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Pacing System Malfunction: Evaluation and Troubleshooting

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1. Introduction

Currently, most of the time spent caring for the patients with permanent pacemaker will be concerned with evaluating the function of the previously implanted pacing system. Pacing system malfunction can be secondary to pacemaker circuitry failure or to lead dysfunction. Various tools are available to help evaluation of the pacing system malfunction, including physical examination, standard 12-lead ECG, ambulatory ECG monitoring, stored telemetry data, chest x-ray, and fluoroscopy.

This chapter will discuss the differential diagnosis, evaluation, and management of the common malfunctions encountered in the single-chamber, dual-chamber, and biventricular pacing system.

2. Single-chamber pacing system malfunction

Although many pacing system malfunction are common to single-chamber and dual-chamber devices, the latter are more challenging. This section will discuss the common malfunctions associated with single-chamber pacing systems. The subsequent sections will focus on specific abnormalities associated with dual-chamber and biventricular pacing systems.

Depending on the presence or absence of pacing artifact, the potential problems identifiable on an ECG can generally be assigned to three categories: 1) pacing artifact present with failure to capture, 2) pacing artifact absent with failure to pace, 3) pacing artifact present with failure to sense.

2.1 Pacing artifact present with failure to capture

To differentiate between different categories of the pacing system malfunctions, it is essential to identify the pacing artifact. This can be done easily in unipolar system with large pacing artifact. However, it can be problematic in dual-chamber devices with diminutive pacing artifact. There are two important issues related to the pacing artifact that can cause great difficulty in differential diagnosis of pacing system malfunction. Some ECG recording systems, such as holter monitoring and in-hospital monitoring units, contain special filter to eliminate baseline noise (Sheffield et al, 1985). These filters can markedly reduce the size of pacing artifact in the unipolar pacing system and effectively erase the pacing stimuli in the

bipolar system. In these situations, recording of the multiple simultaneous or sequential ECG leads with older analog ECG machines would be highly helpful. In addition, newer recording systems (e.g. telemetry system and holter recorder) have unique algorithms for pacemaker pulse detection that generate a discrete artifact in response to any high-frequency signal. Therefore, even nonpacemaker signals may be displayed as pacing artifact. Electromagnetic interference, loose ECG electrode connections, and subthreshold pulses (minute ventilation sensors) are examples of nonpacemaker signals.

The causes of failure to capture with pacing artifact present are summarized in Table 1. The onset of malfunction in relation to implantation of pacing system can provide valuable data to identification of the cause. If failure to capture occurred shortly (hours or days) after implant, the most likely diagnosis is lead dislodgment or perforation. Loss of capture weeks to months after implantation is most probably due to lead maturation process. Currently, this problem is rarely seen because of routine use of the steroid-eluting electrodes. This malfunction occurs between 2 to 6 weeks after implantation. If loss of capture of capture occurs several months to years after implantation, structural injury in the lead insulation or conductor should be suspected. Other less common causes in this period are exit block, metabolic abnormalities, pharmacological agents, misprogramming, and battery depletion.

Lead dislodgment is accompanied by a change in the paced QRS morphology and/or the lead position in repeat chest x-rays. A change in the paced QRS morphology is diagnostically useful when fusion with native QRS complexes is absent. Depending on presence or absence of a visible change in the lead position on a repeat chest x-ray, lead dislodgment can be classified as “macro-dislodgment” or “micro-dislodgment”. Other distinguishing features are normal pacing impedance and elevated thresholds. Incidence of the lead dislodgment has markedly reduced with the introduction of active fixation leads. However, active lead implantation does not guarantee long-term lead stability (Furman et al, 1975). Correction of this malfunction requires surgical intervention. Before reoperation, the cause of dislodgment should be identified. An examination of anchoring sleeve for adequate fixation, looking for adequate amount of current-of-injury in the recorded electrograms at the implant time, and looking for proper lead slack on postimplant chest x-ray would be highly informative. In most cases, lead repositioning is enough but thrombus developed around the lead tip may effectively prevent the lead tip to be fixed at new position. The latter situation, lead dislodgement with no identifiable cause and repeated dislodgment after prior lead repositioning requires new active lead implantation. *Lead perforation* usually occurs at the time of implant or within the first 24 hours (Aggarwal et al, 1995), although this complication has been reported up to 10 months after the device implantation (Haghjoo, 2010). In addition to loss of capture, perforation manifests itself by lead migration to pericardial, pleural, or peritoneal space. Lead perforation can be confirmed by the negative current of injury at implant. *Lead maturation* is an inflammatory reaction that is produced following lead tip contact with endocardium (Furman et al, 1979; Furman et al, 1977). Local tissue swelling displaces the electrode tip from excitable myocardium and, consequently, increases the capture threshold. Although incidence of this problem was significantly reduced in steroid-eluting leads (Mond et al, 1988; Kein et al, 1990), a significant rise in threshold may occur despite the introduction of steroid-eluting leads and other innovation in lead design (Ellenbogen et al, 1999). This process occurs between 2 to 6 weeks after implantation. To minimize the probability of noncapture, a high output setting is recommended during this period. Any threshold rise after this period is considered to be in the chronic phase of lead maturation. If the threshold rise resulted in noncapture, it is

referred to as exit block (Sheppard et al, 1991; Davies & Sowton, 1966). Acute and chronic phase of lead maturation are not associated with a change in paced QRS morphology or lead location in chest x-ray. Acute threshold rise occasionally responds to systemic steroids. A regimen of prednisone, 60 mg (1 mg/kg) per day, is found to be effective in one-half of the patients (Levine, 2005). Exit block does not respond to systemic steroids and require lead repositioning or new lead implantation.

Lead dislodgment
Micro-dislodgment
Macro-dislodgment
Perforation
Lead maturation process
Early inflammation
Late fibrosis- exit block
Mechanical lead injury
Insulation failure
Conductor fracture
Iatrogenic malfunctions
Loose setscrew
Misprogramming
Elevated pacing thresholds
Metabolic abnormalities
Electrolyte disturbances
Myocardial infarction
Cardiomyopathy
Pharmacological agents
Pseudomalfunctions

Table 1. Causes of failure to capture with pacing artifact present

Lead insulation failure is mainly due to extrinsic forces exerted to the lead either at or following implantation. Insulation damage has reported both in silicone- and polyurethane-coated leads (Kertes et al, 1983). Excessively tight suture on the anchoring sleeve, lead passage between clavicle and first rib, and abrasion between overlapping coils or between leads and housing of the pulse generator are three common causes of the insulation defect in clinical practice (Dunlap et al, 1983; Magney et al. 1993). ECG with an insulation failure involving a unipolar lead will show a decrease in amplitude of pacing artifact. An insulation damage involving outer insulation of a coaxial bipolar lead will show an increase in the amplitude of the pacing artifact (unipolarization). Failure of the internal insulation of a coaxial bipolar lead will results in a decrease in the amplitude of pacing artifact. This is best identified with an analog ECG recording system. Chest x-ray is normal in the patients with insulation failure because insulation material is radiolucent. Another specific feature of insulation defect is reduced lead impedance.

Lead conductor fracture is the most common cause of an open circuit. Although the clinician might expect that fracture of a lead would result in the absence of any visible pacing artifact, this is frequently not the case. This occurs because the current passes across the gap in the conductor coil though the fluid in the lead with high resistance. In such circumstances,

energy reaching the heart is subthreshold with no capture, but a stimulus will be present. The ECG with an intermittent conductor fracture may show a varying amplitude artifact if recorded with an analog machine. Lead fracture is associated with infinitely high lead impedance if the insulation remains intact. However, total lead transection (concomitant conductor fracture and insulation defect) may show normal or minimally elevated impedance. Chest x-ray may show conductor fracture (figure 1). In contrast to the unipolar lead, diagnosis of the conductor fracture is very difficult in a coaxial bipolar lead unless there is total disruption of both conductors at the same place. Reprogramming to unipolar mode is a temporary measure in partial fracture of bipolar lead. In pacemaker-dependent patients with complete lead transection, temporary pacemaker implantation is necessary before definitive management. Definitive treatment of conductor fracture is lead replacement.

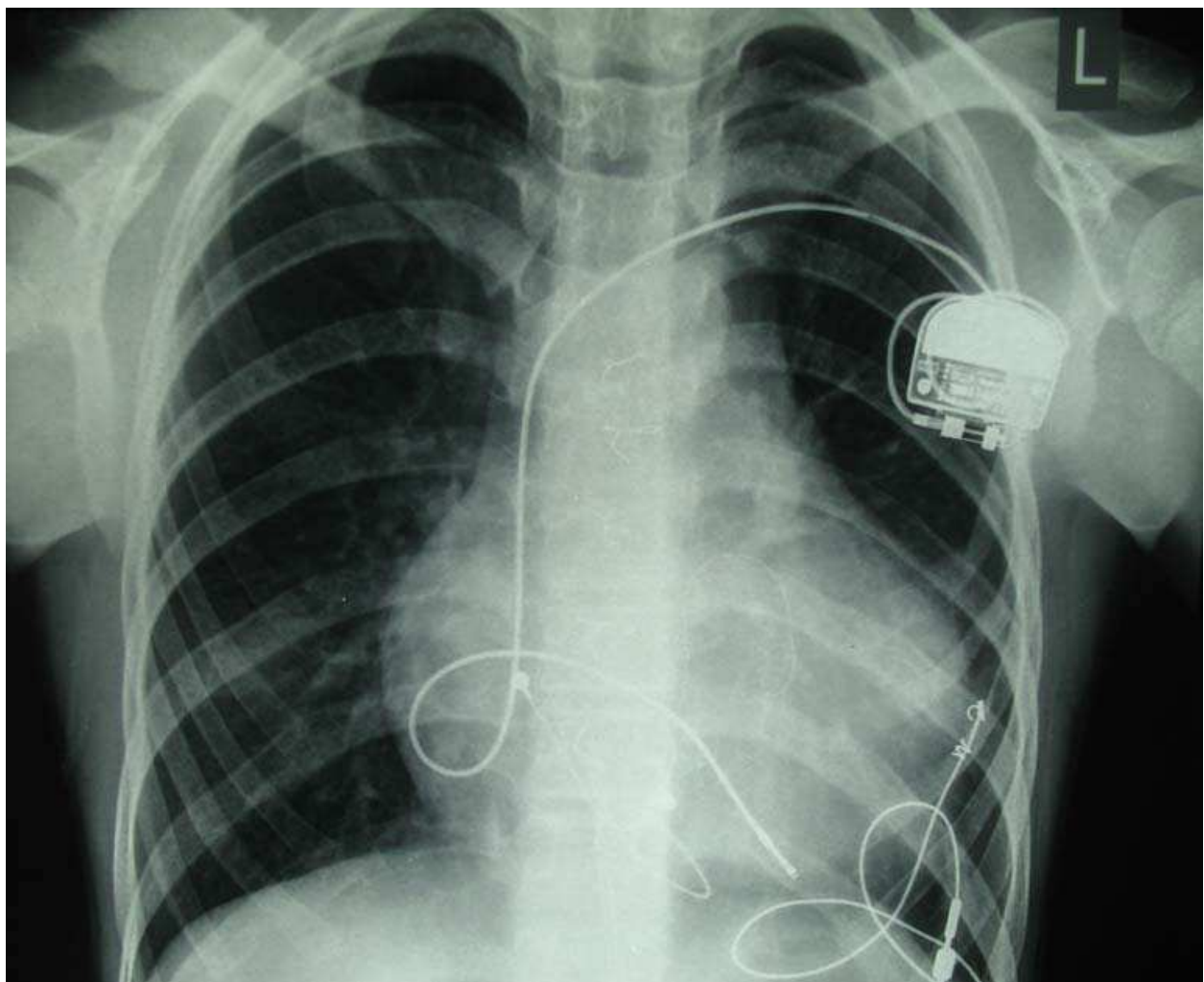


Fig. 1. This chest x-ray illustrates a typical example of conductor fracture with intact insulation. Conductor fracture occurred near to the fixation sleeve.

Loose set screw usually presents with failure to output due to complete disconnection of the lead pin and the connector. Sometimes, minimal contact between the pin and the connector may permit the transmission of the electrical current, but delivered energy is not enough to capture the paced chamber. This problem is produced because of the inadequate tightening of the lead pin within the connector block. The presence of air or fluid within the connector

can produce similar clinical picture. ECG usually may show a decrease in the amplitude of the pacing artifact. Chest x-ray clearly shows the displacement of the terminal pin out of the head of pacemaker. Reoperation with pin reconnection is necessary to correct these malfunctions.

Battery depletion manifests several years after device implantation. Pacemaker-dependency is major determinant of the battery longevity. Although modern pacemakers attempt to maintain the programmed values until the battery is depleted, actual output will fall below the capture threshold and resulting in the loss of capture. ECG and chest x-ray show no abnormalities. Pacing threshold is increased as measured via depleted generator but is normal through the pacing system analyzer. Definitive management requires the generator replacement.

Late rise in the pacing threshold may also occur secondary to the *myocardial infarction, cardiomyopathy, metabolic abnormalities, and pharmacologic agents* (Schlesinger et al, 1980; Hughes, et al, 1975; Preston & Judge 1969; Guarnieri et al, 1988). Any severe electrolyte or metabolic abnormalities can increase pacing threshold. These abnormalities include acidosis, alkalosis, hypoxemia, hypercarbia, hyperkalemia, hyperglycemia, and hypothyroidism. In addition, threshold rise and noncapture may be due to some pharmacological agents such as bretylium, class Ic antiarrhythmic drugs, sotalol, and mineralocorticoids. Myocardial infarction and cardiomyopathy can cause noncapture by producing nonexcitable tissue near the lead tip. Increasing the programmed output, correcting the cause, and the lead repositioning are the therapeutic options in these situations.

2.2 Pacing artifact absent with failure to pace

In this category of pacing system malfunction, the clinician must be certain that a malfunction truly exists before taking any corrective measure. Already diminutive pacing artifact of the bipolar pacing may be further obscured by being isoelectric in a given lead. New pacemaker algorithms for minimal ventricular pacing may also present with absence of the pacing artifact. Similarly, hysteresis and sleep function can mimic this malfunction. Differential diagnosis of pacing artifact present with failure to pace is listed in Table 2. *Oversensing* is the most common cause of intermittent pauses without pacing artifact.

Oversensing
Physiological signals- T-wave, myopotential
Nonphysiologic signals- Electromagnetic interference
Nonphysiologic signals- Make-break signals
Open circuit
Conductor fracture
Loose set screw
Insulation failure
Battery depletion
Pulse generator component malfunction
Pseudomalfuctions
Sleep function
Hysteresis
Algorithms for minimizing ventricular pacing

Table 2. Causes of failure to pace

Oversensing is the sensing of a physiologically inappropriate or nonphysiologic signal. Oversensing is more common in the unipolar pacing. T-waves and myopotentials are major sources of the physiologically inappropriate signals. Myopotential oversensing either inhibits the ventricular output in the ventricular channel or triggers ventricular output when sensed on the atrial channel of a DDD pacemaker. T-wave oversensing can similarly produce long asystole in pacemaker-dependent patients. If the native signal is sufficiently large, reducing the ventricular sensitivity can easily correct this abnormality.

Nonphysiologic electrical signals may also cause oversensing. The potential sources of electromagnetic interference (EMI) are electrocautery, electrolysis, radiofrequency (RF) ablation, cardioversion, defibrillation, lithotripsy, magnetic resonance scanner, positron emission tomographic scanner, high voltage power line, cellular and cordless phone, transcutaneous electrical nerve stimulation, and radiotransmission (AM and FM). Other nonphysiologic source of oversensing is make-break signals. If of sufficient amplitude, these signals can cause significant oversensing and long pauses. The most common sources of make-break signals are loose set-screw, partial conductor fracture, failure of the internal insulation of bipolar lead, loose active fixation screw, and interaction between active and abandoned leads. Diagnosis of the oversensing can be confirmed by applying magnet or programming to asynchronous mode. The oversensing-related pauses are relieved by before-mentioned maneuvers. The definitive treatment differs depending on the sources of oversensing. The lead problems require lead replacement, or repositioning, or correction of the loose connection. Oversensing related to the physiological signals can be treated by reprogramming or lead repositioning. EMI-related oversensing needs avoidance from EMI sources.

Open circuit secondary to conductor fracture or loose set screw are second most common cause of pauses associated with absence of the pacing artifact. As it was mentioned previously, open circuit usually present with loss of capture in the absence of pacing artifact. Measured data telemetry will show infinitely high pacing impedance. Chest x-ray can be diagnostic in unipolar lead but conductor fracture is difficult to detect in case of a bipolar lead. Magnet application has no effect on pauses associated with open circuit. Definitive treatment is the lead replacement or tightening of the pin within the header.

Lead insulation failure can cause failure to capture in the absence of pacing artifact mainly because of oversensing. Again, malfunction persists after magnet application. Reprogramming to unipolar mode can temporarily correct the malfunction until definitive treatment is done.

Pulse generator malfunction is the least common cause of the pacing system malfunction. This malfunction does not respond to magnet application. Pacemaker interrogation shows inconsistent measurements or inaccessible data. Pulse generator replacement is the only effective treatment.

2.3 Pacing artifact present with failure to sense

A proper sensing require good quality signal with adequate amplitude and slew rate. Apparently high amplitude signal may not be appropriately sensed because of the slow slew rate (Furman et al, 1977). Undersensing is characterized by the inappropriate pacing because of underdetection of the intrinsic signals (figure 2). The etiologies of the undersensing are listed in Table 3. As in failure to capture, onset of the undersensing in relation to implantation time may direct the clinician to the correct diagnosis. Early-onset undersensing is most probably due to *lead dislodgment or lead perforation*. Late-onset

undersensing is frequently caused by *mechanical lead problems (insulation failure and partial open circuit)* or *programming errors*. Occasionally, they are attributable to a *change in the morphology of the intrinsic cardiac signal*.

In addition to failure to capture, the *lead dislodgment* is frequently associated with undersensing. In this circumstance, displaced lead will record a signal different from and usually smaller than that recorded at implant. Perforated lead similarly records a smaller signal. Therefore, sensing failure will occur in both situations. Definitive management is the lead replacement.

Lead insulation failure can be associated with sensing failure due to attenuation of the incoming signal. Other corroborating features are increased capture threshold, decreased pacing impedance, increased battery drain, and reduced amplitude of the pacing artifact. Curative treatment will require lead replacement or repair.

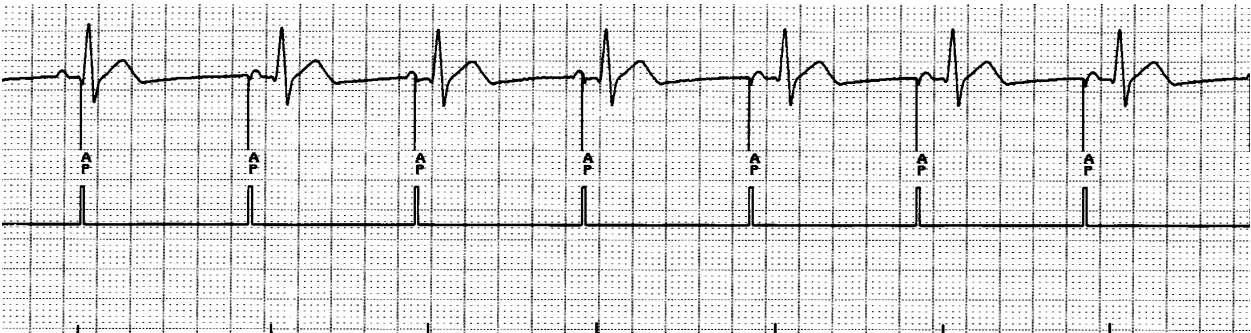


Fig. 2. This tracing shows inappropriate atrial pacing because intrinsic depolarization is not seen by the pacemaker.

Lead dislodgment
Micro-dislodgment
Macro-dislodgment
Perforation
Lead maturation process
Mechanical lead injury
Insulation failure
Partial open circuit
Battery depletion
Elevated pacing thresholds
Metabolic abnormalities
Electrolyte disturbances
Myocardial infarction
Cardiomyopathy
Pharmacological agents
Pseudomalfuctions
Asynchronous mode
Triggered mode
DVI mode
Safety pacing

Table 3. Causes of sensing failure

Partial open circuit is another cause of the undersensing. If the two ends of the conductor are making any contact at all, resistance to incoming signal will reduce the amplitude of the filtered signal and undersensing will occur. Infinitely high impedance is an important diagnostic feature. Chest x-ray is also diagnostic in some cases. Correction of this malfunction requires lead replacement or reconnection of the lead pin within the connector block.

Change in the native signal amplitude occurs secondary to myocardial infarction, cardiomyopathy, metabolic abnormalities, and some pharmacologic agents.

As the *pulse generator begins to become depleted*, intermittent or persistent undersensing may occur. *Stuck reed switch* is another rare cause of the undersensing. Placing *magnet over the pacemaker or triggered mode* are other causes of the failure to sense. In pacemakers using *impedance plethysmography as a rate modulation sensor*, removal of generator out of the pocket will result in the asynchronous pacing in response to a lot of electrical noise. Definitive treatment of the undersensing depends on the underlying cause.

3. Dual-chamber pacing system malfunction

Dual-chamber pacing system malfunctions can be categorized into two groups: 1) general dual-chamber pacing system malfunctions; 2) specific dual-chamber pacing system malfunctions.

3.1 General dual-chamber pacing system malfunctions

All abnormalities previously discussed in the section of the single-chamber pacing system malfunctions can occur with either channel of the dual-chamber pacemakers. However, malfunctions easily detected in single-chamber pacemakers may be very difficult to diagnose in dual-chamber devices. For example, detection of the ventricular loss of capture may be very challenging in the presence of normal atrioventricular (AV) conduction. In this situation, changing the AV interval can be very useful. Occasionally, it is not easy to detect atrial noncapture in the dual-chambers pacing systems. In this situation, programming to AAI mode or increasing AV interval will readily unmask the malfunction. Atrial undersensing may be difficult to recognize in the presence of an intact sinus node function. Oversensing in dual-chamber pacing systems can result in inappropriate inhibition or triggering depending on the channel on which oversensing occurs and the programmed pacing mode. Atrial channel oversensing will result in rapid ventricular pacing whereas oversensing in the ventricular channel will inhibit both atrial and ventricular channels.

3.2 Specific dual-chamber pacing system malfunctions

Crosstalk is the sensing of the far-field signal in the opposite chamber, causing the pacemaker to either inhibit or trigger an output depending on its design. Crosstalk can occur in any dual-chamber mode in which atrium is paced and the ventricle is sensed and paced (DVI, DDI, and DDD). In the presence of complete heart block, inhibition of the ventricular pacing can be life-threatening (Sweesy et al, 1988). Crosstalk is more difficult to detect in the presence of intact AV conduction. Several factors predispose the patients to crosstalk inhibition of the ventricular output: unipolar atrial and ventricular leads, high atrial output, high ventricular sensitivity, and short ventricular blanking and refractory periods. Crosstalk is diagnosed by the ventricular sensing in the absence of a QRS complex in the telemetered marker channels and corrected by the magnet application.

There are several preventive strategies to avoid the crosstalk or its sequel. Use of a bipolar lead with adequate amplitude and pulse width, proper ventricular sensitivity, use of the ventricular blanking period, and safety pacing are the main approaches to prevent the crosstalk (Batey et al, 1985; Barold & Belott, 1987).

Pacemaker-mediated tachycardia (PMT) is characterized by the active participation of the pacemaker in the tachycardia (figure 3). There are several forms of PMT. Typical form of PMT is endless-loop tachycardia (ELT). ELT only occur in patients with P-synchronous dual-chamber pacing (DDD, VDD) and intact retrograde ventriculoatrial conduction. ELT is classically initiated by a PVC. If the PVARP has expired and retrograde P-wave is sensed, the sensed atrial event triggers the ventricular output. This setups a repetitive sequence of the sensed retrograde P-wave, triggering a ventricular output at the end of maximum tracking rate interval (Den Dulk et al, 1982). In addition to PVC, atrial undersensing, atrial oversensing, and atrial noncapture may initiate ELT. PVARP extension after a sensed ventricular event is the main defense against the ELT. If ELT is initiated, PVARP extension and withholding the ventricular output for one cycle are the major modalities to terminate ELT. Second form of PMT is secondary to tracking of atrial signals (atrial tachycardia/flutter/fibrillation) (figure 4) or oversensing on the atrial channel (Greenspan et al, 1984). This is not a pacemaker malfunction and terminated by applying magnet. Third form of PMT is called repetitive nonreentrant ventriculoatrial synchronous rhythm (Love, 2007). Several factors predispose the patient to this kind of PMT. First, intact ventriculoatrial conduction is needed. Second, a sufficiently long PVARP should be programmed to avoid ELT induction. Third, a relatively rapid baseline rate is also required to render the atrial tissue refractory. In this condition, retrograde P-wave is not sensed (functional undersensing) allowing atrial escape interval to be completed and atrial pulse is delivered with no capture (functional noncapture). Then, the ventricular output is delivered at the end of the programmed AV interval. Therefore, this rhythm is characterized by ventricular pacing, atrial undersensing, and atrial noncapture in the presence of normally functioning dual-chamber DDDR or DDIR pacemakers (figure 5). To prevent this rhythm, a new algorithm called noncompetitive atrial pacing (NCAP) has been introduced. Other preventive strategies are providing short AV intervals at high rates or AAI pacing.

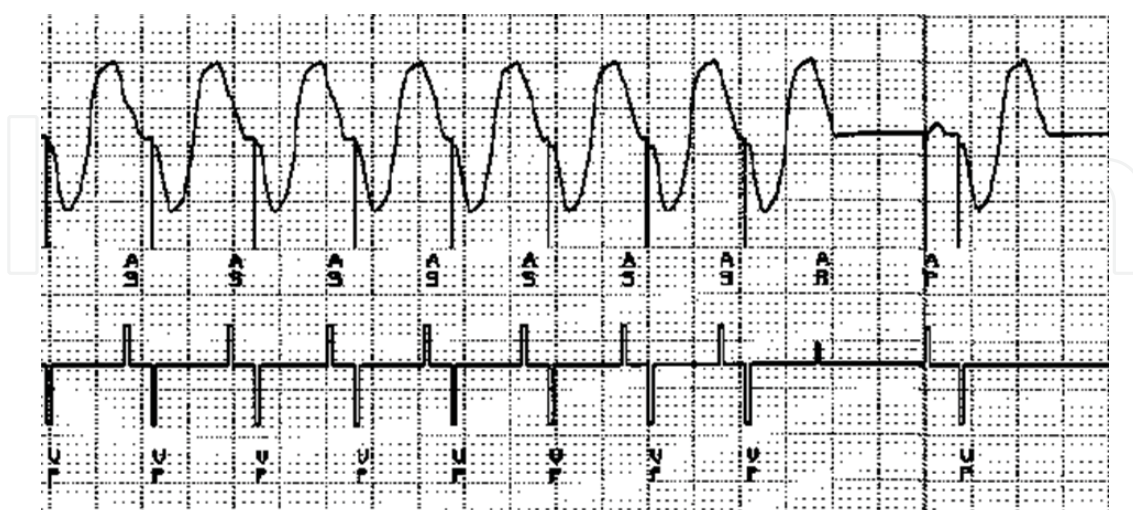


Fig. 3. This tracing was recorded from a patient with dual-chamber pacemakers who presented with frequent palpitations. Pacemaker interrogation showed frequent episodes of pacemaker-mediated tachycardia initiated by a PVC and terminated by a P-wave falling within the PVARP.

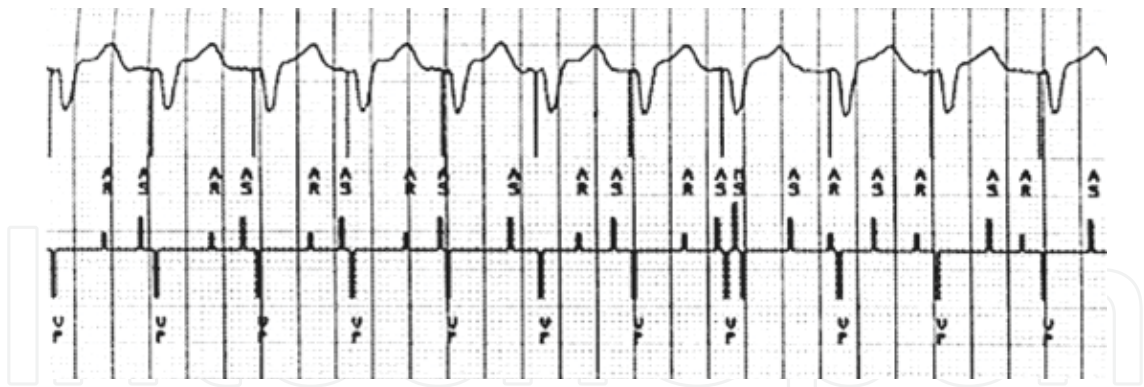


Fig. 4. This figure is recorded from a patient with dual-chamber pacemaker and paroxysmal palpitations. Device interrogation showed tracking of the atrial signals during paroxysmal atrial tachycardias.

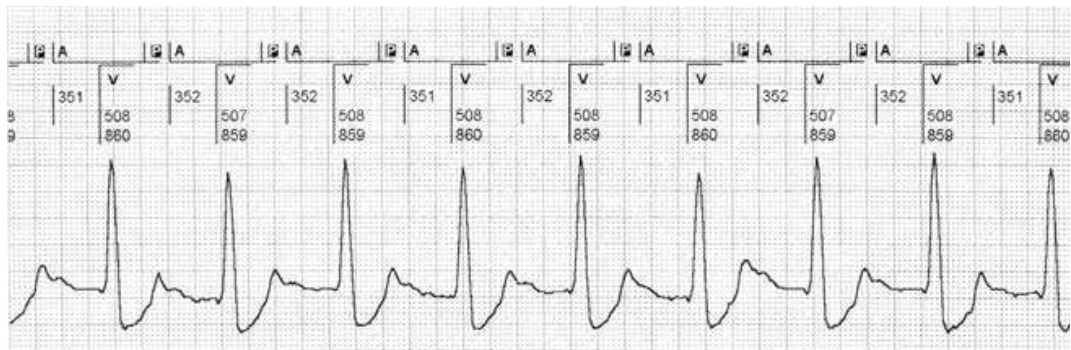


Fig. 5. This figure shows a classical example of repetitive nonreentrant ventriculoatrial synchronous rhythm. This rhythm is characterized by ventricular pacing, atrial undersensing, and atrial noncapture in the presence of normally functioning dual-chamber pacemakers.

4. Biventricular pacing system malfunction

Biventricular pacing system malfunctions can be categorized into two groups: 1) general biventricular pacing system malfunctions; 2) specific biventricular pacing system malfunctions.

4.1 General biventricular pacing system malfunction

This category includes all abnormalities previously discussed in the section of single- and dual-chamber pacemakers.

4.2 Specific biventricular pacing system malfunction

As the left ventricular (LV) lead is not as secure as the right ventricular (RV) lead, LV lead dislodgment is more common than RV lead. However detection of noncapture in the LV lead is very difficult because of the intact RV pacing. Baseline ECG recording after implant would be highly useful in this regard. Therefore, LV lead noncapture mainly present with heart failure decompensation.

5. Pseudomalfunctions

Pseudomalfunctions are defined as unexpected ECG findings that appear to result from pacemaker malfunction but that represent normal pacemaker function. Pseudomalfunctions should be ruled out as the cause(s) of an anomalous ECG strip before corrective measures are taken.

Pseudomalfunctions may be classified into three categories: 1) rate change, 2) anomalous AV interval or refractory period, and 3) mode change.

5.1 Rate change

Rate changes in the presence of normal pacemaker function may occur secondary to magnet operation, timing variations (A-A vs. V-V), upper rate behavior (Wenckebach or 2:1 block), electrical reset, PMT intervention, and rate response. Furthermore, hysteresis, rate drop response, mode switching, and sleep function have varying degrees of impact on the rate. *Magnet operation* varies within different products and from manufacturer to manufacturer, but will usually involve a rate change (increase or decrease depending on manufacturer) when the magnet is applied. *Wenckebach block* has the characteristic Wenckebach pattern of the PR interval gradually extending beat-to-beat until a ventricular beat is dropped. Wenckebach block occurs when the intrinsic atrial rate begins to exceed the upper rate limit. The ventricular response to the intrinsic atrial event cannot exceed the upper rate limit, so the AV interval is lengthened until the upper rate expires and a ventricular pace can be delivered. An atrial sensed event eventually falls into the refractory period and is not seen, and therefore not followed, by a ventricular pace. *Two to one block* is characterized by atrial rates that occur at intervals less than the total atrial refractory period (TARP). Every other P-wave falls into the refractory period and is therefore not preceded by a paced ventricular event. Patients who experience 2:1 block, particularly those who are active at the time the event occurs, will feel the precipitous drop in rate, which is cut in half. *Electrical reset* may occur secondary to exposure to EMI such as electrocautery, defibrillation, etc. These EMI sources can cause both rate and mode changes (back-up mode). As it was mentioned previously, *PMT intervention* is designed to terminate PMT. In Medtronic pacemakers, when PMT intervention is turned ON, the PVARP will be forced to 400 msec after the ninth paced ventricular event. By extending the PVARP, the intent is to interrupt atrial tracking for one cycle and break the PMT. After an intervention, PMT Intervention is automatically suspended for 90 seconds before the pacemaker can monitor for a PMT again. In *rate responsive pacemakers*, physical activity will cause an increase in the pacing rate. However, some patients may experience “false positive” increases in rate from their sensors. In the case of a piezoelectric crystal, the pacemaker may begin pacing at a faster rate if, for example, the patient is either lying on the side that the pacemaker is implanted on or experiencing a bumpy car ride. Similarly, minute ventilation sensors measure the change in respiration rate and tidal volume. If a patient experiences rapid respiration resulting from a cause other than exercise (e.g., hyperventilation), the pacemaker may begin pacing at a faster rate. *Hysteresis* provides the capability to maintain the patient’s intrinsic heart rhythm as long as possible, while providing back-up pacing if the intrinsic rhythm falls below the hysteresis rate. Because hysteresis exhibits longer intervals after sensed events, it may be perceived as oversensing. *Rate drop response* will exhibit pacing at high rates if the detection criteria are met. Rate drop response therapy prevents a precipitous decrease in the rate in patients experiencing cardioinhibitory neurally-mediated syncope. *Mode switching* is used to

prevent the tracking of the atrial signals in paroxysmal atrial tachycardia, atrial flutter, or atrial fibrillation in the DDD(R) and VDD(R) modes. Mode switching is associated both with rate slowing and mode change to DDI(R) or VVI(R). Sleep function is designed to provide a slower rate while patient sleeping.

5.2 Anomalous AV interval and refractory period

AV interval or refractory periods may appear anomalous due to safety pacing, blanking, rate-adaptive AV delay, sensor-varied PVARP, PVC response, and NCAP. *Safety pacing* is designed to prevent inhibition due to crosstalk. This algorithm delivers a ventricular output 110 ms after an atrial paced event. Therefore, safety pacing is manifested as abbreviated AV interval. *Blanking* is the first portion of the refractory period during which the pacemaker is “blind” to any activity. Blanking is designed to prevent multiple detection of a single paced or sensed event by the sense amplifier. However, if the blanking period is too long, it may not sense an event and cause inappropriate pacing. *Rate-adaptive AV delay* is designed to mimic the intrinsic response to increasing heart rate. In a normal heart, PR intervals decrease as the heart rate increases. Conversely, as the heart rate decreases, PR intervals increase. The rate-adaptive AV delay can be programmed to mimic the normal physiologic response of the PR interval to increasing heart rates. When *sensor-varied PVARP* is enabled, the duration of the PVARP will shorten as the rate increases. *PVC response* designates a PVC as a ventricular sensed event following a ventricular event with no intervening atrial event. When a PVC is detected, and PVC response is programmed ON, the PVARP is extended in order to avoid sensing the retrograde P-wave that could occur as a result of the PVC. Pacemaker-defined PVC will initiate a V-A interval. This extended PVARP (if PVC response is ON) and subsequent resetting of the timing interval may appear anomalous on an ECG. *NCAP* can be used in an effort to prevent atrial pacing from occurring too close to a refractory sensed event. If NCAP is programmed ON, the scheduled atrial output will be delayed until at least 300 msec has elapsed since the refractory sensed P wave occurred. PAV interval may then be shortened to maintain a stable ventricular rate.

5.3 Mode change

Change in pacing mode may occur secondary to electrical reset, mode switching, and noise revision. Electrical reset and mode switching have been described in the section of mode change. The portion of the refractory period after the blanking period ends is commonly called the “noise sampling period.” A sensed event in the noise sampling period will initiate a new refractory period and blanking period. Continuous refractory sensing is called *noise reversion* and will cause pacing to occur at the sensor-indicated rate for rate-responsive modes and cause pacing to occur at the lower rate for non-rate responsive modes.

6. Conclusions

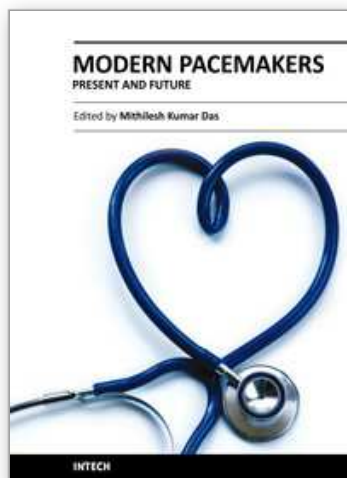
Pacing system malfunctions can be secondary to the pacemaker circuitry failure or to the lead dysfunction. Although primary malfunctions of the pulse generators do occur, they are least common cause of the malfunctions related to the pacing system. More commonly, problems are related to the damaged leads or primary abnormality at electrode-myocardial interface. Not uncommonly what appears to be pacemaker malfunction may actually represent normal functioning of the pacemaker.

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The book focuses upon clinical as well as engineering aspects of modern cardiac pacemakers. Modern pacemaker functions, implant techniques, various complications related to implant and complications during follow-up are covered. The issue of interaction between magnetic resonance imaging and pacemakers are well discussed. Chapters are also included discussing the role of pacemakers in congenital and acquired conduction disease. Apart from pacing for bradycardia, the role of pacemakers in cardiac resynchronization therapy has been an important aspect of management of advanced heart failure. The book provides an excellent overview of implantation techniques as well as benefits and limitations of cardiac resynchronization therapy. Pacemaker follow-up with remote monitoring is getting more and more acceptance in clinical practice; therefore, chapters related to various aspects of remote monitoring are also incorporated in the book. The current aspect of cardiac pacemaker physiology and role of cardiac ion channels, as well as the present and future of biopacemakers are included to glimpse into the future management of conduction system diseases. We have also included chapters regarding gut pacemakers as well as pacemaker mechanisms of neural networks. Therefore, the book covers the entire spectrum of modern pacemaker therapy including implant techniques, device related complications, interactions, limitations, and benefits (including the role of pacing role in heart failure), as well as future prospects of cardiac pacing.

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