A railroad track pattern on an implantable cardiac defibrillator (ICD) interval plot is not an uncommon observation in patients presenting to ICD clinics. It refers to the alternation of ventricular intervals that produces the appearance of a railroad tracks.1,2 In general, this is the result of various types of oversensing, including T wave oversensing and R wave double counting, or caused by triggered repetitive programming changes, such as ventricular safety pacing1,4 or cardiac resynchronization therapy response to premature ventricular complexes (PVCs). Finally, in cardiac resynchronization therapy devices, ineffective ventricular pacing in cardiac resynchronization therapy followed by intrinsic ventricular sensing could produce the same pattern.1,2 Two recent reports3,4 demonstrated that more complex railroad track patterns could occur and involve both atrial and ventricular events, but concluded that they were transient phenomena that resolve on successful ventricular tachycardia (VT) detection. However, in the present article, we review 2 cases of recurrent and sustained railroad tracking that persist for minutes, in which very similar and complex multiple track patterns were observed and required programming measures to resolve them. These 2 cases present a unique opportunity to review the mechanisms and management of railroad track pattern.

Case 1

The patient is an 84-year-old man with hypertension, peripheral vascular disease, hyperlipidemia, prior myocardial infarction, ischemic cardiomyopathy, and paroxysmal VT who received a single chamber secondary prevention ICD in 1999. He later developed paroxysmal atrial tachycardia with tachy-brady syndrome and was upgraded to a dual chamber ICD in 2004. The patient presented to device clinic for routine follow-up. His defibrillator (Medtronic Secura DR dual chamber ICD, Model D224DRG) was programmed DDDR 70 to 110 beats per minute with a paced and sensed atrioventricular (AV) delay of 300 and 270 ms, respectively. He is 100% atrial and ventricular paced. Intrinsic rhythm was sinus with PR prolongation and AV delay of 300–450 ms. Tachycardia therapy zones were as follows: VT monitor zone at 130 beats per minute, VT zone of 167 to 188 beats per minute, and a VF zone at >240 beats per minute. Device interrogation demonstrated normal lead performance. Proportion of pacing was atrial sensed (AS)–ventricular sensed event (VS) 8.6%, AS-ventricular paced event (VP) <0.1%, atrial pacing (AP)-VS 40.6%, and AP-VP 50.8%. He had multiple stored events for nonsustained VT and VF with therapy. There were 41 with short V-V intervals. A representative interval plot (Figure 1) demonstrated a tachycardia within the VT monitor zone (300–450 ms) and a multiple track pattern with alternating short (<100 ms) and 2 intermediate coupling intervals (≈420 and 900 ms, respectively). The electrograms are shown in Figure 2A. What is the cause of the multiple railroad track pattern?

Case 2

The second patient is an 82-year-old male with coronary artery disease, previous myocardial infarction, status post coronary artery bypass grafting in 2008, left ventricular ejection fraction of 20% with New York Heart Association Class II symptoms, primary prevention ICD implant in 2006, history of ICD shocks for sustained VT treated with amiodarone, a Sprint Fidelis™ lead on advisory with normal electric function. The patient presented for routine ICD follow-up. His defibrillator (Medtronic Virtuoso II DR dual chamber ICD, Model D274DRG) was programmed DDDR 70 to 110 beats per minute with a paced and sensed AV delay of 300 and 270 ms, respectively. He is 100% atrial and ventricular paced. Intrinsic rhythm was sinus with PR prolongation (280 ms). Tachycardia therapy zones were as follows: VT monitor zone at 130 beats per minute, VT zone of 167 to 188 beats per minute, fast VT zone at 188 to 240 beats per minute, and a VF zone at >188 beats per minute. Device interrogation demonstrated normal lead performance. Review of transmissions shows 34 episodes of VT in the monitor zone with a cycle length (CL) of ≈420 ms (140 beats per minute). The longest duration was 29 minutes. Figure 3 is the interval plot demonstrating multiple railroad tracks and corresponding electrograms from the episode. As in case 1, a similar complex multiple railroad track pattern is observed in a recurrent and sustained fashion during a slow VT with near identical VT CLs of ≈420 ms. What is the explanation for this pattern and how can we prevent this from happening?
Discussion

Both cases (Figures 1 and 3) demonstrate similar and complex railroad track patterns that occur during a slow VT with a CL of ≈420 ms. In each case, the tachycardia falls within the monitor zone. The complex railroad track pattern consists of an upper track because of AP, two middle tracks because of 2 alternating cycle lengths of sensed ventricular events during VT, and a lower track because of ventricular safety pacing (VSP). As the VT cycle lengths and railroad track patterns are near identical, we will use case 1 as the index example to discuss potential mechanisms underlying this phenomenon.

Mechanisms of Sustained Railroad Tracks

Upper Track: Atrial Pacing During VT as the Initiating Mechanism of Railroad Tracks

Figure 2A shows that AP in case 1 occurs at every other sensed VS event. Atrial pacing accounts for the upper track in the ICD interval plot shown in Figure 1. How does AP initiate during VT? In a typical dual chamber rate-modulating mode (DDDR), AP rate (A-A interval) is determined by the sensor-indicated rate (SIR). AP initiates after an A-A interval of 706 ms (SIR=85 beats per minute) expires in atrial-based timing. However, the presence of 2 consecutive VS events triggers a switch to a ventricular-based timing. In this example, the second consecutive VS event (Figure 2A, asterisk) without a preceding AS or AP event is interpreted as a PVC. As this occurs within the A-A interval, an immediate switch from atrial- to ventricular-based timing results. The switch from atrial to ventricular-based timing in the context of a sensed PVC promotes rate stabilization and prevents subsequent ventricular pacing from violating SIR (706 ms, 85 beats per minute) expires in atrial-based timing. However, the presence of 2 consecutive VS events triggers a switch to a ventricular-based timing. In this example, the second consecutive VS event (Figure 2A, asterisk) without a preceding AS or AP event is interpreted as a PVC. As this occurs within the A-A interval, an immediate switch from atrial- to ventricular-based timing results. The switch from atrial to ventricular-based timing in the context of a sensed PVC promotes rate stabilization and prevents subsequent ventricular pacing from violating SIR (706 ms, 85 beats per minute; A-A interval of 706 ms+AV interval of 300 ms) if atrial timing were to persist. Thus, the A-A interval terminates and an atrial escape interval, also referred to as ventriculo-atrial interval (VAI), is initiated. Panel B shows the VAI interval (406 ms), which is determined by SIR interval (706 ms) minus paced AV delay (300 ms). AP occurs only after a 406 ms VAI expires before the next VS event occurs.

Sustained Tracks

What is the mechanism of persistence of railroad tracks? Unlike previous reports,3,4 railroad track pattern is persistent in the present 2 cases. The pattern terminates only when the tachycardia spontaneously terminates. Railroad tracks persist because AP remains uninhibited during VT and VT persists untreated in the monitor zone. Two separate reports in Medtronic devices3,4 described brief safety pacing episodes during VT, but suggested that after a few seconds, AP would be withheld.3 This is because of a Medtronic algorithm that limits AP by extending the VAI to VT detection CL+30 ms in the case of suspected VT to prevent VT undersensing, as presumably some VT beats could occur in the postatrial ventricular blanking period and not be accounted for, especially when AP and VT rates are close. This algorithm is activated when there are at least 3 V-V intervals that fall in any VT therapy zone. Unfortunately, the algorithm is not activated for VTs in the monitor zone, thus allowing AP to persist in the present 2 cases. As discussed below, AP in turn triggers ventricular safety pacing and culminates in a complex railroad track pattern that terminates only when VT spontaneously terminates (Figures 1 and 3).

Lower Track: Ventricular Safety Pacing

Because the sensor-indicated rate cycle length is a little less than twice that of VT cycle length, AP event occurs just before every other VS event. Because of this fortuitous timing, VS events fall within the cross-talk window (which occurs after the postatrial ventricular blanking period ends) and initiates VSP, which is nominally 80 ms in Medtronic devices. VSP results in critically short V-V intervals (VS-VP) and accounts for the lower track of the multiple track patterns seen on the scatter plots (Figures 1 and 3A). VSP is designed to prevent crosstalk inhibition of ventricular pacing and consequent asystole in patients who are pacemaker-dependent.1 In Medtronic devices, the other potential reason for critically short V-V intervals other than VSP is ventricular sense response in patients with cardiac resynchronization therapy,
which is not the case here. Thus, VSP in this VT example is as a result of the presence of AP, whereas AP occurs because of an enabled rate-modulating pacing mode (DDDR), which uses a hybrid timing cycle to prevent variable and lower than programmed sensor indicated rate in the setting of frequent PVCs.

Middle Tracks: Two Ventricular Tachycardia Cycle Lengths?

Next, why are there 2 middle tracks in the triple track pattern? The 2 middle tracks (Figures 1 and 3A) are the result of a 30 ms variability in VS events (410 and 440 ms). However, measurement of intracardiac electrograms (red dashed lines) demonstrate a stable VT cycle length of 425 to 430 ms (Figure 2A). Interestingly, the longer (440 ms) VS-VS interval always coincides with an AP event (Figure 2A and 2B). This VT interval variability is a result of a delay in ventricular sensing because of the overlap of the initial part of the ventricular tachycardia signal with the postatrial ventricular blanking period. This is supported by the observation that
VS marker has an alternating position in regards to the ventricular EGM. The delay of ventricular sensing (as the initial portion occurs within postatrial ventricular blanking period) inappropriately indicates a shorter timing (410 ms) of the subsequent VS-VS events, whereas the true VT cycle length remains stable at 425 to 430 ms. The left strip in Figure 2A demonstrates that. Figure 2B shows VS timing in relation to V electrograms indicated by blue dashed lines. The 30 ms VT variability resolves (Figure 2A, right strip) when VAI shortens to 390 ms (because of an increase in SIR to 87 beats per minute or 690 ms). At this point, VS after AP event is no longer postponed by postatrial ventricular blanking period and coincides with the normal timing of QRS detection. The pattern changes from double track VS to a single track VS (Figure 1). It is interesting to note that in Case 2 (Figure 3), a near identical rail road track pattern occurs in a patient with a slow VT with similar CLs to case 1, and the same long programmed AV delay of 300 ms.

This combination of events could occur because of a long programmed AV delay, DDDR mode which allows for AP at SIR, and slow VT within monitor zone. Interestingly, the paced AV delay is identical at 300 ms in each case. The long programmed paced AV delay (300 ms) with subsequent short VAI (405 ms) at SIR (85 beats per minute) and the relatively slow VT rate created a situation in which the VAI is shorter than the VT cycle length. This allowed AP to continue during VT. In turn, AP resulted in the appearance of VSP, and the coincident sensor-driven cycle length and VT cycle length resulted in AP coinciding with VS events and delaying sensing of every other VS event. Thus, the root of the problem lies in the change from atrial-based to ventricular-based timing, initiating AP at a relatively rapid sensor-driven rate (85 beats per minute or CL of 706 ms) during a relatively slow VT (CL 420 ms) in a device set to a relatively long AV delay (300 ms), resulting in a short VAI (706 ms minus 300 ms=406 ms) shorter than VT cycle length. In contrast, alternating AP events would not be present if sensor indicated rate results in a VAI equal or longer than VT cycle length of 420 ms (AVI 300 ms+VAI 420 ms=SIR 720 ms or 83.3 beats per minute).

Figure 3. Interval plot and stored intracardiac electrograms from Case 2, demonstrating a slow ventricular tachycardia (VT) with near identical cycle length to case 1, with a similar multiple railroad track pattern. See text for further details.
Railroad Tracks in Other ICDs

Railroad track patterns could potentially occur in Medtronic, St. Jude, Biotronik and Sorin/ELA, but not with Boston Scientific devices. Medtronic, Biotronik, and Sorin/ELA ICDs remain in the same programmed pacing mode even when an event has been classified as a VT, allowing AP to continue after expiration of VAI (ventricular-based timing). St Jude ICDs perform a mode switch to DDI pacing mode in the VT zone, which would still potentially allow for AP. In contrast, Boston Scientific ICDs eliminates AP as it mode switches from DDD(R) to VDI once VT has been detected.

Prevention

To avoid the phenomenon of AP with subsequent VSP during VT, the device could be programmed to indirectly extend VAI>VT cycle length (420 ms) when an atrial-based timing changes to ventricular-based timing (hybrid-based timing). This can be achieved by (1) eliminating rate-modulated pacing response in patients with normal sinus rhythm who might not need it or (2) decreasing MSR to <83 beats per minute; and (3) programming a more physiological (shorter) AV delay with potential adverse effects because of increased ventricular pacing.

Potential Clinical Consequences

Persistent AP during VT accounts for the initiation and maintenance of multiple railroad tracks. There are 2 potential consequences of AP. First, it may lead to undersensing of VT if VS event falls entirely in the PAVB. This did not occur in the present 2 cases because VS occurred toward the end of PAVB and was sensed in the crosstalk window. Second, continued AP during VT may promote further symptomatic and hemodynamic deterioration because of AV dyssynchrony.

Conclusions

In summary, we demonstrated in these 2 cases that a sustained multiple railroad track ICD plot could occur during a slow VT in the setting of a dual-chamber rate-modulating pacing mode and a long programmed AV delay. This combination allows for AP to continue during a relatively slow VT because the switch from atrial- to ventricular-based timing cycles with a long programmed AV interval results in a VA interval shorter than VT cycle length, triggering AP when the VA interval expires without a sensed ventricular event. Continued AP subsequently triggers ventricular safety pacing. Therefore, the pattern can be avoided by methods to avoid AP during VT, either by disabling rate modulating pacing mode or indirectly extending VA interval (by shortening AV delay) to exceed the VT cycle length. The elimination of railroad track plots with these programming changes would need to be balanced against potential detrimental effects of loss of rate response and increased ventricular pacing, respectively.

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References


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