The number of patients who have cardiac pacemakers has increased markedly over the past few decades since the technology was first introduced in the 1950s to prevent Stokes-Adams attacks. The American College of Cardiology and the American Heart Association’s Guidelines for Permanent Cardiac Pacemaker Implantation now lists atrioventricular (AV) node dysfunction, sinus node dysfunction, hypersensitive carotid sinus syndrome, and neurally-mediated syncope (vasovagal syncope), the prevention of tachycardia with long QT syndrome, and hypertrophic cardiomyopathy as indications for permanent cardiac pacing [1]. Recent literature expands the list to include select patients who have congestive heart failure and for the prevention of atrial fibrillation. Advances in technology, expanding indications, and the aging of the population ensure that clinicians will encounter patients with cardiac pacemakers on a regular basis. This article summarizes the electrocardiographic manifestations of the normally functioning permanent cardiac pacemaker, as well as abnormalities associated with pacemaker malfunction.

Pacing modes

As pacemakers have evolved and assumed more functions and capabilities, a five position code has been developed by the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG) to describe pacemaker function [2] (Table 1).

Position I indicates the chambers being paced, atrium (A), ventricle (V), both (D, dual), or none (O). Position II gives the location where the pacemaker senses native cardiac electrical activity (A, V, D, or O). Position III
<table>
<thead>
<tr>
<th>Position</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
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</thead>
<tbody>
<tr>
<td>Chamber(s) paced</td>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
</tr>
<tr>
<td>Chamber(s) sensed</td>
<td>A = atrium</td>
<td>A = atrium</td>
<td>T = triggered</td>
<td>P = simple programmable</td>
<td>P = pacing (antidysrhythmia)</td>
</tr>
<tr>
<td>Response to sensing</td>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td>I = inhibited</td>
<td>M = multiprogrammable</td>
<td>S = shock</td>
</tr>
<tr>
<td></td>
<td>D = dual (atrium and ventricle)</td>
<td>D = dual (atrium and ventricle)</td>
<td>D = dual (inhibited and triggered)</td>
<td>C = communicating</td>
<td>D = dual (pacing and shock)</td>
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<td>Programmability, rate modulation</td>
<td>R = rate modulation</td>
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indicates the pacemaker’s response to sensing—triggering (T), inhibition (I), both (D), or none (O). Older versions of the code only designated these three positions, and pacemakers still are commonly referred to in terms of these three codes. Position IV indicates two things: the programmability of the pacemaker and the capability to adaptively control rate (R). The code in this position is hierarchical. The C, which designates the ability to communicate with external equipment (ie, telemetry), thus is assumed to have multiprogrammable capability (M). Similarly, a pacemaker able to modulate rate of pacing (R) is assumed to be able to communicate (C) and be multiprogrammable (M). Position V identifies the presence of antitachydysrhythmia functions, including the antitachydysrhythmia pacing (P) or shocking (S). The code does not designate how these functions are activated or if they are activated automatically or manually by an external command.

For example, a VOOOO pacemaker is one capable of asynchronous ventricular pacing, with no sensing functions, no adaptive rate control functions, and no antitachydysrhythmia capability. A VVIPP pacemaker paces the ventricle, is inhibited in response to sensed ventricular activity, has simple programmability, and has antitachydysrhythmia-pacing capability. Similarly, a VVIMD pacemaker is a multiprogrammable VVI pacemaker with the ability to pace and shock in the setting of a tachydysrhythmia. A DDDCO pacemaker is a DDD pacemaker with telemetry capability but no antitachydysrhythmia function. From a practical standpoint, most pacemakers encountered in the emergency department or clinic setting are AAIR, VVIR, DDD, DDDR, or back-up pacing modes for cardioverter-defibrillator devices.

**Electrocardiographic findings in a normally functioning pacemaker**

When a pacemaker is active and pacing, small spikes that signify the electrical signal emanating from the pacemaker leads are usually evident on the electrocardiogram (ECG). These low-amplitude pacemaker artifacts may not be visible in all leads. Pacing artifacts are much smaller with bipolar electrode systems than with unipolar leads, and consequently may be difficult to visualize.

Typically, pacing leads used to pace the atrium are implanted in the appendage of the right atrium and leads to pace the ventricles toward the apex of the right ventricle. Atrial pacing appears as a small pacemaker spike just before the P wave. The P wave is usually of a normal morphology. In contrast, the ventricular paced rhythm (VPR) is abnormal (Fig. 1). Because the ventricular pacing lead is placed in the right ventricle, the ventricles contract from right to left, rather than by the regular conduction system. The overall QRS morphology thus is similar to that of a left bundle branch block (LBBB), with prolongation of the QRS interval. In leads V1–V6, the altered ventricular conduction is manifested by wide, mainly negative QS or rS
complexes with poor R-wave progression. QS complexes are seen commonly in leads II, III, and aVF, whereas a large R-wave typically is seen in leads I and aVL. Leads V5 and V6 sometimes have deep S-waves because the depolarization may be traveling away from the plane of those leads. Usually the ventricular lead is placed near the apex, causing the ventricles to contract from apex to base, yielding leftward deviation of the QRS axis on the ECG. If the lead is implanted toward the right ventricular outflow tract, depolarization forces travel from base to apex, resulting in a right axis deviation. Occasionally patients have epicardial rather than intracardiac pacemaker leads. If the ventricular epicardial lead is placed over the left ventricle, the ventricular paced pattern is that of a right bundle branch block.

ST segments and T waves typically should be discordant with the QRS complex, in contrast to the usual ECG pattern—meaning the major vector of the QRS complex is in a direction opposite that of the ST segment/T-wave complex. This is known as the rule of appropriate discordance or QRS complex/T-wave axis discordance for ventricular pacing. This becomes relevant when interpreting the electrocardiogram with VPR in the context of possible cardiac ischemia [3,4].

**AAI pacing**

An AAI pacemaker is one that.paces the atrium, senses the atrium, and inhibits the pacing activity if it senses spontaneous atrial activity (Fig. 2). This mode of pacing prevents the atrial rate from decreasing below a preset level and is useful for patients who have sinus node dysfunction and intact AV node conduction. The timing cycle of the pacemaker begins when it paces the atrium or senses an atrial event. Following initiation of the timing

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**Fig. 1.** DDD pacemaker with atrial and ventricular pacing. Low amplitude atrial and ventricular pacing spikes are best seen in lead V1 and II rhythm strips. The tracing demonstrates the widened QRS complexes typical in ventricular-paced rhythm with a left bundle branch block pattern, left axis deviation and ST segment/T-wave discordance with the QRS complex.
cycle, there is a refractory period in which the pacemaker is insensitive to stimuli. This prevents the pacemaker from sensing the proximate QRS complex and misinterpreting it as an atrial event. Once the preprogrammed refractory period is over, the pacemaker resets to its baseline status and the pacemaker remains silent for the duration of the programmed pacing interval. If, at the end of the interval no atrial activity is sensed, it generates a stimulus and the cycle begins anew. If, following the refractory period, it senses atrial activity, it inhibits itself from stimulating the atrium, and the sensed atrial activity initiates a new timing cycle.

**VVI pacing**

VVI pacing is useful in patients who have chronically ineffective atria, such as chronic atrial fibrillation or atrial flutter (Fig. 3). This mode is similar to the atrial demand (AAI) mode, except that the ventricle is sensed and paced, rather than the atrium. The refractory period is set at a shorter

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**Fig. 2.** AAI pacing. The first two P waves are paced (p), whereas the following three P waves are native and sensed, so atrial pacing is inhibited. The next three P waves are again paced. From Chan TC, et al., eds. ECG in Emergency Medicine and Acute Care. Philadelphia: Mosby, 2004; with permission.

**Fig. 3.** VVI pacing. The first, fourth, and seventh QRS complexes are native, whereas the others are paced (p marks third and fifth beats). Note that in VVI pacing there is no AV synchrony. From Chan TC, et al., eds. ECG in Emergency Medicine and Acute Care. Philadelphia: Mosby, 2004; with permission.
interval than in AAI pacing. Because the atrium is not sensed with VVI pacing, AV dissociation or dyssynchrony may occur in patients who have intact atrial function who suffer from AV node blockade, which has disadvantageous hemodynamic and clinical consequences.

**DDD pacing**

DDD pacing is a form of dual-chambered pacing in which the atria and the ventricles are paced. In DDD pacing the atrium and the ventricle are sensed and paced or inhibited, depending on the native cardiac activity.
sensed (Fig. 4). Other forms of dual-chambered pacing are available, such as DVI and VDD, but DDD is the most common. The principle advantage of dual-chambered pacing is that it preserves AV synchrony. Because of this advantage, dual-chambered pacing is increasingly common.

In DDD pacing, if the pacemaker does not sense any native atrial activity after a preset interval, it generates an atrial stimulus (Fig. 4A). An atrial stimulus, whether native or paced, initiates a period known as the AV interval. During the AV interval the atrial channel of the pacemaker is inactive, or refractory. At the end of the present AV interval, if no native ventricular activity is sensed by the ventricular channel, the pacemaker generates a ventricular stimulus (Fig. 4B, C). Following the AV interval, the atrial channel remains refractory during a short, post-ventricular atrial refractory period (PVARP) so as to prevent sensing the ventricular stimulus or resulting retrograde P waves as native atrial activity. The total atrial refractory period (TARP) is the sum of the AV interval and the PVARP. In a simple DDD pacemaker, the TARP determines the upper rate of the pacemaker (upper rate [beats per minute] = \( \frac{60}{\text{TARP}} \)).

Mode switching

As one might imagine, if a patient who has a DDD pacemaker were to develop supraventricular tachycardia, the pacemaker might pace the ventricles at the rapid rate based on the atrial stimulus (up to the preprogrammed upper rate limit). To prevent this, most DDD pacemakers now use mode switching algorithms, whereby if a patient develops an atrial tachydysrhythmia, the pacemaker switches to a pacing mode in which there is no atrial tracking, such as VVI. On cessation of the dysrhythmia, the pacemaker reverts to DDD mode, thus restoring AV synchrony without being complicit in the transmission of paroxysmal atrial tachydysrhythmias.

Electrocardiographic findings in the abnormally functioning pacemaker

Abnormal function of pacemakers can be life threatening to patients who are pacemaker-dependent. The 12-lead ECG is an indispensable part of the evaluation of pacemaker function. If there is no pacemaker activity on the ECG, the clinician should attempt to obtain a paced ECG by applying the magnet, which typically switches the pacemaker to asynchronous pacing (a small minority exhibit a different preprogrammed effect or no effect) (Fig. 5). This procedure is useful for assessing pacemaker capture (but not sensing), evaluating battery life, treating pacemaker-mediated tachycardia, and assessing pacemaker function when the native heart rate is greater than the pacing threshold. In the latter case, cautious attempts to slow the rate with maneuvers such as carotid massage, adenosine, or edrophonium administration also can be useful [5]. These should be performed with extreme caution in the pacemaker-dependent patient, however.
If routine evaluation yields no pacemaker abnormalities and pacemaker malfunction is suspected, the pacemaker should be interrogated by a company technician (this feature is available on most new pacemakers, those with code C or above in position IV of the NASPE/BPEG Generic Pacemaker Code). Many patients carry a card identifying the make and model of pacemaker. If this is not available, inspection of the pacemaker generator on chest radiographs may reveal useful information. Most manufacturers place an identification number in the generator that is visible on chest radiographs. Additionally, chest radiographs may reveal useful information, such as lead dislodgement, migration, or fracture. Causes of abnormal pacemaker function include failure to pace, failure to capture, undersensing, pacemaker-mediated dysrhythmias, pseudomalfunction, and the pacemaker syndrome.

**Failure to pace**

Pacemaker generator output failure, or failure to pace, occurs when the pacemaker fails to deliver a stimulus in a situation in which pacing should occur. Failure to pace has many causes, including oversensing, pacing lead problems, battery or component failure, and electromagnetic interference (such as from MRI scanning or cellular telephones). Failure to pace manifests on the ECG by an absence of pacemaker spikes at a point at which pacemaker spikes would be expected. In dual-chambered pacing symptoms, isolated atrial or ventricular failure to pace may be evident.

The most common cause of failure to pace is oversensing [6,7]. Oversensing refers to the inappropriate sensing of electrical signals by the pacemaker. Oversensing leads to failure to pace when the inappropriate sensing of electrical signals inhibits the pacemaker from pacing. These abnormal electrical signals may or may not be seen on the ECG.
The most common cause of oversensing is skeletal muscle myopotentials, particularly from the pectoralis and rectus abdominis muscles and the diaphragm [8,9]. In these cases the clinician may be able to reproduce the oversensing by running a 12-lead rhythm strip while having the patient stimulate the rectus and pectoralis muscles. Application of the magnet, which temporarily disables sensing functions, also may be useful. Oversensing caused by skeletal myopotential is almost exclusively a problem encountered in unipolar pacing systems, rather than bipolar pacing systems. Oversensing caused by skeletal myopotentials can be corrected by reprogramming the pacemaker to lower sensitivity or increasing the refractory period.

Oversensing of native cardiac signals also can occur. AV crosstalk occurs, for example, when an atrial sensing system inappropriately senses a QRS complex as native atrial activity. Such oversensing also can be corrected by reprogramming the pacemaker to lower sensitivity of the sensing system or increasing the refractory period. Make-break signals, which are electrical signals produced by intermittent metal-to-metal contact, also can lead to oversensing. Such signals can be caused by lead fracture, dislodgement, or loose connections within the pacemaker generator itself. These lead problems also may cause failure to pace by failing to deliver the pacing stimulus. Battery failure can cause failure to pace, as can primary pacemaker generator failure, although the latter is exceedingly rare. Blunt trauma to the pacing unit can cause failure to pace by damaging the pacemaker or its leads.

Failure to capture

Failure to capture refers to the condition in which a pacing stimulus is generated but fails to trigger myocardial depolarization (Figs. 6–8). On the ECG, failure to capture is identified by the presence of pacing spikes without associated myocardial depolarization, or capture.

Although low current from a failing battery may cause failure to capture as a result of insufficient voltage to trigger depolarization, the most common

Fig. 6. Failure of atrial capture. Atrial and ventricular pacing spikes are visible, but only the ventricular stimuli are capturing. There are no P waves following the atrial spikes (arrow). From Chan TC, et al., eds. ECG in Emergency Medicine and Acute Care. Philadelphia: Mosby, 2004; with permission.
cause of failure to capture is elevation in the threshold voltage required for myocardial depolarization, also known as exit block. Exit block can be caused by maturation of tissues at the electrode–myocardium interface in the weeks following implantation [10]. Tissue damage at the electrode–myocardium interface caused by external cardiac defibrillation is another well-known cause of exit block and failure to capture [11]. Some pacemakers are programmed to provide safety pacing with increased pacing output in the setting of abnormal pacemaker functioning or uncertain native activity (Fig. 8).

Fig. 7. Failure of ventricular capture. There is intermittent native atrial activity (a) and atrial pacing and capture (p) when no native activity is present. There is failure of ventricular capture, however, because no QRS complexes following the ventricular pacing spikes (arrow). The QRS complexes on this tracing are slow ventricular escape beats (v). In the fourth QRS complex, the pacemaker generates a stimulus at the same time a ventricular escape beat occurs, yielding a type of fusion beat (f). From Chan TC, et al., eds. ECG in Emergency Medicine and Acute Care. Philadelphia: Mosby, 2004; with permission.

Fig. 8. DDD pacing with intermittent loss of ventricular capture (arrow). After the third loss of capture event there is a junctional escape beat (J). In the next to last beat, a junctional escape beat is bracketed by two pacing spikes as a form of safety pacing. Rather than inhibiting ventricular pacing (and risk having no ventricular output if the sensed event were not truly a native ventricular depolarization), the AV interval is shortened and a paced output (S) occurs.
Undersensing

Undersensing occurs when the pacemaker fails to sense or detect native cardiac activity. At the time of implantation, the pacemaker is programmed to sense cardiac signals of a certain amplitude and frequency given the conditions particular to that individual lead. Anything that changes the amplitude, vector, or frequency of intracardiac electrical signals can result in undersensing. All of the causes for failure to capture also can cause undersensing, as can new bundle branch blocks, premature ventricular contractions (PVCs), or atrial or ventricular tachydysrhythmias. Most cases of undersensing can be remedied by programming the pacing system to a higher sensitivity.

Assessing for the presence of undersensing can be difficult (Figs. 9 and 10). Unlike failure to capture, there is often no obvious finding on ECG to suggest undersensing, because pacing artifacts may or may not be easily detectable. Instead the clinician must infer from the surface ECG whether the pacemaker is sensing properly and responding with an appropriate output based on its program. For example, in patients who have atrial undersensing, the ECG may reveal native P waves that are not being sensed, with resulting atrial pacing spikes (see Fig. 9).

Pacemaker-mediated dysrhythmias

Pacemakers can be useful to treat or prevent dysrhythmias. The pacemaker itself, however, can become a source of dysrhythmias. Examples include pacemaker-mediated tachycardia (PMT), runaway pacemaker, dysrhythmias caused by lead dislodgement, and sensor-induced tachycardias.

Pacemaker-mediated tachycardia

PMT, also known as endless-loop tachycardia, is a re-entry dysrhythmia that can occur in dual-chamber pacemakers with atrial sensing. In contrast

Fig. 9. Atrial undersensing. In this patient with a DDD pacemaker, the native atrial events (a) are not sensed. If atrial sensing were occurring, atrial pacing (e) would be inhibited. From Chan TC, et al., eds. ECG in Emergency Medicine and Acute Care. Philadelphia: Mosby, 2004; with permission.
to other re-entry dysrhythmias, in PMT the pacemaker itself acts as part of the re-entry circuit.

The atrial channel is programmed with a refractory period (PVARP) immediately after atrial depolarization to prevent sensing of the following ventricular QRS complex or the retrograde P wave that may result from ventricular depolarization. PMT occurs most commonly when a PVC occurs after the PVARP and the atrial channel interprets the resultant retrograde P wave as a native atrial stimulus, which in turn triggers ventricular pacing, which in turn allows the resultant retrograde P wave to again be sensed, and so on. The pacemaker itself acts as the antegrade conductor for the re-entrant rhythm with retrograde VA conduction completing the re-entry circuit loop. It is important to note that PMT cannot exceed the maximum programmed rate of the pacemaker, usually 160–180 bpm. Although a PVC is the most common initiating event, other factors, such as oversensing of skeletal myopotentials or the removal of an applied magnet, also can trigger PMT [12].

On the ECG, PMT appears as a regular, ventricular paced tachycardia at a rate at or less than the maximum upper rate of the pacemaker. Treatment of PMT consists in the application of the magnet, which turns off all sensing and returns the pacemaker to an asynchronous mode of pacing, thus breaking the re-entry circuit. If a magnet is unavailable, PMT can be terminated by achieving VA conduction block with adenosine or vagal maneuvers, which can prolong retrograde and antegrade conduction through the AV node [13–15]. Many modern pacemakers also feature programming to automatically terminate PMT by temporarily prolonging the PVARP or omitting a single ventricular stimulus (Fig. 11).

**Runaway pacemaker**

The runaway pacemaker is an exceedingly rare phenomenon and represents true primary component failure. The runaway pacemaker consists of
inappropriately rapid discharges at rates of up to 400 pulses per minute, potentially inducing ventricular tachycardia or fibrillation. This phenomenon generally is limited to older generation pacemakers; modern pacemakers have a preprogrammed upper rate limit, which the pacemaker cannot exceed. On the ECG the runaway pacemaker appears as a paced ventricular tachycardia, with a rate often exceeding the expected maximum upper limit of a pacemaker. The runaway pacemaker is a true medical emergency. Application of a magnet may induce a slower pacing rate, and emergency interrogation and reprogramming also may be successful. If this fails, emergent surgical intervention to disconnect or cut the leads is necessary [16].

Dysrhythmias caused by lead dislodgement

A lead that has become dislodged may bounce against the ventricular wall and provoke ventricular extrasystoles or dysrhythmias. Definitive treatment of such cases involves removal or repositioning of the lead.

Sensor-induced tachycardias

Many modern pacemakers are equipped with rate-modulation features that attempt to appropriately increase heart rate to meet physiological demands. This capability is designated by an R in the fourth position of the NASPE/BPEG generic pacemaker code. Examples of types of sensors include ones that respond to vibration, respiratory changes, hemodynamic parameters, and acid–base status. Pacemakers capable of rate-modulation may pace inappropriately if the sensors that regulate the pacemaker are stimulated by nonphysiologic parameters [17]. For example, pacemakers that control rate according to vibration sensors may pace erroneously if they are stimulated by loud noises, vibrations from the environment, or sleeping on the side of the implant [18–21]. Temperature sensors may trigger tachycardia in the febrile patient [22]. Sensors that control rate by monitoring
minute ventilation can be triggered by hyperventilation, arm movement, or electrocautery [23,24]. On the ECG, sensor-induced tachycardias appear as paced tachycardias. They are typically benign and cannot exceed the pacemaker’s upper rate limit. If needed, they can always be broken with application of the magnet, which returns the system to asynchronous pacing.

**Pseudomalfinction**

Pseudomalfinction occurs when pacing is actually occurring, but the pacing spikes are not seen. This may happen with bipolar pacing systems and analog ECG recorders, because the voltage in bipolar pacing systems is much smaller than unipolar systems. Pseudomalfinction also occurs when the clinician mistakenly expects the pacemaker to be triggering when it is appropriately inactive.

Rhythms that appear abnormal can occur even when the pacing system is functioning properly. In a DDD pacemaker, AV block rhythms can arise as the native sinus activity increases. As the sinus rate approaches the programmed upper rate limit, the duration of the cardiac cycle, or P-P interval, shortens and becomes less than the TARP. As a result, some native P waves fall within the TARP and go undetected, resulting most commonly in a 2:1 AV block (Fig. 12A).

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**Fig. 12.** Pseudomalfinction. AV block schematic for a normally functioning DDD pacemaker. (A) Sinus rate increases such that the native P-P interval is shorter than the TARP. Every other P wave occurs within the TARP and the paced QRS complex is dropped, resulting in a 2:1 AV block. (B) In this case, the native P-P interval is shorter than the preset upper rate limit interval (minimum cardiac cycle duration for maximum pacemaker rate), but still longer than the TARP. Atrial activity is detected by the pacemaker, but the pacemaker cannot release its ventricular stimulus faster than the upper rate limit resulting in a progressive lengthening of the PR interval until a paced ventricular beat is dropped (Wenckebach).
If the upper rate interval (the cardiac cycle length at the fastest pacemaker rate) is programmed to be longer than the TARP, increased sinus activity actually can lead to a form of Wenckebach rhythm in DDD pacing. This rhythm occurs when the native P-P interval shortens and is less than the upper rate interval but still longer than the TARP. This creates an AV delay as the pacemaker cannot release its ventricular stimulus faster than the upper rate limit allows despite occurring after the TARP. The AV delay increases with successive cycles until a dropped beat occurs, creating a pacemaker-mediated Wenckebach AV block (Fig. 12B). If the atrial rate continues to increase, the P-P interval becomes less than the TARP and a fixed block like that described previously occurs (Fig. 12A).

**Pacemaker syndrome**

Pacemaker syndrome is a constellation of signs and symptoms in patients who have otherwise normally functioning pacemaker systems resulting from suboptimal pacing modes or programming. The pathophysiology of pacemaker syndrome is complex and multifactorial, but the major factor seems to be suboptimal AV synchrony or AV dyssynchrony [25,26]. This leads to unfavorable hemodynamics and decreased perfusion.

Although the diagnosis cannot be made solely from the ECG, the lack of AV synchrony on an ECG in the appropriate clinical situation suggests the diagnosis. Retrograde P waves suggest VA conduction that in the context of AV dyssynchrony may cause atrial overload, part of the pacemaker syndrome. Additionally, the systolic blood pressure may decrease 20 mm Hg or more when the patient goes from a spontaneous native rhythm to a paced rhythm. Treatment consists in upgrading to dual-chambered pacing to restore AV synchrony.

**References**


