulse generator. Since the ATRAS DR ICD was not equipped with an overcurrent detection system, an electrical short circuit between the HV systems should have damaged the internal circuitry of the pulse generator. RV pacing failure seen shortly after shock delivery was caused by the failure of the RV tip conductor that was centrally located in the lead. Thus, it was most likely that a short circuit occurred between the RV tip conductor and one of the following HV systems: the RV coil conductor, the superior vena cava (SVC) coil conductor, or the ICD can (Figure 3). The 3 hypotheses regarding how RV pacing failure developed owing to a short circuit are described below. *Hypothesis 1* is that an electrical short circuit occurred between the RV tip

conductor and the RV coil conductor (Figure 3A). The RV tip electrode functioned as an anode during shock delivery. The electrical current ran from the RV tip electrode to the SVC coil as well as to the ICD can, making a circuit parallel to those between the RV coil and the SVC coil/ICD can. As a result, the shock impedance decreased to 24 Ω . The myocardium surrounding the RV tip electrode was ablated by the shock current, which increased the pacing impedance and pacing threshold. Furthermore, because of the impedance increase in the myocardium surrounding the RV tip electrode caused by the first shock, the shock impedances of the subsequent shocks were increased to the near-normal range. *Hypothesis 2* is that an electrical short circuit occurred between the RV tip conductor and the SVC coil conductor (Figure 3B). In this setting, the RV tip electrode functioned as a cathode. The current ran from the RV coil to the RV tip electrode. The circuit was parallel to that between the RV coil and the SVC coil and to that between the RV coil and the ICD can. The subsequent result was the same as in Hypothesis 1. *Hypothesis 3* is that an electrical short circuit occurred between the RV tip conductor and the ICD can (Figure 3C). The RV tip electrode functioned as a cathode, with the same result as in Hypothesis 2. The Riata lead consists of a centrally located RV tip conductor coil and 3 surrounding pairs of conductor cables: the RV HV defibrillation conductor cables, the SVC HV defibrillation conductor cables, and the ring electrode conductor cables. The central RV tip conductor coil is encased in a tube of polytetrafluoroethylene (PTFE), and each of the 3 pairs of conductor cables is extruded with ETFE. When the lead structure is considered, Hypothesis 3 becomes less likely. Theoretically, it seems impossible to selectively damage the central RV tip conductor without the other surrounding conductors being affected by can abrasion. In addition, the lead damage was not obvious in the pocket. Thus, in this case, breaks in the PTFE insulation and the ETFE insulation of either the RV or the SVC coil conductor were most likely responsible for the short circuiting.

Riata and Riata ST ICD leads are prone to a unique insulation failure with inside-out abrasion with ECs.¹⁻⁴ The prevalence of ECs ranges from 14% to 25% at a dwell time of approximately 5 years.¹⁻⁴ Most leads with ECs seem to be electrically intact with PTFE/ETFE insulation of each cable, and the overall incidence of electrical abnormalities ranges from 1.3% to 6.7% (Table 1).^{1-3,6-8} The pattern of electrical failure is diverse, including nonphysiologic noise and decrease or increase in pace/sense conductor impedance or HV impedance. Sung et al⁶ reported that 14 of 47 failed leads resulted in inappropriate shocks. Among them, an increase in pace/sense conductor impedance and an increase in capture threshold were seen in 1 patient, respectively.⁶ Although they did not mention the clinical manifestation of the lead

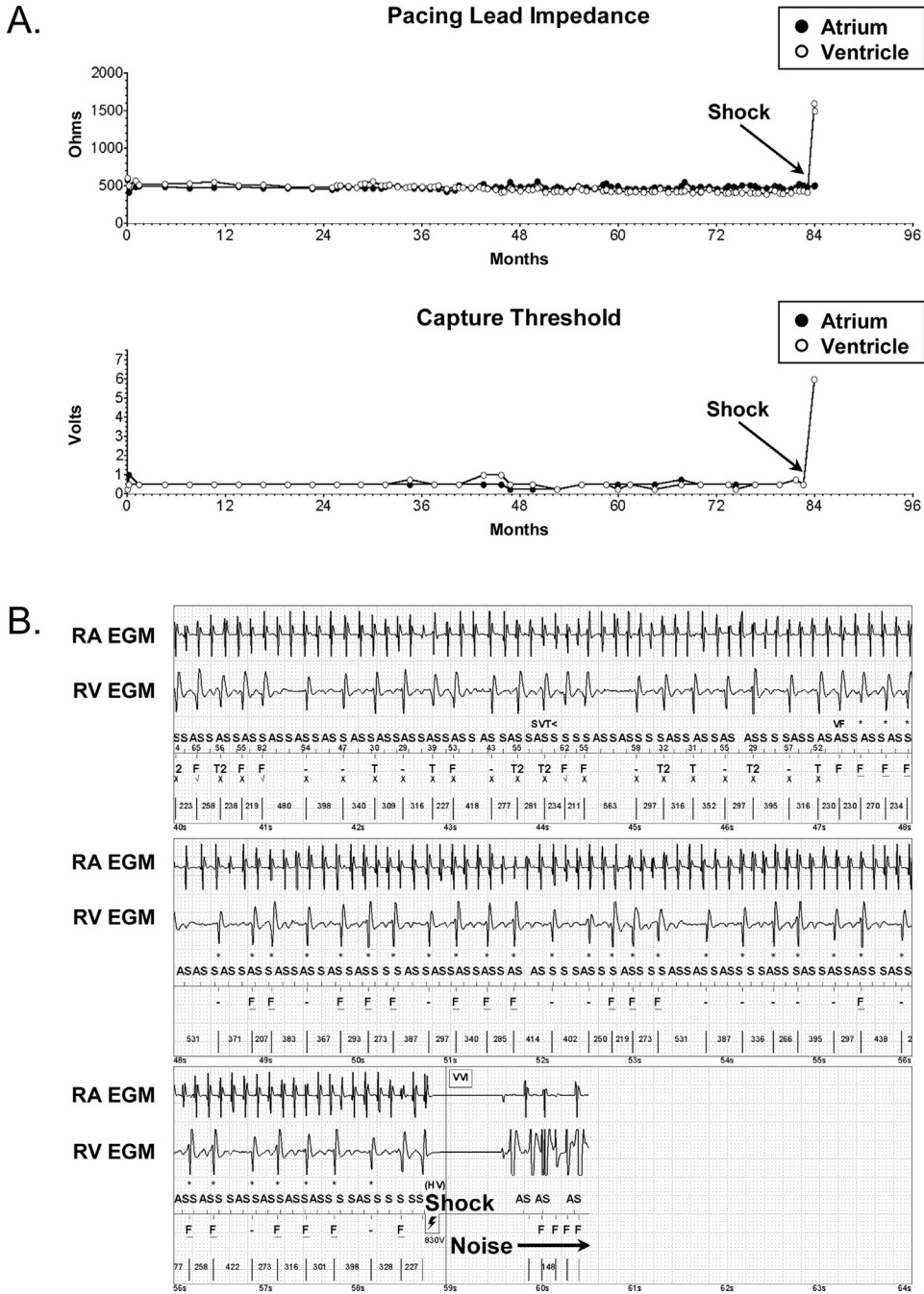


Figure 2 A: Trend of pacing lead impedance and capture threshold. The right ventricular pacing lead impedance and pacing threshold had been stable since device implantation, but markedly increased after shock delivery. B: Stored intracardiac electrograms before and just after the second shock delivery. The shock was delivered inappropriately owing to the rapid ventricular response during atrial fibrillation. No noise or nonphysiologic signals were apparent before shock delivery. Noise appeared shortly after shock delivery on the right ventricular electrogram. RA EGM = right atrial electrogram; RV EGM = right ventricular electrogram.

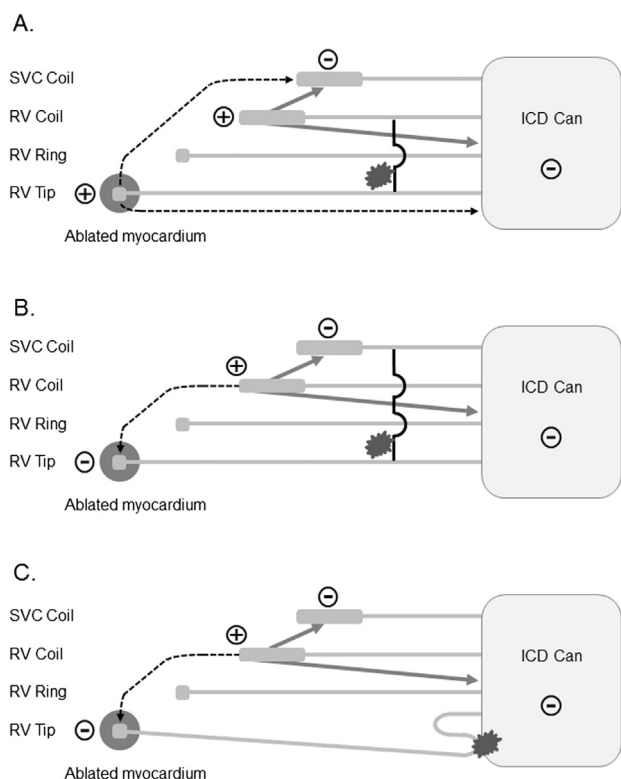


Figure 3 Presumed mechanisms of the electrical short circuit. **A:** Hypothesis 1: An electrical short circuit between the RV tip conductor and the RV coil conductor. **B:** Hypothesis 2: An electrical short circuit between the RV tip conductor and the SVC coil conductor. **C:** Hypothesis 3: An electrical short circuit between the RV tip conductor and the ICD can. Dotted arrows indicate the direction of the short current. The myocardium surrounding the RV tip electrode was ablated by the short current, resulting in an increase in the RV pacing lead impedance and capture threshold. Because the impedance increase occurred in the myocardium surrounding the RV tip electrode owing to the first shock, the shock impedances of the subsequent shocks were increased to the near-normal range. RV = right ventricular; ICD = implantable cardioverter-defibrillator; SVC = superior vena cava.

failures in detail, these 2 leads may have had the pattern of electrical failure similar to that of the present case. It is debatable whether leads with ECs are more likely to fail electrically (Table 1).^{2-4,8} In this regard, a recent longitudinal follow-up study has demonstrated that ECs develop at a rate of 3.7 per 100 person-years and that electrical abnormalities will newly occur with a 4.4 times higher rate in patients with baseline ECs than in those without ECs.¹¹ Another longitudinal study has shown an electrical failure rate of 6.4%/y in the externalized lead.¹² The time-dependent phenomenon of ECs and link to new electrical abnormalities emphasize that fluoroscopic screening of ECs will provide useful information in the management of Riata/ST leads.

It should be noted that neither the EC nor electrical failure had been detected by the routine device checkups before the true shock delivery occurred in the present case. There is no definite explanation for this. However, it is conceivable that the insulation was barely intact, although there should have been partial abrasion of the ETFE and/or PTFE coatings before the high-energy shock current caused a breakdown of the coatings. Another possible explanation is that the delivery of a low current during the HV impedance measurement may not have been enough to reveal a small short circuit that became apparent only through the delivery of a maximum shock. Two previous case reports^{9,10} have described a similar phenomenon in Riata leads, underscoring the concept that a lead problem may become evident only after maximum shock delivery. We should keep in mind that device interrogation, fluoroscopy, and low-voltage shocks may not be able to detect electrical defects even when present. A high-output defibrillation testing is strongly recommended to assess the lead integrity at the time of a generator exchange or when electrical defects are highly suspicious.

Table 1 Incidence of externalized conductors and electrical abnormalities in Riata/ST ICD leads

Study group, year	Lead model	n	EC	Dwell time (y)	EA	EC-EA relation
Liu, 2012	Total	245	53 (21.6%)	5.7 ± 1.5	-	-
	Riata	187	46 (24.6%)	-	-	-
	Riata ST	58	7 (12.1%)	-	-	-
Theuns, 2012	Total	1029	147 (14.3%)	5.0 (median)	47 (4.6%)	Positive
	Riata	482	103 (21.4%)	6.0 (median)	-	-
	Riata ST	547	44 (8.0%)	4.3 (median)	-	-
Sung, 2012	Total	1403	-	-	47 (3.3%), 0.67%/y	-
	Riata	877	-	-	30 (3.4%), 0.61%/y	-
	Riata ST	526	-	-	17 (3.2%), 0.81%/y	-
Hayes, 2013	Total	776	149 (19.2%)	4.8 ± 0.9	10 (1.3%)	Negative
	Riata	517	125 (24.2%)	4.8 ± 0.9	6 (1.2%)	Negative
	Riata ST	259	24 (9.3%)	4.8 ± 0.9	4 (1.5%)	Negative
Parkash, 2013	Total	4358	-	5.0 (median)	201 (4.6%)	-
	Riata	2847	-	-	147 (5.2%)	-
	Riata ST	1,412	-	-	47 (3.3%)	-
	Unknown	99	-	-	7 (7%)	-
Abdelhadi, 2013	Total	1081	27/110 (24.5%)*	-	67 (6.2%)	Positive
	Riata	774	26/81 (32%)*	4.2 ± 2.4	62 (8.0%)	-
	Riata ST	307	1/29 (3.4%)*	3.3 ± 1.7	5 (1.6%)	-
Cheung, 2013	Total	314	-	4.1 (median)	21 (6.7%)	Positive

EA = electrical abnormality; EC = externalized conductor; ICD = implantable cardioverter-defibrillator.

*Not all patients underwent fluoroscopic evaluation of externalized conductors.

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