Heart, Lung and Circulation (2019) **28,** 223–236
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.06.1046

REVIEW

Interpreting the Normal Pacemaker Electrocardiograph

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Received 28 January 2018; received in revised form 6 May 2018; accepted 17 June 2018; online published-ahead-of-print 18 July 2018

Modern cardiac pacing systems have sophisticated software to document, evaluate and record intrinsic and paced rhythms as well as correct pacing abnormalities and rhythm disturbances by applying algorithms, which are generally company specific. To the cardiologist and technologist, these algorithms may be difficult to interpret on both the 12-lead electrocardiograph (ECG) and Holter ambulatory monitoring recordings, which are usually performed because of patient symptoms or physician concern. The tracings may appear bewildering and mimic pacemaker malfunction, thus leading to unnecessary tests or even surgery. This review will define the common programmed pacemaker modes and describe a range of ECG appearances of normal pacemaker function during the application of testing, correcting or therapy algorithms.

Keywords Electrocardiography • Holter monitoring • Cardiac pacemakers

Introduction

The implanted cardiac pacemaker has evolved from simple, non-programmable, asynchronous ventricular pacing to sophisticated multi-programmable dual chamber and biventricular models, all of which now incorporate company specific testing, correcting and therapy algorithms. A consequence of this technological evolution has been the appearance of bizarre pacing rhythms on both 12-lead electrocardiograph (ECG) and ambulatory Holter monitor recordings. Even to the experienced ECG reporter and cardiac technologist, these appearances may suggest pacing malfunction, which may lead to patient anxiety, needless investigations, inappropriate programming and even unnecessary surgery. Today, 12-lead ECGs and Holter recordings are frequently performed in patients with implanted pacemakers. They are indicated to confirm correct pacemaker programming, evaluate palpitations, dizziness or syncope and to determine if there is an appropriate rate response to physiologic demand.

This review of the normal pacemaker ECG will revisit the different modes of cardiac pacing, how to recognise paced from intrinsic rhythms and will also review the ECG appearances of the more common pacemaker company algorithms. The illustrations and discussion will be generic and levelled at a general cardiology reader.

Pacing the Myocardium

By definition, all electrical circuits are bipolar requiring a cathode and an anode [1]. When applied to a conventional cardiac pacing lead, the terms unipolar and bipolar indicate the number of electrodes on the lead. A unipolar lead has a single pole; the cathode at its tip, whereas a bipolar lead has both poles very close to each other at its distal end and within the chamber being paced. For unipolar pacing, the anode lies on the titanium can of the implanted pulse generator.

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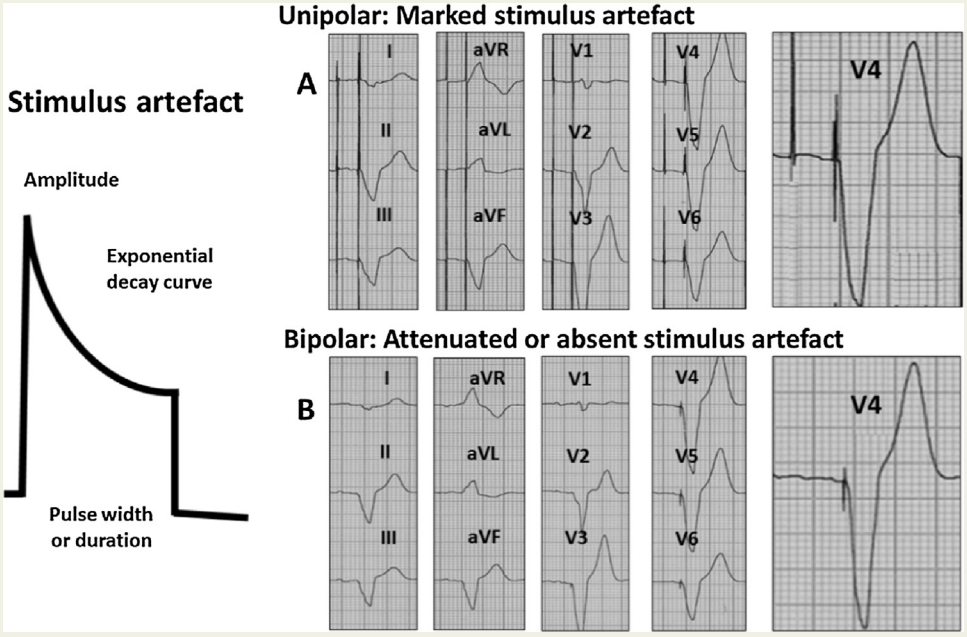

Figure 1 Pacemaker stimulus artefact in 12-lead electrocardiographs (ECGs).
Left: Oscilloscopic appearance of a unipolar stimulus artefact. Initial deflection is followed by an exponential decay curve.
Right A: Unipolar dual chamber pacing. Ventricular pacing is from the apex with an extreme axis deviation and no R waves across the chest leads. A magnified V4 highlights the large stimulus artefacts.
Right B: Same patient, bipolar pacing. No atrial and attenuated ventricular stimulus artefacts best seen in magnified V4.

The ECG of Normal Pacemaker Rhythms

On the ECG tracing, delivery of pacemaker energy to the myocardium can be seen as a stimulus artefact immediately prior to atrial or ventricular depolarisation. It represents about two to five volts delivered for about 0.5 ms and on an oscilloscope appears as an exponential decay curve (Figure 1). With unipolar pacing, the stimulus artefact is large and represents the dissipation of energy through the chest to the implanted pulse generator. Bipolar pacing creates very small or absent stimulus artefacts on the ECG. Figure 1 demonstrates both unipolar and bipolar pacing. The size of both the unipolar and bipolar stimulus artefact is dependent on the programmed voltage and varies with the respiratory cycle (Figure 2A) [2].

Atrial Pacing

Confirmation of atrial pacing, particularly bipolar, can be difficult to confirm on the ECG. Because the lead is usually positioned within the appendage, close to the sinus node, the P wave is often similar in appearance to the sinus P wave (Figure 2A). Pacing from a low atrial position close to the tricuspid valve may mimic junctional rhythm and is very different to the sinus wave (Figure 2B).

Ventricular Pacing

Unlike atrial pacing, recognition of ventricular pacing is generally easy, provided a stimulus artefact can be recognised (Figure 1). The paced QRS is wide with a regular repetition rate. On occasion, fusion beats may occur when there is insufficient time for the pacemaker lead to sense a propagated intrinsic QRS (Figure 3A). With intact atrio-ventricular conduction, the ventricular paced QRS may demonstrate a retrograde P wave due to ventriculo-atrial conduction (Figure 3B).

The 12-lead ECG is helpful in confirming the ventricular pacing site, which traditionally has been the right ventricular apex. There is a left bundle branch block appearance with poor or absent R waves from V4 to V6 (Figure 1). The apical impulse is propagated through both ventricles in a retrograde manner, resulting in a left or extreme axis deviation (Figure 1). The right ventricle can also be paced from non-apical sites. R waves develop in V4 to V6, as the lead position progresses more superior in the right ventricular cavity, whereas the axis becomes more normal (Figure 4) [3].

Today, a well-established non-bradyarrhythmic indication for cardiac pacing in patients with congestive cardiomyopathy is cardiac resynchronisation therapy using biventricular pacing. Clinically, for maximum benefit, the left ventricle is usually paced about 40 to 80 ms before the right ventricle, resulting in a right bundle branch block appearance on the ECG (Figure 5) [4]. Two ventricular stimulus artefacts can often be identified, although today, multipolar left ventricular pacing may result in more than one left ventricular stimulus artefact.

Modes of Cardiac Pacing

With the development of atrial and dual chamber pacing systems, it became necessary to create a simple and easily

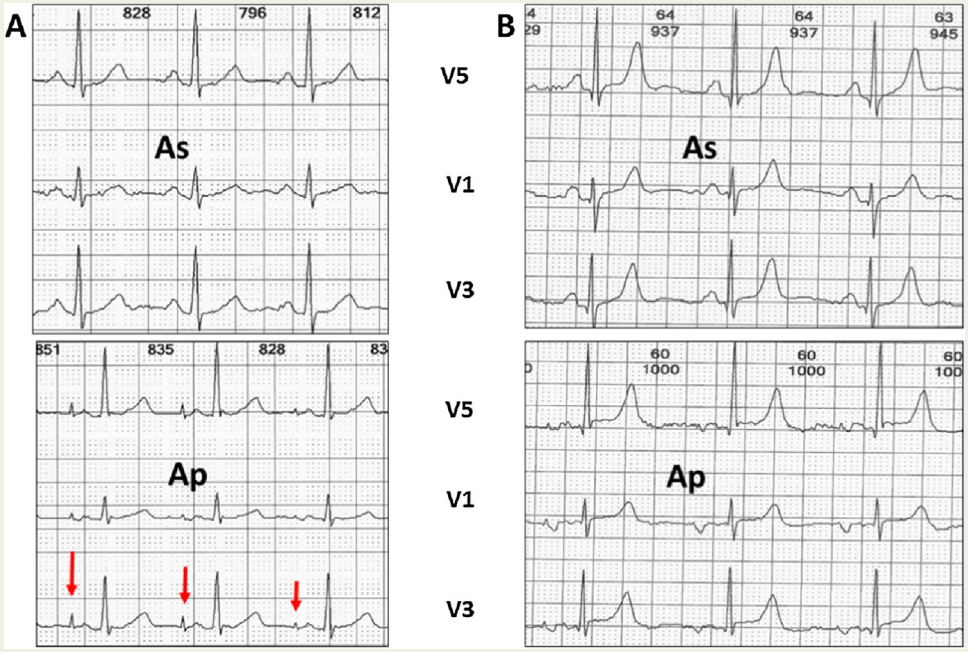

Figure 2 Atrial sensing (As) and bipolar pacing (Ap). Holter monitoring leads V5, V1 and V3.
A. Three channel Holter recordings demonstrating Ap from the atrial appendage similar to sinus P waves (As). The stimulus artefact size varies with respiratory cycle (red arrows demonstrating progressively smaller spikes).
B. Ap adjacent to coronary sinus ostium. Paced P waves are inverted.

recognised classification of the different modes of cardiac pacing (Table 1). Although, there are many theoretical combinations, only five are used clinically (Table 2).

Single Chamber Ventricular

The original cardiac pacemaker had no sensing (VOO) and asynchronous pacing continued unabated, irrespective of the underlying rhythm (Figure 6A). As sensing circuits developed, pacemakers were able to recognise the intrinsic QRS and respond appropriately. The first synchronous designs delivered the full energy of the pacing impulse into the intrinsic QRS (VVT) (Figure 6B). With improvements in sensing circuits, ventricular inhibited (VVI) pacing became established (Figure 6C). Ventricular pacing continues at a minimum programmed pulse repetition rate, unless the pacemaker output is inhibited by intrinsic ventricular rhythms.

Single Chamber Atrial

Although single chamber atrial pacing would appear appropriate in patients with sick sinus syndrome and normal atrio-ventricular conduction, it has never been widely accepted, because of the concern of development of atrio-ventricular block which is about 1% per year with a freedom rate of 88.6% at 10 years [5]. Consequently, when atrial pacing alone is indicated, a dual chamber pacemaker is implanted and programmed appropriately. The most commonly used single chamber atrial mode is AAI pacing (Figure 2), where sensing is only available in the atrium and ventricular ectopics will not be sensed unless there is retrograde conduction (Figure 7). This ECG appearance can be confusing and lead to misinterpretation of otherwise normal function.

Dual Chamber DDD

This is the most widely used pacing mode [6]. There is atrial and ventricular pacing and sensing as well as atrial and ventricular synchronisation with an electronic PR interval, called the atrio-ventricular delay (Figure 8A). In patients with heart block and a normal sinus mechanism, there will be predominantly atrial sensing followed by ventricular pacing (Figure 8A left), whereas patients with sick sinus syndrome generally have fixed atrial pacing followed by ventricular sensing. With DDD pacing, there is a low rate, usually programmed 60 or 70 beats per minute (bpm) and if the sinus rate falls below this level, atrial pacing commences (Figure 8A right).

Dual Chamber VDD

There is sensing in both chambers, but only ventricular pacing (Figure 8B). This is only suitable for patients with complete heart block and a normal sinus mechanism. Unlike DDD pacing, if the sinus rate falls below a programmed lower rate, there will be no atrial pacing, but rather ventricular pacing and loss of atrio-ventricular synchrony. The absence of atrial pacing in the presence of atrial sensing may suggest pacemaker malfunction.

Dual Chamber DDI

There is normal pacing and sensing in both chambers, but a fixed ventricular rate, irrespective of the intrinsic atrial rate. The ECG appearances are confusing as the rhythm appears to be single chamber VVI with intermittent atrial pacing

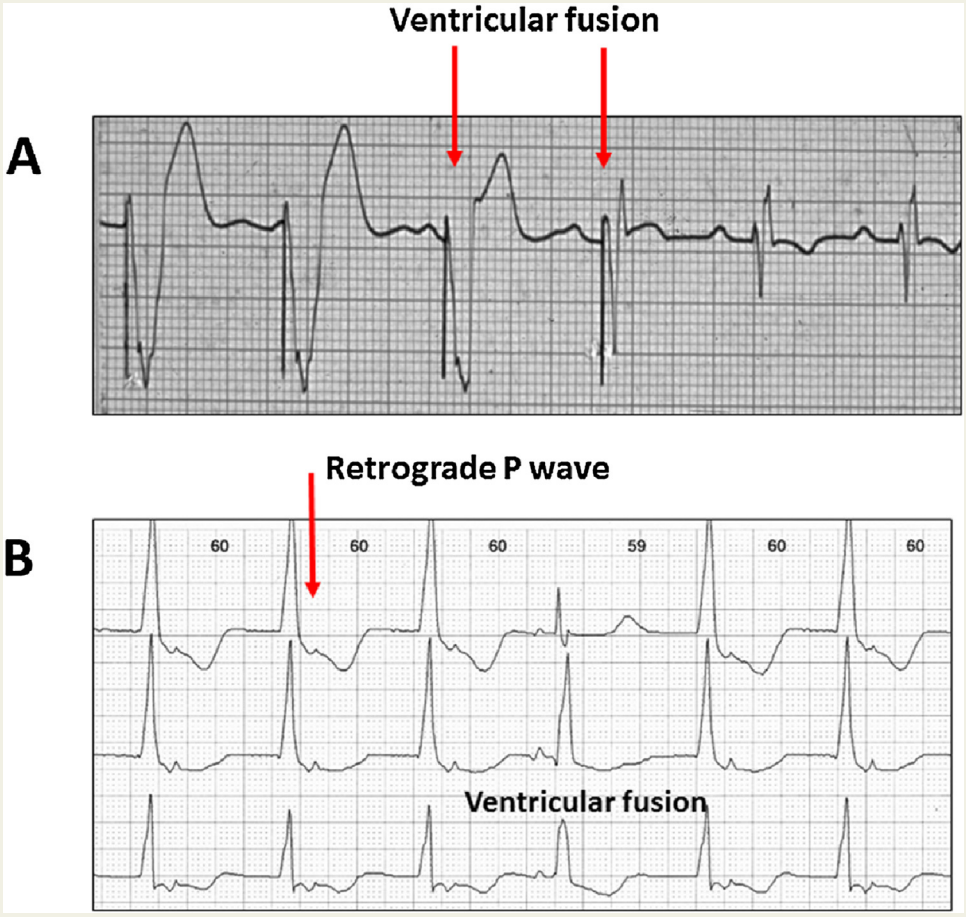

Figure 3 **A**. Unipolar ventricular inhibited pacing with fusion. The first two beats are paced, the last two sinus. Middle complexes are fusion beats. The sinus rate is gradually increasing, but too early to be sensed by the apical ventricular pacing lead and a stimulus artefact is present (red vertical arrows). The first complex is mainly paced whereas the second predominantly sinus. The contribution of each can be judged by the T wave size.
B. Bipolar ventricular pacing with retrograde conduction. Retrograde P waves in the ST segment (red vertical arrow) result in the atria contracting against closed atrio-ventricular valves. There is a sinus beat followed by ventricular fusion and no retrograde conduction.

(Figure 8C). DDI pacing was originally indicated for paroxysmal atrial tachyarrhythmias to prevent rapid ventricular pacing [7]. Modern pacemakers now have automatic mode switching algorithms which have a more physiologic response, although, DDI pacing may be indicated if mode switching is unreliable.

Rate Adaptive Pacing

Pacemaker recipients with sick sinus syndrome manifesting as atrial bradyarrhythmias, are unable to increase their pacing rate with physiologic demand. This has been corrected by sensors that emulate changes in cardiac output in a linear fashion in response to physical exertion (Figure 9). Although many sensors have been developed, only three have stood the test of time; activity, minute ventilation and closed loop stimulation. These sensors are now routinely incorporated into otherwise conventional pacemaker systems with standard bipolar leads. By far, the most popular sensor is an accelerometer within the pulse generator which detects activity, whereas minute ventilation and closed loop stimulation are more physiologic sensors which can be used in specific circumstances. When a rate adaptive sensor is programmed ON, the letter R is added to the code although when the sensor is programmed OFF, it will appear in parenthesis (Tables 1 and 2).

Pacemaker Algorithms

As the complexity of both single and dual chamber pacing evolved, a number of intrinsic rhythm and pacemaker interactions emerged, which were corrected by an array of both generic and company specific algorithms, and although familiar to the pacemaker specialist, nevertheless produce confusing patterns on the ECG and particularly Holter recordings, mimicking pacemaker malfunction. The ECG features of a number of the more common algorithms will be discussed, but beforehand, it behoves us to review the

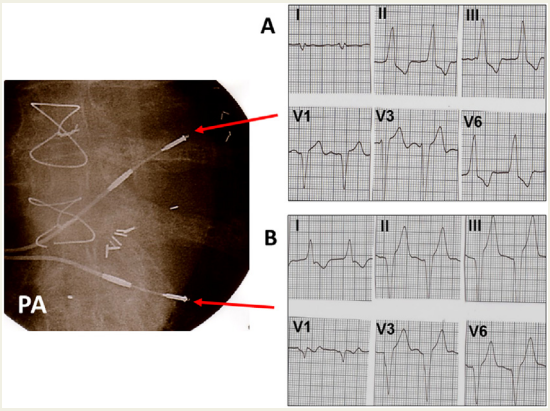

Figure 4 Electrocardiographs (ECGs) with ventricular pacing from two sites in the right ventricle.
Left: Postero-anterior (PA) fluoroscopic image of two bipolar leads in the right ventricle. *Right:* **A**. The top lead is in the right ventricular outflow tract. There is a near iso-electric lead 1 and tall R waves in leads II and III. There is a tall R wave in V6.
B. The bottom lead is at the right ventricular apex. There is a left axis deviation and no R waves in the V leads.

basic pacemaker dual chamber timing cycles in order to understand the complexities of these algorithms.

Pacemaker Timing Cycles

Timing cycles comprise a complex set of rules which govern pacemaker sensed and paced events. The specific rules as well as terminology differ between companies and a comprehensive description lies well outside the confines of this manuscript. The most basic dual chamber timing cycles are demonstrated in Figure 10. There is a low rate interval, divided into two zones; the atrio-ventricular delay and the ventriculo-atrial interval. The atrio-ventricular delay duplicates the PR interval of the unpaced ECG and may also be terminated prematurely by a conducted ventricular sensed event. The ventriculo-atrial interval is divided into the post ventricular atrial refractory period (PVARP) and the atrial tracking interval. Because of the potential for inappropriate sensing in both atria and ventricles, a number of refractory periods are mandated within the cycle of which the PVARP is both the longest and programmable. It is designed to prevent inappropriate sensing of retrograde P waves or large far-field R waves by the atrial channel. The atrium is, therefore, refractory from the time of atrial pacing or sensing to the end of the PVARP, with this period known as the total atrial refractory period. Actual sensing in these zones is governed by either total refractoriness (blanking periods) or if "atrial sensing" occurs, specific algorithms may come into play.

At the end of the PVARP is the atrial tracking interval, where there is normal atrial sensing. At rest, this period is generous, allowing sensing of a normal sinus mechanism or ectopy. With exertion, the zone gradually disappears until an upper atrial sensed rate is achieved. This upper rate can be extended by programming both a rate adaptive atrio-ventricular delay and a rate adaptive PVARP, which allows these intervals to shorten in a physiologic manner (Figure 9).

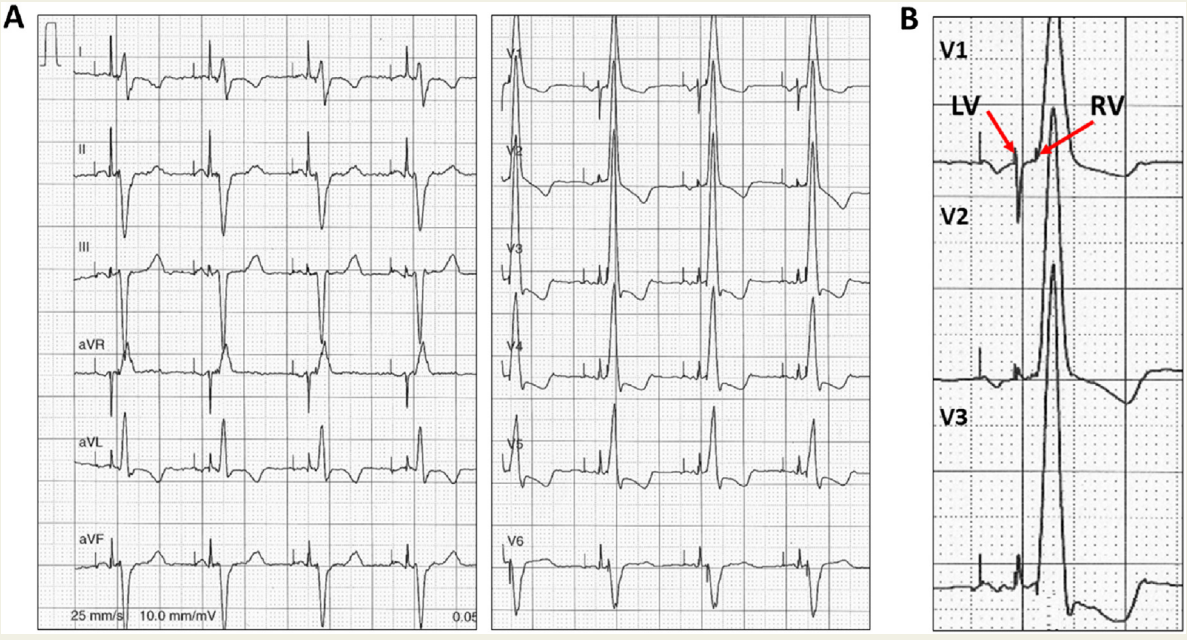

Figure 5 12-lead electrocardiograph (ECG). Bipolar, dual chamber biventricular pacing.
A. Right bundle branch block indicating left ventricular pacing before right.
B. Magnified view of leads V1 to V3 confirming left ventricular stimulus artefact (LV) followed 80 ms later by the right ventricular stimulus artefact (RV).

Table 1 **Four letter pacemaker identification code.**

First letter (chamber paced)	Second letter (chamber sensed)	Third Letter (chamber response)
A — atrium	*A* — atrium	*O* no response
V — ventricle	*V* — ventricle	*I* — inhibited
D — atrium and ventricle	*D* — atrium and ventricle	*T* — triggered
O no pacing or sensing	*O* no pacing or sensing	*D* — atrium and ventricle

Fourth letter "R" identifies a rate adaptive function programmed ON whereas (R) identifies that a rate adaptive function is available.

Table 2 **Pacemaker modes most commonly used clinically.**

Single chamber:	
VVI(R)	Single chamber. Ventricular pacing (V), ventricular sensing (V), inhibited by intrinsic ventricular rhythms (I) and rate adaptive function available (R).
AAI(R)	Single chamber. Atrial pacing (A), atrial sensing (A), inhibited by intrinsic ventricular rhythms (I) and rate adaptive function available (R).
Dual chamber:	
DDD(R)	Dual chamber. Atrial and ventricular pacing (D), atrial and ventricular sensing (D), inhibited by intrinsic atrial and ventricular rhythms as well as atrium triggering ventricle after a programmable atrio-ventricular delay (D) and rate adaptive function available (R).
VDD	Dual chamber. Ventricular pacing (V), atrial and ventricular sensing as well as atrium triggering ventricle after a programmed atrio-ventricular delay (D). Rate adaptive function (R) rarely required as it results in VVIR pacing.
DDI(R)	Dual chamber. Atrial and ventricular pacing (D), atrial and ventricular sensing (D) with limited atrio-ventricular synchrony, resulting in lower rate ventricular pacing irrespective of the intrinsic atrial rate (I) and rate adaptive function available (R).

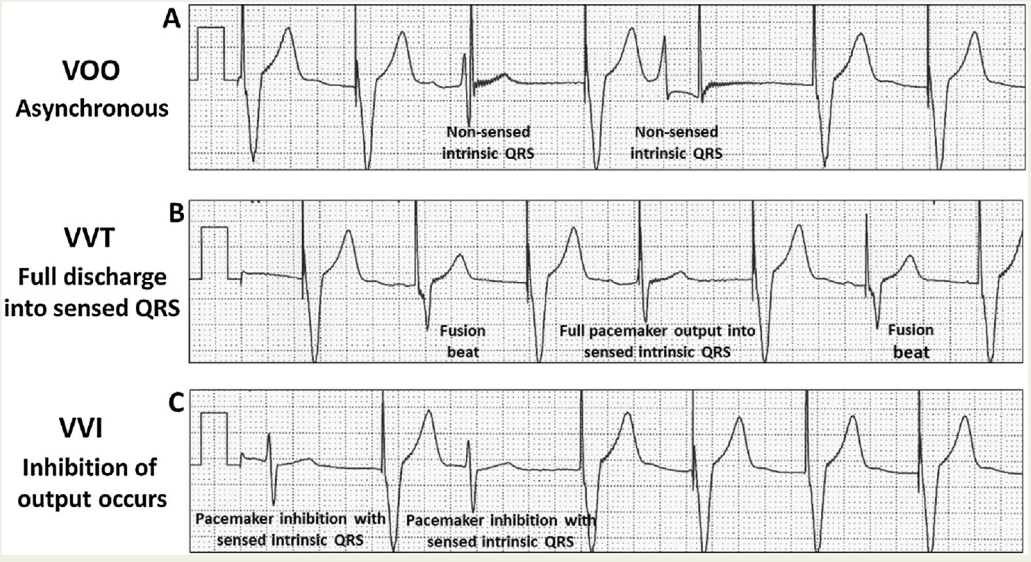

Figure 6 Electrocardiographs (ECGs) lead II; unipolar ventricular pacing modes from the same patient.
Above. VOO (asynchronous). No sensing. Stimulus artefacts are delivered irrespective of intrinsic rhythm and may fall within the refractory periods, thus not contributing to ventricular depolarisation.
Middle. VVT (triggered). Full output is delivered into the sensed intrinsic beat. Fusion beats are present.
Below: VVI (inhibited). Intrinsic QRS complexes are sensed and inhibit the next stimulus artefact.

Figure 7 Electrocardiograph (ECG) lead II. Unipolar AAI pacing with a prolonged atrio-ventricular delay (horizontal red arrow). Within two ventricular ectopics (vertical red arrows) are atrial stimulus artefacts. Although normal pacemaker function, atrial pacing appears asynchronous as there is no retrograde conduction.

The Recognition of Pacemaker Algorithms on the ECG

The array of algorithms required to correct inappropriate dual chamber intrinsic rhythm and pacemaker interactions are complex and usually company specific. The features are in continual development and differ between companies and even between models from the same company. The presentation of these algorithms as well as the perceived ECG abnormalities of conventional pacing modes can be categorised into groups which correspond to the way they present on the ECG.

Pacing Repetition Rate Below the Programmed Low Rate

In theory, with single chamber pacing, the low programmed pulse repetition rate cannot be violated. There is, however, a programmable function called hysteresis which allows the sensing window to be extended so as to encourage the patient's own rhythm (Figure 11) [8]. This may result in confusing pauses being present before ventricular pacing occurs.

In order to encourage intrinsic rhythms during rest or sleep, the pacing rate can be programmed lower at these times to mimic the physiologic bradycardia of the normal heart. Called sleep or rest rate, the function is either clock or sensor based and the rate of onset and offset may be gradual or rapid. Even if clock based, the sensor is used as a cross-check if the patient becomes roused or active during sleep hours. In turn, sensor based algorithms are also time based in that they require a certain period of rest before the sleep function is activated. An example of a sensor based sleep rate 24-hour Holter monitor is shown in Figure 12. Because the programmed low rate is violated, this may be interpreted as pacemaker malfunction.

Rapid Pacing Rhythms

When interpreting pacemaker Holter monitor reports, brief "inappropriate" rapid ventricular pacing is frequently seen and difficult to interpret. Most of these are short runs of a supraventricular tachycardia, which the ventricular channel follows until the programmed upper limit. Another is pacemaker mediated tachycardia, which is an endless loop re-entry tachycardia that occurs in patients with dual chamber pacemakers who have retrograde conduction. The pacemaker acts as the anterograde limb of an accessory pathway and, in order to create the substrate for a pacemaker mediated tachycardia, retrograde conduction must be sensed outside the PVARP which then triggers ventricular pacing, close to or at the upper rate. Triggers for retrograde conduction include atrial or ventricular ectopics (Figure 13A),

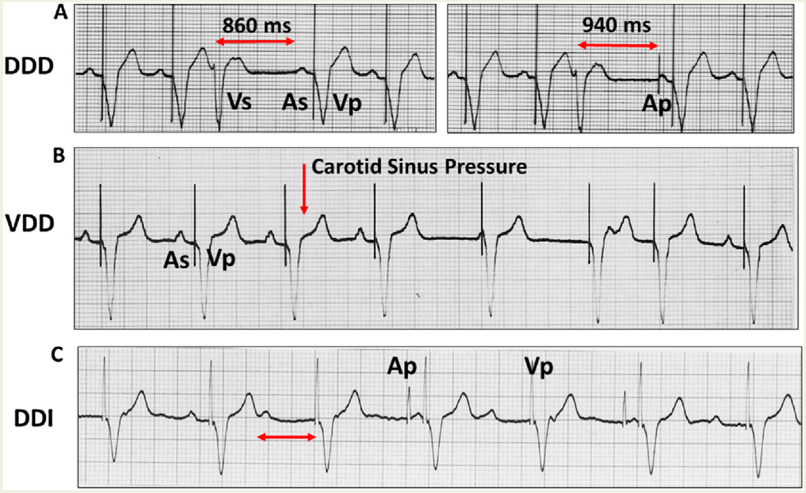

Figure 8 Electrocardiograph (ECG) leads II. Dual chamber pacing.
A: DDD. Atrial pacing (Ap), sensing (As) and ventricular pacing (Vp), sensing (Vs).
Left panel: Following a Vs (ventricular ectopic), As-Vp occurs after 860 ms.
Right panel: Following a Vs, Ap-Vp occurs after 940 ms which is the low rate.
B: VDD. As-Vp. Carotid sinus pressure demonstrates sinus slowing, resulting in VVI pacing as the sensed sinus rate falls below the low rate limit.
C: DDI. As-Vp with a limited ventricular tracking response to atrial sensing. Vp cannot violate the programmed ventricular rate of 65 bpm (920 ms). Although P waves are sensed, there may be a significant delay before the next Vp (red vertical arrow). Atrial pacing (Ap) occurs, if no P waves are sensed. Although the rhythm suggests VVI pacing, atrial pacing is also present.

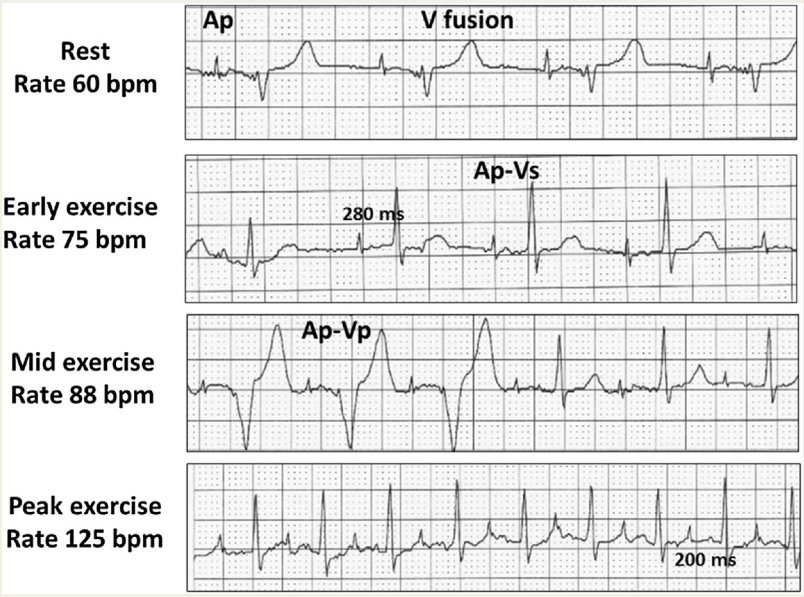

Figure 9 DDDR pacing during exercise. At rest, there is atrial pacing (Ap) and ventricular (V) fusion. As the Ap rate increases to 125 bpm, there is both ventricular pacing (Vp) and ventricular sensing (Vs). The atrio-ventricular delay shortens from 280 ms to 200 ms during exercise.

intermittent loss of atrial capture or sensing (Figure 13B), generally coupled with a prolonged atrio-ventricular delay and/or short PVARP.

The presence of a true endless loop tachycardia is rare today as each pacemaker company has one or more algorithms designed to prevent, recognise and treat pacemaker mediated tachycardia. Once detected, there are a set number of sensed tachycardia sequences, so that the pacemaker can exclude intrinsic tachyarrhythmias. Ventricular pacing is then withheld or the PVARP is extended for one beat, to block the retrograde conduction and break the incessant tachycardia cycle (Figure 13B).

In patients with proven neuro-cardiogenic syncope, dual chamber pacing has been shown to be beneficial in those with

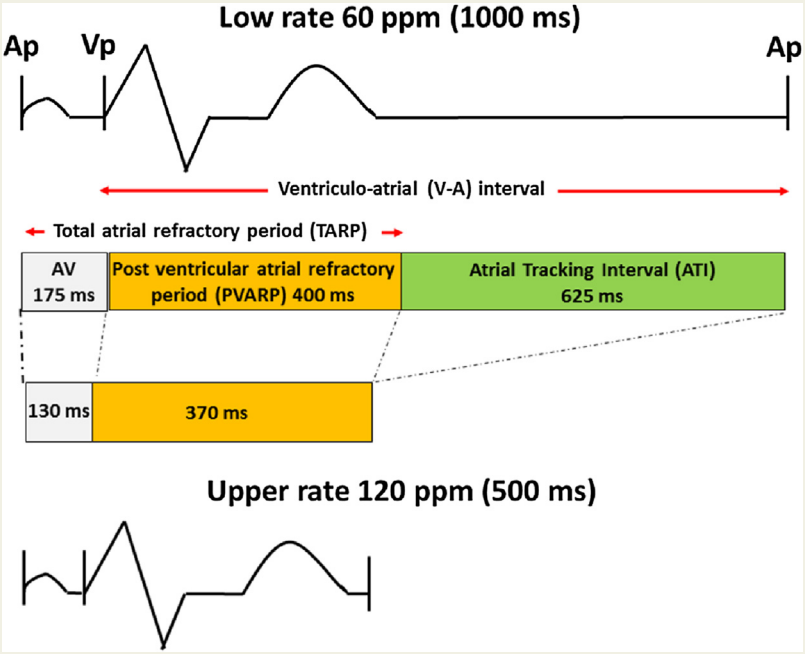

Figure 10 The basic dual chamber timing cycles at rest (above) and upper rate (below). See text for details. Abbreviations: Ap, atrial pacing; Vp, ventricular pacing; AV, atrioventricular delay

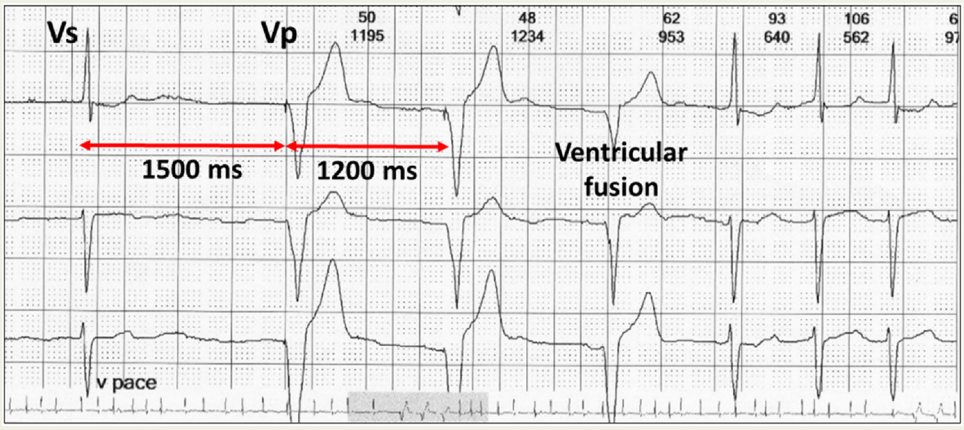

Figure 11 Three channel Holter recording demonstrating hysteresis with ventricular pacing (Vp) and atrial fibrillation (Vs). The pacing rate is 50 bpm (1200 ms). Following a Vs, the rate falls to 40 bpm (1500 ms) to encourage the intrinsic rhythm. Ventricular fusion is frequently seen with hysteresis.

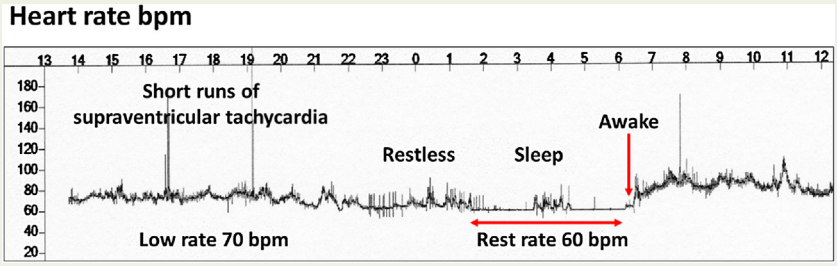

Figure 12 Holter monitor heart rate histogram over 24 hours, demonstrating rest rate function of a pacemaker programmed DDD (St Jude Medical/Abbott, Sylmar CA, USA). The low rate is 70 bpm, whereas rest rate is 60 bpm. During daytime, the heart rate ranges between 70 and 110 bpm with occasional short runs of supraventricular tachycardia. At 23:00 hours until 01:30, the patient is restless. From then until 06:00, rest rate is activated with occasional movement. At 06:00 hours, the patient wakes and rest rate is terminated.

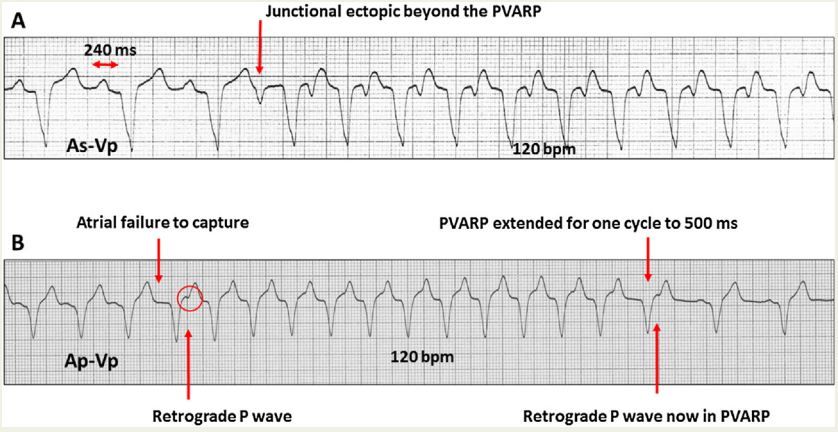

Figure 13 Pacemaker mediated tachycardia.
A. Electrocardiograph (ECG) lead II, DDD pacing with endless loop tachycardia. There is atrial sensing (As) and ventricular pacing (Vp) with a prolonged atrio-ventricular delay of 240 ms (red horizontal arrow). A sensed non-conducted junctional ectopic (red vertical arrow) lies outside the PVARP, followed by ventricular pacing which sets up the endless loop tachycardia at 120 bpm.
B. DDD pacing, lead II demonstrating an automatic algorithm to terminate pacemaker mediated tachycardia. There is initially atrial (Ap) and ventricular (Vp) pacing. Following atrial failure to capture (red downward vertical arrow), there is a retrograde P wave (red circle) which initiates a pacemaker mediated tachycardia at 120 bpm. The pacemaker determines that this is not an intrinsic tachycardia and extends the PVARP to 500 ms for one beat, terminating the tachycardia.

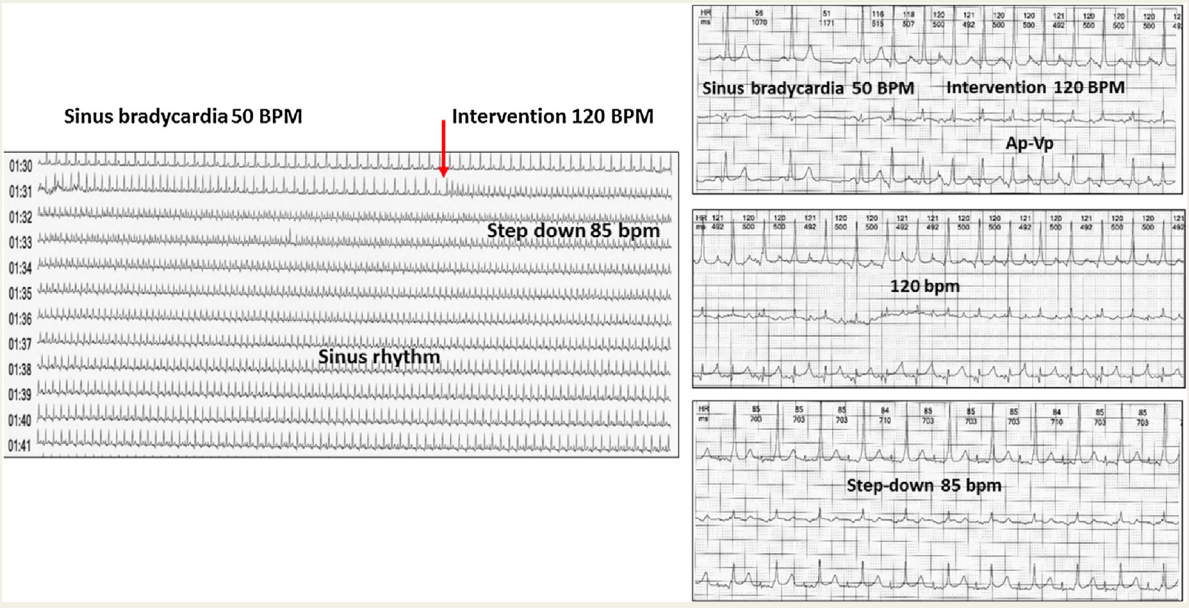

Figure 14 Three channel Holter recording to demonstrate pacemaker therapy to correct sudden bradycardia (Medtronic Inc. Minneapolis MN, USA).
Left: Full disclosure demonstrating abrupt onset of dual chamber pacing as intervention therapy for sinus bradycardia. After 2 minutes, there is step down pacing to 85 bpm followed by the reestablishment of sinus rhythm.
Right: Sinus bradycardia with abrupt rapid dual chamber intervention pacing (Ap-Vp) with step-down pacing in the bottom panel.

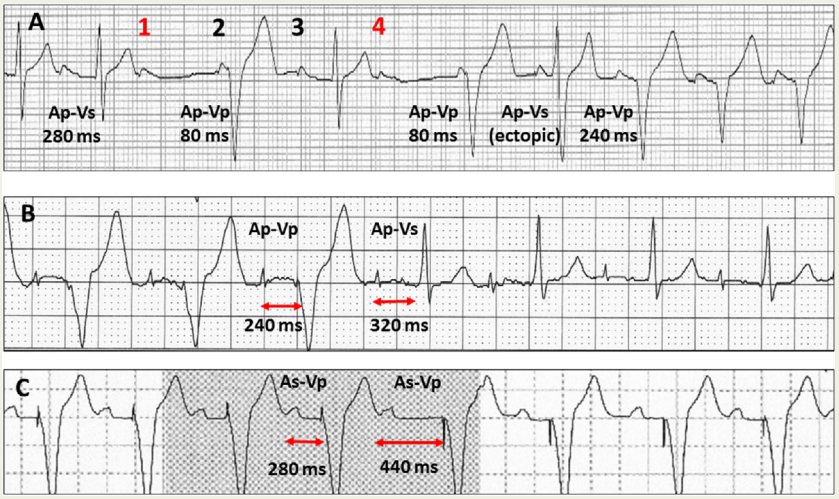

Figure 15 Single channel, Holter monitor recordings illustrating algorithms to minimise ventricular pacing.
A. Managed Ventricular Pacing [MVP^{TM} AAI(R) – DDD(R), Medtronic Inc.]. Atrial pacing (Ap) and ventricular sensing (Vs) with continual ventricular monitoring in the atrio-ventricular delay. Failure of atrio-ventricular conduction (1 in red), results in Ap-Vp with an atrio-ventricular delay of 80 ms. If two out of four paced or sensed P waves fail to conduct (1 and 4 in red), then DDD(R) pacing (Ap-Vp) is established at the programmed atrio-ventricular delay of 240 ms.
B. Successful atrio-ventricular delay extension algorithm. There is Ap-Vp with a 240 ms atrio-ventricular delay. Extension to at least 320 ms results in atrio-ventricular conduction (Ap-Vs) (St Jude Medical/Abbott).
C. Unsuccessful atrio-ventricular delay extension. With As-Vp, the atrio-ventricular delay is extended from 280 ms to 440 ms and Vp remains. As-Vp then continues (St Jude Medical/Abbott).

predominant bradycardia [9]. The algorithm common to all manufacturers is based on bradycardia recognition and appropriate rate intervention. On the Holter monitor, the characteristic appearances are those of accelerated pacing in the presence of a bradycardia and then gradual rate reduction (Figure 14).

Pacing Pauses

The presence of pauses in pacing on the Holter monitor usually denotes pacemaker malfunction such as a high threshold exit block or over-sensing of intrinsic or extracorporeal signals. Because of the potentially deleterious effects of chronic ventricular pacing on left ventricular function [10], there has, in recent years, been a concerted effort to design algorithms to minimise ventricular pacing in patients with intact atrio-ventricular conduction. One group, programmed "AAI(R)-DDD(R)" is effectively AAI(R) pacing with

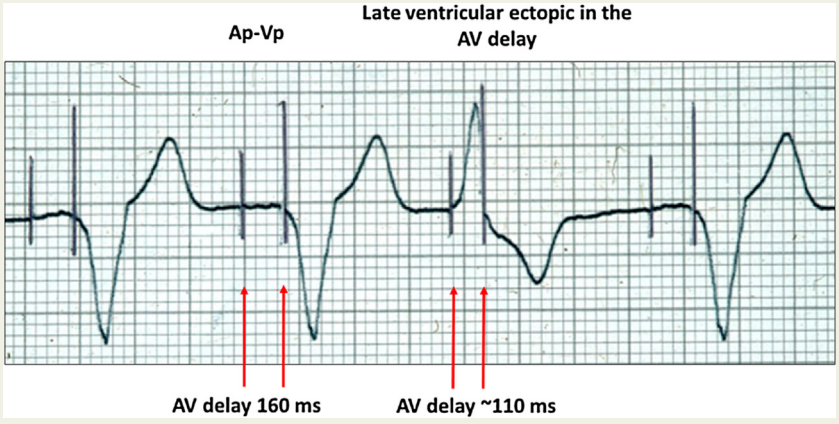

Figure 16 Electrocardiograph (ECG) lead II, ventricular safety pacing. Atrial (Ap) and ventricular (Vp) pacing with atrio-ventricular delay 160 ms (first set of red vertical arrows). A late ventricular ectopic is sensed in the ventricular safety pacing window during the atrio-ventricular delay, resulting in shortening to ~110 ms (second set of red vertical arrows).

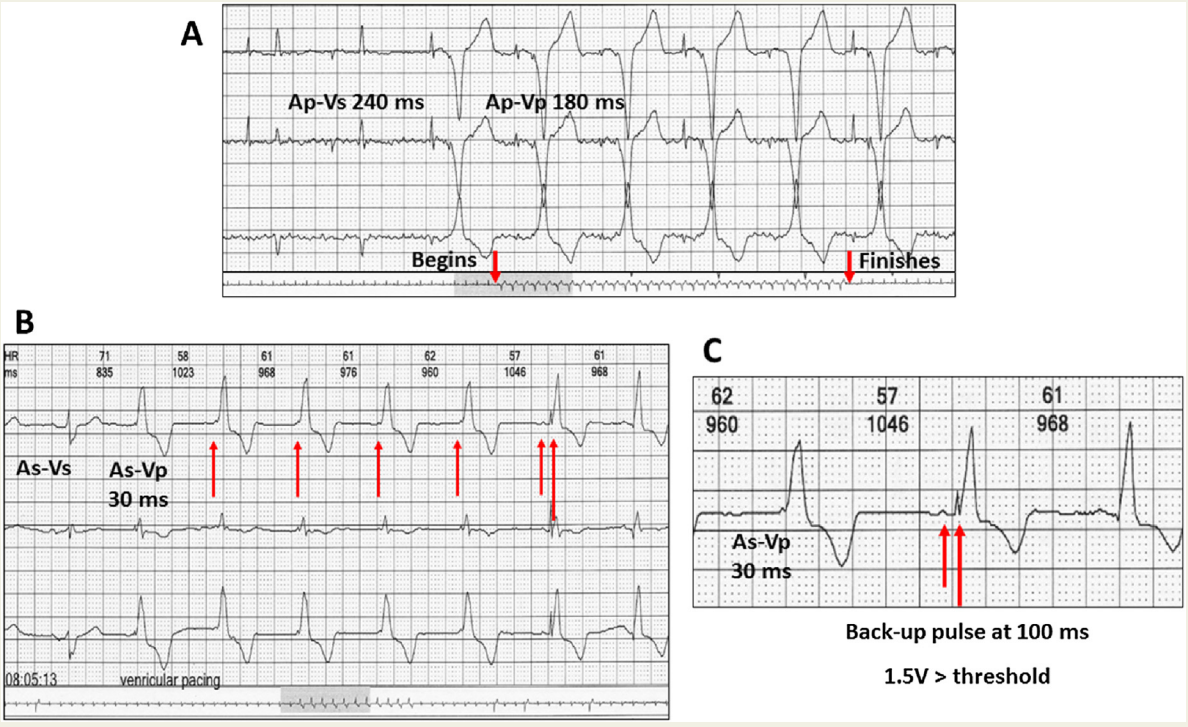

Figure 17 Three channel Holter recordings. Auto-threshold ventricular testing algorithms.
A. Atrial pacing (Ap), ventricular sensing (Vs) with 240 ms atrio-ventricular delay. At 1:00 AM, there is an abrupt onset of ventricular pacing (Vp), when the atrio-ventricular delay shortens to 180 ms. The condensed electrocardiograph (ECG) strip below (red vertical arrows) shows the period of testing (Medtronic Inc).
B. Atrial and ventricular sensing (As-Vs). There is an abrupt onset of As-Vp, when the atrio-ventricular delay shortens to 30 ms. After six beats, a back-up pulse occurs 100 ms after loss of ventricular capture (1.5 volts greater than threshold). As-Vp follows at the programmed atrio-ventricular delay (St Jude Medical/Abbott).
C. Magnified view of panel B at the time of loss of ventricular capture.

continual monitoring in the atrio-ventricular delay. Failure of atrio-ventricular conduction may result in an ''inappropriate'' pause before re-establishment of DDD(R) pacing (Figure 15A).

Varying Atrio-Ventricular Delay

This is a common finding on pacemaker Holter monitor recordings and may be due to a number of causes. As discussed under timing cycles, dual chamber upper rate can be extended by programming a rate adaptive atrio-ventricular delay and, as the atrial pacing or sensing rate increases, the atrio-ventricular delay shortens in a physiologic manner (Figure 9).

Another means of minimising ventricular pacing in patients with intact atrio-ventricular conduction, is an atrio-ventricular delay extension algorithm. If dual chamber

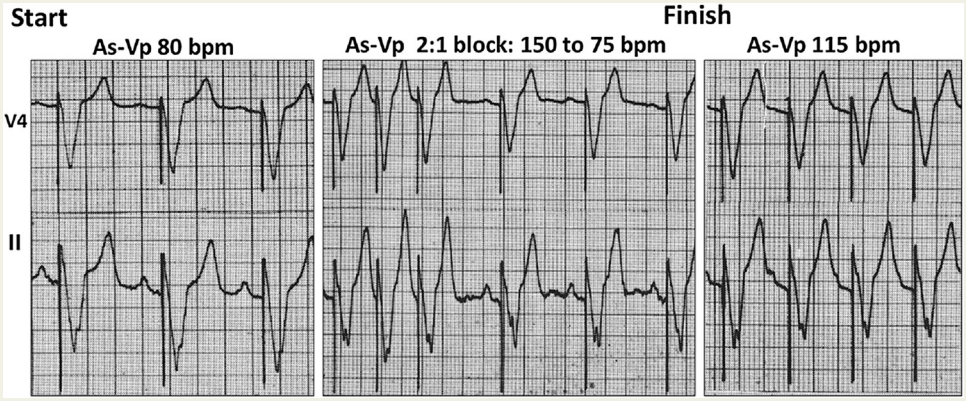

Figure 18 Two channel Holter recording with DDD pacing, demonstrating upper rate behaviour during exercise. There is atrial sensing (As), ventricular pacing (Vp) with an upper rate of 150 bpm. When the sinus rate exceeds 150 bpm, the ventricle cannot follow 1:1 and exhibits 2:1 block with Vp falling abruptly to 75 bpm. When the exercise is terminated, the sinus rate falls and atrio-ventricular synchrony is re-established.

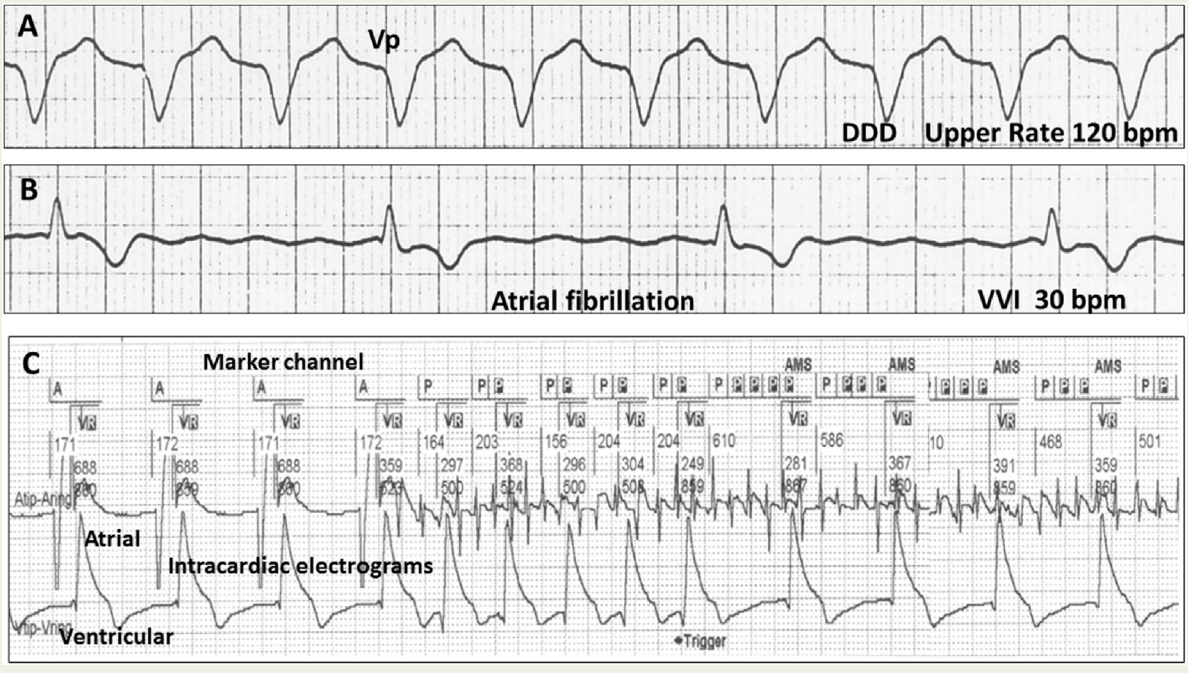

Figure 19 Mode switching. DDD pacing with atrial fibrillation.
A. DDD with upper rate ventricular pacing (Vp) at rest (120 bpm).
B. VVI 30 bpm. Same patient. The rhythm is atrial fibrillation with complete heart block.
C. Retrieved saved electrogram demonstrating onset of atrial fibrillation and mode switching (St Jude Medical/Abbott). The marker channel above and atrial and ventricular intracardiac electrograms below. There is atrial (A) and ventricular pacing (VR). Atrial fibrillation potentials (P) are chaotic within and outside the refractory period. There is accelerated ventricular pacing for five beats and mode switching (AMS®) then occurs.

ventricular pacing is present, then, at regular scheduled intervals, the atrio-ventricular delay is extended and, if ventricular sensing occurs, then the atrio-ventricular extension remains (Figure 15B). If ventricular pacing continues, then the algorithm is deactivated and the programmed atrio-ventricular delay re-established (Figure 15C).

At a time when only unipolar leads were available and skeletal muscle or extracorporeal sensing was common, inappropriate sensing of the atrial stimulus artefact or noise in the atrio-ventricular interval could result in ventricular inhibition with asystolic pauses, referred to as crosstalk. Today, most pacemaker manufacturers have a safety pacing window within the atrio-ventricular delay, where sensing, particularly of ventricular ectopics results in shortening of the atrio-ventricular delay to a value between 95 to 120 ms, although potentially shorter with faster pacing rates or very short programmed atrio-ventricular delays (Figure 16).

Capture threshold is the minimum amount of voltage required to consistently capture the paced chamber. In recent years, pacemaker companies have developed specific auto-threshold algorithms that test for ventricular capture thresholds. The threshold testing algorithms can be recognised on the ECG as there is forced ventricular pacing generally using very short atrio-ventricular delays (Figure 17A). With failure to capture, there is a back-up stimulus artefact which may be recognised on the ECG (Figure 17B,C). This back-up pulse may occur every time the voltage is reduced or only when failure to capture is recognised. The delay and polarity of this back-up pulse varies between manufacturers. Once the thresholds are determined, there is automatic readjustment of the voltage output.

Loss of Atrio-Ventricular Synchrony

During exercise, the sensed sinus rate may reach and then exceed the upper rate limit as discussed in the timing cycles. The result is a loss of atrio-ventricular synchrony, seen initially as an atypical Wenckebach sequence and then a 2:1 block (Figure 18) [11]. Clinically, there can be almost instantaneous halving of the ventricular rate, which the patient describes as like running into a brick wall and may be interpreted on the ECG as failure of atrial sensing.

One of the limitations of DDD(R) pacing is the sensing of atrial tachyarrhythmias resulting in ventricular pacing at the upper rate limit (Figure 19A). The atrial tachyarrhythmia can

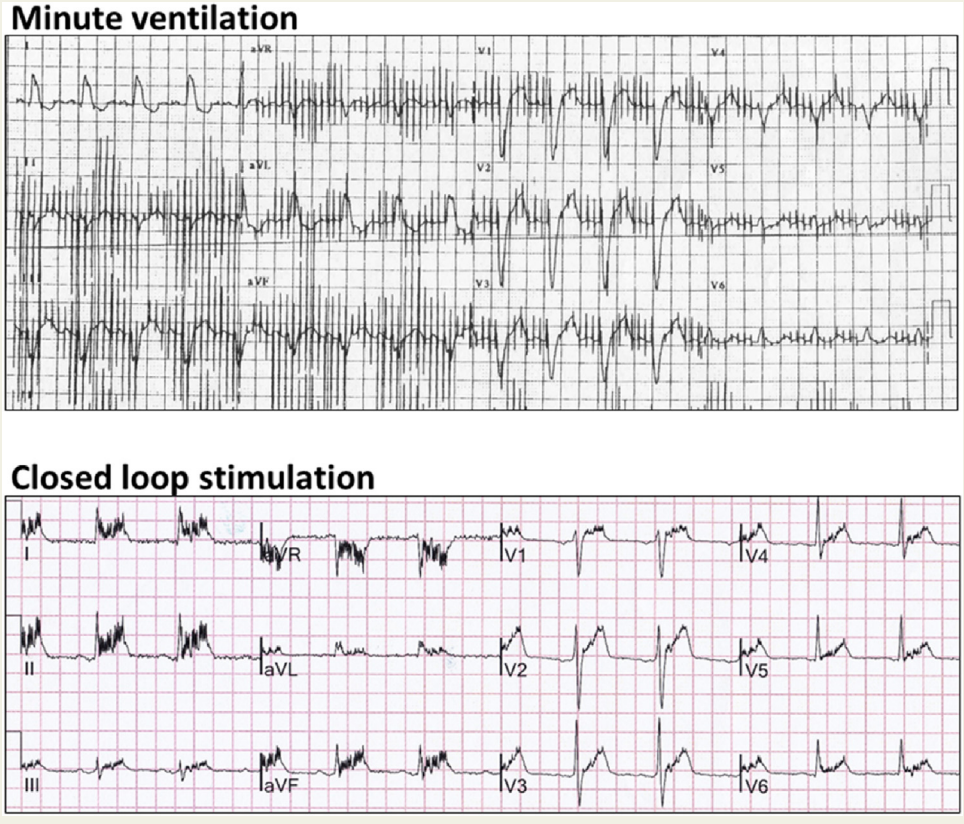

Figure 20 Persistent electrocardiograph (ECG) artefact from rate adaptive sensors.
Above: Minute ventilation sensor. The sensor measures changes in transthoracic impedance which reflect changes in minute ventilation. A very small subthreshold current is delivered across the chest wall every 40 ms resulting in marked artefact throughout the cardiac cycle (Boston Scientific, St Paul, MN, USA).
Below: Closed loop stimulation. The sensor delivers 16 bipolar subthreshold impulses commencing 50 ms after the onset of the sensed or paced QRS. The intracardiac impedance measurements reflect changes in the cardiac contraction dynamics (Biotronik, Berlin, Germany).

be diagnosed by programming VVI pacing with a slow pulse repetition rate (Figure 19B). To correct this pacemaker tachycardia, an algorithm called mode switching counts the number of sensed atrial events, both in the PVARP and the atrial tracking interval and following the application of company specific formulae, the pacemaker reverts to VVI(R) or DDI(R) pacing (Figure 19C). The ventricular rate is now dependent on the sensor to increase the ventricular rate. Atrial monitoring, however, continues during the tachyarrhythmia and once the arrhythmia reverts, DDD(R) pacing recommences.

Persistent ECG Artefact

Minute ventilation and closed loop stimulation are highly physiologic pacemaker sensors used with rate adaptive cardiac pacing. They both require the generation of frequent sub-threshold currents, either transthoracic or intracardiac, which may be detected on the ECG as artefact (Figure 20).

Conclusion

Electrocardiographs and Holter recordings are frequently performed on patients with implanted cardiac pacemakers and may demonstrate an array of poorly understood appearances, often mimicking pacemaker malfunction. The objective of this review was to provide a window into the ECG patterns of normal pacemaker function and outline the range of common company specific algorithms that test, correct or provide therapy.

Disclosures

Associate Professor Harry G Mond is Medical Director of Cardioscan Pty Ltd.

No external financial support.
No acknowledgements.

References

[1] Mond HG. Unipolar versus bipolar pacing — poles apart. Pacing Clin Electrophysiol 1991;14:1411–24.
[2] Mond HG. The Cardiac Pacemaker. Function and Malfunction. Clinical Cardiology Monographs. New York: Grune and Stratton; 198393–8.
[3] Rosso R, Medi C, Teh AW, Thuy TH, Feldman A, Lee G, et al. Right ventricular septal pacing: A comparative study of outflow tract and mid ventricular sites. Pacing Clin Electrophysiol 2010;33:1169–73.
[4] Barold SS, Herweg B. Usefulness of the 12-lead electrocardiogram in the follow-up of patients with cardiac resynchronization devices. Part II. Cardiol J 2011;18:610–24.
[5] Hiroshi M, Yuichi U, Rinya K, Akihiko U, Takashi M, Yasushi T, et al. Long-term clinical performance of AAI pacing in patients with sick sinus syndrome: a comparison with dual-chamber pacing. Europace 2004;6:444–50.
[6] Mond HG, Proclemer A. The 11th world survey of cardiac pacing and implantable cardioverter-defibrillators: Calendar year 2009. Pacing Clin Electrophysiol 2011;34:1013–27.
[7] Barold SS. The DDI mode of cardiac pacing. Pacing Clin Electrophysiol 1987;10:480–4.
[8] Rosenqvist M, Vallin HO, Edhag KO. Rate hysteresis pacing: How valuable is it? A comparison of the stimulation rates of 70 and 50 beats per minute in patients with sick sinus syndrome. Pacing Clin Electrophysiol 1984;7:332–40.
[9] Baron-Esquivias G, Morillo CA, Moya-Mitjans A, Martinez-Alday J, Ruiz-Granell R, Lacunza-Ruiz J, et al. Dual-chamber pacing with closed loop stimulation in recurrent reflex vasovagal syncope. The SPAIN study. JACC 2017;70:1720–8.
[10] McGavigan AD, Mond HG. Selective site ventricular pacing. Curr Opin Cardiol 2006;21:7–14.
[11] Mond HG, Vohra J. The electrocardiographic footprints of Wenckebach block. Heart Lung Circ 2017;26:1252–66.