



Pacemaker Follow Up Guidelines for Physicians

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Abstract

Use of pacemakers has become fairly common even in developing country like India. Implantation of appropriate pacemakers, their optimal programming to exploit the various available function and their trouble shooting requires advanced training and should preferably be carried out by specifically trained cardiologists or cardiac electrophysiologists. However internists and general physicians should familiarize themselves with certain basic functions of pacemaker, and must acquire the knowledge needed to follow up the patients with pacemakers. They also should be able to assess the patients and ascertain whether the particular patient needs to be referred to the trained cardiologist for further evaluation or replacement of pacemaker. This article is aimed at providing such information to the noncardiologist physicians. ©

INTRODUCTION

Implantation of pacemakers has become fairly common even in developing country like India.

Every recipient of a pacemaker needs to be monitored for the span of his/her life. The purpose of such continued surveillance includes

- Ensuring optimal functioning of the device and exploiting the full capabilities of the device to improve patients well being.
- To detect, diagnose and correct any abnormalities that arise in the pacing system
- To improve longevity of the device by appropriate programming.
- To assess and predict the elective replacement time of the pulse generator
- To appropriately manage various situations arising in patient's lifetime that may interfere with functioning of the pacing system.
- To educate and reassure the patient and the family
- To generate database of pacing systems and to train medical personnel.

Evaluation of a patient with pacemaker has been transformed from the relatively simple job of evaluating single chamber asynchronous devices to monumental task of trouble shooting dual chamber, rate modulation, and multi sensor devices. Considering the complexities of newer pacemakers and the need for special knowledge and skill required to provide such services to pacemaker patients, the periodic follow up is best done by cardiologist or other medical personnel specifically and sufficiently trained in pacemaker care in the setting of appropriately organized pacemaker clinics.

However practicing internists and physicians are often called upon to evaluate/manage pacemaker patients with other medical issues and are expected to have sufficient knowledge and skill to follow up these patients. This article is aimed at providing such information to the non-cardiologist physicians. Detailed description of various pacemaker-timing cycles, analysis of pacemaker ECG's, interrogation of the pacemakers and optimizing their programming etc are beyond the scope of this article.

Early Post Implantation Assessment

At this stage the evaluation is mainly directed at verification of the

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wound healing and early detection and management of complications related to implantation. As most patients are discharged in 24 to 48hrs after implantation, a physician's visit is preferable by 10-14 days after the implantation. A superficial wound gape may be left alone to heal but if there is deeper gaping with exposure of pulse generator or the leads, then patient may have to be referred to the implanting cardiologist for resuturing. Similarly superficial epidermal serous discharge or a minor cuticular stitch seroma can just be treated by appropriate cleaning and dressing. But deeper dermal pus collection or discharges which increase by squeezing the pacemaker pocket are ominous enough to suggest the pocket infection and likely to necessitate referral to implanting cardiologist for reexploration, explantation and reimplantation etc. Uninfected, boggy hematomas are to be generally left alone as they get absorbed over a period of 4-6 weeks. Needle aspiration should definitely be avoided, as it more often tends to introduce infection. Tense, painful, expanding hematomas especially when patients are receiving oral anticoagulants sometimes merit opening of one edge of the wound under aseptic precautions for evacuation of hematomas followed by resuturing under aseptic precautions.

Other early complications for which the physician has to be on the look out include pneumothorax, haemothorax, pericardial effusion and rarely cardiac tamponade, pericarditis with resultant chest pain, lead displacements which could be macro displacements (easily visible displacements seen on chest X Ray or detected by 2D Echo), or micro displacements (satisfactory lead position on X Ray /Fluoroscopy but with loss of capture or loss of sensing). Vigilant clinical examination, 12 lead ECG with out and with magnet application, chest X Ray in PA and lateral views as well as Fluoroscopy and 2D Echocardiography help in arriving at appropriate diagnosis. Worsening of heart failure is sometimes seen after pacemaker insertion due to inappropriate pacing modes or inappropriate programmed parameters.

Timing of Subsequent Visits

The capture threshold starts rising after the implantation for 2-6 weeks and then decreases again to reach a chronic plateau. Follow up visit with the cardiologist between 2-4 months after implantation allows reprogramming of the pacemaker settings based on the chronic threshold to maximize the longevity of the pacemaker. This visit also helps the cardiologist to verify the clinical benefits of pacing and allows him to reprogram the pacemaker appropriately. For example a Tread mill test or 24 hr Holter will help in appropriate programming of rate response, the minimum or maximum paced rates, or the mode switch to be programmed "ON" in a patient with dual chamber pacer with brief episodes of atrial flutter or atrial fibrillation. Subsequently patients with single chamber pacemakers need once a year and patients with dual chamber pacers need twice a year follow up with the cardiologist's pacing clinic.

Common Symptoms During Subsequent Follow-up

Specific history pointing to decreased cardiac out put or cerebral hypo perfusion need to be elicited as they may point out either pacemaker malfunctions or inappropriate programming. Syncope, presyncope or dizziness could either result from loss of capture, pacemaker inhibition due to oversensing etc. or could be continued manifestation of underlying cardiac disease eg. Calcific aortic stenosis. Exercise fatigue and /or generalized weakness can often result from failure to capture or pacemaker syndrome as well as due to inappropriately programmed pacing rate or rate response or due to progressive worsening of underlying cardiac or pulmonary diseases such as coronary artery disease (CAD) valvular heart disease, COPD etc. Worsening of dyspnoea, orthopnoea or PND can be the result of pacemaker syndrome in a patient with VVI pacemaker or due to inappropriate programmed AV delay in a dual chamber pacemaker. It could also suggest worsening of LV function in a patient with single chamber pacing from RV apex. Palpitation is a frequent complaint by the patients and could suggest spontaneous extra-systoles, supra-ventricular tachycardias, pacemaker mediated tachycardia or due to rapid ventricular pacing etc. Diaphragmatic pacing from RV apex or by stimulating the phrenic nerve during atrial or coronary sinus pacing can result in distressing twitches, which vary with body positions. Similarly jerky muscle twitching can occur by use of unipolar pacing mode or due to lead insulation break.

Pacemaker Syndrome

This often under diagnosed syndrome refers to constellation of symptoms such as dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea (PND), and sometimes frank pulmonary edema, fatigue, presyncope or syncope and sometimes awareness of uncomfortable large neck vein pulsations. Usually it is seen among elderly patients with diastolic dysfunction of the heart, as they are dependent on "atrial kick". This syndrome is seen in patients with VVI pacemaker and sinus rhythm with AV dys-synchrony, or in those patients with DDD pacemakers with inappropriately long or too short AV delays, resulting in atrial contractions not preceding the ventricular contraction in the appropriate time window. Patients with DDD pacemaker with programmed long AV delays, due to "Electronic Wencheback" effect also can present with pacemaker syndrome.

When the AV delay is too prolonged it reduces atrial contribution to ventricular filling, as atrial contraction tends to inappropriately synchronize with rapid filling phase of ventricular diastole: it also reduces duration of ventricular diastole and may result in actual diastolic mitral regurgitation all of which either necessitate higher ventricular filling pressures or result in higher atrial pressures giving rise to symptoms of dyspnoea, orthopnoea and PND. Reduced

ventricular filling also contributes to symptoms of low output symptoms like fatigue. When the AV interval is too short or when the atrial contraction intermittently occurs close to ventricular systole or when there is VA conduction there can be a situation where atrium contracts against a fully or partially closed AV valve. Resultant pulmonary venous hypertension not only causes the congestive symptoms but also can kick off atrial baroreceptor activation resulting in reflex peripheral vasodilation and hypotension, precipitating presyncope /syncope as well as symptoms like weakness and fatigue. Recognition of these symptoms is important to improve patients overall well being by suitable change in the pacing mode from VVI to atrial sensed and /or paced modes like AAI/VDD/DDD and with Echo guided adjustment of AV delay.

Some of the other symptoms with which patients present include neck pulsation due to cannon waves in VVI pacing mode or symptoms and signs of venous thrombo occlusion such as SVC or subclavian venoocclusion. Persistent pain at the pulse generator site often is a result of inappropriate pocket fashioned in the dermal layer instead of the subcutaneous plane. Sometimes it can be severe enough to warrant explantation and reimplantation. Rarely patients present with chronic pericardial effusion due to lead migration/perforation and even with late pericardial constriction due to earlier Dressler's syndrome or lead migration.

PHYSICAL EXAMINATION

Examination of The Pacemaker Site

Presence of signs of inflammation like erythema, induration and pain in the first 6 months after the implantation suggests likelihood of pocket infection and warrant immediate antibiotic therapy. Well-established pocket infection eventually will require explantation of the device, pocket debridement, antibiotic therapy and reimplantation. The skin above the pacemaker should normally be freely mobile. If adherent it may eventually lead to chronic pain or erosion. Easy rotation of the pulse generator on manipulation indicates a loose pocket or repeated manipulation by the patient (Twiddler's syndrome). This can eventually result in lead displacement. Improperly placed unipolar pulse generator in the pocket (face down) or an insulation break of the lead within the pocket can result in skeletal muscle twittings during the pulse generator manipulation.

Systemic Examination

While elevated JVP could be due to persistent or worsened heart failure, apulsatile full neck veins with edema of chest wall and dilated venous collaterals with upper limb edema could suggest superior venacaval thrombosis. Irregular cannon waves may normally be seen

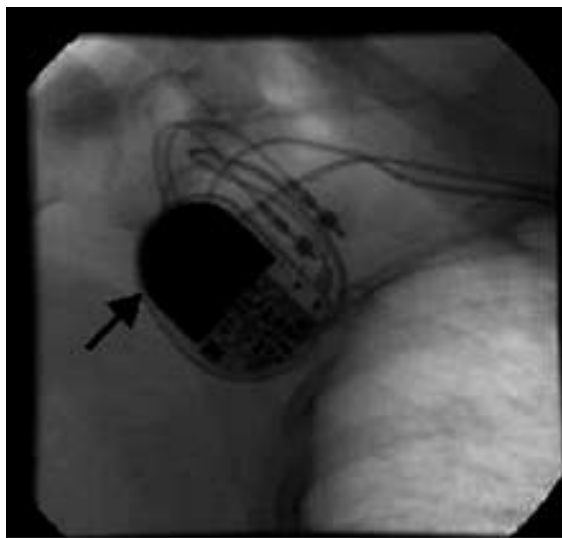


Fig 1 A. Post implantation X Ray

Shows atrial lead displacement due to Twiddler's syndrome. Note the rotated position of the pulse generator (Arrow mark) 3months after implantation

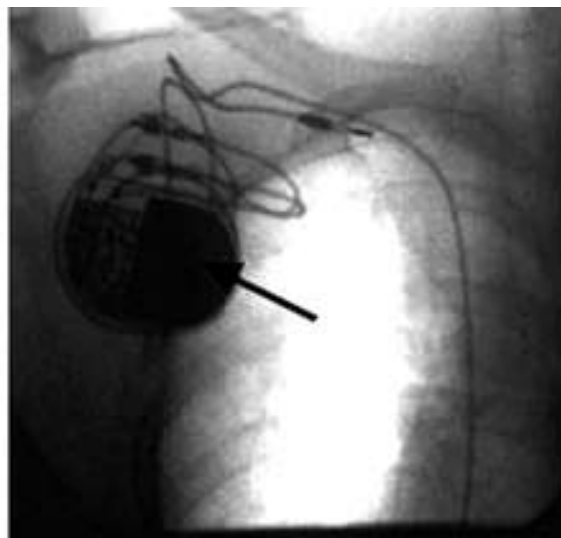


Fig1B. X Ray after 3 months



Fig2A : Application of magnet over a medtronic sigma VVI pacemaker increases pacing rate to 100/min for next 3 beats and then programmed rate is resumed. Note the tiny bipolar pacemaker artifact.

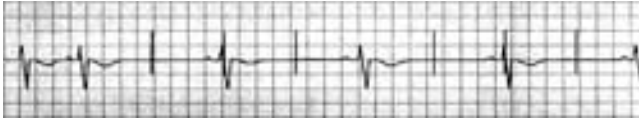


Fig 2B : Pacemaker ECG showing true capture lose. The pacing spike is falling after the T waves (beyond refractory period of the ventricle) and get fails to capture this ventricle.

in patients with VVI/VVIR pacemakers. However regular cannon waves suggest short AV interval or VA conduction. In such patients careful elicitation of symptoms of pacemaker syndrome is essential. Rarely thrombosis of the intracardiac part of the pacing leads can manifest with signs of thrombo-embolic pulmonary hypertension or right ventricular inflow obstruction. Pulse rate lesser than minimum programmed rate could result from pacemaker malfunctions like failure to pace/failure to capture or due to special programmed functions like Hysteresis. Small volume pulse with low pulse pressure should alert the physician to seek history suggestive of pacemaker syndrome. Which muscle twitching during arm movements occur in unipolar pacing or as a result of lead insulation breakage. Diaphragmatic pacing can be demonstrated by change of posture or by deep breathing. Arm movements like lifting or pressing the arms together can be used to identify myopotential inhibition when used in combination with ECG monitoring.

On auscultation, first heart sound may be variable due to AV dysynchrony (VVI mode) or "Electronic Wencheback" in dual chamber pacing modes. As RV pacing mimicks left bundle branch block (LBBB) the S_2 is often paradoxically split. Scratchy systolic murmur can occur due to lead induced insignificant tricuspid regurgitation and rarely lead movement induced non-ejection clicks or loud musical systolic "whoops" may be heard. "Pacemaker sound" sometimes can be heard due to skeletal muscle or diaphragmatic contraction. These physiological sounds should be documented and need to be differentiated from sounds like pericardial rubs, late onset tricuspid regurgitation murmur due to lead endocarditis etc.

X Ray Chest and Fluoroscopy

X Ray chest is an integral part of pacemaker follow up and when needed can be complimented with fluoroscopy to ascertain lead positions, (number of leads, sites being paced, position in the paced chamber) lead configuration (unipolar/bipolar/single pace dual chamber sensing leads), lead connectors, pulse generator position, pulse generator manufacturer identity, integrity of lead body, lead insulation defects, faulty connections, loose set screws, cardiac chamber enlargement, pulmonary congestion etc. Comparison with post implantation X Ray helps in detection of lead displacement, migration etc.

Electrocardiogram

Twelve lead (preferably the simultaneous and digitally acquired) ECG recorded with and without magnet placed over the pulse generator should always be recorded. Clear identification of spontaneous as well as paced P waves, QRS complexes and ST – T waves help in analysis of pacemaker function, ascertaining underlying rhythm, as well as to diagnose post implantation events like myocardial infarction etc. Underlying rhythm recognition is important as non recognition of underlying AF in patients with VVI or DDD pacemakers result in

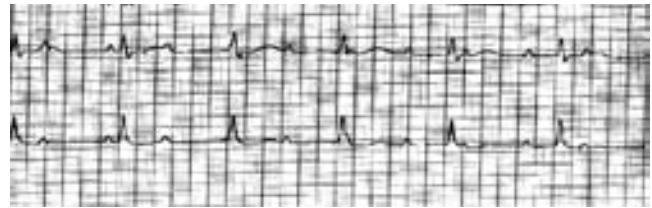


Fig 2C : Pacemaker ECG showing psuedo capture loss. The pacemaker spike has fallen in the absolute refractory period of the ventricle. The pacemaker was in asynchronous mode (CDD).

non initiation of oral anticoagulation and resultant higher risk of cardio-embolic stroke.

Magnet Application

On placing the magnet over this pulse generator the response varies in different models (Table1).

Most models turn into asynchronous mode with maximum outputs and pace at a prefixed rate. Magnet rate below the prescribed fixed rate for the particular model indicates elective battery replacement time and end of life (E O L) of the pulse generator can be diagnosed. Magnet mode also ascertains capture at maximum output and in some models it also helps to establish the capture threshold by Threshold Marginal Test. Familiarity of the magnet function in a given model of pacemaker is essential for interpretation. Magnet application also helps in terminating pacemaker-mediated tachycardia (PMT), in slowing runaway pacemaker, and to treat pacemaker inhibition due to cross talk or over sensing.

Pacemaker malfunction ECG Analysis

This requires adequate information about the patients' original cardiac disease/ rhythm disorders and indication for pacing, as well as adequate knowledge of the pacemaker model used, site paced, parameters programmed etc. For example- in RV apical pacing, paced QRS complex with RBBB morphology could suggest undesirable situation like lead migration, lead perforation into pericardium, perforation into left ventricle, in advertent lead placement into LV through patent foramen ovale, or lead placement into coronary sinus tributary. It could also be seen in about 10% of patients despite the lead being in desirable RV apical position due to RV apical portion being activated by left bundle branch, and this knowledge can avoid unnecessary panic. Key questions one has to ask during analysis of pacemaker ECG for malfunction are mentioned in (Table 2). Using the systematic approach pacemaker Failure, Capture failure, sensing failure etc, can be identified (Tables 3 and 4). Definite diagnosis and confirmation of these conditions necessitates pacemaker interrogation using specific programmers and need referral of patient to cardiologist with expertise for the same.

SPECIAL CONSIDERATIONS

Diagnosis of myocardial infarction (MI) in Paced Patients

Right ventricular pacing results in paced QRS morphology resembling LBBB. Hence one can rely on the criteria to diagnose MI in the presence of LBBB in paced patients also. However the pacing artifacts often mask the Q waves especially in unipolar pacing

Table 1 : Key questions to be asked during analysis of pacemaker ECGs

Is there pacing artifact
Which chamber is being captured?
Is there capture failure? – True or Pseudo?
Is there sensing failure? - True or Pseudo?
What is the rate ?
Is it fixed/ variable ?
Is the rate & mode different from programmed rate?
Is there indication of ERI or EOL?

mode. Presence of pacing artefact-small Q with large R in avL, I, v5-v6 & stimulus artefact-small r with large S wave in v₁ has – high specificity (and low sensitivity) for diagnosing extensive anterior wall MI. Cabrera’s sign (late notching of ascending limb of QRS in left precordial leads) also is highly specific for extensive anterior MI in RV paced patients. ST elevation especially when concordant with paced QRS complexes is diagnostic of MI. The changes due to MI may be best seen in spontaneous unpaced beats or on inhibition of ventricular pacing with chest wall stimulation. Post pacing ST – T changes in non-paced beats are however nonspecific findings seen often as a result of T wave memory.

External Cardioversion and defibrillation

External cardioversion can potentially damage the pacing system especially due to high current flow through the lead as well as due to large electro magnetic fields generated. Damage to lead tip-myocardial interface can result in failure of sensing and / or pacing. Although pacers are designed to withstand energy up to 400 Joules without any damage, all pacing systems should be interrogated to check their integrity after external cardio version / defibrillation. During cardioversion preferably the paddles should be placed perpendicular to the axis between the pacemaker and the lead tips. Bipolar pacemaker with shorter antenna has lesser chances of malfunction after defibrillation.

Electromagnetic Interference (EMI)

Pacing system malfunction can occur as a result of EMI. The potential dangers include electrical burn of lead myocardial interface, ventricular / atrial tachycardia or fibrillation, component failure of the pulse generator, change of output / rate / mode, inhibition of pacing by sensed events / myopotential inhibition etc. Common potential sources of EMI that occur in the hospital settings and precautions to be followed during their use in patients with pacemaker are indicated in (Table 5).

CONCLUSION

Use of pacemakers has become fairly common even in developing country like India. Implantation of appropriate pacemakers, their optimal programming to exploit the various available function and their trouble shooting requires advanced training and should preferably be carried out by specifically trained cardiologists or cardiac electrophysiologists. However internists and general physicians should familiarize themselves with certain basic functions of pacemaker, and must acquire the knowledge needed to follow up the patients with pacemakers. They also should be able to assess the patients and ascertain whether the particular patient needs to be referred to the trained cardiologist for further evaluation or replacement of pacemaker. This can be achieved by familiarizing oneself with the expected and commonly reported symptoms in patients with pacemakers, carrying out diligent physical examination for the normally expected and unexpected abnormal findings, carefully studying the chest x ray, and analyzing the ECG in detail. Use of magnet ECG helps in timely detection of early replacement indicators and end of life of the pacemaker facilitating timely referral

Table 2 : Causes of pacing failure

True Malfunction	Pseudomalfunction
Battery failure	Bipolar spike not seen
Circuit failure	Hysteresis (single and dual chamber devices)
Lead fracture	Search hysteresis
Internal insulation failure (bipolar lead) with “dead short”	Automatic mode switching
Oversensing	Unrecognized intrinsic activity
Improper positioning of lead in header	Pacemaker-mediated tachycardia
Incompatible lead or header	prevention and termination algorithms
Lack of anodal connector contact during implantation	
-Unipolar lead in bipolar device	
-Bipolar lead in device programmed unipolar	
-Air in pacemaker pocket (unipolar device)	

Table 3 : Causes of capture failure

True capture loss	Pseudo capture loss
Pulse Generator Causes	Recording Artifact
-Battery/ Circuit Failure	Pacing during Refractory Period
-Air in the Pocket (Unipolar)	
-Low Output Programmed	
Connector Causes	
-Improper Pin Insertion	
-Improper set screw	
-Incompatible Connector	
Lead Causes	
-Fracture /Insulation break	
-Dislodgement - Micro / Macro	
-Migration / Perforation	
Elevated Threshold	
-Early / Late	
-Exit Blocks	
-Metabolic / electrolytes	
-Drugs	
-MI	

Table 4 : Drugs increasing the capture threshold

Increase threshold	Possibly increase threshold
Bretylium	Beta-blockers
Encainide	Lidocaine
Flecainide	Procainamide
Moricizine	Quinidine
Propafenone	
Sotalol	

for pulse generator change. Any suspicion of malfunction as well as optimization of programming requires further referral of the patient to trained cardiologists for pacemaker interrogation, detailed evaluation and troubleshooting.

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Table 5 : Common causes of EMI and precautions to be taken during their use in paced patients

Medical Equipment	Precautions
1. Unipolar Electrocautery:	Not to be used preferably or > 6 inches away from the site.
2. Bipolar Electrocautery	-To be greater than 6 inches away from PG. -Ground plate to be closest to the operating site and farthest from PG. -Use for brief periods only -Monitor arterial pressure in case patient is dependent on pacer. -Preferably patient should not be pacemaker dependent.
3. Short Wave Diathermy	Should be avoided in patients with pacers
4. Ionizing Radiation:	Should be avoided
5. MRI	-To be avoided preferably If essential can be done with 1.5 Tex A MRI.
6. Transcutaneous Electrical	Generally safe Electrodes not to be parallel to pacing vector
7. Mobile phone:	To be kept 6 inches away. Post "2000" pacemakers are protected with inbuilt filter.
8. Lithotripsy:	Rate response in pacemaker with peizo electric crystals to be avoided.