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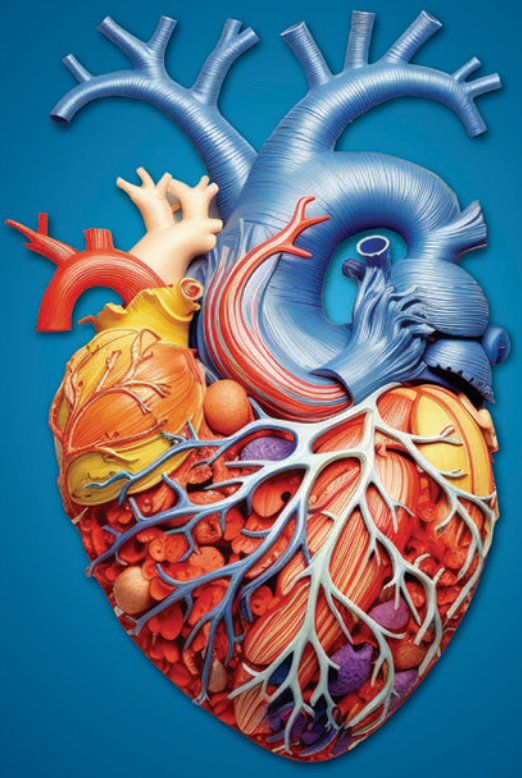

THE CARDIOLOGY COMPANION

A SOCRATIC APPROACH TO CLINICAL INSIGHTS

Prabir Kumar Das

3rd Edition

Foreword
Abdul Malik

JAYPEE

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CHAPTER

16 Cardiac Pacing (Including Implantable Cardioverter Defibrillator, Cardiac Resynchronization Therapy and EP Study)

"Pacemaker evolved primarily as a life saving device for patients with complete heart block, now used as an aid to alleviate symptoms in heart failure."
—History of Cardiology

INTRODUCTION

Pacemakers and other implantable devices for the management of cardiac arrhythmias have undergone tremendous growth and evolution since the introduction of transvenous pacing in the late 1950s. Advancement of electronics, computer technology, power source (i.e., battery), and miniaturization of pacemaker generators have contributed to the rapid development of the field of pacing. The use of implantable devices extends from bradycardia management to tachycardia (antitachycardia pacing) to management of sudden cardiac death (SCD) by implantable cardioverter defibrillator (ICD) and heart failure management by cardiac resynchronization therapy (CRT).

Q1. What is a pacemaker? What are the basic components of a pacemaker generator?

Ans. A pacemaker is a device that delivers battery-supplied electrical stimuli over leads with electrodes in contact with the heart.

Essential components of a pacemaker generator are:

- *Power source (battery):* Lithium-iodine is the chemical commonly used, having a longer end of life with more predictable behavior.
- *Circuitry:*
  - Output circuits—control programmable features of the pulse, including amplitude and pulse width
  - Sensing circuits—process the intracardiac electrocardiogram (ECG), including external electromagnetic interference (EMI)
  - Timing circuits—control the pacing intervals and sensing and refractory periods. They may be altered by input from the sensing circuits.
- *Others (present in some generators):*
  - Telemetry circuits—allow communication between an external programmer and the pulse generator for pacemaker programming and retrieval of information

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- Microprocessor—computer chips with memory. These allow downloading of new features by means of telemetry and increased storage for diagnostic data.
- Sensor circuits—for rate-responsive pacing

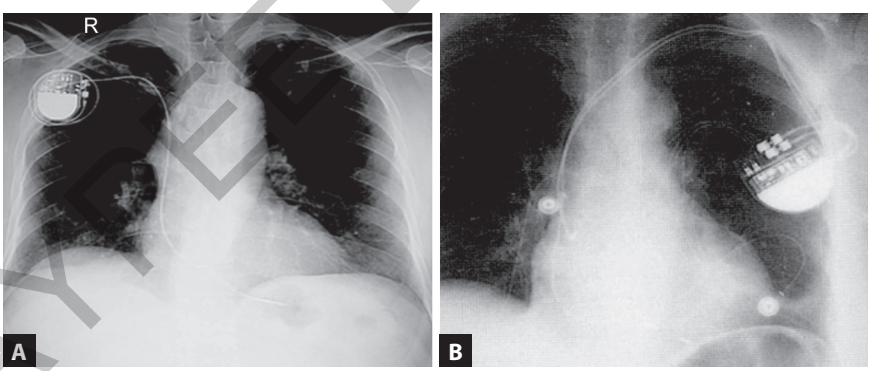
Q2. What are the types of pacemakers?

Ans. Categories of pacemaker varies according to its duration of pacing, polarity and chamber paced **(Box 16.1)**.

BOX 16.1: Types of pacemakers.

- According to the duration of pacemaker function—temporary and permanent
- According to the polarity of the pacemaker lead:
  - *Unipolar:* Leads have one terminal (–); the device can act as the positive terminal
  - *Bipolar:* Has two terminals at the lead tip; the current path is short, and the pacing signal is small on ECG. Extracardiac potentials are less likely to interfere with function
- According to the chamber paced:
  - Single-chamber pacemakers—carry a single lead within the RA or RV. Single-chamber atrial (e.g., AAI) pacemakers are used in sick sinus syndrome. Single-chamber ventricular (e.g., VVI) pacemakers are used in atrial fibrillation with symptomatic bradycardia **(Figs. 16.1A and B)**
  - Dual-chamber pacemakers–carry two leads in the RA or RV. This allows sequential pacing of atria and ventricles (e.g., DDD) or ventricular pacing in synchrony with the patient's intrinsic atrial rhythm in CHB (VDD mode)

(CHB: complete heart block; ECG: electrocardiogram; RA: right atrium; RV: right ventricle)



Figs. 16.1A and B: Single-chamber (VVI) pacemaker (A); dual-chamber (DDD) pacemaker (B).

Q3. How are pacemaker units coded?

Ans. Pacemaker units have been classified to enable operators to identify the capabilities of individual units. The three-letter code is now in general use:

First letter—relates to the chamber(s) paced:

O = none
A = atrium
V = ventricle
D = dual (A+V)

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Second letter—relates to the chamber(s) sensed:

O = none
A = atrium
V = ventricle
D = dual (A+V)

Third letter indicates the pacemaker's response to the sensed impulse:

O = none
T = triggered
I = inhibited
D = dual (T+I)

To cope with the facilities available on new programmable units, the three-letter code has been expanded to a five-code:

Fourth letter—relates to programmability and rate modulation:

O = none
P = simple programmable
in = multiprogrammable
C= communicating
R = rate-adaptive sensor

Fifth letter—relates to antitachyarrhythmia function:

O = none
P = pacing (antitachyarrhythmia)
S = shock
D = dual (P+S)

Q4. What are unipolar and bipolar pacemakers? Give their advantages and disadvantages?

Ans. Pacemaker may be unipolar or bipolar according to the electrode configuration of the pacing lead or the configuration of the pulse generator.

Unipolar: Cathode (negative) is on the lead tip, and the anode (positive) is the pacemaker can. It results in a large sensing antenna. A large pacemaker artifact (spikes) on the ECG is caused by the proximity of the circuit to the ECG electrodes.

Advantages: Better sensing of ventricular extrasystoles, low-amplitude signals, and shifted axis.

Disadvantages:

- Oversensing of external signals, especially myopotentials (i.e., pectoralis muscle activity)
- Skeletal muscle stimulation—twitching may occur
- Large pacemaker spikes on ECG may obscure native wave structure.

Bipolar: The electrodes are at the stimulating end of the lead—the cathode (negative) at the distal tip and the anode (positive) at the proximal ring.

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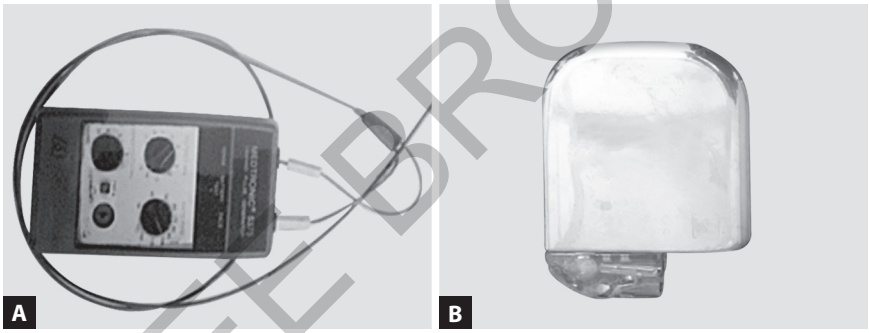
It results in a smaller sensing antenna. A smaller pacemaker artifact (spikes) on the ECG results.

Advantages:
- Less myopotential oversensing
- Less skeletal muscle stimulation
- Smaller pacemaker spikes on ECG do not obscure native wave structure.

Disadvantages:
- More complex lead design is more susceptible to malfunction and failure.
- Small pacemaker spikes on ECG may be difficult to see.

Q5. Compare and contrast between temporary and permanent pacemaker.

Ans. Both are electronic devices to treat symptomatic bradycardia producing hemodynamic compromise **(Figs. 16.2A and B)**. TPMs are external devices used for short periods of time. PPMs are internally implanted devices intended for long-term control **(Table 16.1)**.



Figs. 16.2A and B: TPM generator (A) and PPM generator (B).

TABLE 16.1: Comparison between temporary and permanent pacemaker generators.

Points	*TPM*	*PPM*
Generator type	Bipolar	Unipolar or bipolar
Location of the generator	External; portable	Internal, subcutaneous pocket in subclavicular location
Route and method of insertion	Femoral vein, subclavian, internal jugular, median basilic, by Seldinger technique	Cephalic or subclavian, axillary, basilic vein (sometimes)
Chamber paced	Single chamber—RV apex	Single or dual chamber
Duration of pacing	7–14 days	>10 years (in simple demand pacemaker)-depending of battery life*

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Points	*TPM*	*PPM*
Power source	Alkaline battery	Lithium-iodine battery
Reuse of the generator	Always done	Usually not done
Indications	As a bridge to PPM, especially for patients who are unable to undergo immediate PPM or for patients whose bradycardia is severe and hemodynamically unstable	Symptomatic bradyarrhythmias of long duration

*Depends on the current output required for capture, the requirement for incessant or intermittent pacing, and the number of chambers paced.

Q6. What are the indications for temporary pacing?

Ans.

- *In acute myocardial infarction (MI):*
  - Asystole and complete heart block (CHB)—in anterior or inferior infarction with hemodynamic instability
  - Second-degree atrioventricular (AV) block—Mobitz Type II AV block, usually in association with anterior infarction. It carries a high risk of developing complete AV block. Prophylactic pacing is done.
  - Bundle branch block—Patients with evidence of trifascicular disease* or nonadjacent bifascicular disease** complicating MI (should be prophylactically paced).
  - Others—symptomatic bradycardia (sinus pause >3.5 seconds) and transient asystole not responding to atropine
- As a bridge to permanent pacing in permanent symptomatic bradycardia, e.g., in CHB and sick sinus syndrome
- *During cardiac surgery:* Surgery adjacent to the AV node and bundle of His, e.g., aortic valve replacement (AVR) for calcific aortic stenosis (AS) (calcium extending into the site of the AV node). Tricuspid valve surgery in Ebstein's anomaly, ostium primum atrial septal defect (ASD), and AV canal defects, and corrected transposition of the great artery (TGA).
- Prophylactically—during right heart catheterization in patients with LBBB, electric cardioversion in patients with sick sinus syndrome
- Miscellaneous—drug overdose (e.g., digoxin, β-blocker, verapamil); termination of refractory tachyarrhythmias with overdrive pacing during electrophysiology (EP) study.

*Trifascicular disease: Right bundle branch block (RBBB) and left anterior hemiblock (LAHB) or left posterior hemiblock (LPHB) plus long PR interval, alternating RBBB and left bundle branch block (LBBB).

**Nonadjacent bifascicular disease: RBBB plus new LPHB.

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Point to ponder:
Right bundle branch block in anterior myocardial infarction-left anterior descending (MI-LAD) supplies both RBBB and the anterior superior division of left bundle branch (LBB). The onset of RBBB, especially with left-axis deviation, often precedes infranodal heart block. An abrupt transition can occur from 1:1 conduction to Mobitz Type II block or complete asystole. The lack of escape rhythm makes this particularly dangerous and warrants TPM even with only the onset of RBBB in anterior MI.

Q7. What are the indications of permanent pacing?
Ans. In general, permanent pacing is instituted for persistent or intermittent symptomatic bradycardia. It is indicated in long-standing conditions resulting from the failure of impulse formation and conduction.

- Acquired chronic complete AV block—symptomatic (Stokes-Adams attack) or with ventricular rate <40 beats/min, irrespective of symptoms (asymptomatic is unusual)
- *Mobitz Type II AV block:* Progressive, predictable, and may result in Stokes-Adams attack.
- Chronic bundle branch block—symptomatic patients with bifascicular block and patients with trifascicular disease (such as RBBB with alternating LAHB/LPHB, LBBB with alternating RBBB, or LBBB plus long PR interval)
- *Post-MI:* Persistent complete AV block or Mobitz type II AV block following anterior infarction. If such a block regresses during a hospital stay, subsequent Holter monitoring may help identify subjects at risk who need permanent pacing.
- Sick sinus syndrome—when it is documented in association with symptomatic bradycardia or symptoms that arise from drug-induced bradycardia (used to control tachyarrhythmias).
- Congenital complete AV block—in the presence of rate-related symptoms, failure of the AV node to respond to exercise, or a daytime ventricular rate <50 beats/min (carries a higher long-term risk of syncope or sudden death). Asymptomatic children may survive into adult life when permanent pacing is easier to insert.
- Carotid sinus hypersensitivity with recurrent syncope

Q8. What are the indications of epicardial lead placement in PPM?
Ans. Here, a pacing wire is attached to the epicardium with a thoracotomy or by subxiphoid route. The permanent unit is usually intra-abdominal (extraperitoneal below the rectus muscle). It is less reliable in the long term; wire displacement and fracture may occur due to kinking and vigorous movement.

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Epicardial lead placement is done:

- In infants weighing <7–8 kg, small children—where rapid growth makes transvenous pacing difficult
- When the chest is already open, i.e., in the course of a cardiac operation, when heart block develops during surgery
- When transvenous access cannot be obtained
- If endocardial lead placement cannot be achieved

Q9. What are the steps in PPM implantation?

Ans. The subclavian method is commonly used in PPM implantation. The subclavian vein passes over the first rib behind the medial third of the clavicle to join the internal jugular vein in forming the innominate vein. It lies just anterior to the subclavian artery and the apical pleura. The head turned to the right (for left-sided puncture). Ensure adequate hydration. The patient is not to inhale deeply with head down and do Valsalva maneuver during puncture **(Fig. 16.3)**.

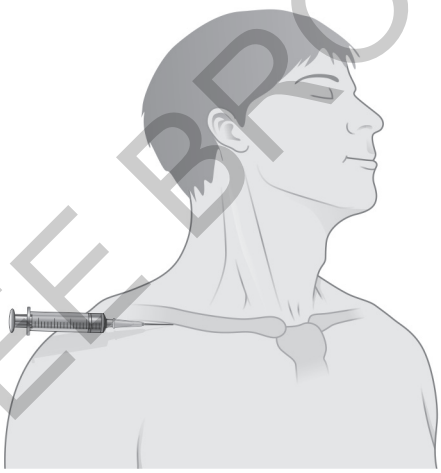

Fig. 16.3: Subclavian puncture for PPM implantation.

- Infiltrate 5–10 mL of 1% lignocaine into subcutaneous (SC) tissue 1–2 cm below the clavicle and two third distance from the sternoclavicular to the acromioclavicular joint; infiltrate toward the suprasternal notch.
- Another 5–10 mL of lignocaine 1% given SC along the line of the planned incision, the line of lead insertion, and in the area of the pacemaker pocket.
- Introduce the access needle just lateral to the midclavicular line at an angle of 15° both to the long axis of the clavicle and to the chest wall. Advance the needle slowly toward the suprasternal notch, keeping close to the underside of the clavicle (horizontally and nearly parallel to the

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clavicle) and above the first rib until the vein is entered at a depth of 4–5 cm. Vary the angle slightly if the vein is missed at the first pass.

- Flexible j-shaped guidewire is inserted (using Seldinger technique). The guidewire is left in situ and secured with the help of an artery forceps.
- Two separate punctures are done for dual-chamber pacemaker.
- Incision and pocket is made (by blunt dissection).
- Plastic sheath with a central dilator is fed over the guidewire and gently pushed to its full length into the vein to dilate it.
- Inner dilator is removed, leaving a port entry into the vein; the orifice is covered with fingertip to stop blood leakage.
- The lead is fed into the superior vena cava (SVC) immediately under fluoroscopy. The introducer sheath peeled out (after pulling it out of the vein) from the lead.
- Lead is now manipulated into the appropriate place. J-curve made at distal 3 cm of stylet (supplied with the pacemaker), fed into lead, and advanced into right atrium (RA).
- Lead advances until it passes through tricuspid and pulmonary valves.
- Stylet is taken out, and a soft, straight stylet is placed within 1–2 cm of the lead tip. Retract the lead slowly until it starts to fall toward the floor of the right ventricle (RV). Advance the lead smoothly until it reaches the RV apex. Leave enough curve on the lead that it does not straighten with deep breaths.
- In dual chamber pacing, the atrial lead (j-shaped) is positioned in the RA appendage (located anterosuperiorly)
- Lead is fed into the SVC/RA junction using a soft, straight stylet. Retract the stylet, and the lead should curl up slightly. Lead advanced until it slips into the RA appendage. It should "flick" left and right in AP view because of appendage contraction. The position is confirmed in the right anterior oblique (RAO) 45° view. Leave enough "belly" so that the lead does not straighten out or fall into the body of the RA.
- Active lead fixation is done by rotating the lead hub, braced over a stylet when passive lead fixation is unstable, the lead is displaced, or when the apical position gives inadequate electrogram or threshold values (lead thresholds are usually quite high immediately after placement owing to tissue injury, but fall over 5–10 minutes).
- Electrogram (atrial and ventricular), threshold, and lead impedance are tested to see lead stability. Pacing at 10 V ("Ten volt test") is done to exclude diaphragmatic or phrenic nerve pacing. The threshold is rechecked if lead movement is suspected during device positioning.
- Pocket is made medial to incision in plane of prepectoral fascia. The pocket should be large enough to accommodate the pacemaker and lead without allowing undue movement.

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- Leads home pushed and secure appropriate screw-on device header. Coil spare leads under the pacemaker and orient the device face-up.
- Pocket is closed using a layer of dissolvable sutures between the muscle and the opposing SC layer. One or two further layers of SC suturing are used to oppose skin edges. Skin closure is ideally done using subcuticular dissolvable sutures (to give a good cosmetic result).

Q10. What is the programmability of pacemakers?

Ans. It is the noninvasive, stable, reversible change in the operating parameters of an implanted pacemaker generator. Programmability is the means by which the cardiologist chooses the particular type of pacing program that is most suitable for an individual patient. Pacemaker programming is accomplished by activation of the programming head positioned over the implanted pulse generator after making the desired changes in programmable parameters. When three or more functions are altered, the system is called multiprogrammable. A handheld programmer is necessary.

These are:

- Pacing rate
- Energy output (with an adequate safety margin, exceeding the threshold voltage)
- Sensitivity (with an adequate safety margin, exceeding the threshold voltage for detecting sensed ECGs)
- *Hysteresis:* It refers to the escape interval from the sensed beat to the next paced beat. It is longer than the pacing rate, e.g., 50–70 hysteresis indicates that pacing at a rate of 70 beats/min will occur when the sinus rate falls below 60 beats/min; the sinus rate will emerge when the rate exceeds 70/min. It is particularly useful in sick sinus syndrome with a sinus rate marginally slower than the pacing rate. It is not desirable in atrial fibrillation (AF).
- Lead polarity—unipolar may be converted to bipolar (immune to external signals)
- Others—refractory period, AV delay, etc.

Advantages of programmability: (1) Selection of the most appropriate pacing parameter or mode; (2) Noninvasive diagnosis and correction of pacing malfunctions; and (3) Extension of pulse generator longevity by reduction of energy output.

Q11. What do you mean by sensing failure and oversensing?

Ans. Sensing failure means the pacemaker fails to notice an intrinsic cardiac impulse; hence, it is not inhibited. This is because the pacing unit is too insensitive or the R wave of the intrinsic ECG is too small or the slew rate is too slow. In a programmable pacing unit, the sensitivity may be increased, e.g., R wave sensitivity increased from 2 to 10 mV. A porous tip electrode may offer better sensitivity **(Fig. 16.4)**.

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Fig. 16.4: Undersensing in VVI mode (pacemaker tries to pace because it has not sensed the intrinsic rhythm).

In oversensing, the pacemaker is inhibited by electrical signals other than the R wave, e.g., physiological myopotential signals from skeletal muscle (usually pectoral) or nonphysiological EMI, e.g., leak from microwave ovens, etc. In this setting, the pacemaker must be reprogrammed to a lower level **(Fig. 16.5)**.

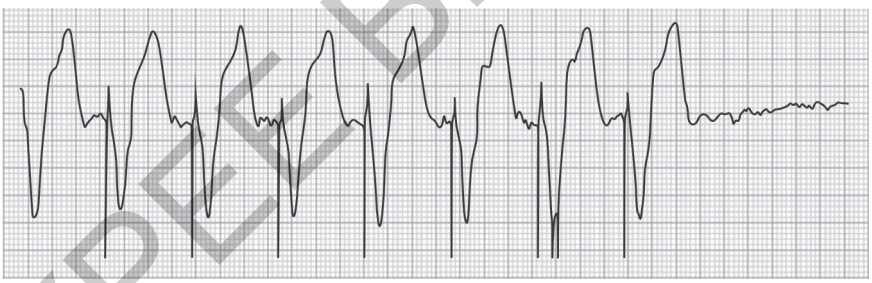

Fig. 16.5: Oversensing in VVI mode (pacemaker inhibited when it should pace).

Q12. What is pacemaker syndrome and pacemaker-mediated tachycardia (PMT)?

Ans. Pacemaker syndrome is a sign and symptom caused by inadequate timing of atrial and ventricular contractions. It is seen with VVI pacing because of an abnormal wave of depolarization. The retrograde ventriculoatrial (VA) conductions cause atrial contraction against closed mitral and tricuspid valves. The resulting low cardiac output (COP) and blood pressure (BP) produce vertigo, lightheadedness, and occasional syncope. This problem can be rectified by pacing the atrium prior to the ventricle using DVI or appropriately programmed DDD pacing. The problem can be exaggerated with VVIR pacing.

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Pacemaker-mediated tachycardia—wide complex (paced) tachycardias sustained by the continued active participation of the pacemaker. PMT may appear, at first glance, to be ventricular tachycardia (VT), especially for pacemakers with bipolar leads. The pacing artifact may be difficult to discern on an ECG tracing.

PMT may be:

- When a dual chamber pacemaker attempts to track the rapid atrial rate during an atrial tachyarrhythmia, producing rapid ventricular pacing
- When oversensing occurs in the atrial channel, such as myopotential
- Endless loop tachycardia (ELT) occurs when a repetitive sequence of sensing of retrograde atrial activity triggers a ventricular pace beat.

Q13. Give the various indications of commonly used pacemaker units?
Ans.

- *VVI:* Here spontaneous QRS potentials are sensed by the pacemaker, and subsequent pacing stimulus is inhibited. A pacing stimulus then occurs at a set rate unless further spontaneous QRS potentials fall within that period **(Fig. 16.6)**. Devices are cheap. VVIR devices are suitable for those with AF (heart rate responds to activity and exercise).



Fig. 16.6: ECG tracing in VVI mode.

It is the unit of choice in patients with AV block and AF, and sick sinus syndrome with atrial paralysis. It may be used in symptomatic bradycardia. Patients with AV block and persistent sinus node function will lose atrial contribution to ventricular filling as the atrium often contracts against closed AV valves. Retrograde VA conduction complicates the problem and produces pacemaker syndrome. Programmable VVI units may partly overcome this by being programmed to a lower rate or with hysteresis. VVI pacing in sinus rhythm may also induce AF by retrograde atrial contraction.

- *DVI (AV sequential pacing):* Both atrium and ventricles are paced, but only spontaneous ventricular activity is sensed **(Fig. 16.7)**. Spontaneous atrial

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activity is ignored. After a spontaneous ventricular impulse is sensed, the unit resets to one V-A interval and fires an atrial impulse followed by a ventricular impulse. Spontaneous P wave occurring within this V-A interval is ignored.



Fig. 16.7: ECG recording in DVI mode.

It can be used in complete AV block, pacemaker syndrome (atrial pacing prior to ventricular pacing prevents retrograde conduction), and sinus bradycardia. It cannot be used in AF.

- *VDD (P-synchronous pacing):* Single ventricular lead but also has sensing electrodes in RA to allow tracking atrial rhythm. Implantation is simpler than dual-chamber devices. P wave sensing results in ventricular pacing after an appropriate AV delay. If the atrial rate becomes very rapid, a specific pacemaker protects the ventricle from a rapid-paced rate. If a spontaneous P wave does not appear, the generator paces the ventricles on demand at its programmed rate. Spontaneous ventricular impulses inhibit the pacemaker, when it is reset to fire after a standby period **(Fig. 16.8)**.

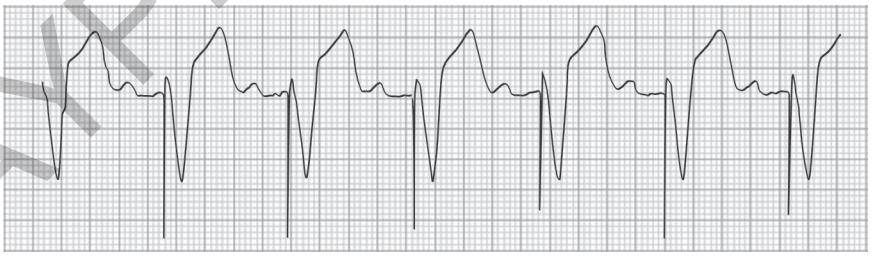

Fig. 16.8: ECG tracing in VDD mode: Atrial rhythm is sinus. After AV delay (programmed), a ventricular stimulus is delivered. Thus, the ventricular paced rate is the same as the sinus rate.

VDD is excellent for CHB in the presence of normal sinus node function. If AF develops, the pacemaker reverts to VVI mode (also occurs if the spontaneous atrial rate falls before the escape rate).

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Disadvantage: Absence of atrial backup pacing in the event of sinus bradycardia. PMT may occur in the presence of retrograde conduction.

- *DDD:* Fully automated unit that paces and senses both the atrial and ventricular on demand. During sinus bradycardia, atrial (and, if necessary, ventricular) pacing occurs. During AV block, ventricular pacing occurs following either a spontaneous or a paced atrial impulse. Thus, ventricular pacing can occur in sequence with sinus activity (tracking), and normal rate response to exercise is maintained despite AV block **(Fig. 16.9)**.

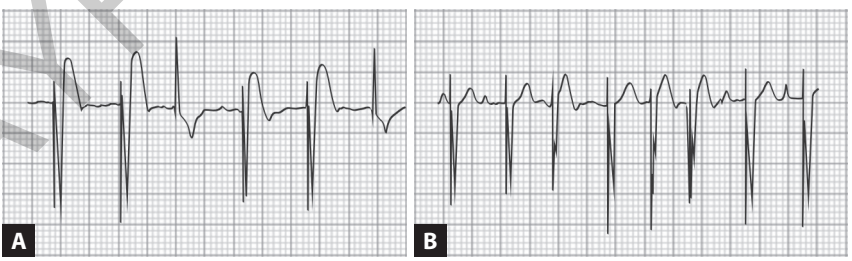


Fig. 16.9: DDD mode (ventricular pacing is triggered by sensed atrial activity).

DDDR devices are available for patients with chronotropic incompetence. It can be used in AV block with intact sinus node function and in sick sinus syndrome with additional AV node disease (an advance on the VDD). If AF develops, it also reverts to VVI mode (mode switching).

Q14. What is the difference between inhibited and triggered pacing?

Ans. Inhibition and triggering are the modes of response of the pacemaker unit to the sensed impulse. Inhibited mode is the demand pacing system. Pacing is inhibited by the sensed spontaneous cardiac impulse (atrial or ventricular). The pacemaker fires a stimulus only after a preset interval if no further impulse is sensed **(Figs. 16.10A and B)**.



Figs. 16.10Aand B: Ventricular inhibited pacing (A) and ventricular triggered pacing (B).

In triggered mode, a sensed spontaneous R wave results in an immediate pacing stimulus fired into the R wave (the heart is refractory and obviously not paced).

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Ventricular triggered pacing may be used:
- To avoid electromyography (EMG) inhibition
- When a temporary wire is inserted to cover a failing permanent

Q15. What do you mean by physiological pacing? How is it achieved?
Ans. Utilization of atrial systole to increase left ventricle (LV) end-diastolic volume and COP by either sensing and pacing in synchrony with ventricular pacing is called physiological pacing. Such pacing may improve COP in patients with borderline LV function. In sick sinus syndrome, it may prevent stagnation of blood in a flaccid left atrium, thus avoiding systemic embolism.

It may be achieved by establishing cardiac synchrony in the following ways:
- Atrioventricular sequential pacing—pacing of the atrium followed at a preset interval by pacing of the ventricle. This allows physiologic atrial transport into the LV as in DVI pacing.
- Rate-responsive pacing—via sinus node conduction, i.e., depends on the sinus node as the physiological sensor. Sinus rate is altered according to hemodynamic requirement, and because of AV synchrony, ventricular pacing rate alters accordingly. Example: VDD and DDD.

Physiological pacing system has limitations and cannot be strictly physiological at high heart rates. Some of the limitations are: More expense, shorter battery life, complexity of the unit and programming equipment, occasional pacemaker-medicated tachycardia, and limitations of pacemaker potential on disease progression, e.g., development of sick sinus syndrome in VDD and AF in DDD (should be converted to VVI by programming or automatic mode switching).

Q16. How do environmental factors influence pacemaker function?
Ans.
- *Cellular telephones:* These may interfere with cardiac pacemakers while transmitting or receiving calls. Patients with pacemakers are advised not to carry cellular telephones near the pacemaker site (e.g., shirt pocket) and to hold the telephone to the contralateral arm during use.
- *Electronic article surveillance:* The electromagnetic field may cause pacemaker interference, primarily inhibition of pacemaker output. Patients with unipolar dual-chamber pacemakers are particularly susceptible.
- *High-voltage power lines and electric substations:* These may cause inhibition or asynchronous pacing in a unipolar pacemaker if the patient is quite close to the electric field.
- Metal detectors may be set off because of the detection of a pacemaker; there is generally no appreciable interference with pacemaker function.
- *Magnetic resonance imaging (MRI):* The magnetic force may close the pacemaker and cause asynchronous pacing. One radiofrequency signal

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of MRI may inhibit pacing, cause rapid pacing, or cause reversion to reset mode. Unipolar pacemakers are more susceptible to interference from MRI. In general, MRI is avoided in patients with pacemakers.

- *Cardioversion or defibrillation:* The shock may damage the pulse generator or cause the device to be reset. If necessary, the patch electrodes are positioned as far from the pulse generator as possible. A pacemaker evaluation is performed after the procedure.
- *Diathermy:* It may cause pacemaker interference or damage if applied to the region near the pulse generator.
- *Dental equipment:* Some types may cause pacemaker inhibition, particularly for unipolar pacemakers. Vibrations may increase the pacing rate of activity-sensing, rate-adaptive pacemakers.
- *Transcutaneous electric nerve stimulation (TENS):* It is safe for patients with bipolar pacemakers. Patients with a unipolar pacemaker may need a reduction in sensitivity.
- *Electrocautery:* It may cause temporary inhibition of pacemaker output because of oversensing of EMI; electrocautery is used sparingly and in short bursts. The cautery electrode is placed at a distance from the pacemaker site.
- *Extracorporeal shock wave lithotripsy (ESWL):* The shock waves can interfere with or damage a pacemaker. Hence, the pulse generator must be as far as possible from the focal point of the lithotripsy shock waves. Activity-based rate-responsive pacemakers with piezoelectric crystals may be damaged, and the shock wave may cause oversensing and subsequent nonphysiologic rapid pacing rates. Such pacemakers are reprogrammed with the rate-adaptive features deactivated before the procedure. Shock waves may be misinterpreted as atrial activity; therefore, dual-chamber pacemakers are programmed to VVI mode to avoid rapid ventricular pacing.

Q17. What are the indications of pacemaker battery failure? How can battery life be increased?

Ans. The end of life of most pacemaker batteries is indicated by:

- Slowing of the basic pacing rate
- Increasing pulse width/pulse duration
- Decreasing output voltage

Battery life may be increased by: Avoiding low lead impedance with a large electrode tip, avoiding wide pulse width*, avoiding fast pacing rate and constant pacemaker use, and avoiding complex circuitry. Generally, the more complex the pacemaker, the shorter the expected battery life. If a pacemaker with hysteresis mode is available, the takeover rate may be set lower than the basic pacing rate, conserving battery life. A pacemaker may have its battery life prolonged by reducing rate, pulse width, and output. However, reducing

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pulse width, and/or output voltage should not be performed until enough time has elapsed from implantation to allow for the establishment of the chronic threshold (e.g., 3 months).

Q18. How does a magnet help?
Ans.

- *Diagnostic:* Placing a magnet over the pacemaker units converts them to a faster fixed-rate mode (VOO). This is done to test battery life and satisfactory pacing if there is competition at a slower demand rate. Failure pacemaker stimulus output may produce a pause during a paced rhythm. If the pause resolves with the application of the magnet, the diagnosis of oversensing is most likely. If the pause does not resolve, one of the other causes is considered.
- *Therapeutic:* If a patient is at frequent risk from external interference, he or she may use a magnet to switch the pacemaker to fixed-rate mode, during which it is immune to external signals.

ICD, CRT, EP STUDY

These are special pacing devices used for the management of life-threatening arrhythmias and heart failure. Because of the inability of antiarrhythmic agents to control life-threatening arrhythmias, ICD is being introduced. CRT is designed to bring synchrony in cardiac contraction in heart failure by pacing both ventricles. Often ICD and CRT are combined, i.e., combo device.

Q1. What are the electrotherapies for cardiac arrhythmias?
Ans.

- Direct current electrical cardioversion
- Implantable electrical device, i.e., ICD—monitor rhythm, deliver pacing stimuli, and low- and high-energy shock
- Ablation therapy—destroy myocardial tissue by delivering electrical energy through electrodes on a catheter placed next to an area of the myocardium integrally related to the onset or maintenance of arrhythmia or both.

Q2. What is an ICD? What are the components?
Ans. It is an implantable device that detects life-threatening arrhythmias as well as delivers defibrillating shock to combat that arrhythmia, thus preventing SCD. In light of the evidence for inefficiency and hazards of antiarrhythmic agents for the prevention of SCD, ICD is now a preferred therapeutic option.

Components of ICD are **(Fig. 16.11)**:

- Implantable cardioverter defibrillator box containing a battery and capacitors to store energy to deliver a defibrillation shock

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- Ventricular lead and atrial paced-sense lead contain shocking coil and a bipolar pacing electrode at the tip. These sense electrogram and deliver low voltage pulses to pace the heart if the rate is too low(anti-bradycardia pacing) or overdrive, if the rate is too fast, e.g., VT (antitachycardia pacing)
- Microprocessors—contains an algorithm to interpret the sensed electrograms to: (1) Detect tachycardia, (2) discriminate VT/ventricular fibrillation (VF) from nonlife-threatening arrhythmias, e.g., AF, and (3) deliver energy. Some of these are programmable.

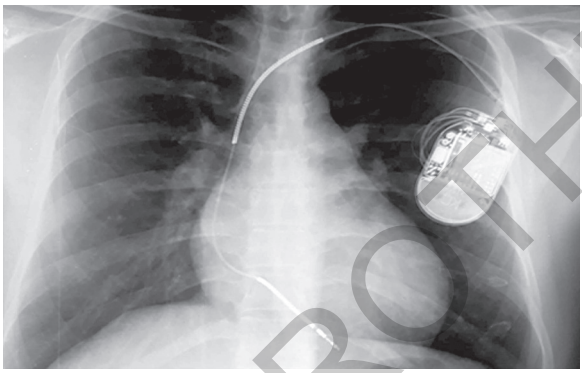

Fig. 16.11: ICD with shocking coil and generator in situ.

Q3. How does an ICD device work?

Ans. An ICD device works by:

- Delivering a defibrillating shock
- Bradycardia pacing (single and dual chamber) and rate-adaptive option
- Antitachycardia pacing (ATP)—terminates a rhythm disturbance without the delivery of a shock
- Incorporating resynchronization therapy, i.e., cardiac resynchronization therapy with defibrillator (CRT-D).

Q4. Enumerate some cardiac conditions where an ICD is beneficial.

Ans.

- Survivors of SCD secondary to VT or VF, where all reversible factors are excluded (secondary prevention)
- Sustained VT and significant structural heart disease, e.g., ischemic cardiomyopathy and dilated cardiomyopathy
- Syncope of undetermined origin and VT or VF inducible on EP study (secondary prevention)
- Sustained VT with reasonable LV function (primary prevention)
- Previous MI with poor LV function [ejection fraction (EF ≤35%)] and inducible VT on EP study (primary prevention)
- Long QT syndrome and Brugada syndrome with history of VT or syncope

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- Hypertrophic cardiomyopathy (HCM) or arrhythmogenic right ventricular cardiomyopathy with one or more risk factors for SCD.

Q5. What are the indications of ICD?

Ans. Implantable cardioverter defibrillator is indicated for patients with heart failure with reduced ejection fraction (HFrEF) who are at increased risk of ventricular arrhythmia and SCD. It is indicated for both primary and secondary prevention of SCD.

- *Primary prevention:* (1) Both ischemic and nonischemic cardiomyopathy and EF ≤35% with New York Heart Association (NYHA) class II or III symptoms on guideline-recommended therapy (GRT) for at least 3 months. (2) Patients who are at least 40 days post-MI and with EF ≤35% and NYHA class II symptoms or more while on GRT.
- *Secondary prevention:* For survivors of cardiac arrest and patients with sustained ventricular arrhythmia, irrespective of left ventricular ejection fraction (LVEF)

Q6. What are the complications unique to ICDs?

Ans. Besides the complications associated with the placement of PPM, complications unique to ICD are:

- Failure to deliver appropriate energy when indicated
- Delivery of inappropriate therapy

Q7.What are the limitations of ICD implantation?

Ans.

- Implantable cardioverter defibrillator is only offered to patients who have a reasonable expectation of survival beyond a year in the absence of frailty and/or advanced comorbid conditions.
- ICD shocks (appropriate or not) can lead to anxiety and even post-traumatic stress disorder. Hence, the decision of ICD implantation should be judiciously taken.
- ICD is not a disease-modifying therapy; heart failure progresses even with ICD in situ. Deactivation may be needed at a point when it becomes harmful rather than beneficial.
- ICD should not be implanted in patients with NYHA class IV symptoms who are candidates for LVAD or cardiac transplantation.

Q8. What are CRT, CRT-P, and CRT-D?

Ans. Cardiac resynchronization therapy is a biventricular pacing device for heart failure patients to bring the contraction and relaxation of ventricles into a coordinated manner, thus increasing COP and reducing heart failure symptoms. Patients with heart failure and LBBB exhibit dyssynchrony, i.e., uncoordinated contraction and relaxation of ventricles, because of delayed and heterogeneous depolarization of the LV. By pacing LV and RV simultaneously **(Fig. 16.12)**, CRT improves LVEF and reduces ventricular

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dimensions (reverse remodeling). CRT has been shown to improve quality of life, exercise capacity, and EF in patients with advanced congestive heart failure (CHF). As this is achieved by biventricular pacing, it is also called CRT-P. When defibrillation facilities, such as ICD, are incorporated into CRT, it is called CRT-D (often leveled as a combo device).

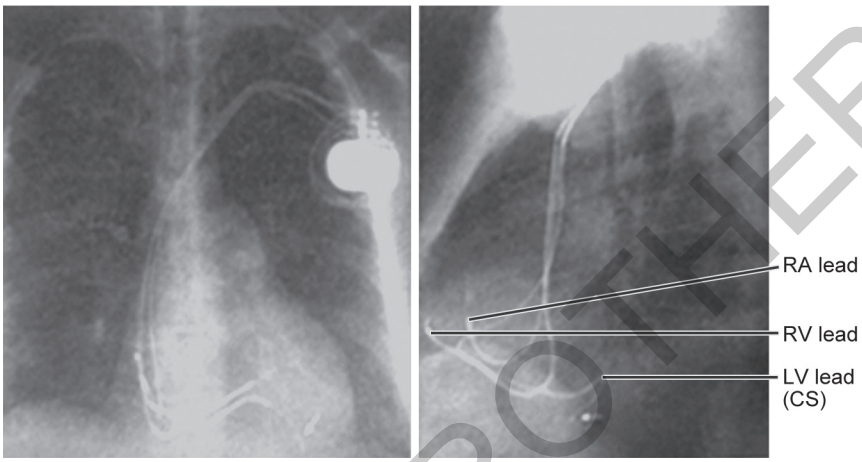

Fig. 16.12: CRT-D chest radiograph showing leads in RA, RV, and LV (the LV lead is placed in the posterolateral branch of the coronary sinus). (CRT-D: cardiac resynchronization therapy defibrillator; CS: coronary sinus; LV: left ventricle; RA: right atrium; RV: right ventricle)

Q9. How does CRT work?

Ans. The device paces the LV and the RV simultaneously, thus improving LVEF and reducing death and hospital admissions in heart failure.

Q10. What are the indications of CRT?

Ans. It is recommended for patients in normal sinus rhythm with LVEF ≤35% and an LBBB with QRS of ≥120 ms. American Heart Association, American College of Cardiology, and Heart Rhythm Society (AHA/ACC/HRS) guidelines (2008) recommend that CRT is indicated in patients with NYHA class III–IV heart failure established on optimal medical therapy, QRS duration >120 ms, LVEF ≤35%, and those in sinus rhythm.

It may also be beneficial in heart failure patients with NYHA class III–IV established on optimal medical therapy, AF with QRS duration >120 ms, and LVEF ≤35%. Also NYHA class III–IV patients and LVEF ≤35% who require frequent RV pacing.

Q11. What is EP study? Give its importance.

Ans. It is the method of recording and/or stimulating cardiac electrical activities by introducing multipolar catheter electrodes and positioning them at various intracardiac sites. An EP study is often combined with catheter ablation to treat the identified arrhythmia.

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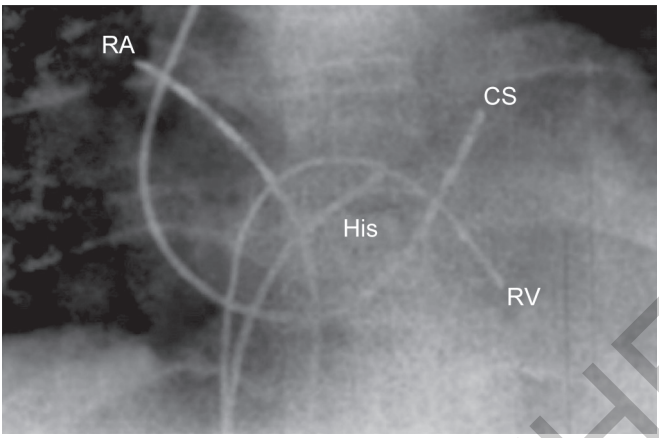

Fig. 16.13: EP study and SVT ablation—fluoroscopic image showing quadripolar catheters in high RA, RV, His bundle, and a decapolar catheter in high RA, RV, His bundle and a decapolar catheter in coronary sinus.

The importance of EP studies is:

- Diagnostic—type of clinical rhythm disturbances and its electrophysiologic mechanism
- Therapeutic—to terminate a tachycardia by electrical stimulation or electroshock, to evaluate the effect of therapy by determining whether a particular intervention modifies or prevents electrical induction of a tachycardia or an induced tachyarrhythmia, and to allocate myocardium involved in the tachycardia to prevent further episodes.
- Prognostic—to identify patients at risk of SCD

Q12. What is catheter ablation? When is it indicated?

Ans. It is the catheter-based antiarrhythmic therapy achieved by ablating or damaging the tissue responsible for arrhythmia by application of radiofrequency energy. Catheter placed in RA, RV, near His bundle, and in coronary sinus via femoral vein **(Fig. 16.13)**. The radio frequency (RF) energy creates a burn by resistively heating the tissue. A cryocatheter that freezes the tissues can also be used for ablation. Acoustic and laser-based ablation systems are also evaluated. For a successful ablation, accurate diagnosis of arrhythmia, mapping to determine the precise location of tissue causing arrhythmia, placement of ablation catheter on site, and application of effective RF energy are needed. Ablation lesion heals by fibrosis. For many arrhythmias, the risks are sufficiently low, and catheter ablation has become an important common therapy.

Indications of catheter ablation:

- Supraventricular tachycardias (SVTs)—AV nodal re-entrant tachycardia (AVNRT), AV reciprocating tachycardias using an accessory pathway,

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focal atrial tachycardia, inappropriate sinus tachycardia (modifying S.A. node function), atrial flutter, etc.

- Atrial fibrillation—AV junction ablation (for rate control) and atrial ablation (for maintaining sinus rhythm)
- Ventricular tachycardia—idiopathic focal VT, idiopathic interfascicular re-entrant tachycardia, VT after MI, and bundle branch reentrant VT

Q13. What is myocardial mapping?

Ans. It is the method of recording potentials directly from the heart that is spatially depicted as a function of time in an integrated manner. The mapping system displays the location of the catheter and a three-dimensional representation of the anatomic as well as electrophysiologic character of the tissue. It can display continuous catheter position without the need for fluoroscopy, thus reducing radiation exposure. Integration of cardiac MRI, computed tomography (CT), and echocardiography is needed when extensive ablation is required over complex anatomy, e.g., extensive left atrial ablation in AF. Direct cardiac mapping by catheter electrodes can be used to identify and localize the areas responsible for rhythm disturbances in patients with SVT and VT for catheter or surgical ablation or resection.

Indications are: Wolff–Parkinson–White (WPW) syndrome with accessory pathways, AV node and His bundle ablation in AV node re-entry, and the site of origin of VTs

Q14. What are the indications of an EP study?

Ans.

- Atrioventricular block—to determine the site of block (which cannot be determined by routine ECG), patients with PPMs having symptoms
- Intraventricular conduction disturbances—an HV interval >35 ms raises the possibility of trifascicular block, and an HV interval >80–90 ms indicates an increased risk of AV block.
- Sinus node dysfunction—symptoms attributable to bradycardia or asystole (presyncope or syncope) for whom noninvasive tests provide no explanation.
- Tachycardia—to differentiate aberrant supraventricular conduction from VT
- Unexplained syncope
- Palpitation.

SUGGESTED READING

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