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Electrocardiographic Troubleshooting of Implanted Cardiac Electronic Devices

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Additional information is available at the end of the chapter

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1. Introduction

1.1. Evaluation of CIED function

Pacemakers, ICDs and cardiac resynchronization devices are implanted and followed mostly by cardiac electrophysiologists. Detailed diagnostic data (pacing statistics, lead function, arrhythmia episode intracardiac electrograms etc.) are available using manufacturer-specific programmer devices or remote follow-up (Figure 1). However, patients may present with suspected cardiac or arrhythmia-related symptoms when these measures are not immediately available. Using conventional diagnostic methods basic device function can be evaluated and correlation with the clinical presentation may be assessed (McPherson, 2004). In certain cases, such as with transient events, these may be the only diagnostic clues available as current CIEDs do not have full Holter capability – only episodes of significance, as determined by the device, are stored.

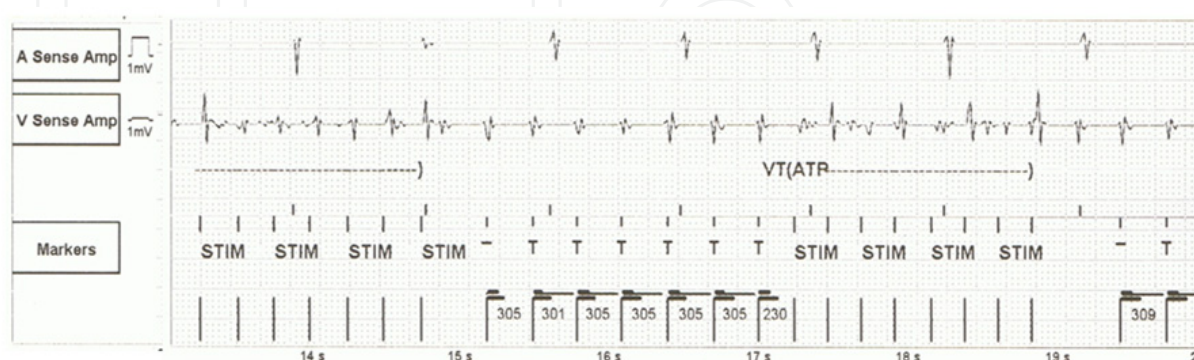


Figure 1. Device interrogation provides detailed information about intracardiac signals, their interpretation and device response. The tracing depicts an episode of ventricular tachycardia, where the implanted cardioverter-defibrillator attempted burst antitachycardia stimulation.

Basic evaluation of CIED function requires a 12-lead ECG and review of past medical records to identify device type and settings. If prior records are not available, a simple chest X-ray may provide important clues (pacemaker, ICD, or CRT; lead locations) (Jacob, 2011). In case intermittent or transient malfunction is detected and device interrogation does not provide clear answer, Holter monitoring or an event recorder may be required. If a programmer is available, diagnostic tests should be performed according to the guidelines (Wilkoff, 2008). Patient symptoms, if any, should be assessed, whether they can be signs of a possible device malfunction.

2. Electrocardiographic evaluation of device function

A 12-lead ECG may raise the suspicion of device malfunction. Careful evaluation of patient-related factors is required as these interact with device function (Table 1). Occasionally, very advanced forms of electrophysiological abnormalities may be identified as the devices generally do not prevent natural progression of underlying pathophysiology. In case an arrhythmia or device malfunction is suspected on a telemetry recording, a full 12-lead ECG is recommended to avoid misinterpretation (Figures 2-6). Artifacts may severely impact interpretation and tracings with good technical quality should be obtained (Figures 7-10). Atrial rhythm and characteristics of atrioventricular/ventriculoatrial conduction should also be assessed (Figures 11-17).

ECG feature	Importance – diagnostic clues and possible interaction with devices
Atrial rhythm	
Bradycardia	Should be paced unless there is oversensing or no atrial lead present
Premature beats	Blocked PACs should elicit different response than sinoatrial block if atrial sensing is present
Atrial flutter	May be tracked with high ventricular rate
Atrial fibrillation	May be undersensed, leading to ineffective atrial pacing
Atrioventricular conduction	
Variable AV conduction interval	May lead to fusion and pseudofusion beats
Complete heart block	May be intermittent or unidirectional
Retrograde conduction	May lead to pacemaker-tachycardia or pacemaker syndrome
Ventricles	
Native QRS morphology	Assess biventricular capture during cardiac resynchronization If atrial pacing only, may be used to identify ischemia, etc.
Premature beats	May trigger safety or sense response pacing or activate rate smoothing algorithms

Table 1. Important ECG features that should be assessed when evaluating CIED function.

Certain conditions, such as acute heart failure may require adjustment of device settings, even without device malfunction – pacemaker algorithms do not provide optimal hemodynamics for all situations. Unfortunately, evidence-based approach is limited due to scarcity of data (Lahiri, 2011).

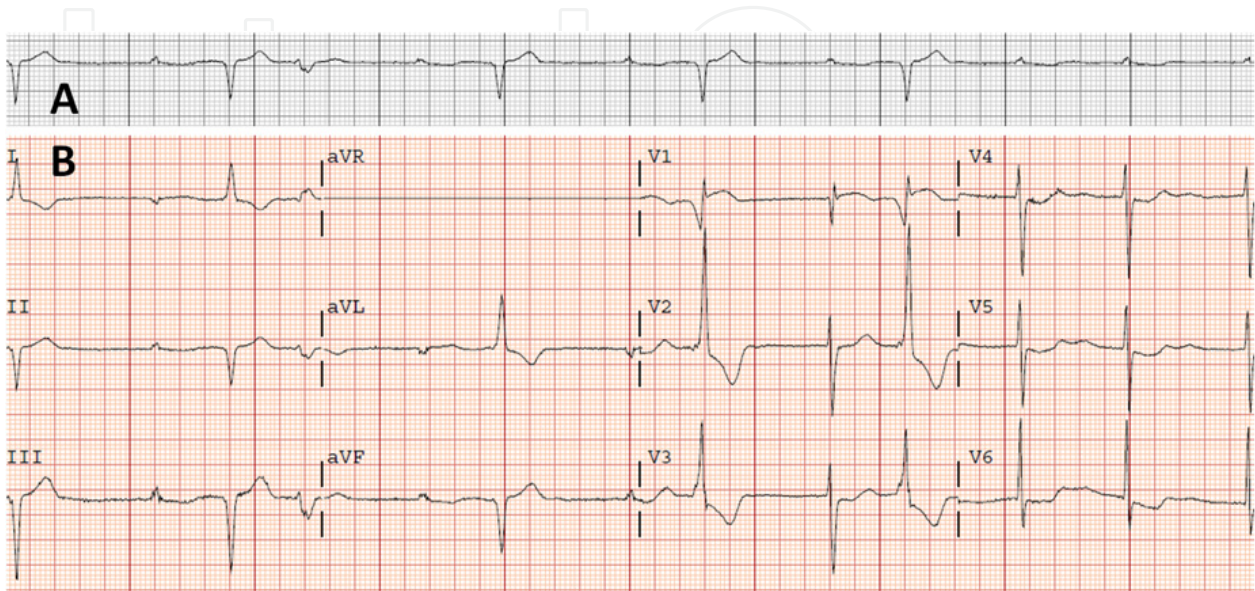


Figure 2. Rhythm strip suggestive of high degree AV block (A). 12 lead ECG obtained at the same time actually shows that the low amplitude signals are QRS complexes and the higher amplitude ones are PVCs (B).



Figure 3. Artifacts masking AV block. High frequency artifacts mimic fast, irregular ventricular rate, resembling atrial fibrillation (A1). However, these artifacts are not present on the simultaneous tracing in a different lead (A2). Once the artifacts disappear, P waves are easily recognizable with high degree AV block (B1, B2).

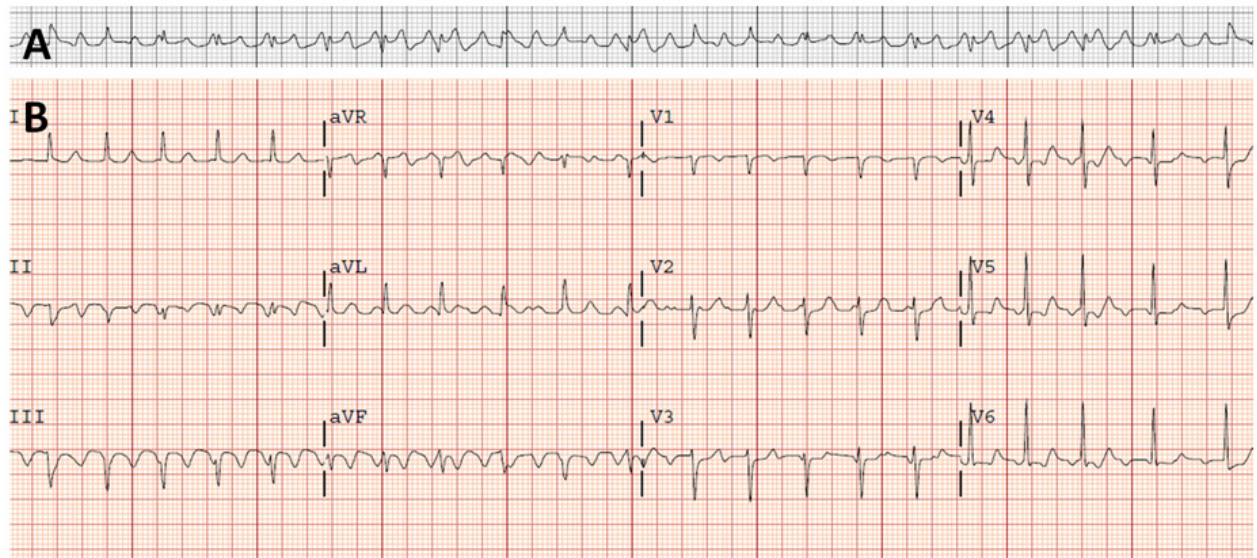


Figure 4. Atrial flutter may mimic ventricular tachycardia in a rhythm strip (A), however, 12-lead ECG clearly identifies the flutter with dominantly 2:1 conduction (B).

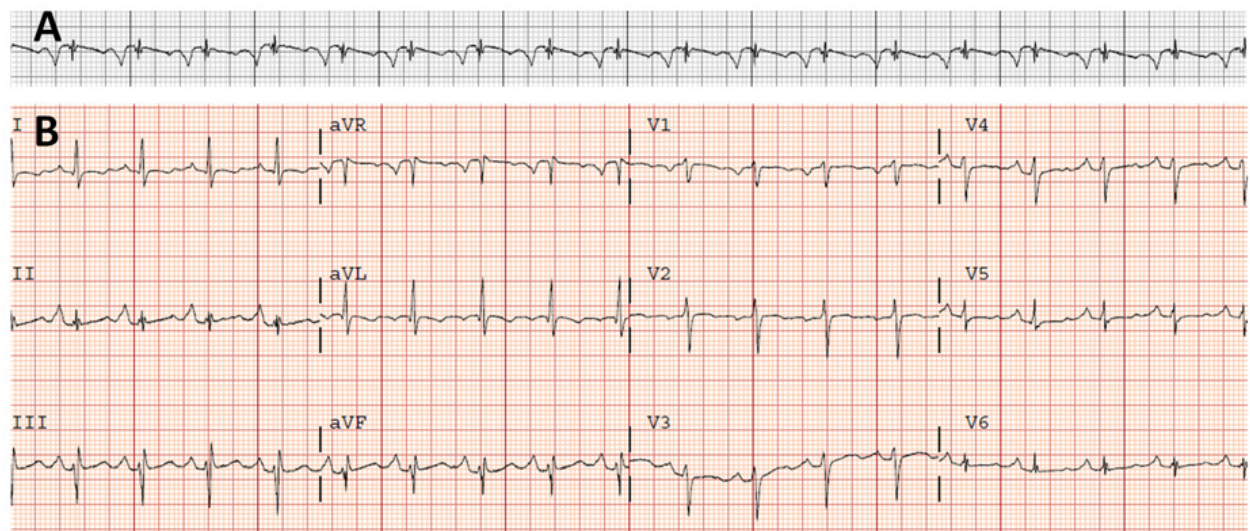


Figure 5. Rhythm strip suggestive of atrial pacing with prolonged AV interval (A). 12 lead ECG shows no evidence of pacing, however, P pulmonale is present and the QRS is low amplitude in II (B).

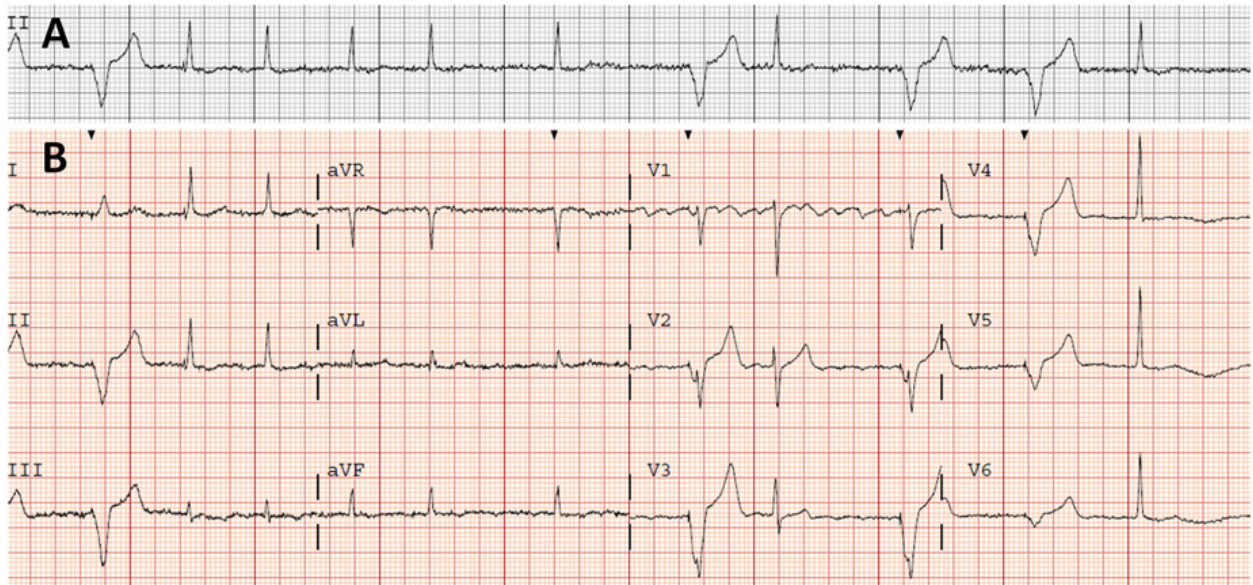


Figure 6. The rhythm strip suggests atrial fibrillation with PVCs or escape beats (A). Simultaneous 12 lead ECG shows evidence of VVI pacing at 60/minute (B). Even when pacing spikes are not visible, wide QRS beats with constant coupling interval, and no R-R cycle longer than this interval should suggest ventricular demand pacing.

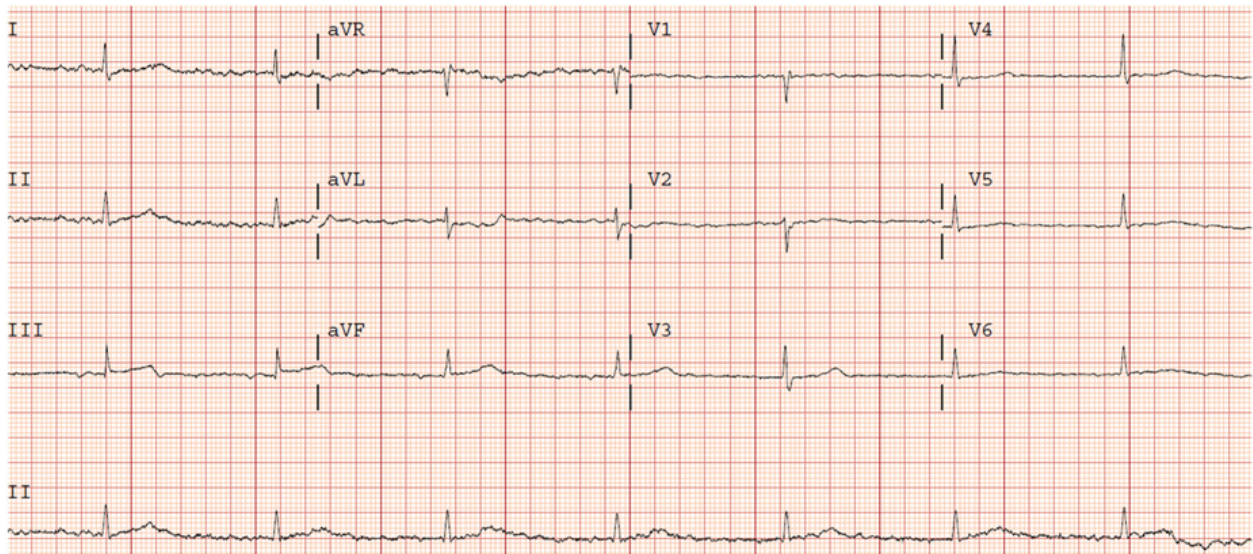


Figure 7. Low amplitude, high frequency artifact masking sinus or ectopic atrial bradycardia. The rhythm may be confused with atrial fibrillation and junctional rhythm, however, P waves can be identified in III and aVF.

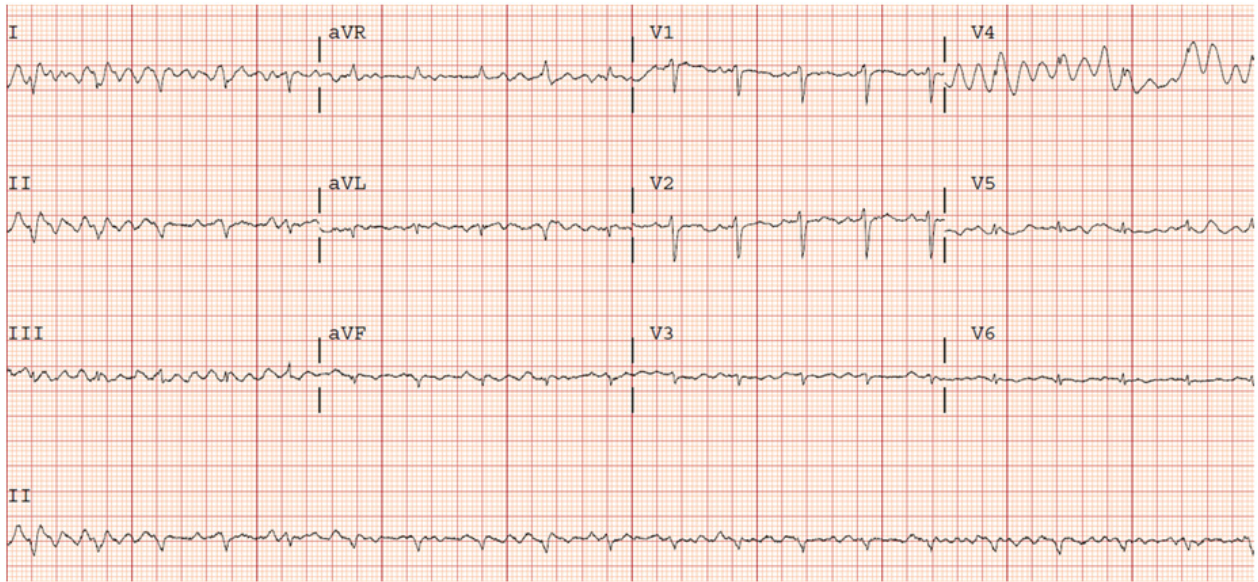


Figure 8. High amplitude artifacts with low ECG voltage may be misinterpreted as atrial flutter or fibrillation. However, sinus tachycardia is easy to recognize in V1 and V2.

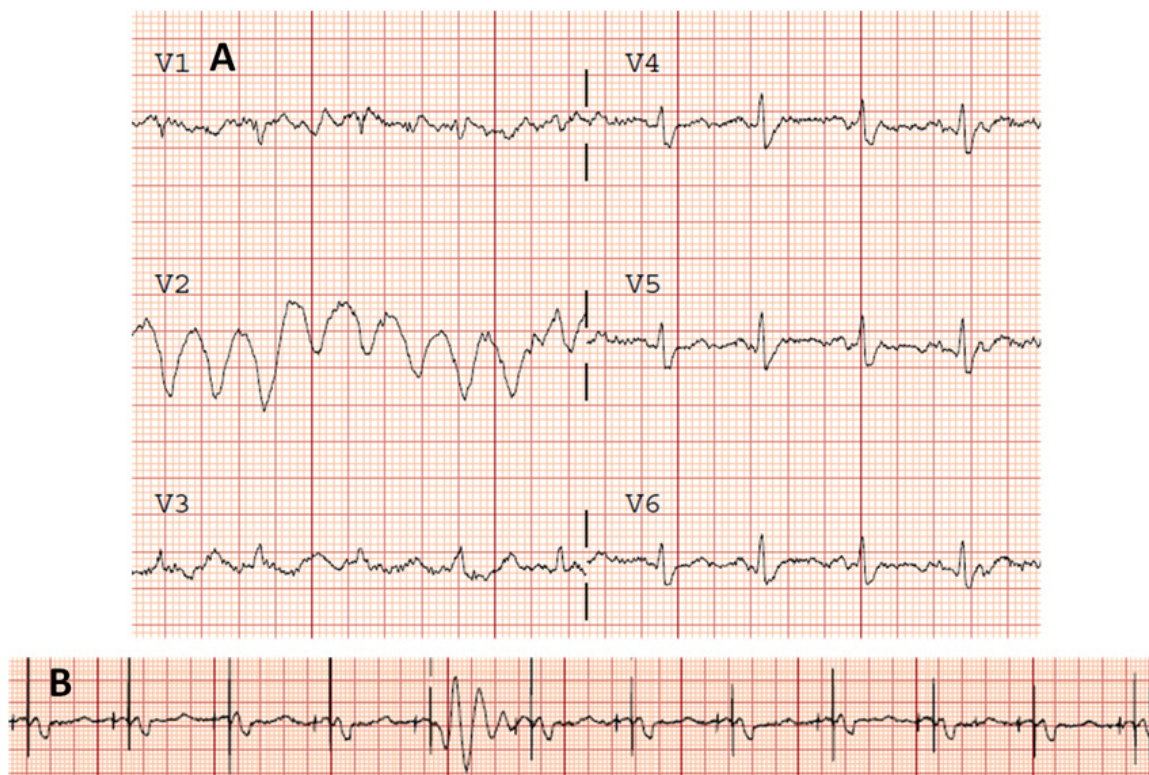


Figure 9. Artifacts suggestive of NSVT. However, the simultaneous V1 and V3, and the following V4-6 leads show that the underlying rhythm is sinus (A). Biventricular pacing is not affected by the artifact – this would be unlikely with any true ventricular arrhythmia (B).

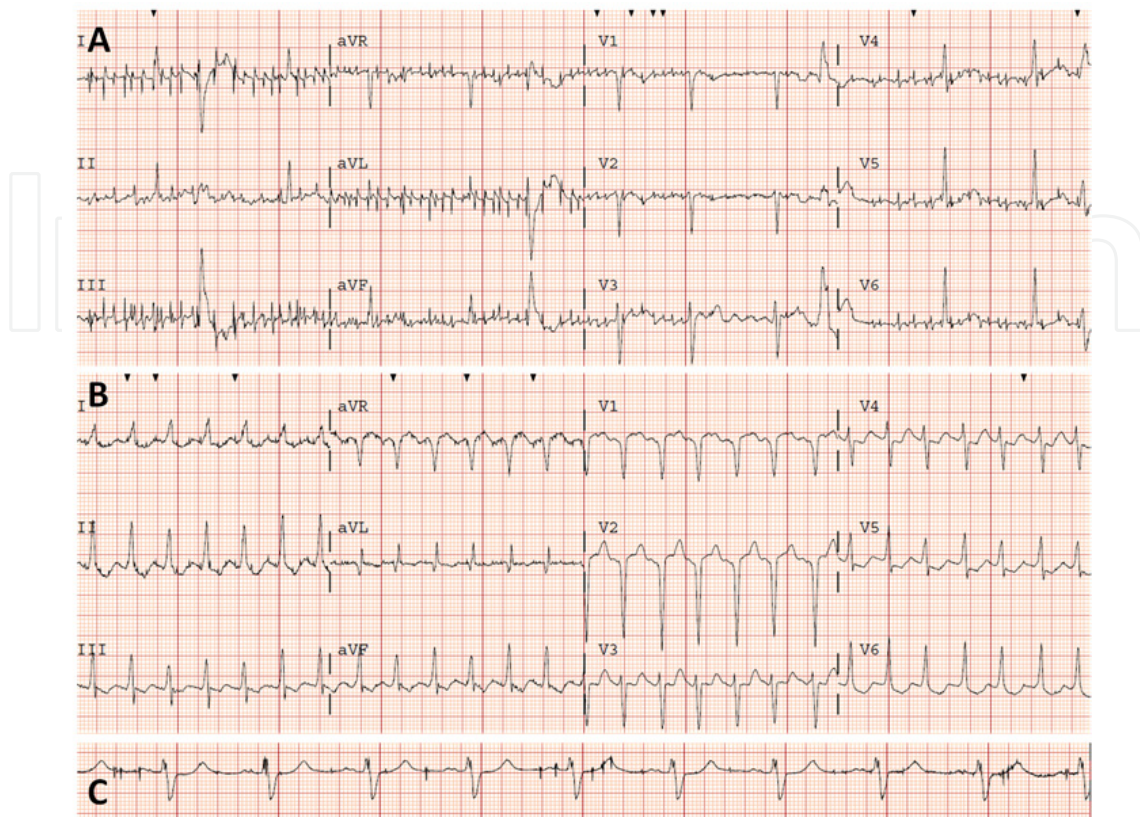


Figure 10. High frequency artifacts causing false detection of pacing in automated ECG device. Although the pacemaker-spike gain and marker functions of the ECG systems may be very helpful to identify small pacing spikes, these systems may be overcalling artifacts. This patient does not have a pacemaker, the ECG improperly identifies some artifacts as pacing (black triangles on top (A)). In some cases, the artifacts may be less obvious (B), or may closely resemble pacing spikes (C).

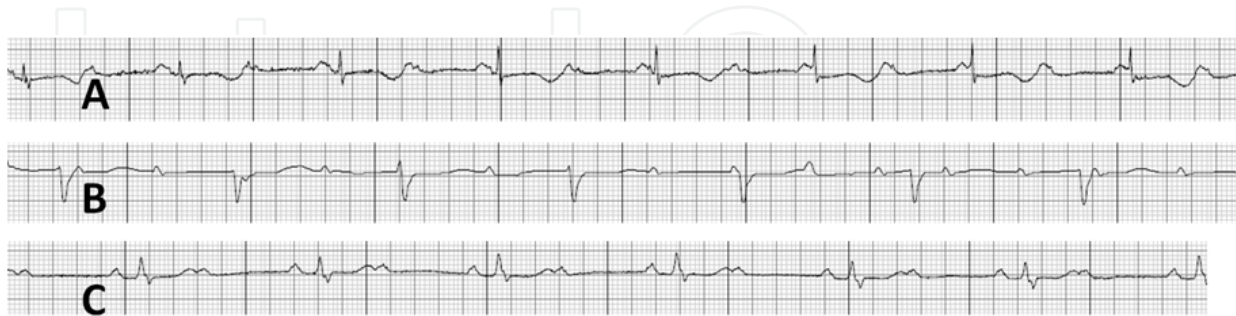


Figure 11. Rhythm strip suggestive of 2:1 AV block (A). However, the QRS complexes are „creeping in” on the preceding P waves – there is complete AV dissociation, more typical for complete heart block with junctional escape rhythm. A similarly difficult tracing (B), suggestive of first degree AV block. As the atrial rate accelerates, complete AV dissociation becomes apparent. (C) True 2:1 AV block – the PR interval following the conducted P waves is constant. Note that the P-P interval slightly irregular (short-long), which may represent ventriculophasic sinus arrhythmia or atrial bigeminy.

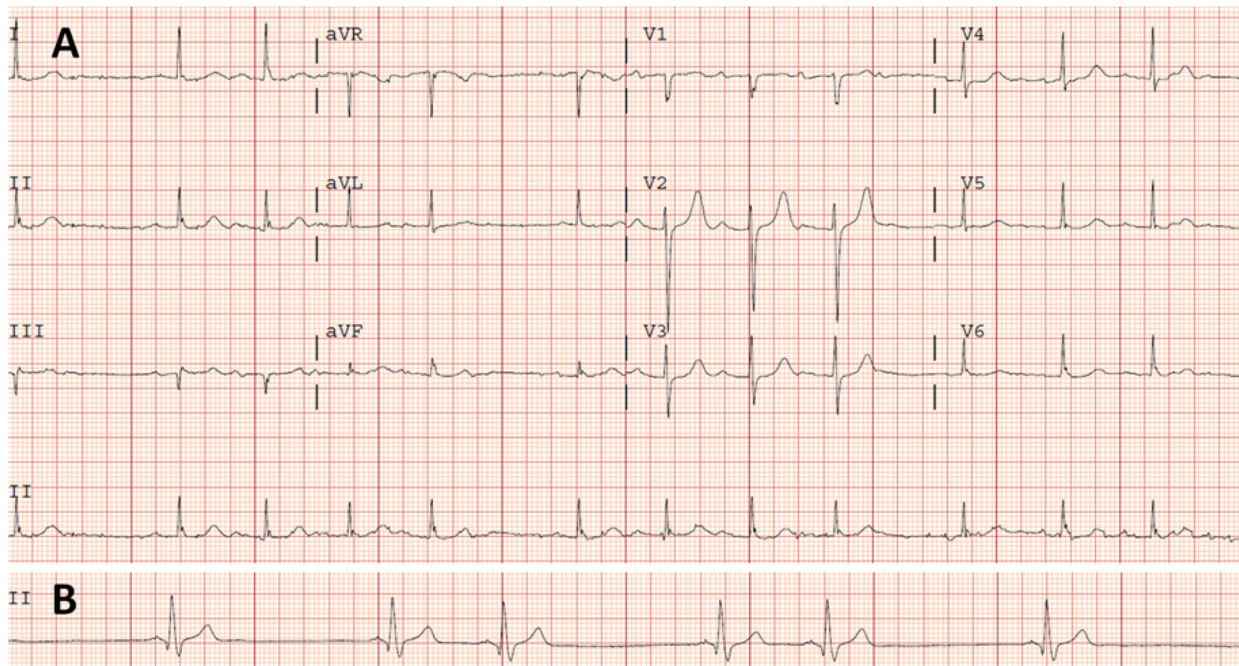


Figure 12. Blocked premature atrial beats (PACs) should be identified as they elicit a different response during atrial pacing (inhibition) than sinus arrest (pacing). In (A), V1 gives the clue for the arrhythmia mechanism – blocked PACs. In (B), there are no visible early P waves – this is 3:2 sinoatrial block.

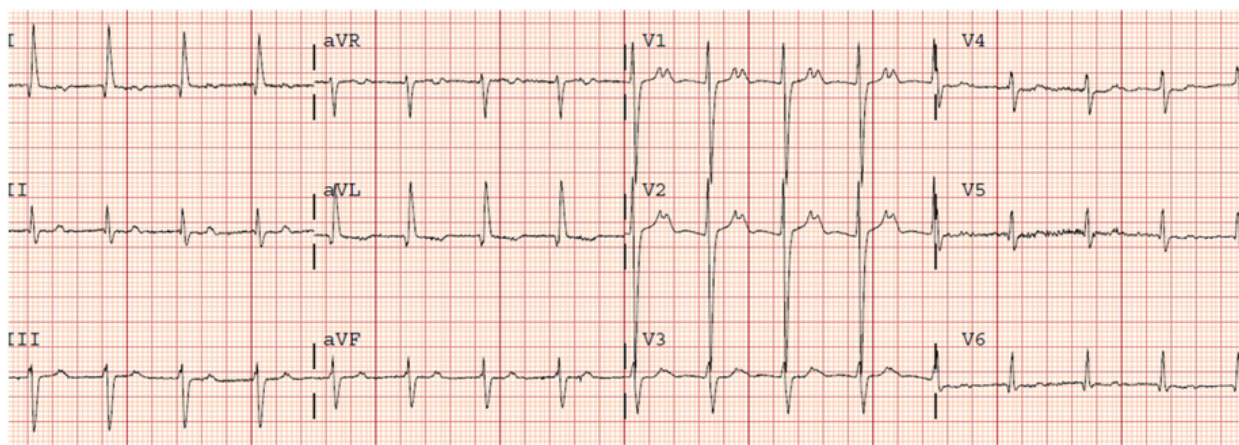


Figure 13. Sinus tachycardia with 1st degree AV block resembling junctional tachycardia. P waves with constant PR interval can be seen in V1-2.

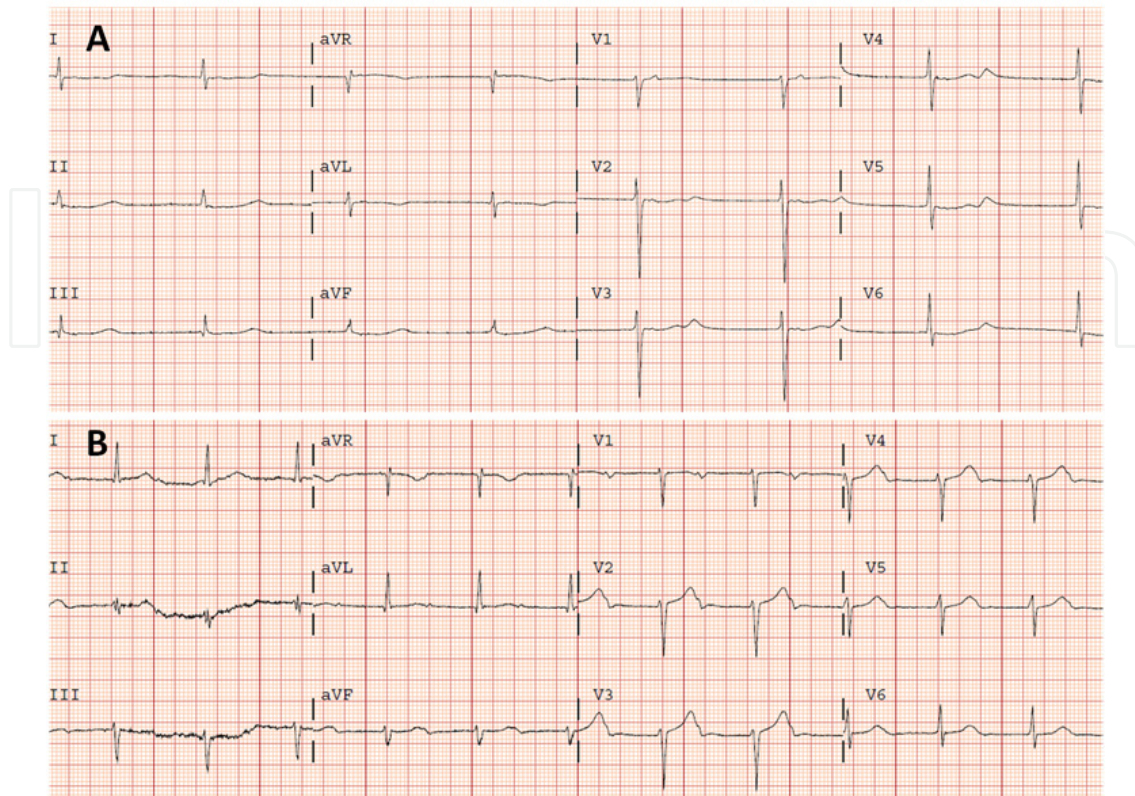


Figure 14. Junctional rhythm with 1:1 VA conduction. Retrograde P waves are visible in V1, which may be misinterpreted as T waves. Note, however, the prolonged QT in all other leads (A). Very long (640 ms) first degree AV block may be confused with junctional rhythm, however, P waves are seen in V1 (B).

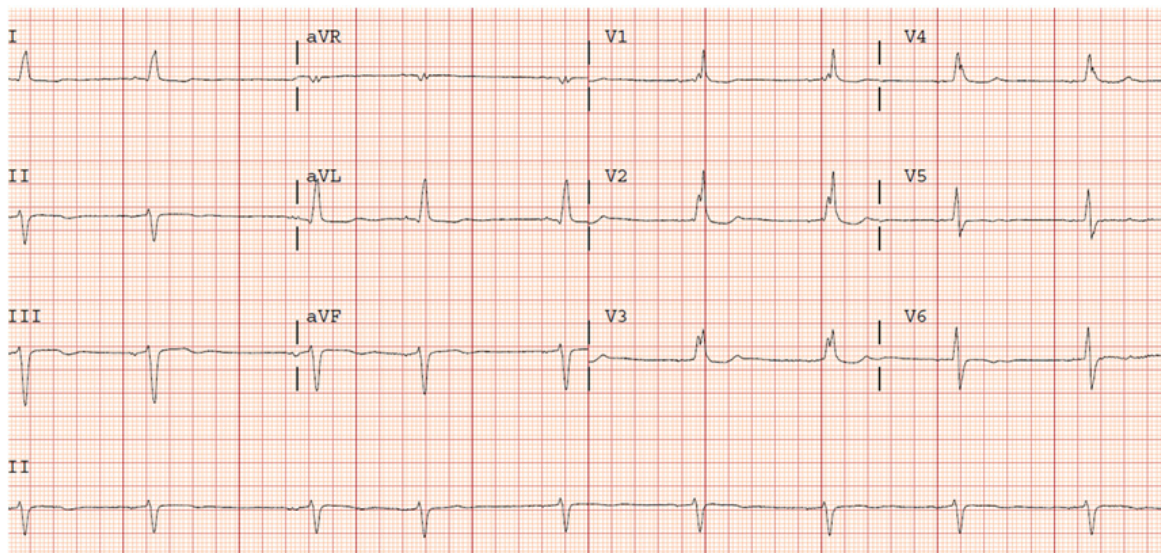


Figure 15. Irregular atrial rhythm resembling atrial fibrillation. The actual rhythm is most likely sinus with PACs, The P waves are of low amplitude, however, they can be identified in III and aVL with 1:1 relationship to QRS and with constant AV delay.

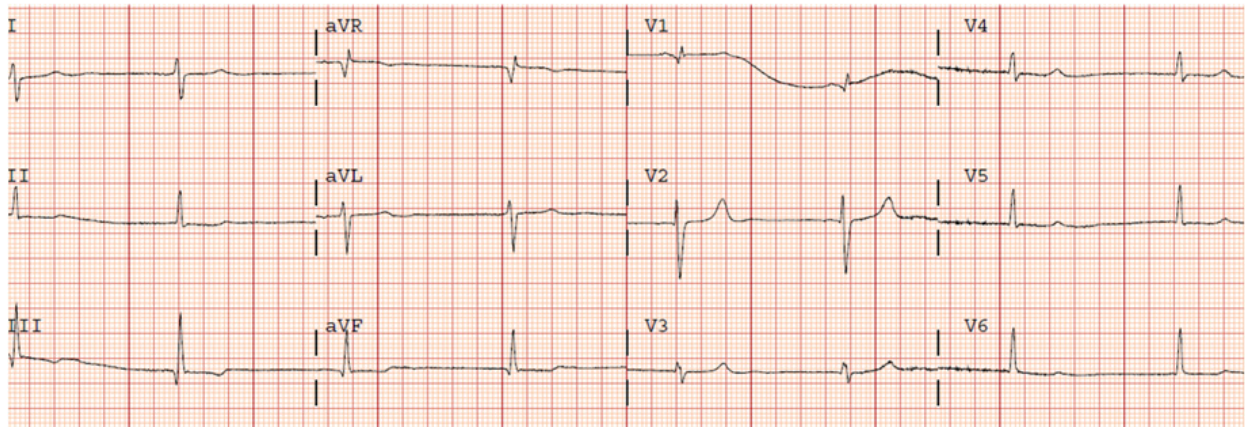


Figure 16. Regular bradycardia with narrow QRS would suggest junctional rhythm, however, P waves are seen before each QRS in V1 – the driving focus is atrial. Advanced atrial conduction disease is not uncommon in pacemaker recipients, leading to low amplitude, fragmented P waves.

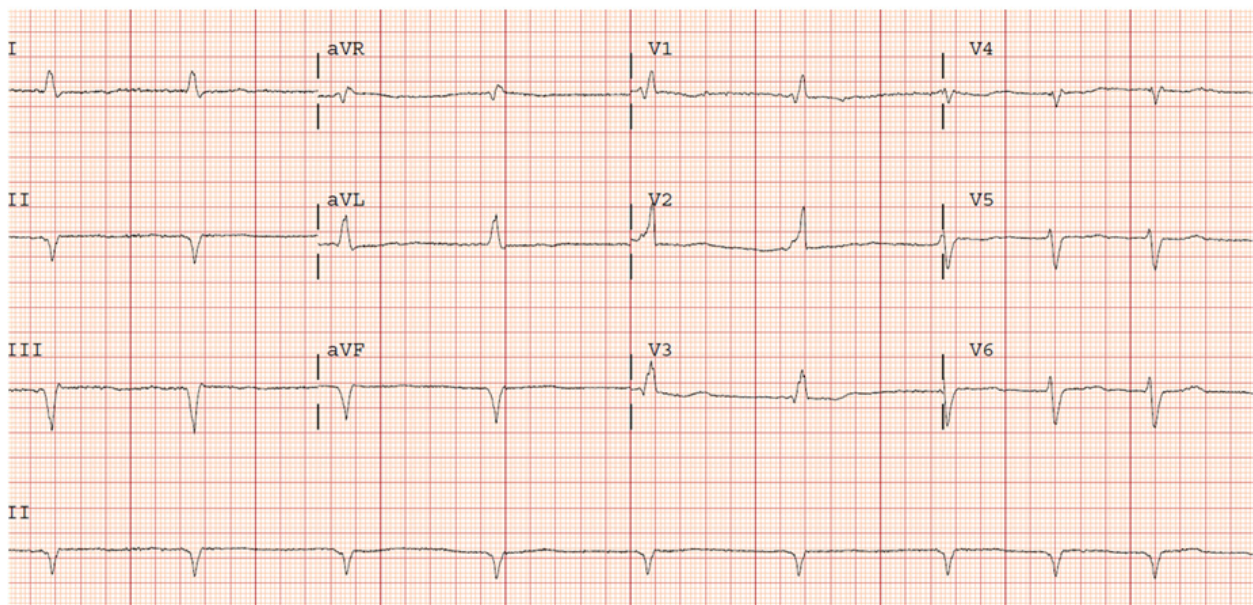


Figure 17. Atrial fibrillation with junctional escape. The regular rhythm may be misinterpreted as pure junctional rhythm, however, the ventricular rate changes when atrioventricular conduction improves and conducted activity overtakes junctional escape. Proper identification of atrial rhythm is important when evaluating pacemaker function – atrial fibrillation should suppress atrial pacing, while atrial pacing should take place with pure junctional rhythm, if an atrial lead is present.

3. Unexpected findings with normal device function

Certain artifacts or interaction of pacemaker algorithms with underlying rhythm may lead to electrocardiographic findings, which may be difficult to distinguish from abnormal function (Balachander, 2011). P/QRS morphology, timing and response to pacing spikes should be addressed, when analyzing the ECG. With ubiquity of bipolar systems, spikes may be difficult to identify (Figure 18). In addition, myocardial depolarization has a vector, which may be isoelectric in certain leads, or may be delayed by intraatrial or intraventricular conduction delay, suggesting ineffective stimulation (Figure 19). Spike morphology may be affected by automatic signal gain function of the ECG system or issues with digital sampling (Figure 20). „Anticipated” spikes may be missing due to very small variations in heart rate, inhibiting demand pacing (Figure 21).

Variable signal morphology may be caused by fusion beats (when the resulting signal morphology is the sum of activation from the pacemaker and spontaneous/conducted activation) or pseudofusion beats (pacing occurs when the myocardium is already refractory from spontaneous/conducted activation, Figures 22-24). Identification of the pacing site is crucial to prove appropriate device function (Figures 25-28).

Occasionally, pacing mode may be difficult to identify based solely on the ECG (Figure 29). It may change due to algorithms trying to minimize right ventricular stimulation (Figures 30-32), rate smoothing function (Figure 33), or arrhythmia – mainly, atrial fibrillation (Israel, 2002).

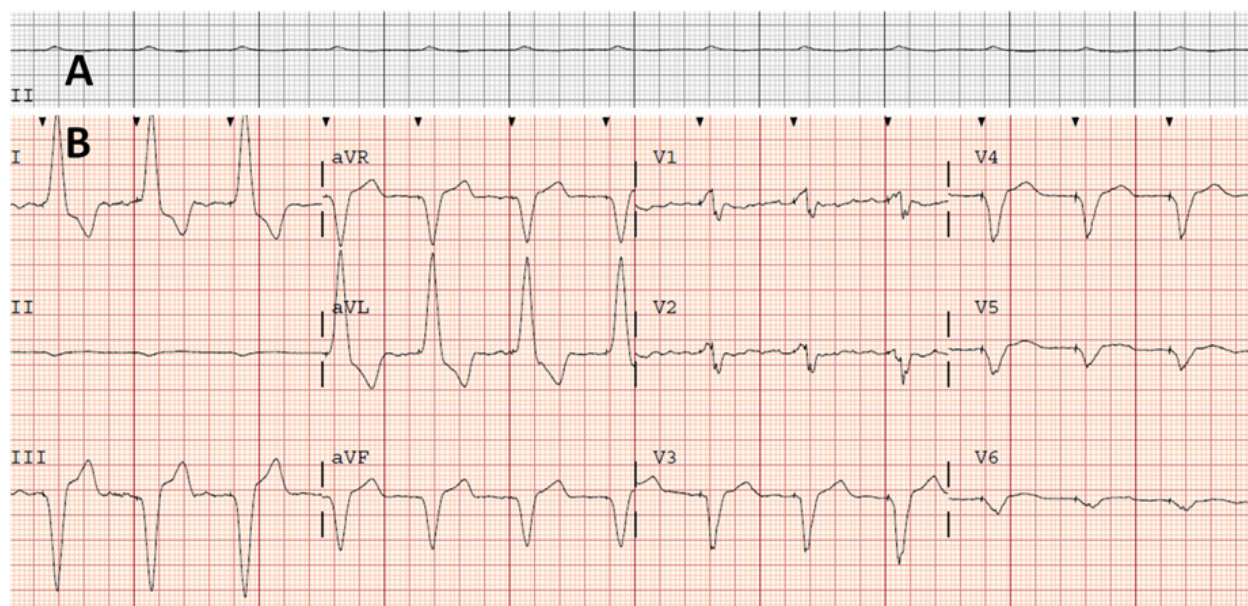


Figure 18. Rhythm strip suggestive of complete heart block and absence of pacing (A). Simultaneous 12 lead ECG shows appropriate ventricular stimulation – the vector of the myocardial activation is close to isoelectric in II.

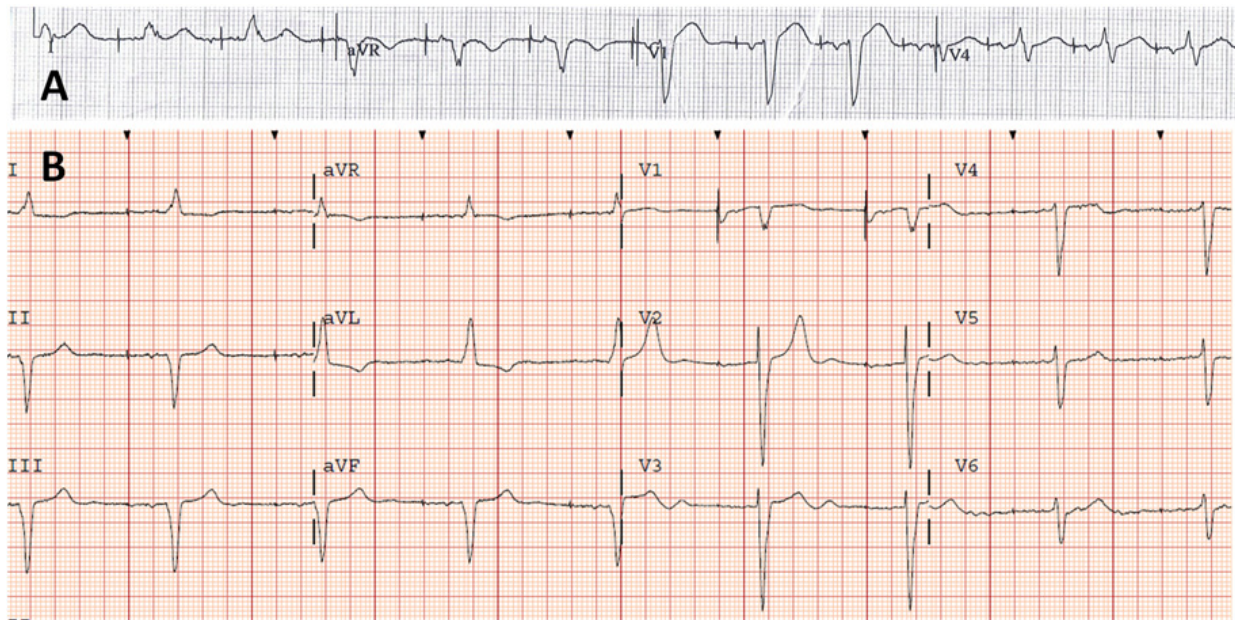


Figure 19. P waves are not seen in I despite effective atrial pacing – the atrial depolarization vector may be isoelectric in certain leads, depending on the atrial pacing site and pathologic conditions. Note that there is a delay in each lead from the atrial spike to the P wave, suggestive of conduction delay (A). Intra-atrial conduction delay may present even in regions far from the pacing electrode – note instant capture in V1, however, significantly prolonged, fragmented P wave in the frontal leads (200 ms) (B).

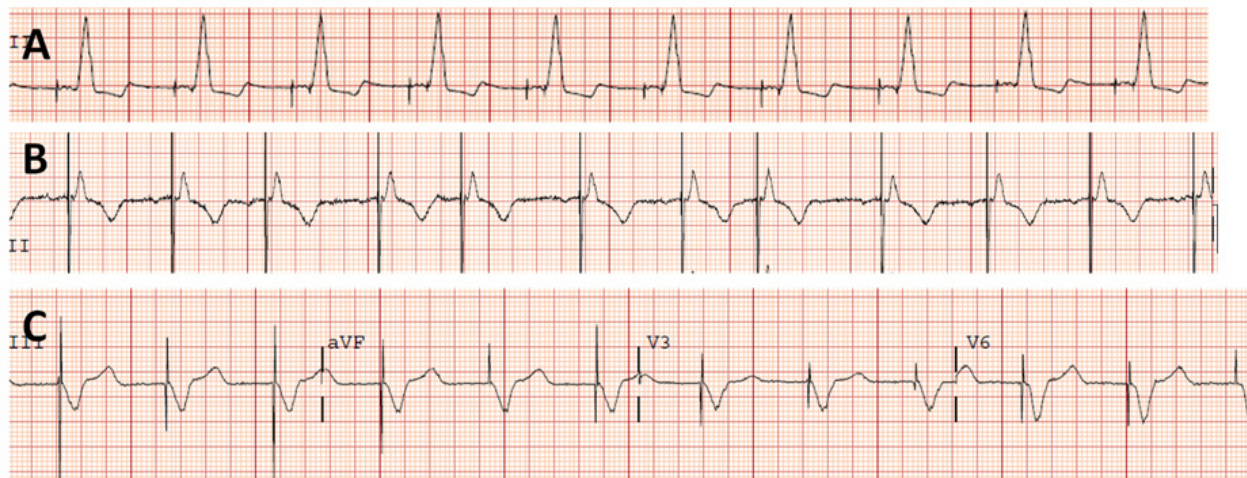


Figure 20. Variable spike morphology (A). This is a normal finding as the spike morphology is affected by the digital sampling of the ECG system and whether it uses pacemaker signal identification/amplification. There is no clinically useful correlation between the spike height and pacing energy. Generally, unipolar pacing (B) leads to much higher amplitude signals than bipolar (A). Spike height may vary not just between different ECG leads, but even with each beat (C).



Figure 21. Sinus rhythm competing with AAI pacemaker – there is appropriate inhibition of atrial pacing when the P-P interval is shorter than the basic pacing cycle length.

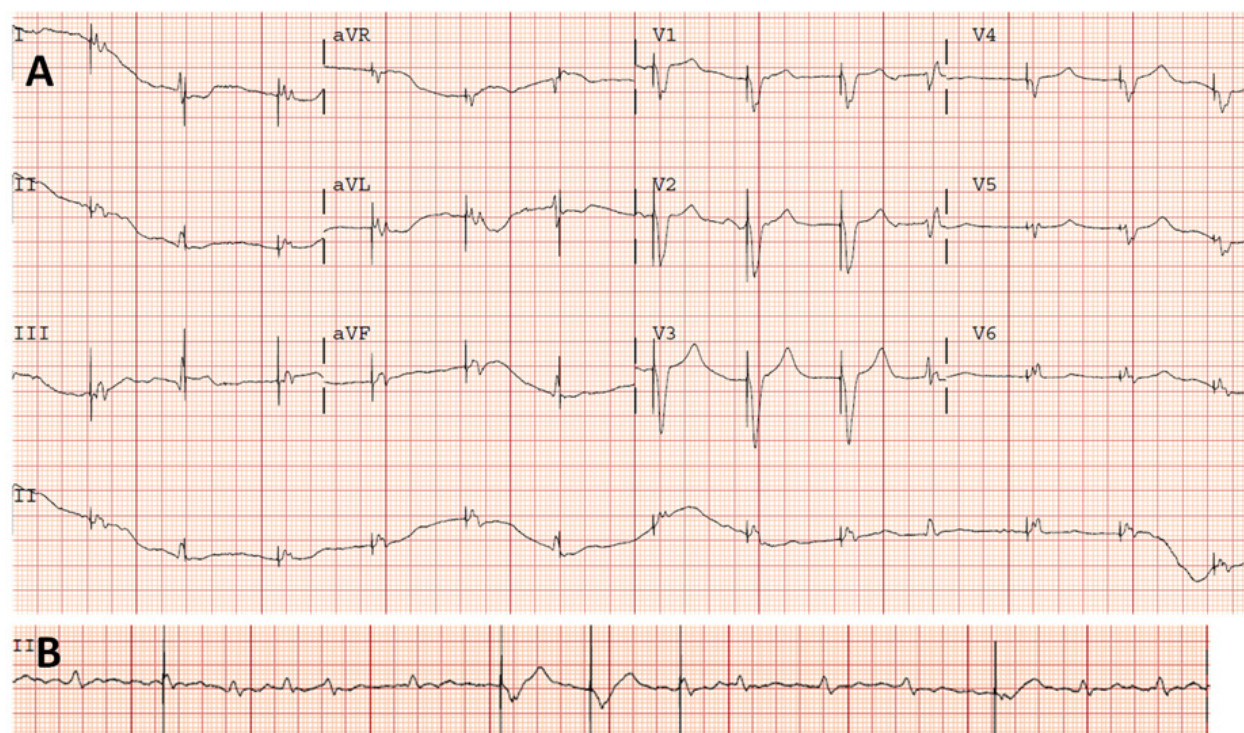


Figure 22. Pseudofusion beats during ventricular stimulation – this is a normal phenomenon as detection of ventricular activation is delayed due to lead tip position (usually right ventricular apex – the ventricles may be partially depolarized, when signal is sensed in this region). Beats 2 and 6 show pseudofusion, ventricular pacing is delivered after the ventricles are depolarized and refractory to further stimuli. Beat 10 is sensed appropriately and pacing is inhibited, as the coupling interval is somewhat shorter - this makes ventricular undersensing very unlikely as the cause for pseudofusion (A). Fusion and pseudofusion beats are very common during atrial fibrillation due to the wide range of coupling intervals (B).

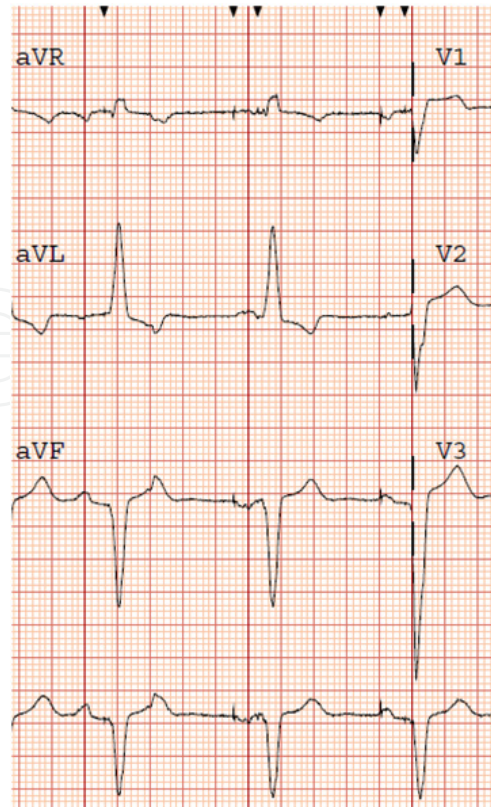


Figure 23. Fusion can be also encountered in the atria, although may be more difficult to identify due to lower signal amplitudes. Undersensing of PACs should be excluded and may require longer tracings or device interrogation.

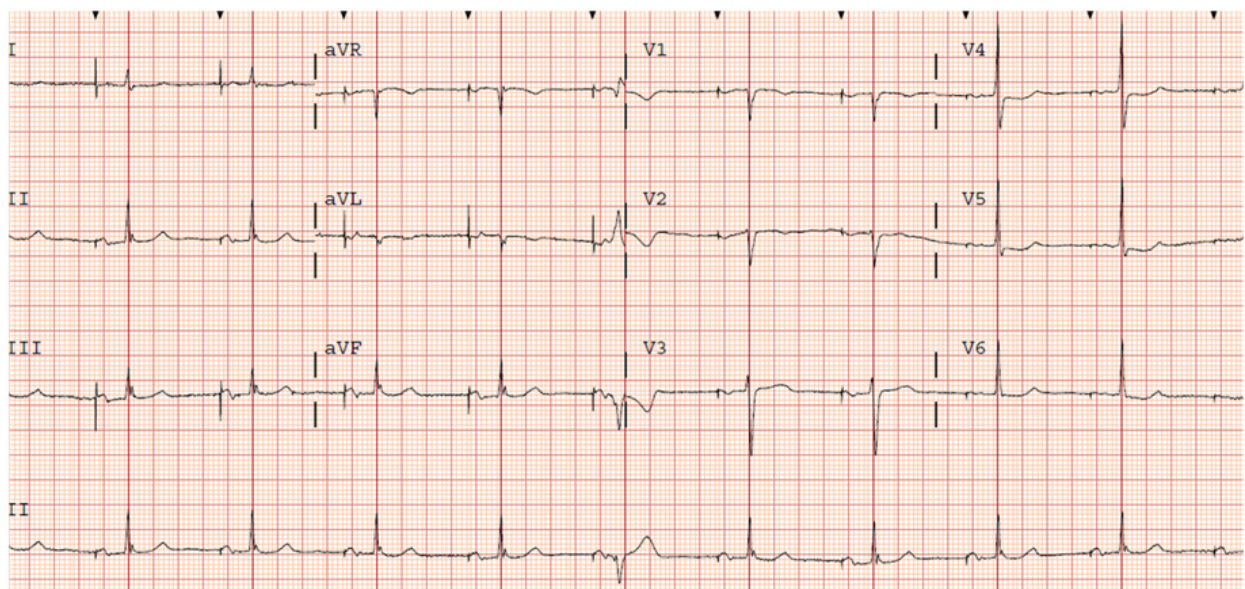


Figure 24. Wide QRS beat encountered during regular atrial pacing with short PR interval. This is most likely a premature ventricular contraction (PVC), fusing with the atrial paced, spontaneously conducted beat.

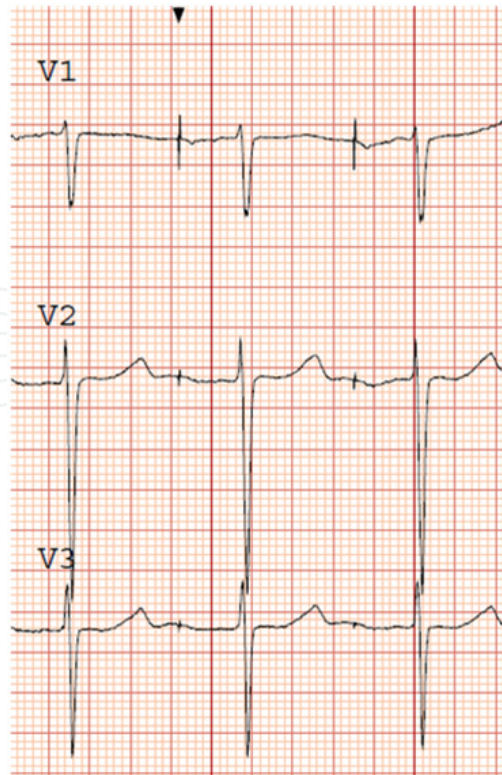


Figure 25. Pacing spikes fall into the U waves in V2 and V3 and are not followed by apparent capture. However, in V1 atrial capture is clear.

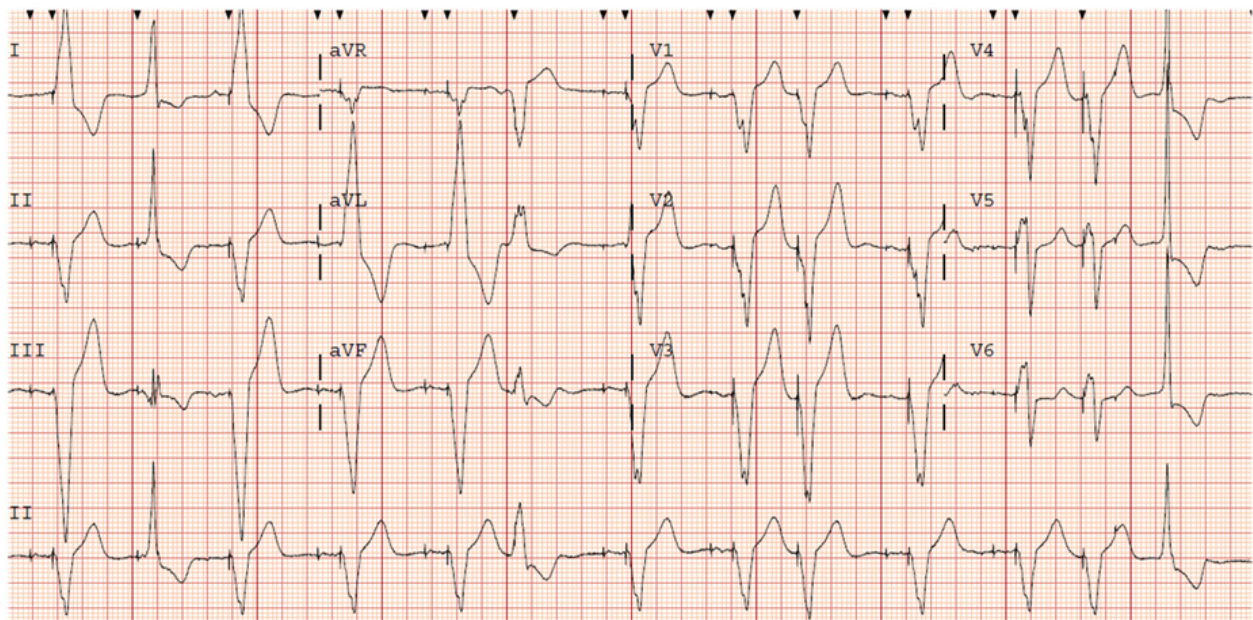


Figure 26. Pseudo pseudofusion – a spike appears immediately before (3rd spike) or within a QRS (9th spike). However, these are atrial spikes and the tracing represents appropriate DDD pacemaker response to frequent premature ventricular and atrial beats. Origin of pacing spikes should be identified based on their timing and sequence to avoid misinterpretation as undersensing or ineffective capture.

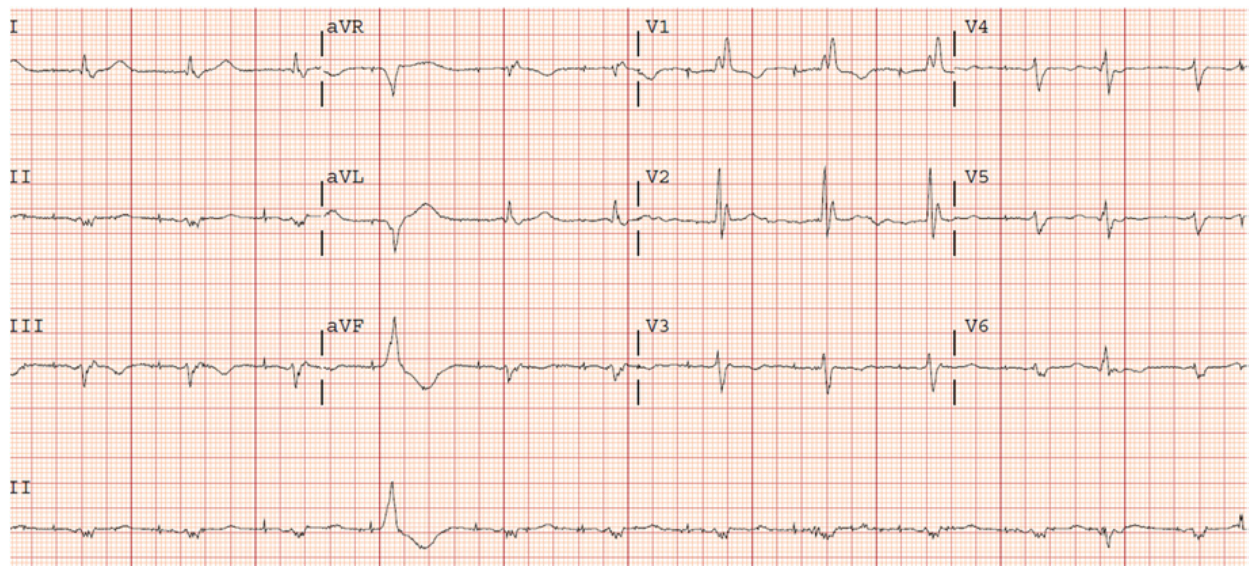


Figure 27. Identification of pacing site is important to avoid misinterpretation. The 4th spike seems to be non-capturing and is followed by a wide QRS beat with an 80 ms delay. However, this is an atrial stimulus as it is apparent by reviewing the consecutive beats. The wide QRS beat is a PVC, which does not have any correlation with atrial pacing. The P waves are of low amplitude and difficult to identify, however, regular atrial pacing followed by regular ventricular activation with the same atrioventricular delay suggests consistent atrial capture.

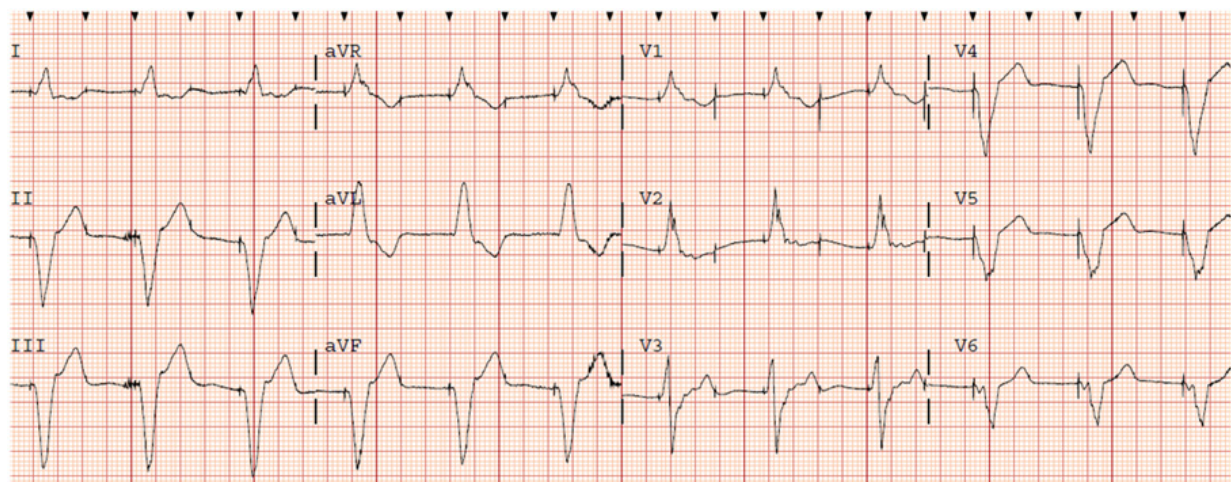


Figure 28. High frequency pacing may occasionally be a sign of serious pacemaker malfunction (runaway pacemaker) or appropriate response to an arrhythmia (tracked sinus/atrial tachycardia). In dual chamber systems, proper identification of pacing spikes is necessary for troubleshooting. In this tracing, 150/minute pacing seems to be capturing 2:1. Note, however, that the pacing is regularly irregular (short-long) and the spike morphology is alternating in V1 – every other one is an atrial spike. The patient is in atrial fibrillation, which is undersensed and the DDD pacemaker is delivering dual chamber stimulation at 70/minute with an AV delay of 400 ms.

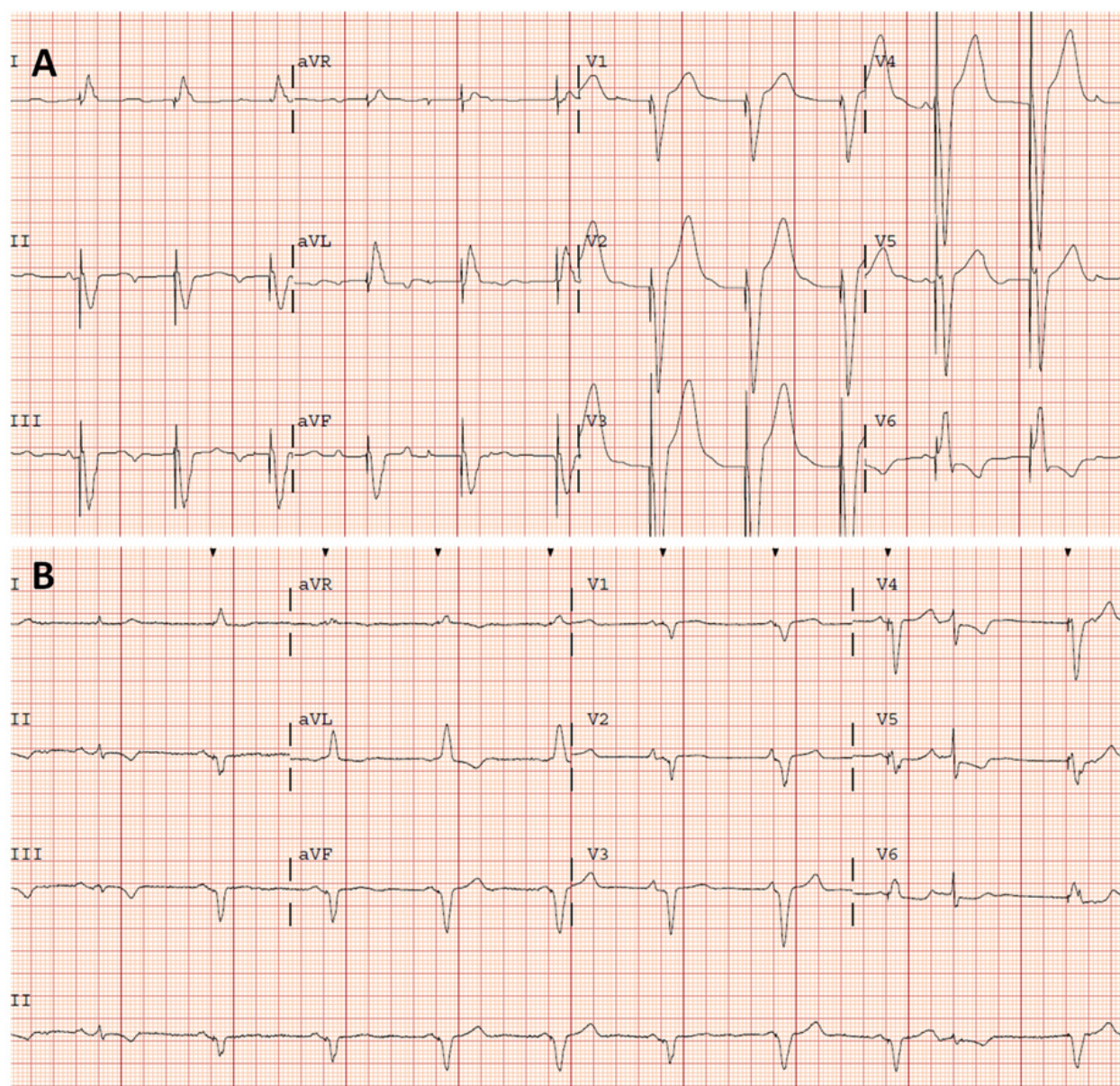


Figure 29. Pacing mode may be difficult to identify from surface ECG. (A) In III and aVF it may appear that the atrial activity is tracked to the ventricles. However, the ventricular rate is completely regular despite variable P-P intervals. AV dissociation is seen in V4 and V5. This device is a VVI pacemaker in a patient with complete heart block. (B) Isorhythmic dissociation between sinus rhythm and VVI pacing – close inspection of the PR intervals reveals that the atrial activity is not tracked

If pacing spikes are seen during tachycardia, most common causes are atrial tachyarrhythmia tracked by the pacemaker, or true PM mediated tachycardia (caused by retrograde conduction or atrial oversensing of ventricular events, leading to endless loop tachycardia). Rate response function may also cause transient increase in pacing rate. The differential is usually difficult based on surface ECG alone, unless initiation and termination can be clearly identified. Device interrogation is strongly recommended (Ip, 2011). Transient changes in rhythm may elucidate the mechanism of a suspected malfunction (Figure 34).

Both atrial and ventricular tachyarrhythmias may raise the concern of device malfunction. If no spikes are seen, the rhythm is likely not related to pacing and patient-related issues should be suspected (Figures 35-38). Underlying rhythm should be identified: atrial fibrillation/flutter may be difficult to recognize with asynchronous pacing, but still pose a thromboembolic risk (Figures 39-40).

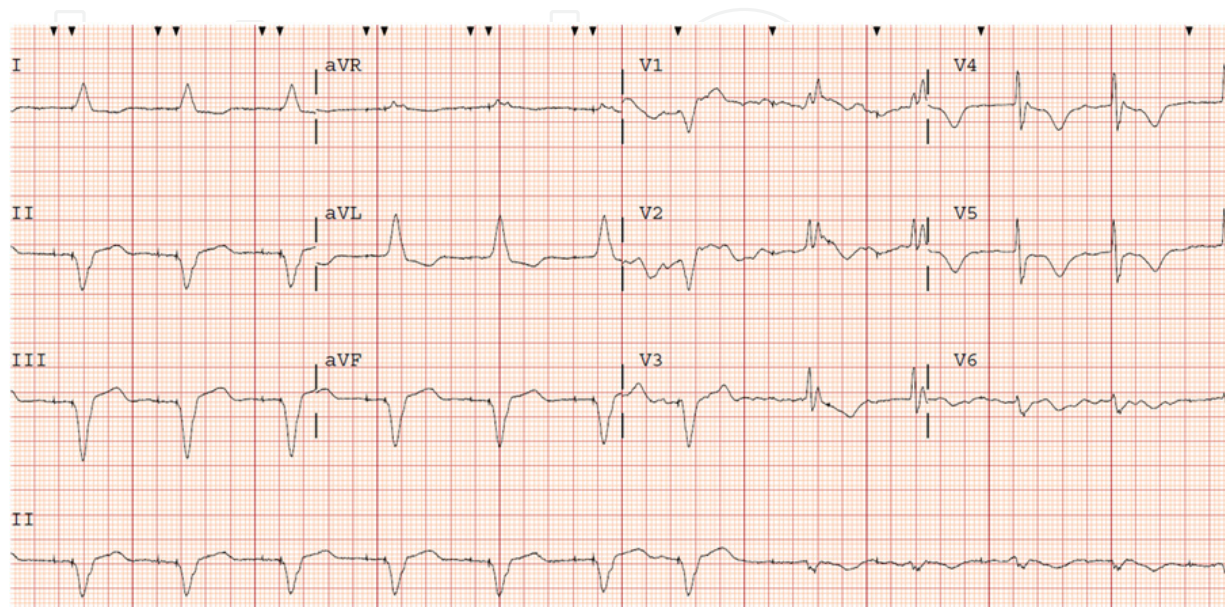


Figure 30. Response to a premature atrial beat with managed ventricular pacing algorithm (Medtronic, Inc). The DDD pacemaker is delivering atrioventricular stimulation, then an atrial-sensed ventricular pace following a premature atrial beat. Following this beat, an atrial stimulus is delivered with mode switch to ADI. As atrioventricular conduction is detected, the device continues with atrial stimulation and allows spontaneous conduction with prolonged AV delay.

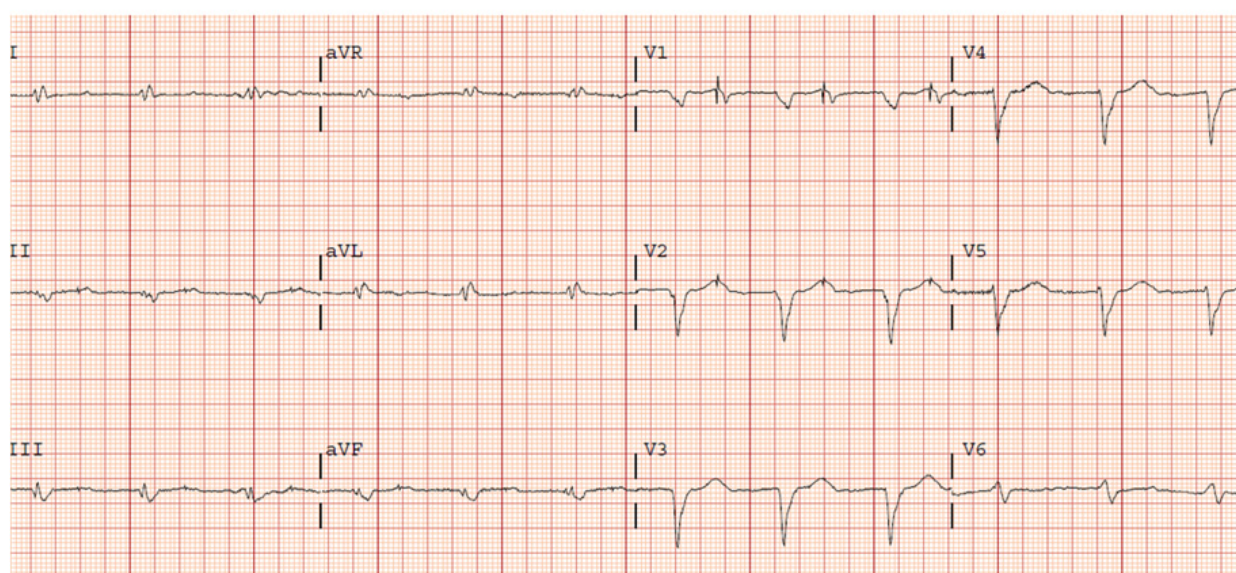


Figure 31. Managed ventricular pacing may maintain very long AV interval, if the 1:1 atrial:ventricular ratio is maintained. In this case, atrial pacing spikes occur after the QRS, in the T waves. V1 shows atrial capture with an AV delay of 460 ms.

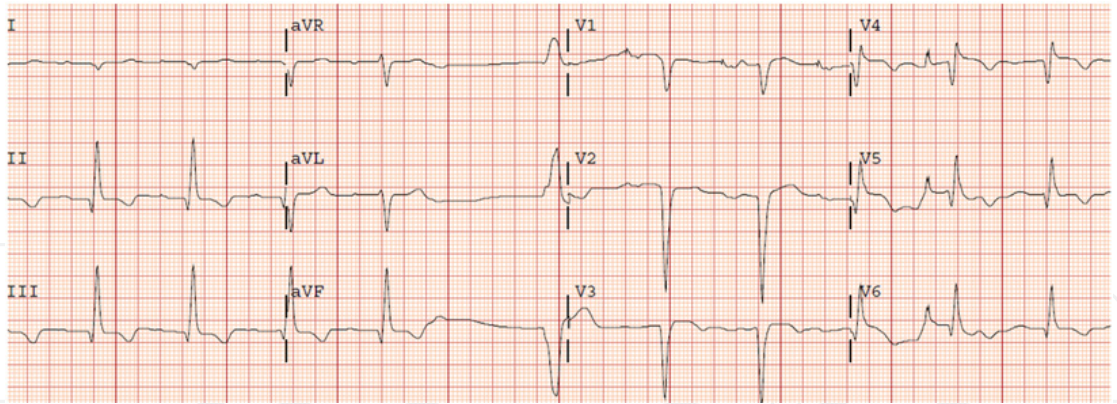


Figure 32. Heart rate may drop down to $\approx 50\%$ of the basic rate for one cycle with managed ventricular pacing – in this case a late blocked PAC is followed by an atrial stimulus, followed by a ventricular stimulus with very short AV delay (wide QRS beat, the spikes are not visualized in these leads). V1 gives the clue that the rhythm is paced at 70/minute. The artifact in V4-6 is not related to pacing.

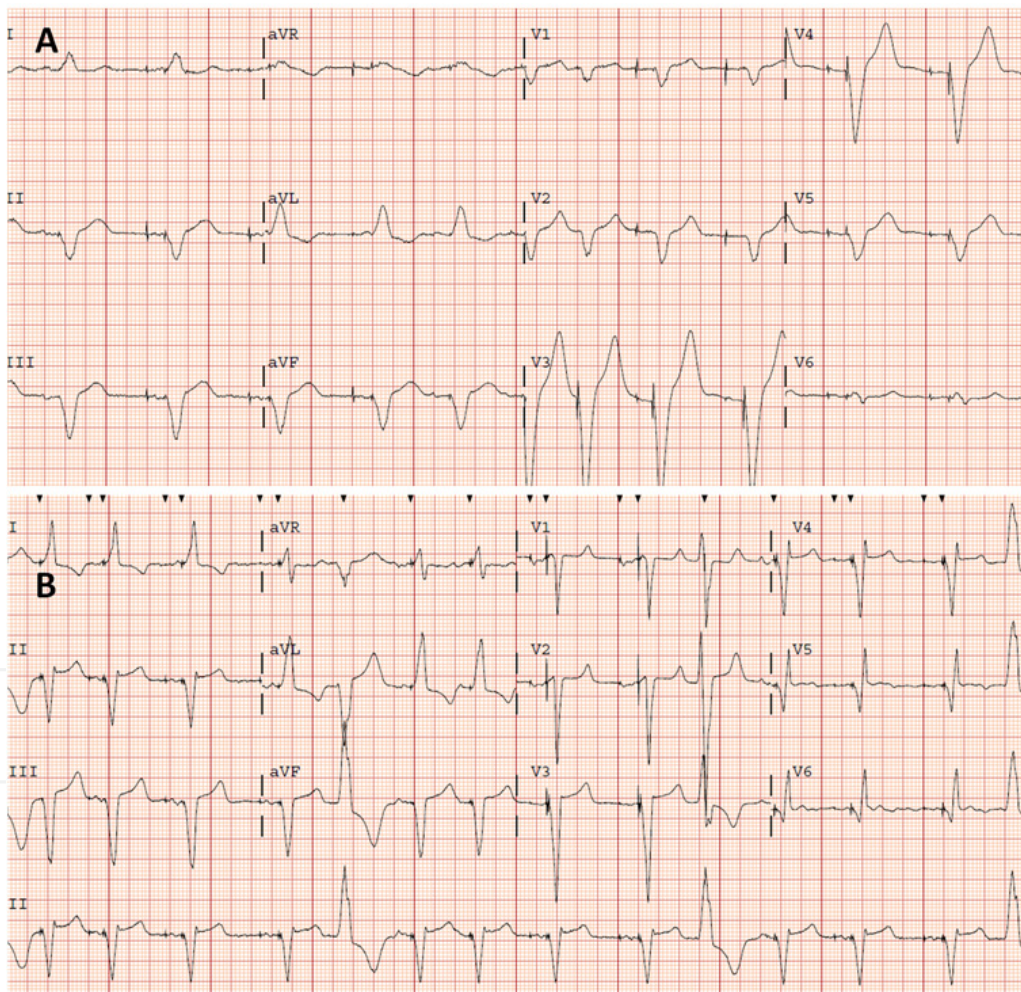


Figure 33. Rate smoothing with a DDD pacemaker (A). Following the premature beats, the atrial pacing rate is gradually decreased to the basic pacing rate. Irregular pacing caused by rate smoothing in a biventricular system (B). All premature beats are sensed (ventricular and atrial), and either a sense response pace or an tracked biventricular pace is delivered. The basic pacing rate is gradually decreased after these over a few cycles, the lowest pacing rate on this tracing is 65/minute.

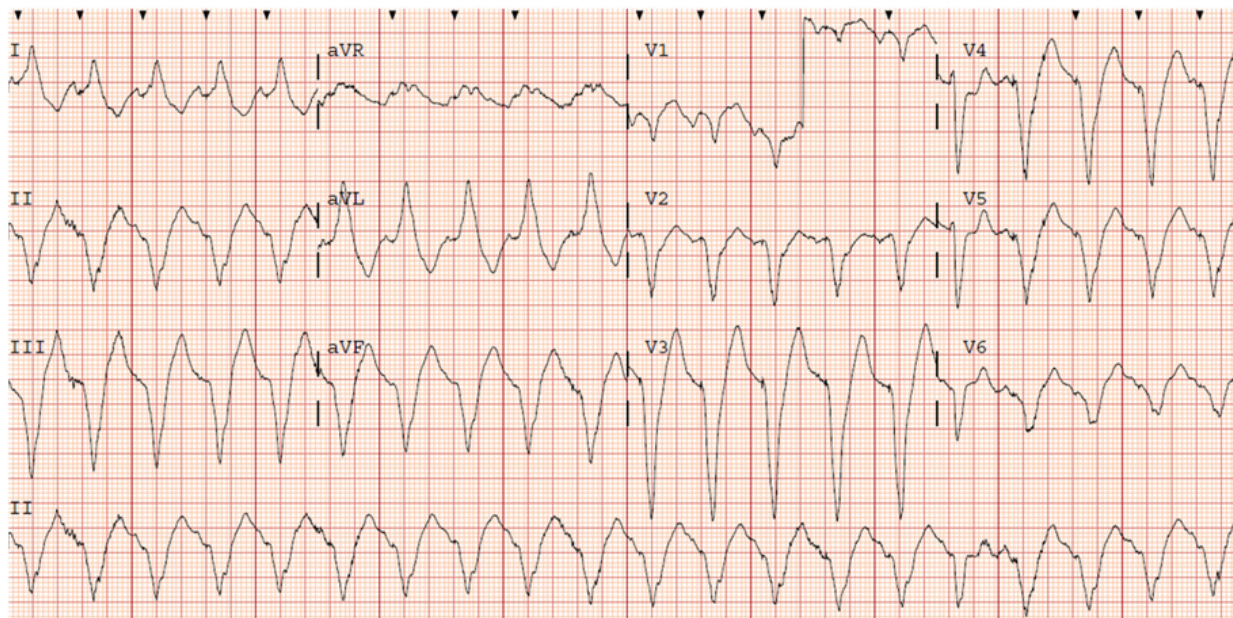


Figure 34. Sudden changes in regular tachycardia may elucidate the mechanism. The premature beat unmasks a P wave, followed by a ventricular paced beat – this is a supraventricular tachycardia tracked by the dual chamber pacemaker.

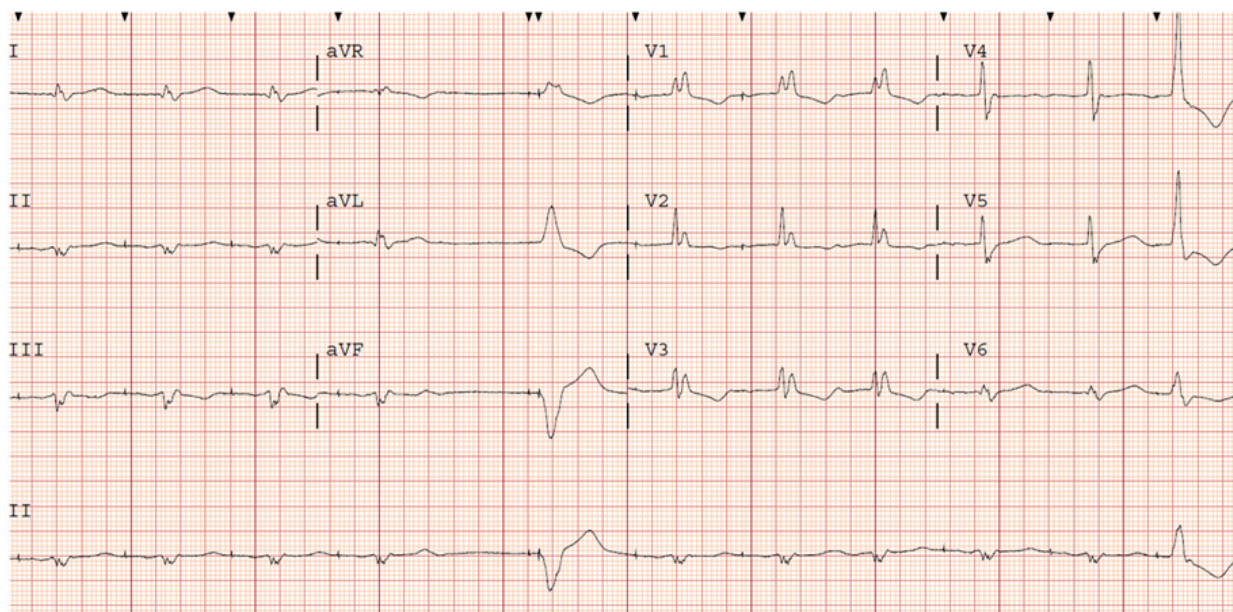


Figure 35. Hidden premature atrial beat mimics pacemaker malfunction. The 5th atrial spike is delayed, suggestive of oversensing, however, the 4th T wave in the rhythm strip is different from the previous three, suggestive of a buried premature atrial beat, with AV block. The 5th atrial spike actually comes right on time as the pacing cycle was reset by the premature atrial beat. Additionally, a ventricular pace is delivered by the managed ventricular pacing algorithm.

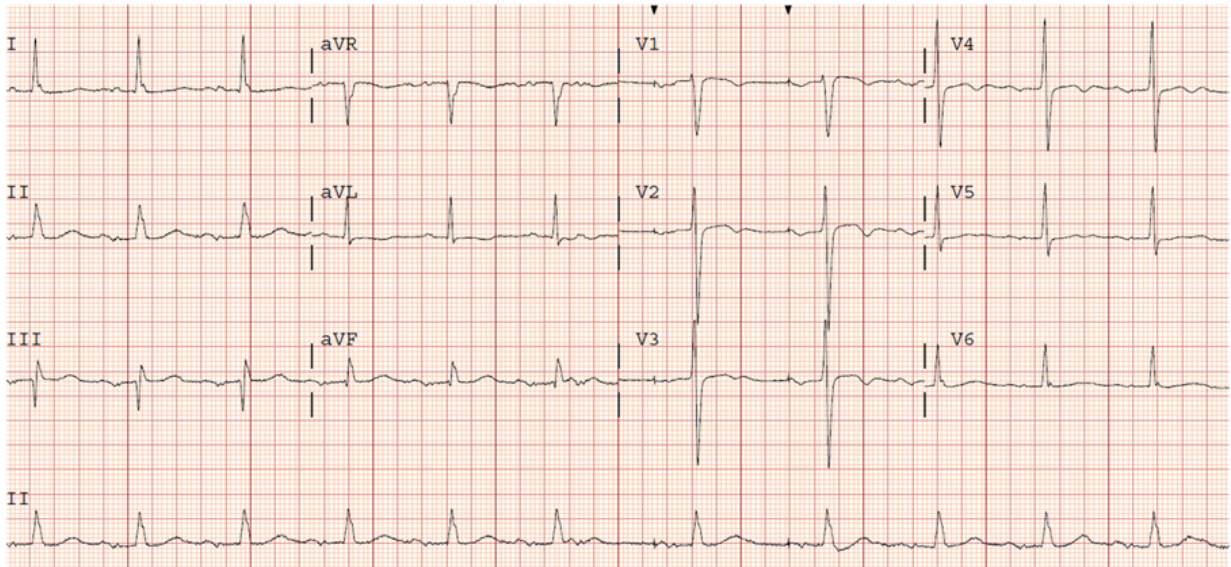


Figure 36. Blocked PAC without tracking with a DDD pacemaker. This is normal function, as the blocked PAC (after the 6th QRS) comes very early and was sensed in the post-ventricular atrial refractory period. Instead, an atrial stimulus is delivered later to maintain the basic rate. As there is spontaneous AV conduction, ventricular pacing is inhibited. After 2 atrial paced beats, sinus rhythm takes over again.

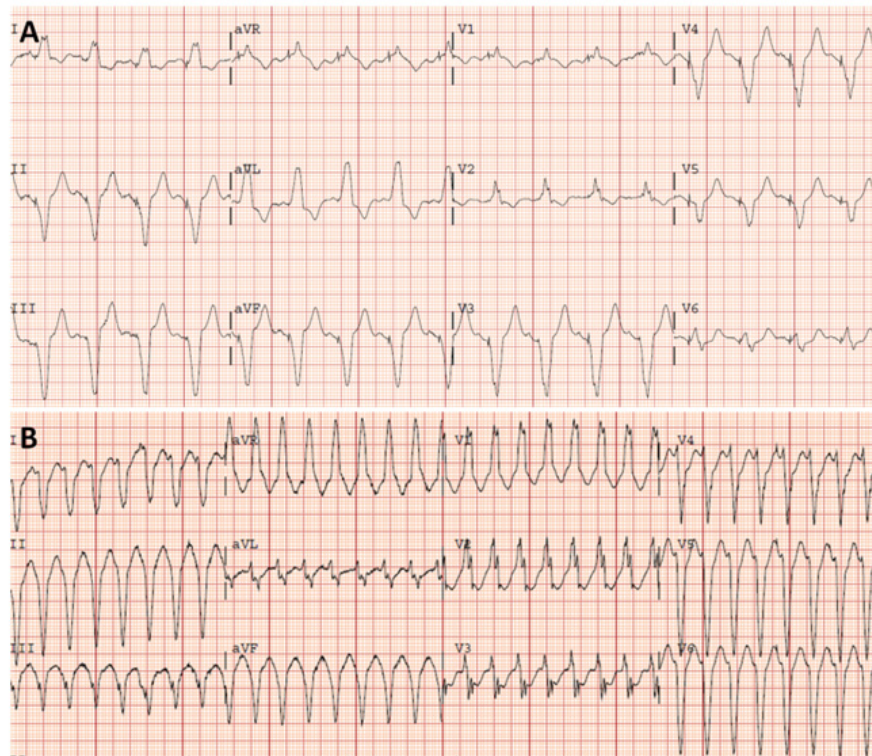


Figure 37. Wide QRS tachycardia in pacemaker recipients. Sinus tachycardia with appropriate sensing and ventricular pacing (A). With bipolar pacing the spikes may not be visible in all leads and the rhythm may be misinterpreted as ventricular tachycardia – especially in telemetry tracings. When ventricular rate is higher than the upper tracking rate (if known) or the QRS morphology is not compatible with usual pacing sites, VT (B) or SVT with aberrancy should be suspected.

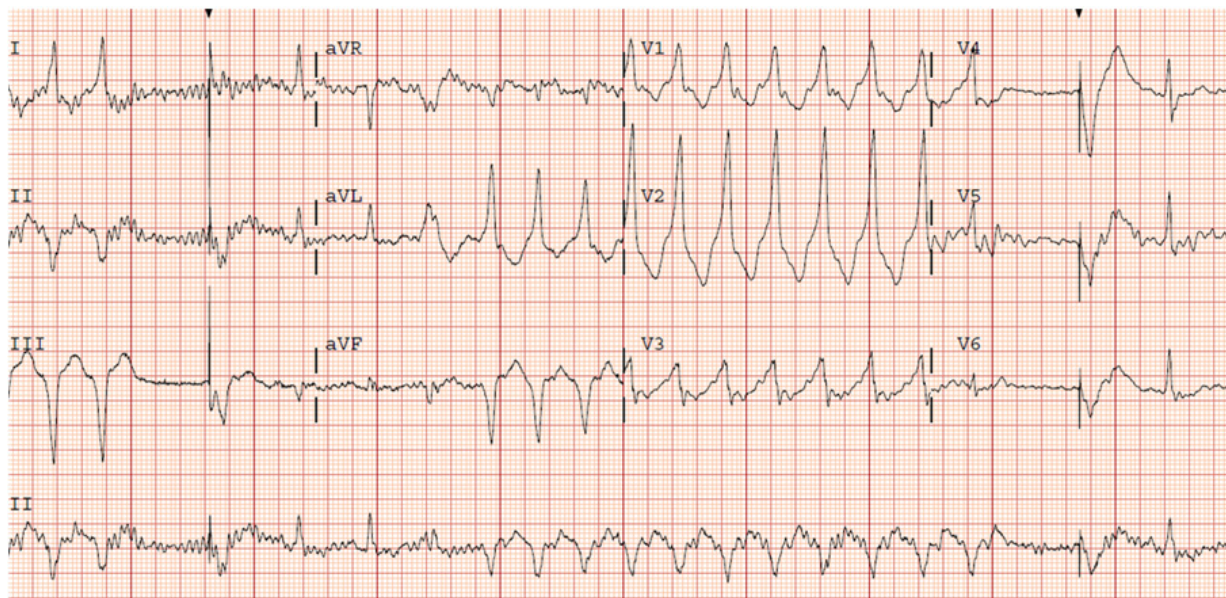


Figure 38. Non-sustained wide QRS tachycardia in a patient with a VVI pacemaker. Note appropriate demand pacing and the absence of spikes during the tachycardia – this is not pacing-related, but a true non-sustained ventricular tachycardia.

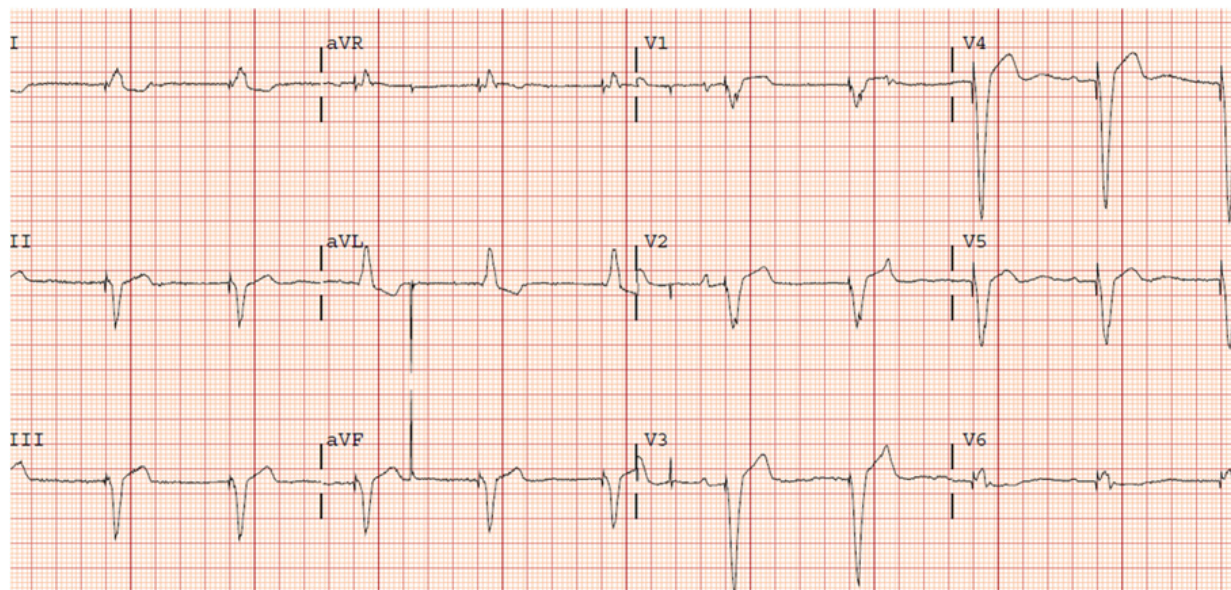


Figure 39. VVI pacing without retrograde conduction. An underlying, slow regular atrial rhythm is seen in V2 and V5. The spikes after the 3rd and 5th beats are artifacts.

ECG analysis should always include assessment of QRS and ST-T, even in patients with paced rhythms. Atrial pacing preserves normal ventricular activation, so it may be interpreted without interference from the device. Underlying conduction blocks may mimic paced beats (Figures 41-42). If artifacts limit interpretation, comparing multiple simultaneous ECG leads may be helpful (Figure 43).

In patients presenting with symptoms suspicious for pacemaker syndrome (hypotension, shortness of breath, dizziness, most commonly in an intermittent pattern), atrioventricular

activation sequence and presence of ventriculoatrial conduction should be assessed. If these are compatible with PM syndrome, device settings should be adjusted (or the device should be upgraded), to restore AV synchrony and avoid atrial contraction during ventricular systole (Figures 44-45).

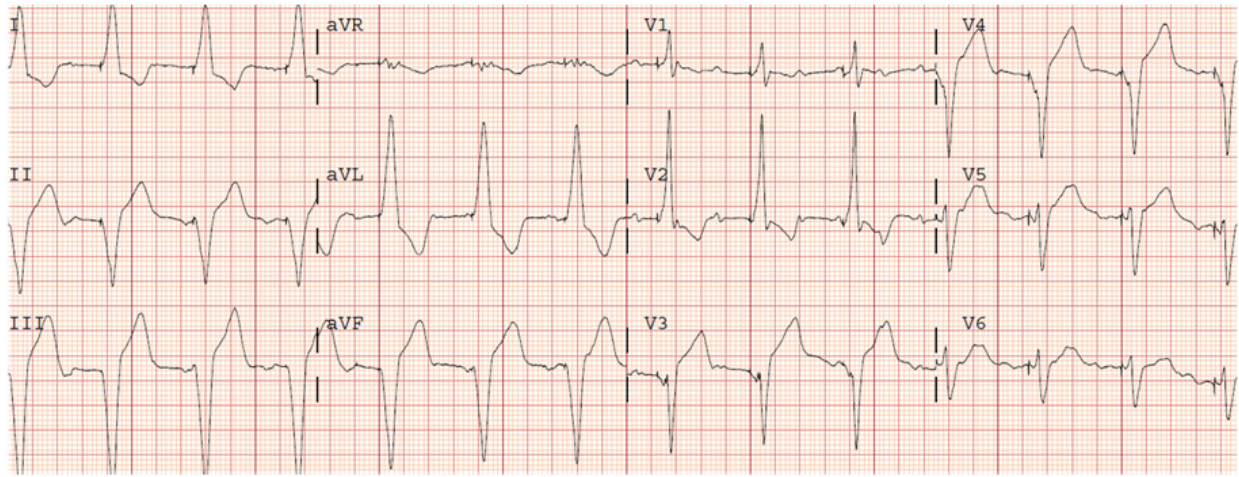


Figure 40. Underlying rhythm is atrial tachycardia or slow atrial flutter with a cycle length around 320 ms. This is neither spontaneously conducted, nor tracked by the pacemaker to the ventricles, 80/minute ventricular stimulation is seen.

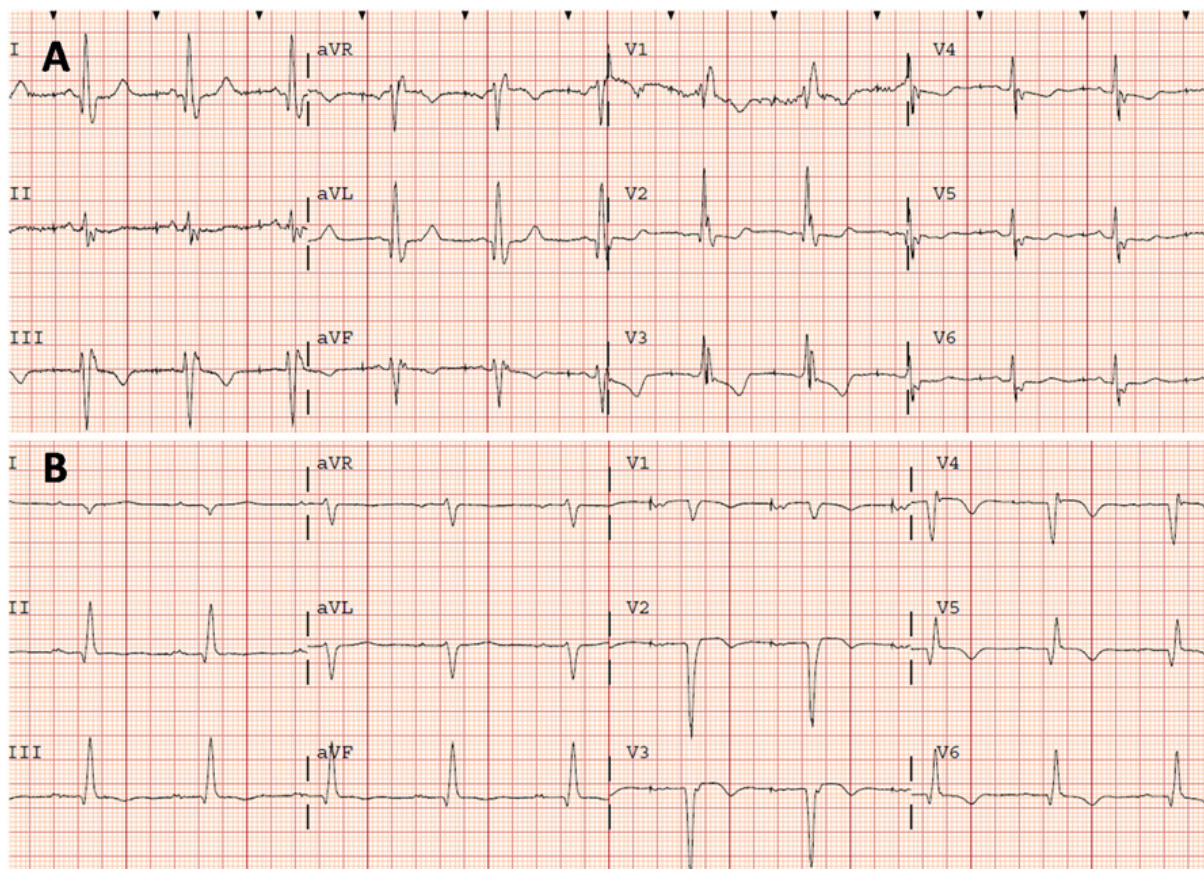


Figure 41. Atrial pacing preserves normal ventricular activation sequence, conventional ECG criteria may be used to identify ischemia, blocks, hypertrophy. RBBB (A), remote anterior MI (B).

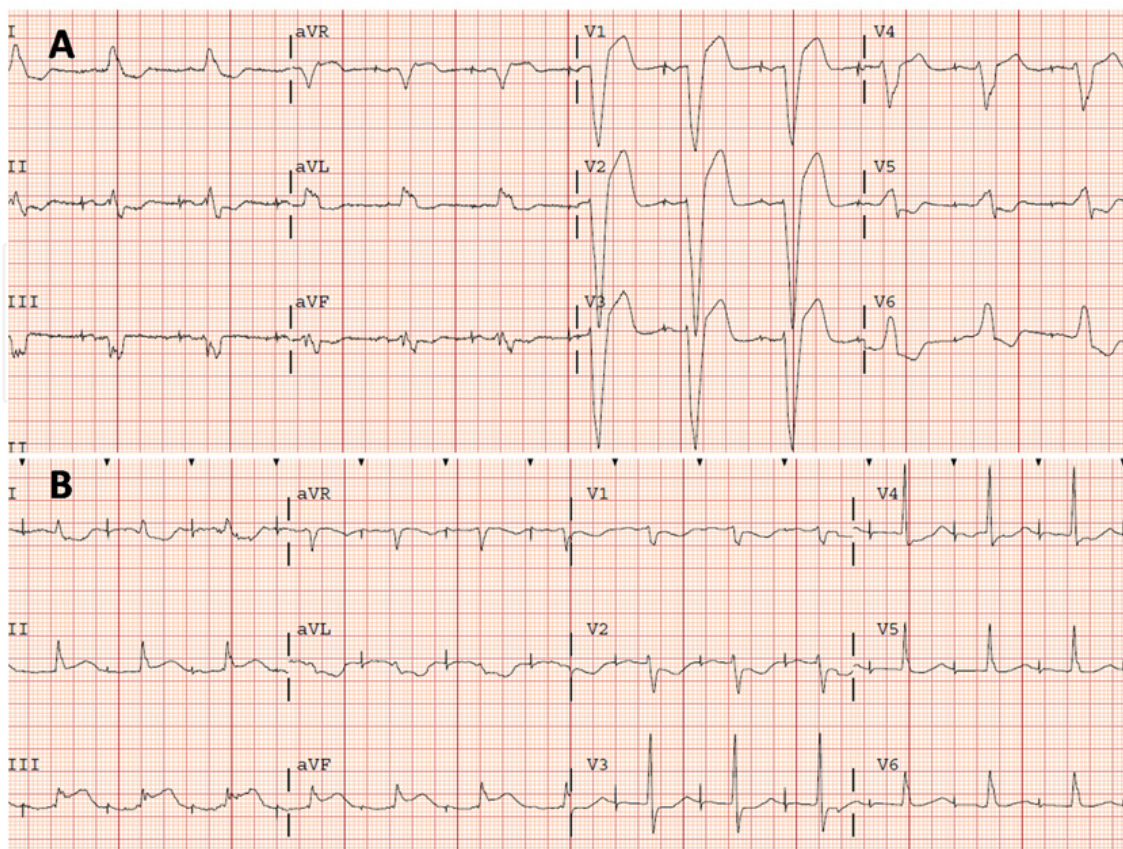


Figure 42. Preexisting LBBB may be confused with dual chamber pacing. Close inspection of the QRS complexes reveals atrial pacing and typical LBBB (A). Acute inferior MI with atrial pacing – typical ST elevation with reciprocal changes (B).

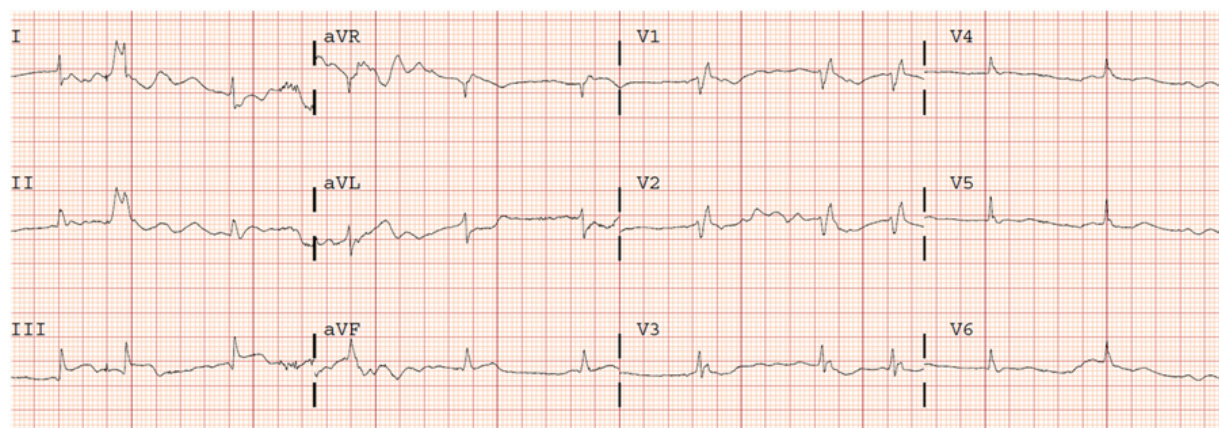


Figure 43. Artifact suggestive of ventricular undersensing with a recently placed temporary right ventricular lead. There appears to be a pacemaker spike shortly after the first QRS with a captured beat, suspicious for undersensing. However, the morphology of this “paced” beat is not typical and note that in III there is a 120 ms delay between the “spike” and the QRS and no change in depolarization/repolarization compared to non-paced beats – this would be impossible with a ventricular paced beat. This phenomenon was caused by an artifact causing a high amplitude, positive deflection in I and II, imitating a LBBB pattern, and there was actually no pacing – the artifact was gained as a spike by the ECG. There are multiple artifacts in I, II and aVR, suggestive of noise coming from the right upper extremity ECG electrode.

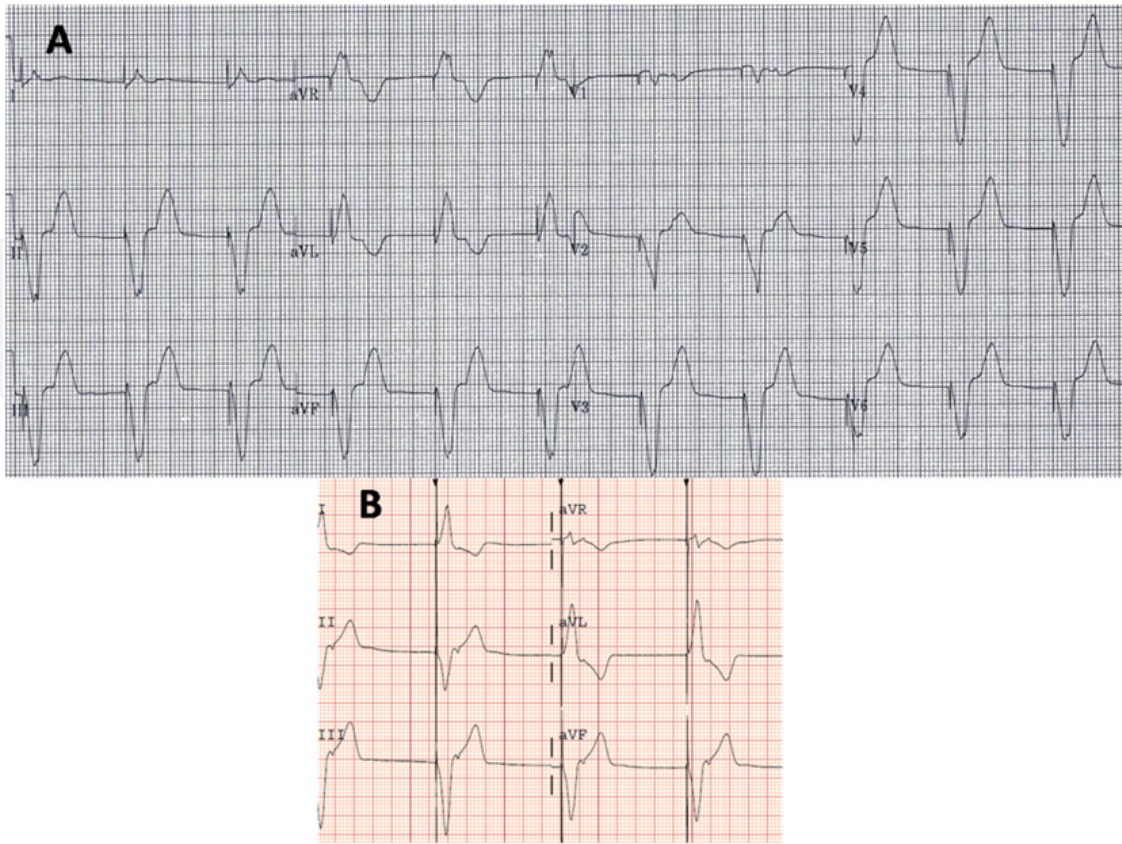


Figure 44. Mode switch due to battery depletion (A). The patient with a DDD PM presented with sudden onset complaints typical for pacemaker syndrome. ECG shows 65/minute ventricular stimulation with 1:1 VA conduction (best seen in V1) – this pacemaker converted to VVI 65/min backup mode when battery condition reached end-of-service. (B) is a more typical presentation of VVI stimulation with 1:1 retrograde conduction – without significant atrial conductive system disease, retrograde P waves are easily recognized

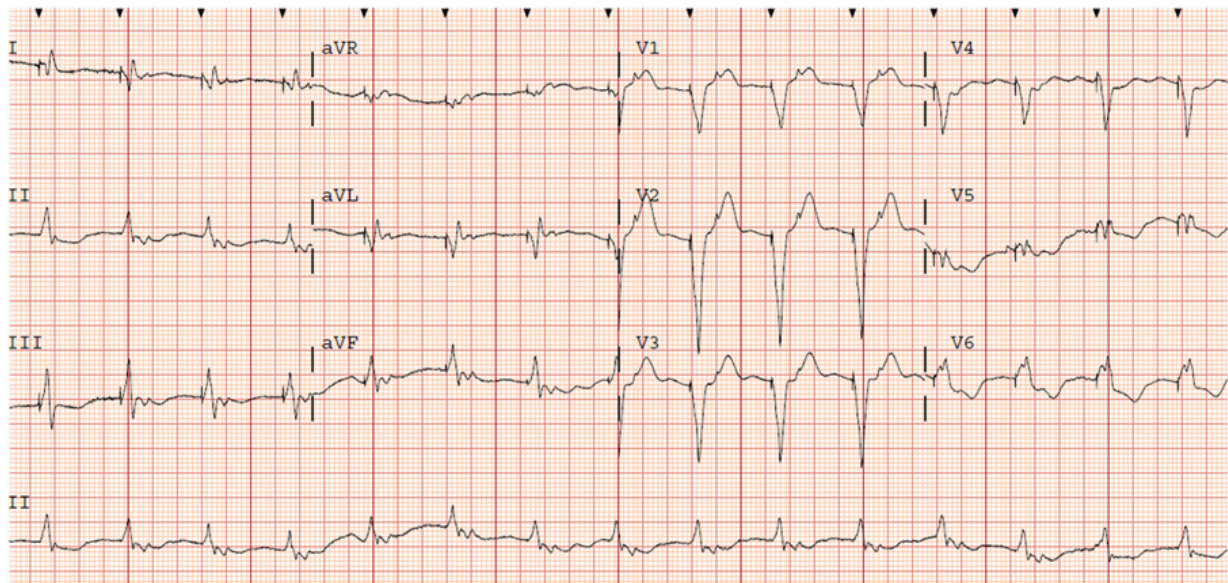


Figure 45. Retrograde conduction may be intermittent even during VVI pacing at constant rate – in this case, it starts after the 2nd beat and ends 3 beats before the recording ends.

4. Pacemaker or lead malfunction

Most common pacemaker and lead related malfunctions, that should be promptly identified and corrected, include oversensing, undersensing and ineffective stimulation. These may be related to inappropriate settings that may be easily corrected with a programmer, however, pacemaker lead related issues (dislocation, fracture, insulation failure) may present similarly and require hardware revision. If true pacemaker dysfunction cannot be ruled out with certainty based on ECG, device interrogation should be performed – this is especially important, if the patient was exposed to factors with potential device interaction, such as MRI, therapeutic irradiation, trauma, or drugs with known effect on pacing threshold (Goldschlager, 2001).

Pure undersensing may be identified by a pacing spike that comes early compared to the anticipated timing, with appropriate capture, if the paced chamber is not refractory. Transient arrhythmias, such as premature ventricular beats, may lead to intermittent undersensing, as their intracardiac signal amplitude may be low (Figure 46). Atrial fibrillation is often undersensed and elicits different behavior in AAI and DDD systems (Figures 47-48).

Loss of capture is easily recognized, however, post-pacing artifacts should not be misinterpreted as capture (Figure 49). Complete lead fracture usually leads to exit block with no visible spikes, while lead dislocation or insulation failure may manifest in various ways (Figures 50-54).



Figure 46. Ventricular undersensing in a patient with VVI pacemaker after AV node ablation for AF. The first PVC was detected and the pacing cycle was reset, however, the second PVC with a different morphology was not detected and inappropriate ventricular pacing occurred in the refractory period of the ventricles.

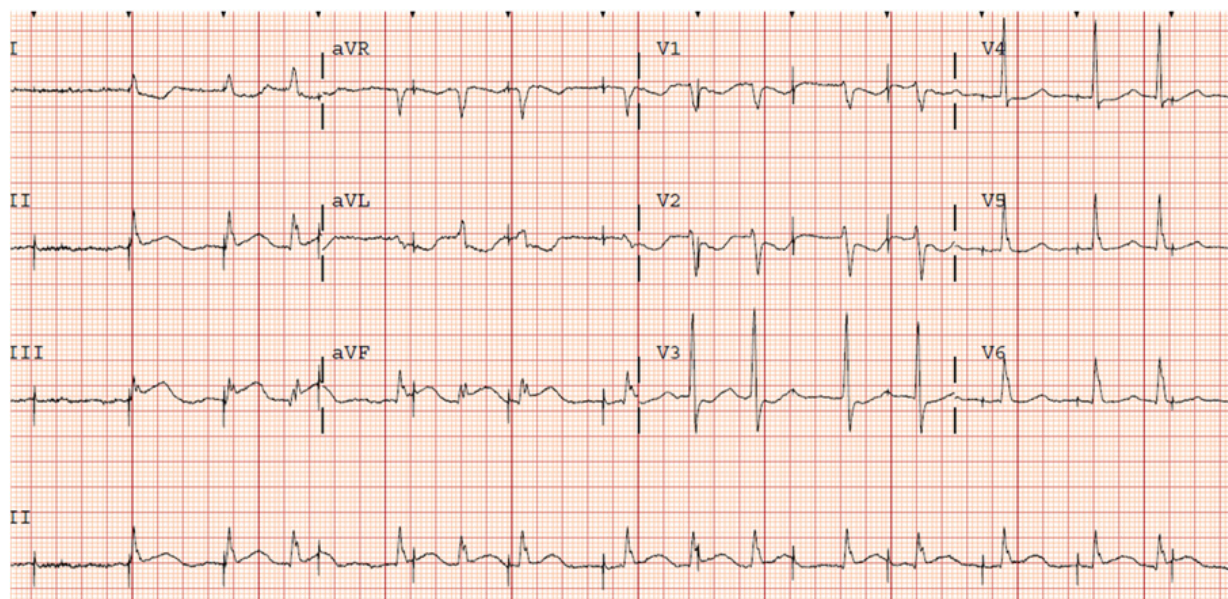


Figure 47. Undersensing of atrial fibrillation with an AAI pacemaker – there is asynchronous atrial pacing without capture. Pseudo pseudofusion beats are seen (2nd, 3rd). VVI pacing would give a similar picture in case of complete sensing failure and loss of capture.

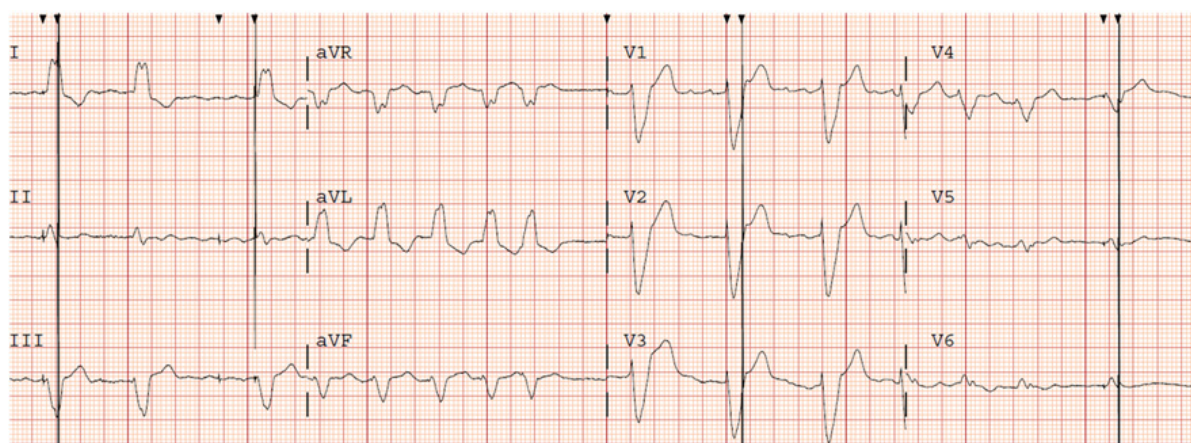


Figure 48. Undersensing of atrial fibrillation with a DDD pacemaker. When the ventricular rate during AF falls below the basic pacing rate, the PM delivers an atrial stimulus, which is not capturing as the patient is in AF. If conduction does not occur after the preset AV delay, a ventricular stimulus is delivered. If conduction occurs after the atrial spike within the ventricular safety period, a ventricular safety pace is delivered, which is not capturing as the ventricles are refractory.

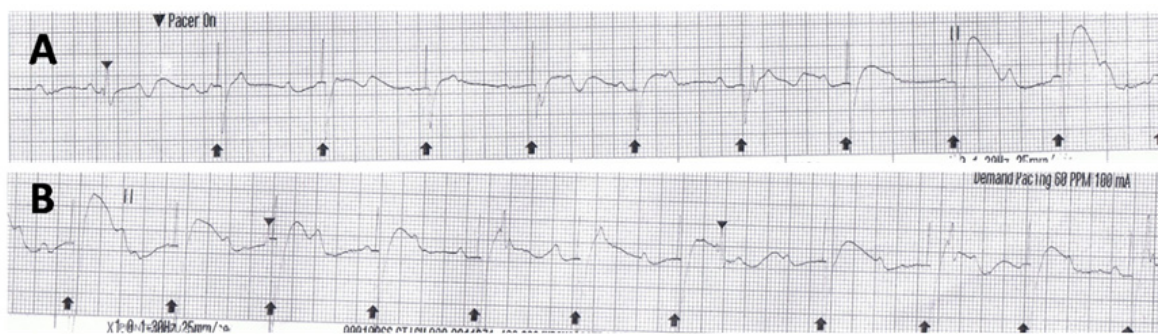


Figure 49. Pseudocapture during temporary external pacing. Transcutaneous pacing was initiated due to complete heart block (underlying rhythm is sinus tachycardia). An escape beat is seen (marked with a black triangle), then pacing is initiated and pacing energy is increased rapidly, causing progressively increasing post-pacing artifacts, which may be misinterpreted as capture (A). However, the slow escape rhythm is still visible between the spikes (best seen after the 6th spike). Later, dissociation between pacing and ventricular rhythm is even more evident despite marked post-pacing artifacts (B).



Figure 50. Atrial lead dislocation of a DDD pacemaker. The atrial activity is not sensed, which leads to asynchronous pacing without atrial capture, followed by ventricular pacing with capture. The 3rd beat is a sinus beat conducted with prolonged AV delay, which is sensed in the ventricular safety pacing interval, so a ventricular safety pace is delivered without capture – the ventricle is refractory at this time.



Figure 51. (A) Undersensing and ineffective pacing with a unipolar pacemaker. Both single chamber atrial and ventricular pacemakers would present similarly in case of lead dislocation. (B) Complete failure of sensing and pacing in a dual chamber pacemaker. There is asynchronous dual chamber pacing, without capture in either chamber. Unipolar pacing causes notable post-spike artifacts, which should not be confused with cardiac electrical activity. (C) Intermittent loss of capture with a ventricular pacemaker. Sensing appears to be normal as each spontaneous QRS resets the pacing cycle. The last spontaneous beat comes very early after the pacing stimulus and is likely not detected due to sensing in the blanking period.



Figure 52. Lead dislocation in a recently implanted single chamber ICD. There is no ventricular sensing, so the pacing is at 40/min, asynchronous to the intrinsic rhythm. There is also lack of capture – attention should be paid when assessing capture as spikes 1-3 come very early when the ventricles may still be refractory. However, spike 5 should have lead to capture.

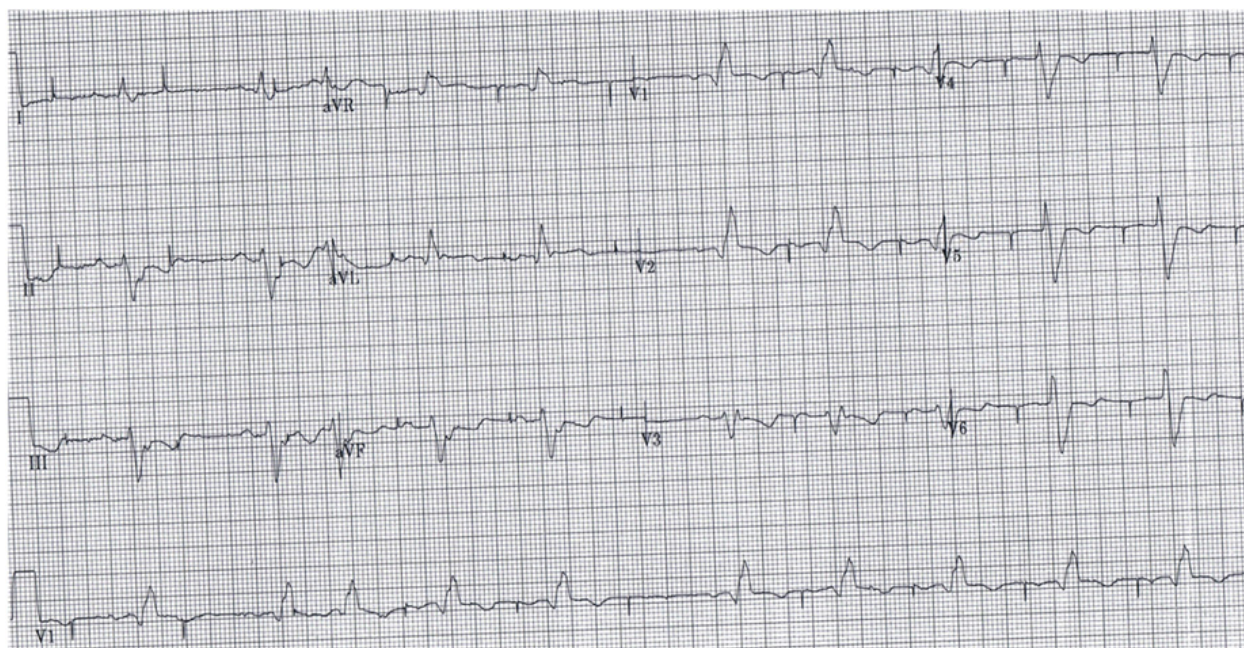


Figure 53. Intermittent loss of atrial capture during AAI stimulation. There is also intermittent undersensing - the atrial activation before the 3rd spike was not detected by the device. This scenario is suspicious for lead dislocation.

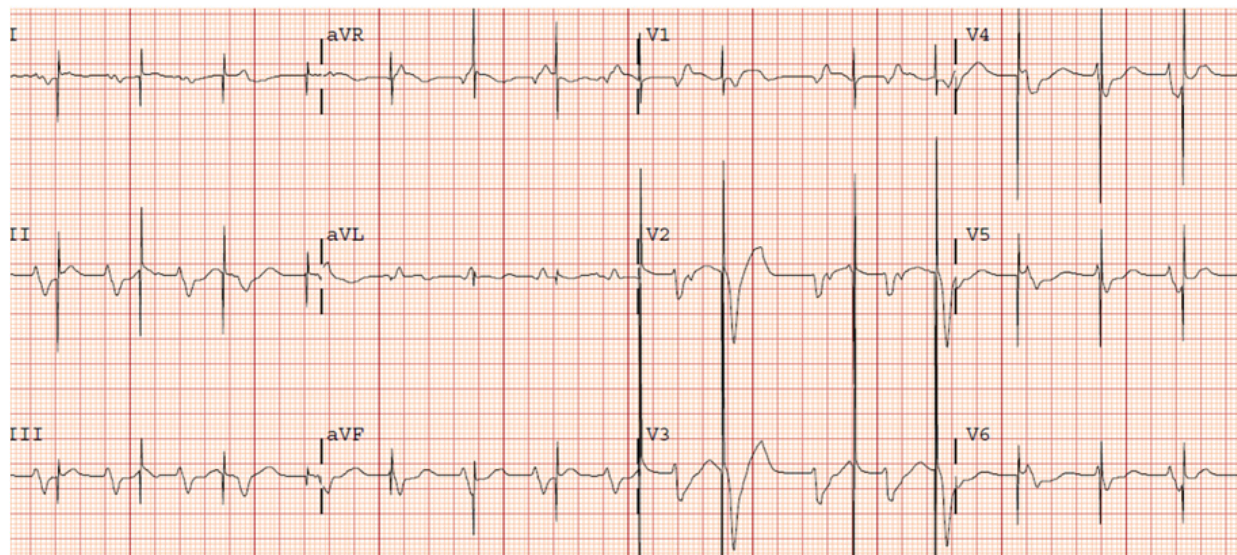


Figure 54. Loss of sensing with oversensing in a ventricular pacemaker. The first few beats may be misinterpreted as atrial pacing, however, the spike to QRS interval is not constant. Fusion and paced beats are seen when pacing occurs during an excitable period. Transient oversensing caused delayed pacing (3rd spike in V3). This scenario is suggestive of lead dislocation or failure.

6. Evaluation of ICD function

Implantable cardioverter defibrillators have multiple therapeutic zones (bradycardia, „physiological“, ventricular tachycardia and fibrillation - VT, VF), that should be taken into account when interpreting ECG changes. While issues due to undersensing or ineffective capture usually manifest similarly to a pacemaker, oversensing may lead to inappropriate therapy due to false VT/VF detection.

As ICD therapies may cause severe patient distress or proarrhythmia, prompt device interrogation and expert consultation is required after such events, unless appropriate device behavior is evident (Figures 55-56). Even when appropriate therapies have been delivered, the patient has to be fully evaluated and appropriate measures should be taken to reduce the risk of arrhythmia recurrence (Mishkin, 2009). In cases when inappropriate therapy is suspected and the risk of recurrence is high (atrial fibrillation with rapid ventricular rate, oversensing), a magnet may be applied to temporarily inhibit tachyarrhythmia therapies, until the device may be interrogated and appropriately reprogrammed (Figure 57). Continuous monitoring is required in the meantime as the patient will not be protected from malignant tachyarrhythmias while in magnet effect.

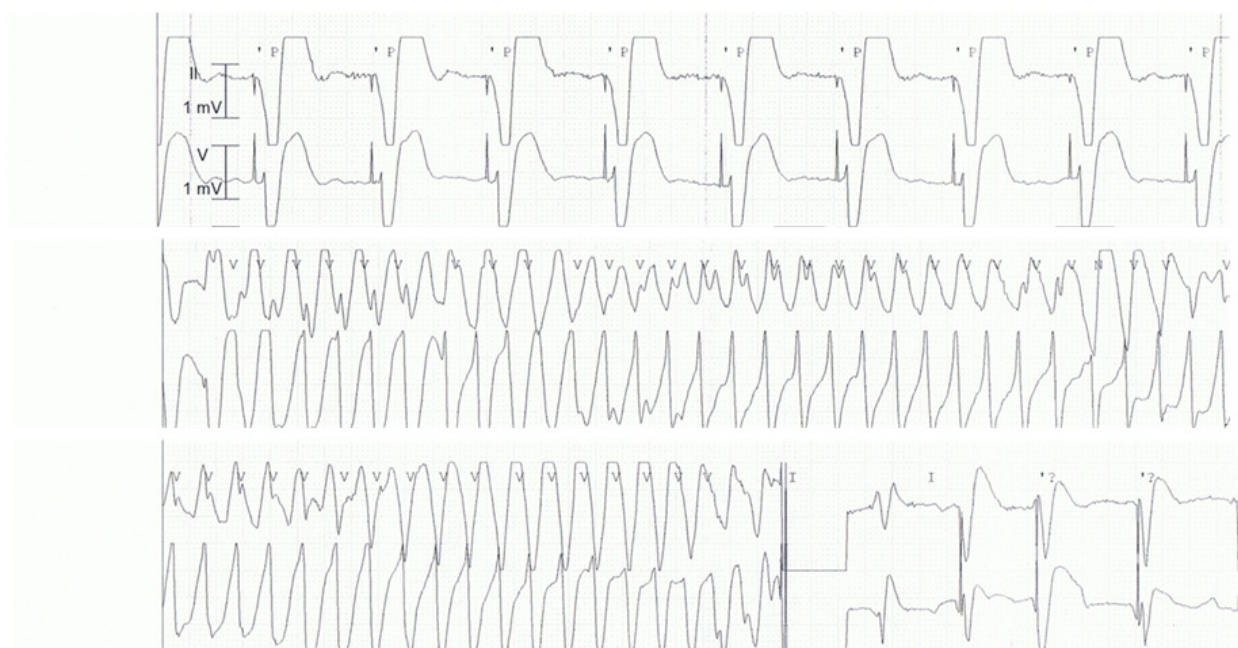


Figure 55. Appropriate ICD function recorded on telemetry. Following ventricular paced rhythm, rapid polymorphic ventricular tachycardia develops, which is terminated by a single endocardial shock after appropriate detection.

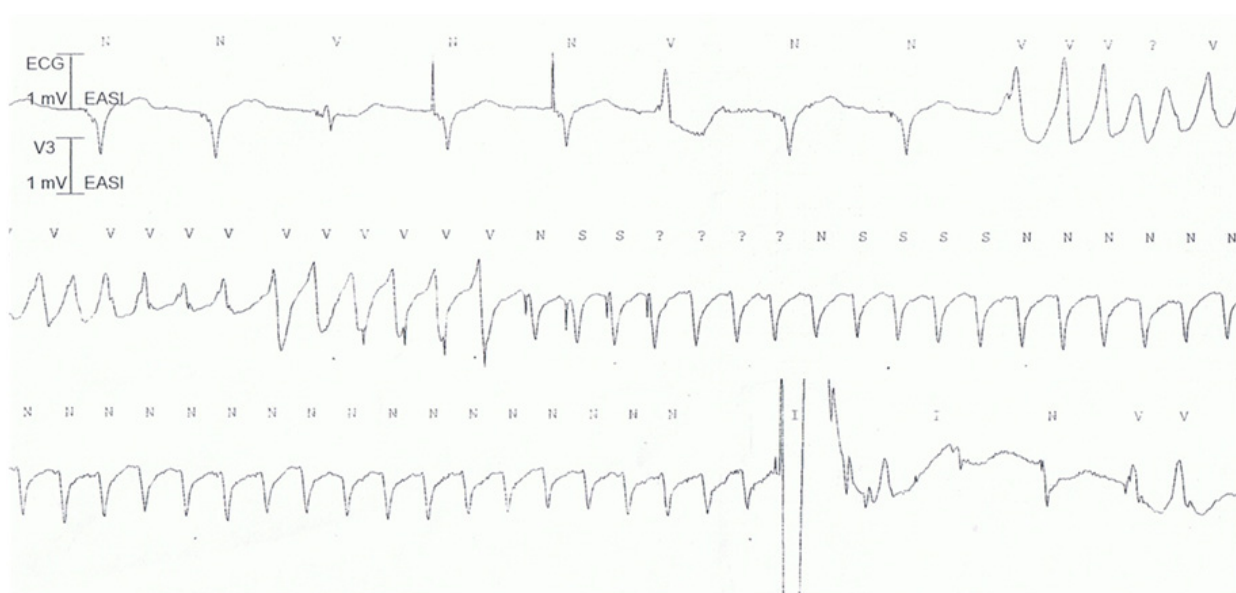


Figure 56. Appropriate ICD function recorded on telemetry. Following ventricular paced rhythm, rapid polymorphic ventricular tachycardia develops, then burst antitachycardia stimulation is attempted, however, fails to terminate the arrhythmia, although changes it to monomorphic VT. The tachyarrhythmia is terminated by an endocardial shock.

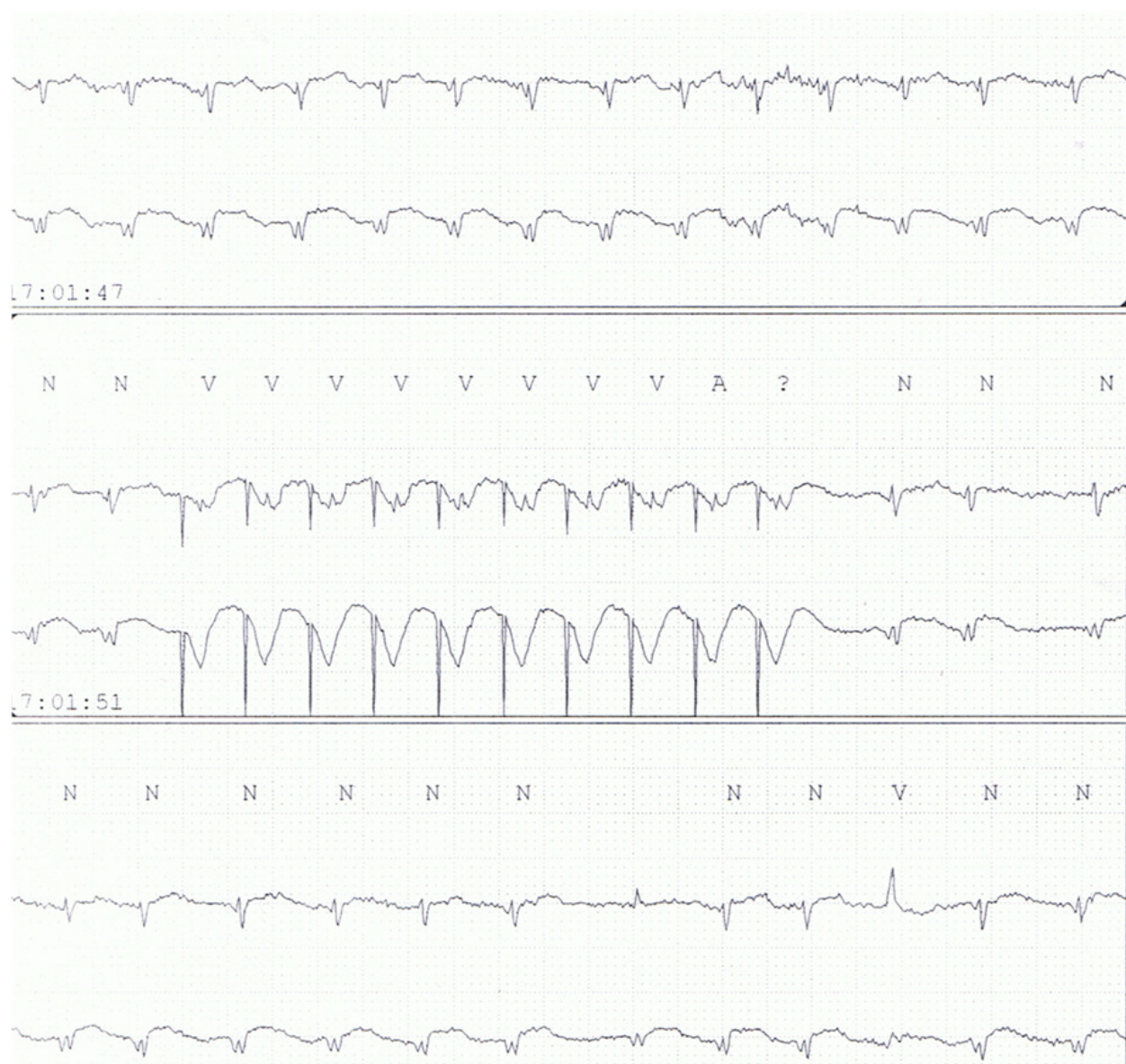


Figure 57. Atrial fibrillation with rapid ventricular rate sensed as ventricular tachycardia – inappropriate burst antitachycardia pacing burst was delivered. The patient is at risk of further inappropriate therapies as the underlying rhythm did not change.

7. Evaluation of cardiac resynchronization devices

Consistent biventricular capture is required to maintain cardiac resynchronization. Paced QRS morphology may vary based on underlying conduction abnormalities, lead location, interventricular delay and the amount of myocardium captured by each lead, relative to each other. Typically, right axis deviation and atypical RBBB pattern is present. If interventricular delay is set greater than 0 ms, usually two pacing spikes can be seen prior to the QRS (Figure 58). In rare cases, conventional RV pacing may mimic biventricular paced QRS morphology (Figure 59). QRS morphology may change due to variable fusion with conducted beats either from variable AV delay or atrial fibrillation (Figures 60-61).

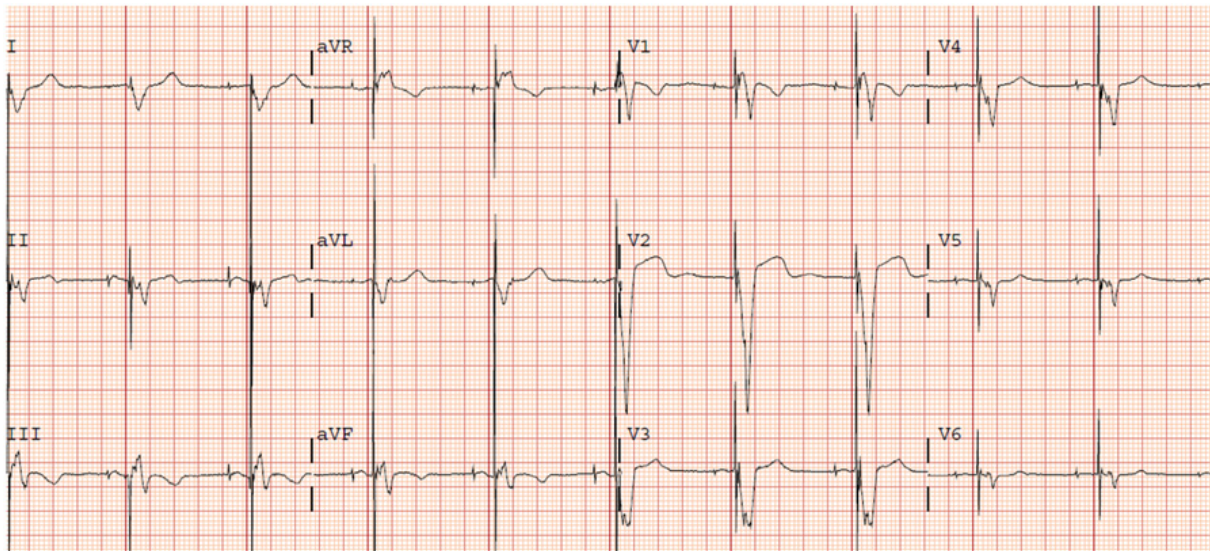


Figure 58. Typical atrioventricular pacing. The paced QRS usually shows right axis deviation and an atypical RBBB pattern in V1. Two distinct pacing spikes, representing right and left ventricular stimulation with a delay around 20 ms, can be best seen in II and V3 on this tracing.

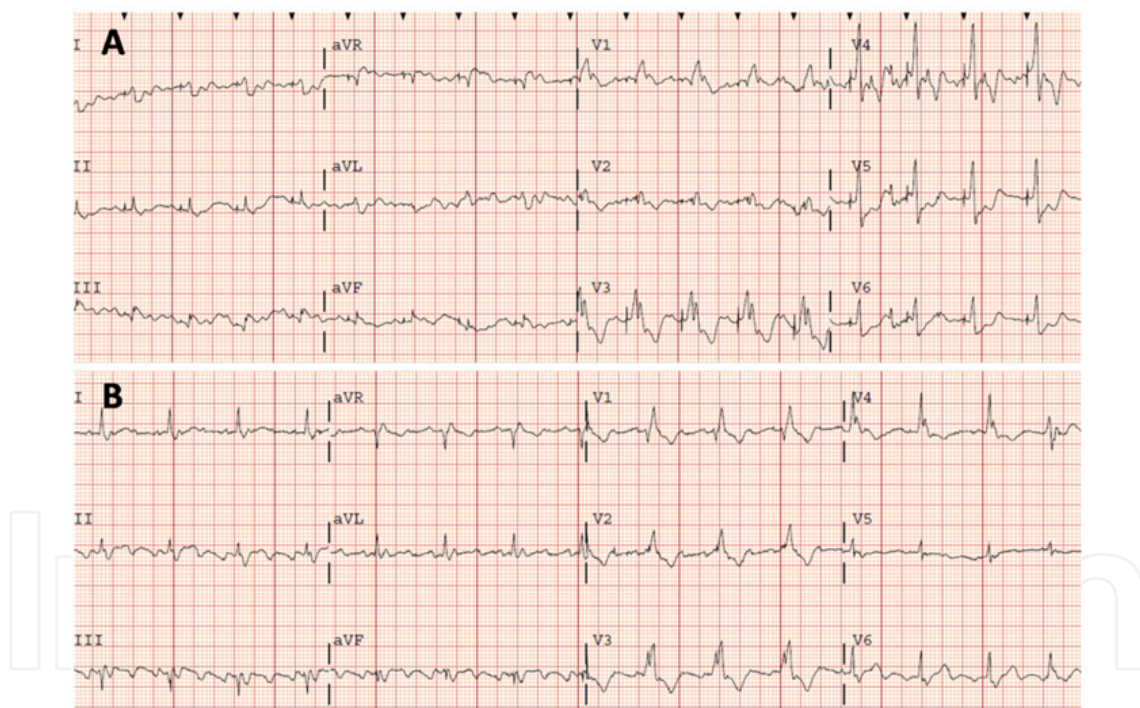


Figure 59. Although biventricular pacing may be recognized in most cases, underlying RBBB may mimic this QRS morphology during right ventricular pacing, especially, if fusion is present – this patient has a DDD pacemaker (A). His previous ECG showed atrial flutter with RBBB (B).

Sense response pacing is an algorithm that was designed to maintain the benefits of biventricular stimulation with premature beats or fast AV conduction – in case a ventricular event is sensed, a pacing stimulus is delivered simultaneously to decrease ventricular activation time. The resulting QRS morphology is affected by the origin of the premature beat and the amount of fusion (Figures 62-64).

Loss of left ventricular lead capture changes QRS morphology, so it becomes similar to RV pacing. A full 12-lead ECG should always be obtained during follow-up (Barold, 2011a and Barold, 2011b). Comparison with previous tracings is recommended as biventricular paced QRS morphology varies individually (Figure 65). Undersensing or oversensing may be more difficult to identify with resynchronization devices, than with conventional pacemakers, due to the algorithms designed to maintain biventricular pacing (Figure 66-67). In uncertain cases, device interrogation should be performed to prevent loss of resynchronization.

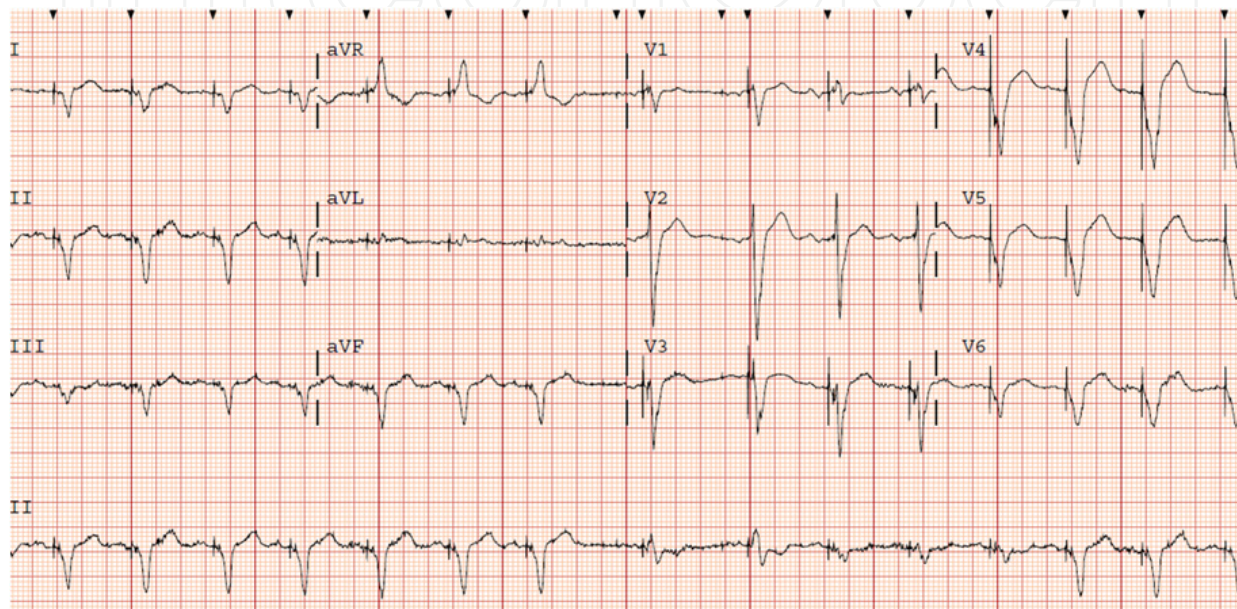


Figure 60. Variable fusion during biventricular pacing due changes in the atrial rate – the degree of ventricular fusion is different for atrial sensed and atrial paced beats, due to different atrioventricular delay, affecting how much of the ventricular myocardium can be activated through the native conduction system during biventricular pacing.

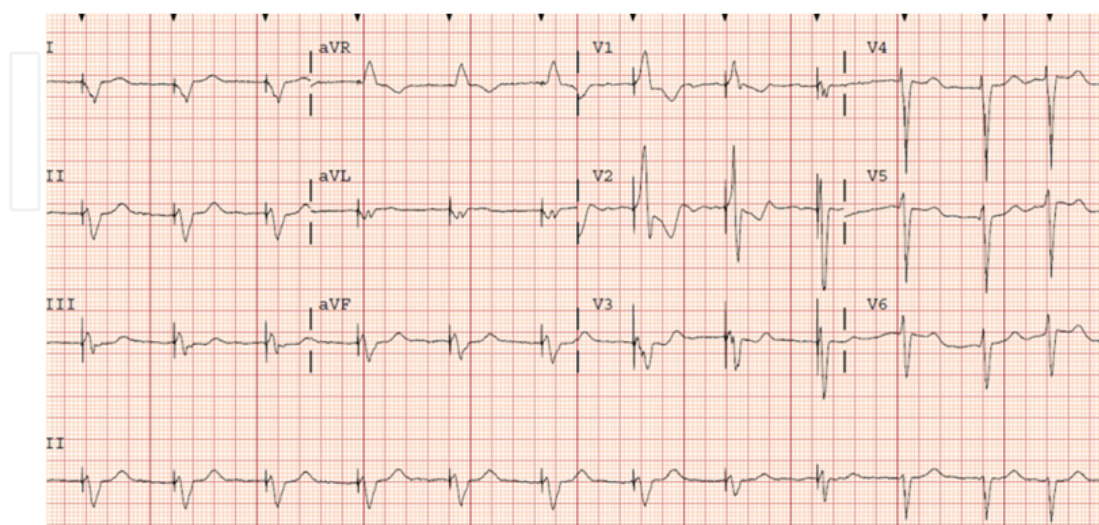


Figure 61. AF with biventricular pacing. When the ventricular rate increases, first it leads to more fusion, then to sense response pacing – appropriate response of the system.

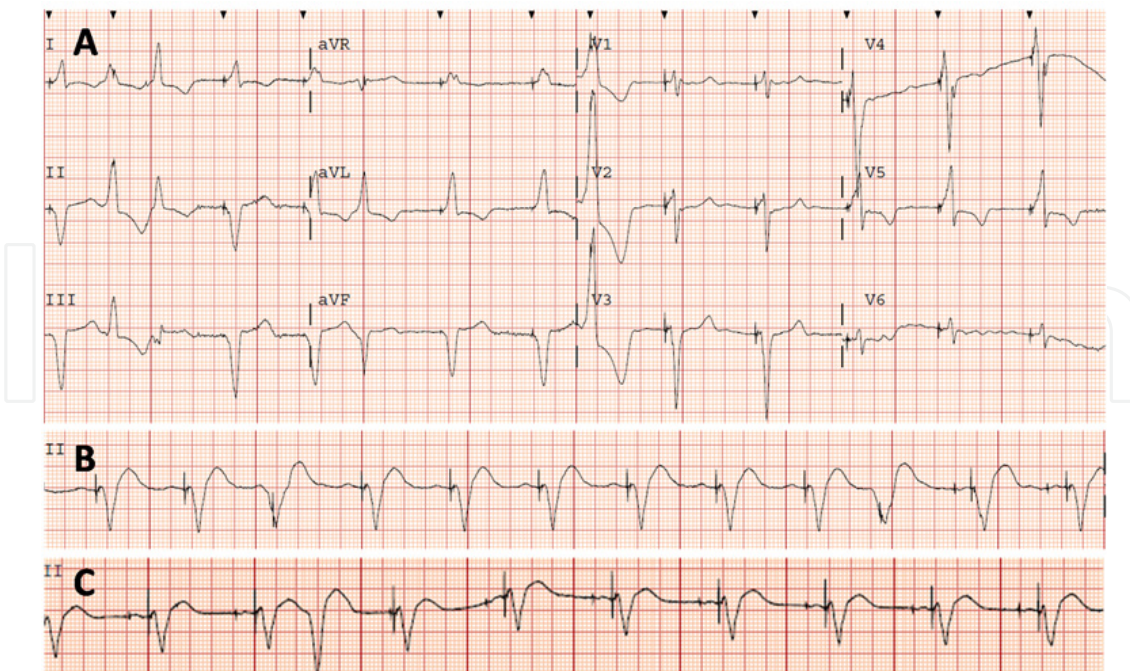


Figure 62. Sense response pacing during biventricular stimulation – each ventricular sensed event (PVC, rapidly conducted AF) leads to simultaneous pacing, aiming to maintain optimal hemodynamics of biventricular pacing. Due to the various origin of these early beats, the result can be fusion of even pseudofusion. Despite irregular rate and variable QRS morphology, this tracing shows appropriate biventricular pacemaker function (A). This function may be easier to evaluate when the underlying rhythm is regular, such as in sinus rhythm (B). There is an appropriate sense response pace for each premature beat. Depending on the coupling interval of the premature beat and the atrial rate, this may lead to post-event atrial pacing, if the compensatory pause exceeds the basic pacing rate. Very early PVCs do not trigger sense response pacing if that would exceed a maximal tracking rate (C).

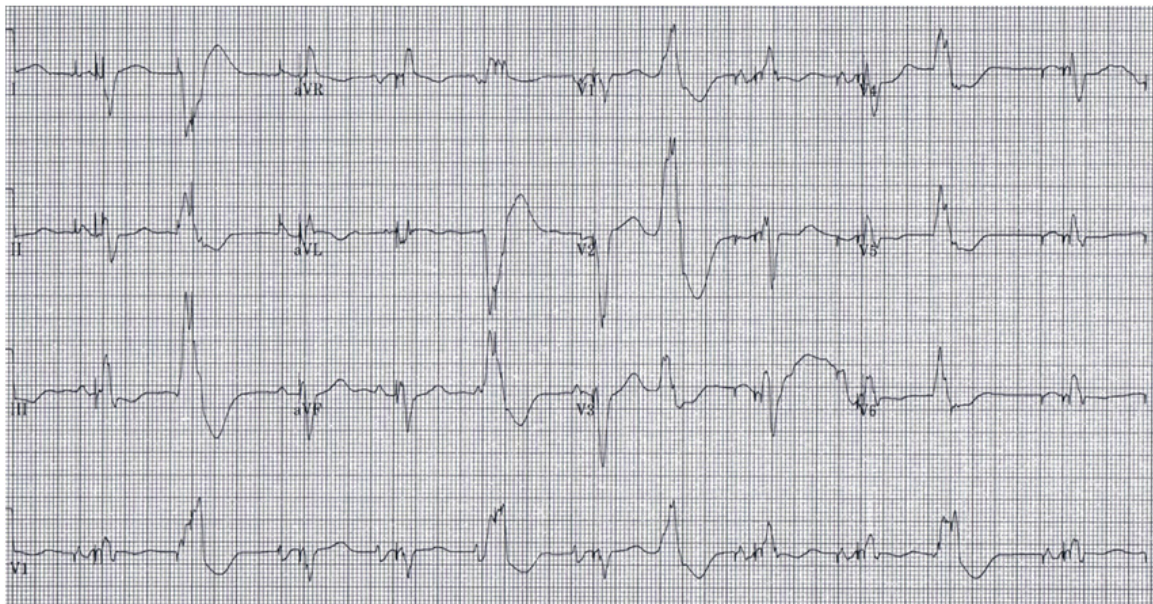


Figure 63. Atrioventricular pacing with irregular ventricular rhythm due to frequent PVCs. Appropriate device behavior with sense response pacing during PVCs (best seen during the 1st PVC). There is notable interventricular delay between the right and left ventricular stimulation (60 ms).

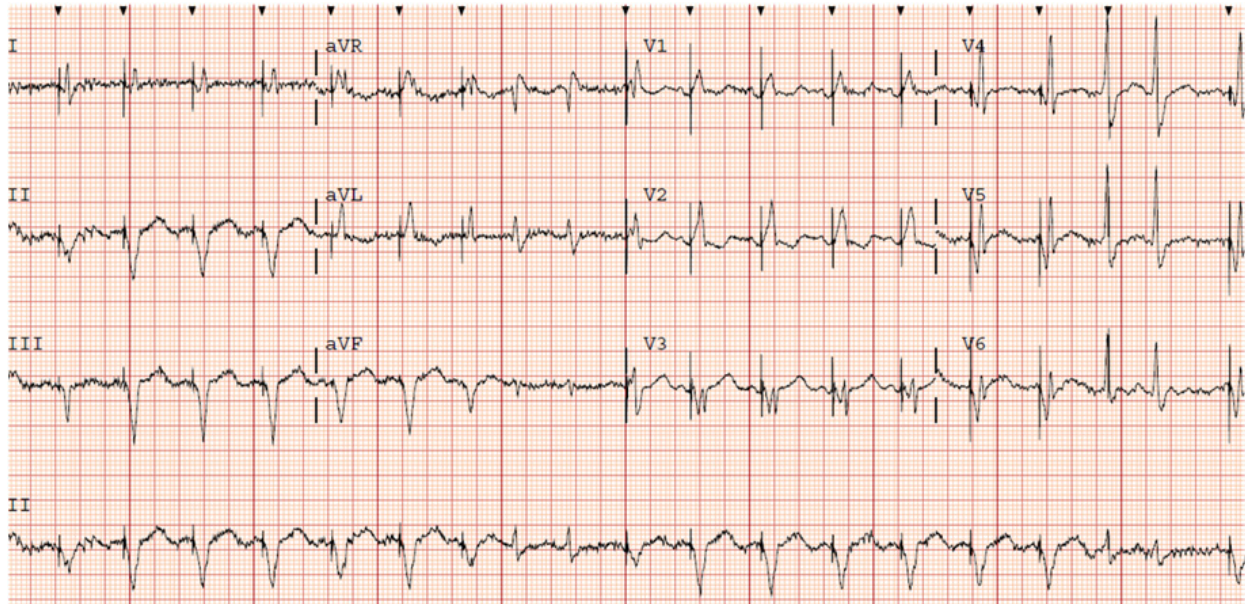


Figure 64. Biventricular (sense response) pacing ceases above the upper biventricular tracking rate – this is appropriate pacemaker function, however, may lead to rapid deterioration if the tachycardia persists.

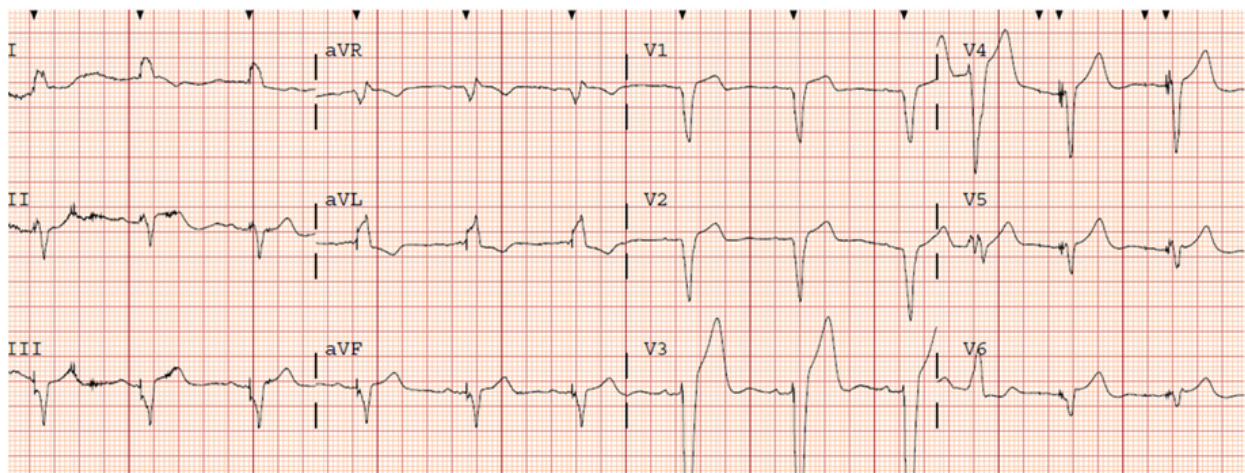


Figure 65. LBBB QRS morphology in a patient with a biventricular system should raise the suspicion of left ventricular non-capture. Other causes include suboptimal LV lead placement or too long RV-LV delay – these may diminish the amount of myocardium activated by the LV lead during biventricular pacing. In this case, biventricular pacing with 40 mm VV delay is apparent in V4.

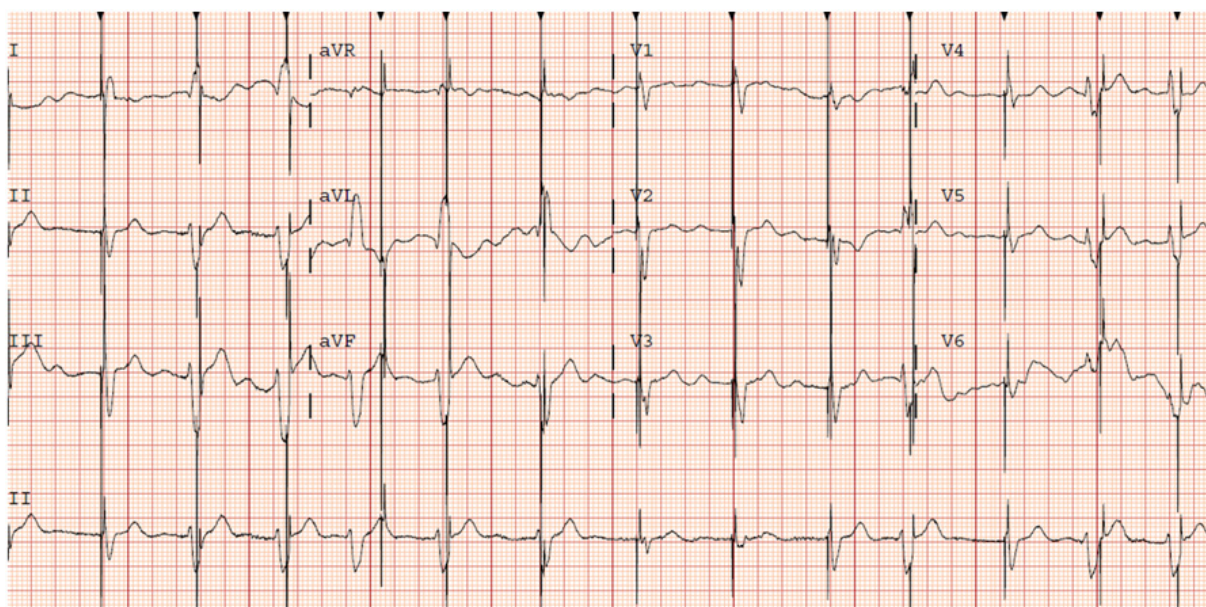


Figure 66. Intermittent ventricular undersensing in a biventricular system. Most ventricular beats are biventricular paced at 75/minute or sense response paced (occurring faster than 75/min). However, there are few spikes coming late (instead of a sense response pace), at 75/minute – the ventricular activation was not detected by the device. The undersensing is intermittent, as the sense response paced beats present on this tracing require a sensed event.

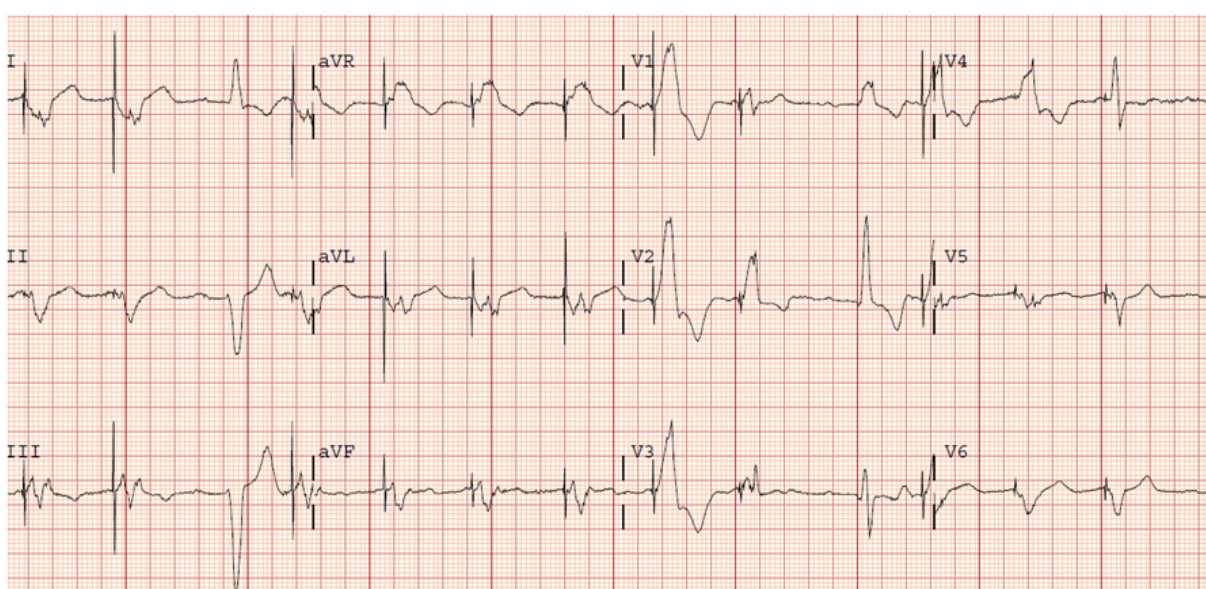


Figure 67. Pacing below the basic rate in an atrioventricular system is always abnormal – algorithms are designed to maintain the ventricular rate, track premature beats and provide sense response pacing. This patient with a biventricular defibrillator had a fracture of the right ventricular sensing/pacing/shock ICD lead, leading to intermittent ventricular oversensing and multiple inappropriate shocks.

7. Conclusion

Conventional 12-lead ECG is an important tool to evaluate CIED function. A systematic approach is required to identify appropriate device function and to decide whether further investigation is necessary. As advanced devices, such as implantable cardioverter-defibrillators and cardiac resynchronization systems become more abundant, even common malfunctions and pseudo-malfunctions may be more difficult to identify, due to the presence of special pacing algorithms. In uncertain cases, review of prior patient data, device interrogation and expert consultation is required.

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