AUTOMATIC ADJUSTMENT OF VENTRICULAR ANTI-TACHYCARDIA PACING AND INDIVIDUALISED DEVICE THERAPY

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Automatic adjustment of Ventricular anti-tachycardia pacing and individualised device therapy: A case report of a new algorithm of ATP and efficacy for incessant slow ventricular tachycardia

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Introduction:
Intrinsic ATP™ (iATP) is an automated ventricular anti tachycardia pacing (ATP) algorithm that provides individualized therapy. Each ATP attempt, if unsuccessful, is sequentially adjusted based on the post-pacing interval. This algorithm was tested and proven to be beneficial in virtual modelling studies and patient cohorts. Here we describe a case where manually programmed and attempts with conventional ATP failed to terminate a slow incessant ventricular tachycardia (VT) but iATP proved clinically more efficacious.

Case Report:
An 81 year old gentleman was admitted to our coronary care unit with light-headedness and palpitations coincident with episodes of slow ventricular tachycardia. He had a background history of having coronary artery bypass grafting in 1997 with 3 venous grafts, severe left ventricular systolic dysfunction, and paroxysmal atrial flutter.

On clinical examination he had a blood pressure of 124/81, pulse of 128 bpm. His oxygen saturation was 98% on air. He was alert, responsive and appeared comfortable at rest. Median sternotomy and saphenous venous harvest site scars were well healed. Cardiovascular and respiratory examinations were otherwise unremarkable.

Initial work up showed normal renal function, electrolytes and normal full blood count. A chest radiograph revealed globular cardiomegaly, without pulmonary congestion. ECGs were consistent with ventricular tachycardia and an inferobasal left ventricular exit site given the remote history of inferior infarction and the QRS morphology. High sensitivity troponin T was mildly elevated (23.5 ng/l). NT-pro-BNP was not assayed.

Intravenous amiodarone was infused at 300mg over 1 hour followed by 900mg over 24 hours. DC cardioversion was performed and an externalised dual chamber pacemaker was implanted to manage intermittent complete AV block. The pacemaker was programmed DDI at 80/min and paced AV delay of 300ms. After 2 days of amiodarone loading the incessant VT settled and the patient was 100% Atrial and ventricular paced.

His transthoracic echocardiogram demonstrated severe LV (Left Ventricular) systolic dysfunction with an LVEF 25-30%, LVOT VTI was 14.2cm and stroke volume calculated as 59mls with a cardiac output of 3.5L/min. The end diastolic volume was 157mls. His inferior segments were had thinned and akinetic with severely hypokinetic infero-septal and inferolateral regions and otherwise global hypokinesis. The RV was dilated with reduced longitudinal function and a hypokinetic RV (Right ventricular) apex. There was moderate, functional tricuspid regurgitation. Coronary angiography demonstrated occluded venous grafts, a chronic total occlusion of the right coronary artery and severe left anterior descending artery disease. Percutaneous coronary intervention to the left anterior descending artery was performed. His chronic total occlusion of the right coronary artery
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was not revascularized, given the likelihood of non-viability. A sub-pectoral cardiac resynchronisation defibrillator (CRT-D) was implanted (fluoroscopy on supplementary figure 1). VF induction and defibrillation threshold testing were not performed in view of suspended anticoagulation and recent atrial flutter. He was programmed AAI to DDD with a base rate of 50 bpm. He was set for backup CRT as his intrinsic QRS was narrow with a duration of 106ms when not in AV block. His initial tachycardia programming was a single treatment zone for ”VF” at rate >188bpm, 30/40 with 3 sequences of iATP prior to and during charging followed by full output (40J) shocks (6). A monitor zone was programmed at 102bpm with 130beats needed for detection as the VT was well tolerated and recurrence was to be checked at follow up. Prior to discharge he remained hemodynamically stable but unfortunately was unable to tolerate amiodarone. He developed an intractable and disabling tremor and was subsequently discharged on bisoprolol 10mg od.

2 weeks after discharge, his VT recurred after amiodarone cessation and as this was untreated by the device (in monitor zone) manual ATP was attempted. The VT was unaffected by multiple sequences of traditional ventricular ATP (Burst and Ramp ATP with various numbers of pulses, coupling intervals and R-R% adjustments). ATP was delivered by RV pacing. The most aggressive manual ATP attempted was a Ramp sequence of 12 pulses at 78% R-R interval and decrementing by 30ms; minimum interval 150ms.

Programming of intrinsic ventricular ATP (iATP) a novel company specific algorithm on following the above failed attempts proved successful at terminating the incessant VT at the first attempt. 1 minute later however, the VT reinitiated. He was left in sustained, well tolerated ventricular tachycardia for a total of two weeks. The patient was started on sotalol and up titrated to a dose of 160mg bd. Following this, with iATP reactivated (figure 2), the VT was pace terminated by the device, with sustained restoration of Ap Vs (Atrially paced, ventricularly sensed) rhythm (figure 3 and supplementary figure 2). He remained stable, with intermittent recurrence of VT that was managed with iATP treatment; there were seven total further episodes of ventricular tachycardia treated with iATP (at most 3 cycles per episode) in the month that followed. He was then seen by an electrophysiology team at a tertiary centre who opted to continue monitoring over an ablation due to how well the algorithm worked and how clinically asymptomatic he remained. No episodes of acceleration to faster cycle length VT or degeneration to VF have occurred in this gentleman. He is clinically stable currently and routinely followed up at the heart failure and complex device clinic.

Discussion:

Anti-tachycardia pacing (ATP) is a mode of treatment offered for termination of ventricular tachycardia by intracardiac defibrillators. ATP allows for painless treatment of ventricular tachycardia with less battery consumption and possibly less detrimental effects on the myocardium. It involves bursts of pacing, usually 5-15 pulses, with the goal of penetrating and interrupting the ventricular tachycardia circuit. Studies have shown ATP capable of terminating up to 90% of ventricular tachycardias with a cycle length of >300ms (rate of <200bpm). There is a risk of acceleration which is defined as a 10% or greater increase in the VT cycle length after ATP or degeneration to VF; these may occur in up to 10% of ATP therapy. The modes of traditional ATP are either burst or ramp protocols. Burst pacing is a train of impulses with equal interstimulus intervals. Ramp is a train of impulses with decrementing interstimulus intervals.

Studies have compared burst vs ramp overdrive pacing for both slow (<188bpm) and fast ventricular tachycardia. Overall, there is not a clear difference in the efficacy of burst or ramp ATP, in ischemic and non-ischemic cardiomyopathies. In most cases, the choice of programming is based on clinician’s preference or manufacturer nominal settings. Ramp sequences may have a greater
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likelihood of acceleration, especially in some patient cohorts, for example arrhythmogenic cardiomyopathy. Non-programming factors that play a role in the effectiveness of ATP include the lead position and its proximity to the tachycardia circuit (septal vs apical lead positions related to individual tachycardia exit sites), as well the biochemical status of the patient (electrolytes, pH), medications (antiarrhythmic drugs) and acute ischaemic events. Due to the array of confounders, it is exceedingly difficult to tailor ATP specifically to each patient and as such most patients receive empiric programming, not necessarily specific to their underlying conditions.

The alterations that can be made to the standard ATP algorithms include: 1) Changing the coupling cycle- defined as the delay between the last sensed and the first paced ventricular event. The shorter the coupling interval, the higher the chance of accelerating the tachycardia. 2) Changing the duration and/or number of sequences of ATP or the amplitude of the pulses. 3) Changing the site of the stimulus- only possible in CRT devices which can deliver ATP via the LV lead. 4) Device manufacturer specific sequences such as Sweep pacing in Boston Scientific devices, Burst + from Biotronik, and the new iATP algorithm from Medtronic.

iATP is a novel Medtronic ICD algorithm that offers specific and individualised ATP therapy that may be more efficacious in some cases. iATP delivers patient and arrhythmia specific therapy by using calculations based on the tachycardia cycle length and origin to determine the most accurate pacing timing to terminate the tachycardia.

The iATP algorithm involves automated anti-tachycardia pacing where each sequence ‘learns’ from the prior sequence and alters the therapy. The algorithm designs initial ATP sequences based on recent heart rate data. Subsequent sequences are based on analysis of the post-pacing interval (PPI) of the prior ATP sequence, to determine whether or not the train of impulses reached the VT circuit or not, or lost capture. The iATP S1 coupling interval is set to 88% of the VTCL. The number of S1s is computed from pacing electrode-to-VT circuit travel time, assuming a long travel time for initial sequences. If reset is not achieved, additional S1 pulses are subsequently added. If the initial sequence achieved reset, iATP estimates actual travel time and minimises the number of S1 pulses in subsequent bursts to the number needed for reset.

The S2 impulse is intended to advance VT circuit timing and close the excitable gap. In contrast to the longer S1 coupling interval (88%VTCL), the S1-S2 coupling is intended to be as short as possible without loss of capture. Using heart rate data analysis, the initial S2 is delivered just beyond the predicted myocardial effective refractory period, and decremented by 20-30ms with each unsuccessful attempt, provided capture is achieved. Once loss of capture occurs, the S2 is restored to the shortest coupling interval that captured the myocardium. An S3 pulse is then added, and S3 pulse is decremented thereafter to continue searching for efficacy until minimal coupling of 160ms has been attempted. By this time, if shocks are programmed, shock therapy would be delivered. If criteria for adjustment of the clinical arrhythmia (>10% change in VTCL) are met at any time during iATP therapy, the algorithm is reset to the initial S1 impulse determination stage.

iATP was tested in a clinical setting in patients who had DR-ICDs or CRT-ICDs, a history of ≥1 ICD-treated ventricular tachycardias (VT)/ventricular fibrillation episode or sustained monomorphic ventricular tachycardias (SMVT). A total of 669 SMVTs from 49 patients were adjudicated with an overall termination rate of 80.1%.

iATP was also tested in a virtual scenario using MRI and EP data via CARPEntry simulation. Re-entrant VT was generated with 259 unique ATP scenarios from 7 scarred hearts. In this study iATP was found...
Automatic Adjustment of Anti-Tachycardia Pacing to be 17% more effective than traditional ATP in terminating tachycardia without increasing the rate of acceleration. This suggested that iATP may be more successful with complex scar related tachycardias.

In addition to patient and arrhythmia specificity, iATP is easier to program with less room for error from pacing physicians or physiologists.

One form of treatment not explored in this case was that of low energy shocks (<2J). Generally, when programming low energy shocks, it counts towards total shocks delivered and can therefore limit the amount of high energy shocks that can be delivered in sequence. This coupled with the risk of acceleration and ventricular fibrillation limit the use of low energy shocks however it would have been considered in the absence of successful treatment by iATP.

**Conclusion:**

This case demonstrates a real-world example of Medtronic’s iATP algorithm successfully terminating incessant slow ventricular tachycardia that was otherwise refractory to both pharmacological and traditional ATP therapy.
References:


Images:

Figure 1. Ecg showing Ventricular tachycardia at a rate of 120 beats per minute.
Figure 2. Image taken from Medtronic programmer demonstrating successful use of the iATP algorithm. There were three sequences of iATP delivered which eventually led to termination of the tachycardia.

There was a termination of the VT by the first attempt and it restarted prior to meeting the episode termination rule (8 consecutive RR’s outside enabled therapy zones, not counting the PPI (Post pacing interval) and the one after it as the redetection algorithm initializes on that event) – there were 7 events then an event in the VT zone. Because the PPI was long (paced at 1200ms – longest possible cycle) the closed loop analysis is “Break” so if there’s a redetection and no change in the VT, iATP will not decrement S2 on the next sequence. If S2 continued to decrement eventually you’d lose capture and stop working – or increase acceleration risk. This is unique to iATP.

In this case the Non-reset limit was 1035ms, making the break lower limit 1135ms so the PPI of 1200 was called a break (correctly) the re-detected VTCL was within 10% of the prior so S2 wasn’t decremented.
Figure 3. Image taken from Medtronic programmer demonstrating the successful conversion of VT to sinus rhythm due to iATP. There are two ventricular paced beats after conversion then intrinsic rhythm takes over (as per device programming).
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Key Teaching Points:

1) Antitachycardia pacing (ATP) is a safe, pain free, and effective method of terminating ventricular tachycardias.
2) There are several different algorithms of ATP, the most encountered ones are burst and ramp. Burst pacing is a train of impulses with equal interstimulus intervals. Ramp is a train of impulses with decrementing interstimulus intervals.
3) While ATP is an important tool in the arsenal of treating ventricular arrhythmia, it may not work for all tachycardias- Consider the substrate being treated as well.
4) In some cases, such as arrhythmogenic right ventricular dysplasia ramp pacing was found to be much less effective than burst pacing in terminating tachycardia as well as having a higher rate of acceleration.
5) Intrinsic ATP is a new algorithm present in Medtronic defibrillators that allows for the delivery of patient and arrhythmia specific therapy by using calculations based on the tachycardia cycle length and origin to determine the most accurate pacing timing to terminate the tachycardia.