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Cardiac Perforation Associated with a Pacemaker or ICD Lead

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1. Introduction

Cardiac perforation after pacemaker or implantable cardioverter-defibrillator (ICD) implantation is an infrequent complication. The reported rates of perforation are 0.1-0.8% after pacemaker implantation and 0.6-5.2% after ICD implantation (Khan et al., 2005; Mahapatra et al., 2005). From 1994 to 2009, total 1026 permanent pacemakers and ICDs were implanted in my lab, and two cases (0.2%) developed cardiac perforation without pericardial effusion. However, the incidence rate may be underestimated because some type of perforation does not produce any symptom. Asymptomatic perforation is known to be relatively common (up to 15%) and this can be found incidentally by chest computed tomography (CT) scan (Hirschl et al., 2007).

Although acute presentation is common, delayed presentation can be possible (Khan et al., 2005). The clinical importance is that perforation can lead to longer hospital stays, pacing failure, cardiac tamponade, cardiogenic shock and death (Aizawa et al., 2001; Ellenbogen et al., 2002; Garcia-Bolao et al., 2001; Gershon et al., 2000). In this chapter, information on the overview and management strategy for cardiac perforation will be provided.

2. Cases

2.1 Pacemaker lead perforation

A 64-year-old woman presented to the emergency room with left chest pain which was aggravated with left lateral decubitus position. Four days ago, she underwent implantation of a single chamber permanent pacemaker due to sick sinus syndrome. A passive fixation lead was implanted in the right ventricle. The electrocardiogram and pacemaker analysis revealed sensing and capture failure. Device Interrogation demonstrated unmeasurable capture threshold and normal impedance. Chest X-ray findings indicated lead displacement (Fig. 1). Transthoracic echocardiography excluded pericardial effusion but could not confirm the ventricular lead tip position.

Cardiac CT scan revealed the abnormal lead tip position in the anterior chest wall and confirmed the diagnosis of pacemaker lead perforation (Fig. 2). The displaced lead was removed by open-chest surgery. Surgical exploration showed that the pacemaker lead had perforated the RV and the parietal pericardium without developing hemopericardium. The perforated myocardium was repaired, and a new epicardial pacemaker lead was successfully implanted.

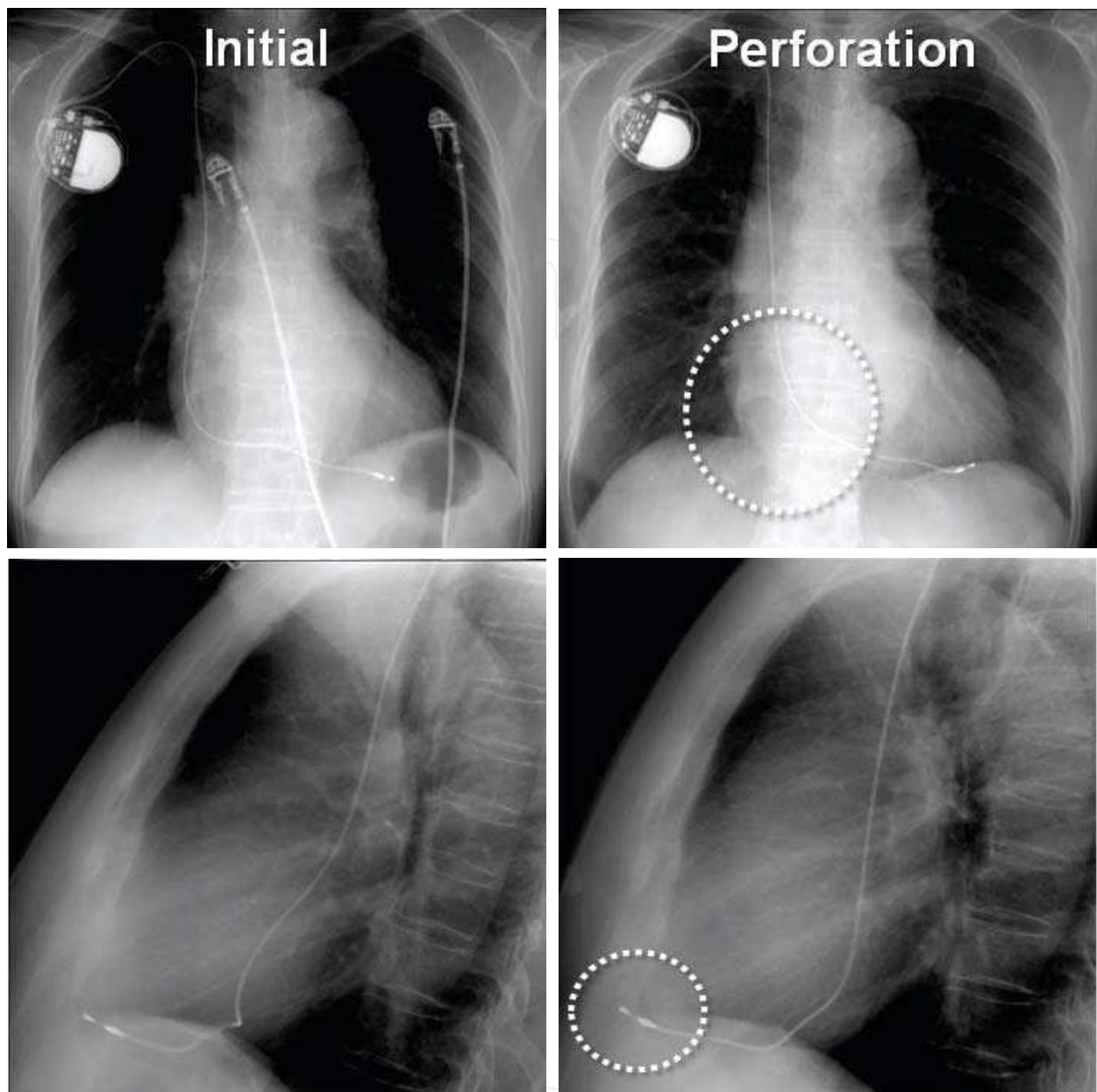


Fig. 1. Initial and perforation chest X-ray. In comparison with the initial images, curvature of the lead in the atrial portion (dotted circle in the posteroanterior view) and the lead tip position (dotted circle in the lateral view) were changed.

2.2 ICD lead perforation

A 32 year-old man with dilated cardiomyopathy underwent ICD implantation due to spontaneous and sustained ventricular tachycardia. Four days after implantation, he experienced one episode of ventricular tachycardia (VT), which was terminated successfully by antitachycardia burst pacing. After the event, he felt left chest pain, which was aggravated by deep inspiration. He visited the device clinic 13 days after the VT event, and device interrogation revealed that the lead parameters were changed as follows: threshold 0.4 volt \rightarrow capture failure despite 5-volt pacing (Fig. 3); impedance 1080 ohm \rightarrow 440 ohm; intrinsic sensing 7.0 mV \rightarrow 3.2 mV. Chest x-ray indicated lead tip displacement, therefore

lead repositioning was performed. The migrated lead was removed from the RV apex safely by simple traction under fluoroscopy and echocardiographic monitoring with surgical backup support. Then the lead was re-implanted in the septal area.

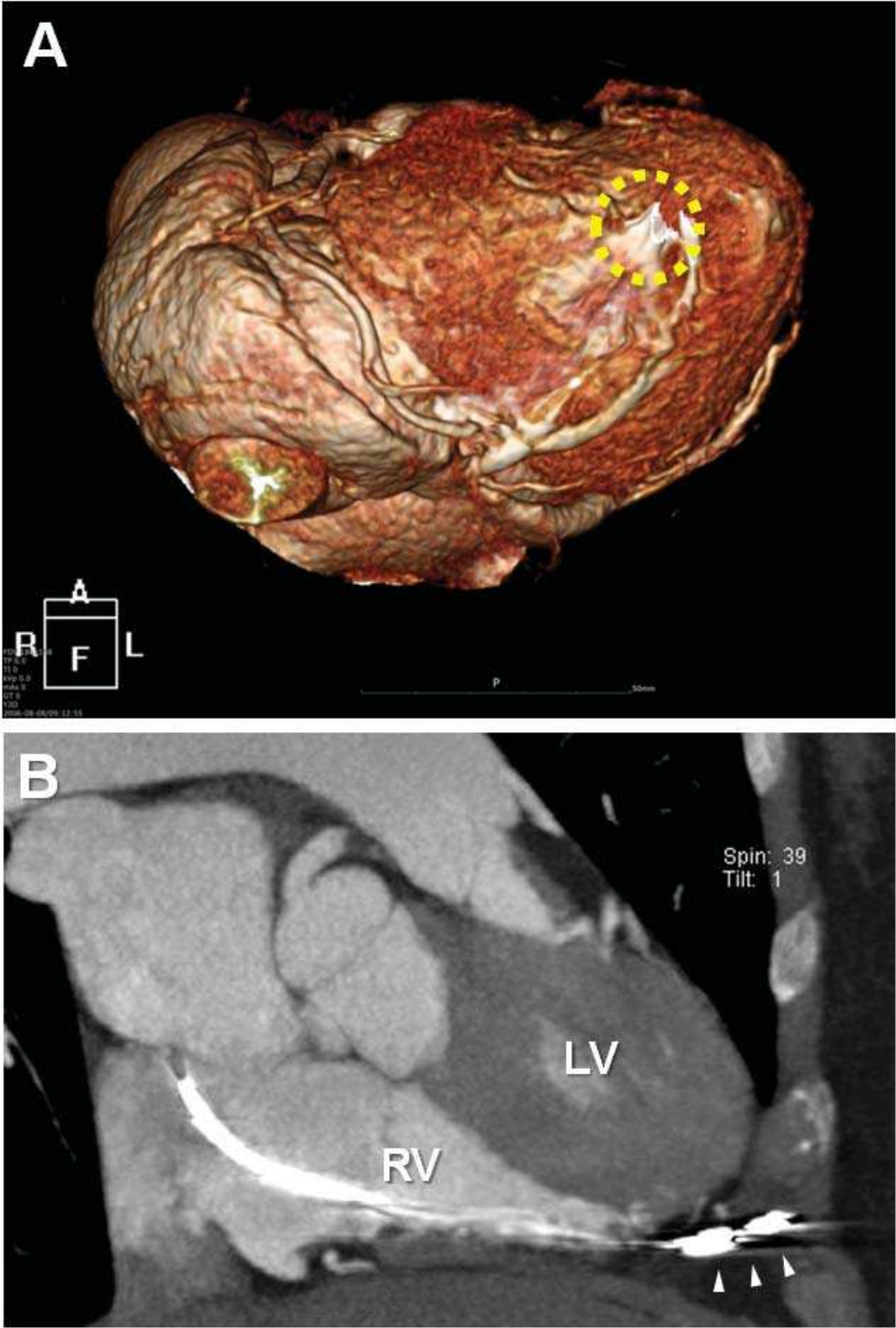


Fig. 2. Multi-detector CT scan images. A, Distal portion of the lead (dotted circle) is observed in the 3D reconstruction image. B, Arrowheads indicate the pacemaker lead penetrating RV apical myocardium and extending to anterior chest wall. LV, left ventricle; RV, right ventricle.

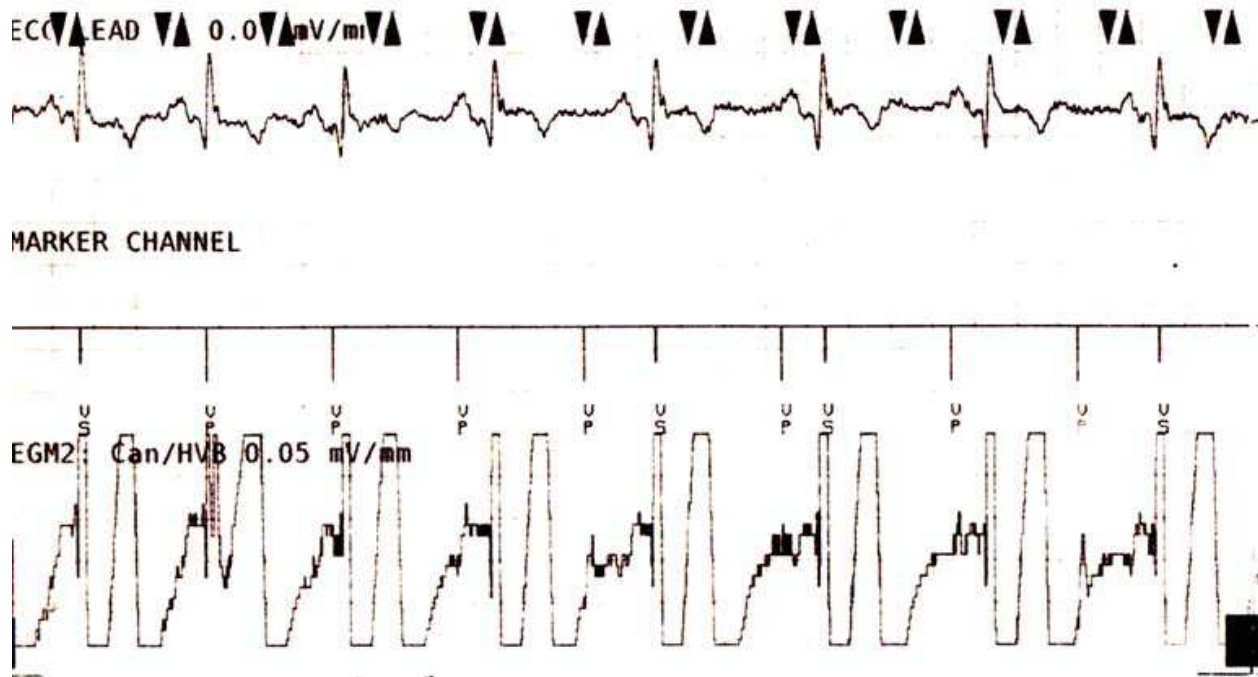


Fig. 3. Capture failure of the ventricular lead. VP, ventricular pacing; VS, ventricular signal sensing.

3. Clinical manifestations

Cardiac perforations are presented with acute, subacute, or delayed manifestations. In general, acute perforation is defined as event within 24 hours after implantation. Delayed perforation is defined by the event which presents at least one month after pacemaker/ICD implantation (Ellenbogen et al., 2002; Rydlewska et al., 2010). Acute perforations usually present with more severe clinical conditions. Delayed perforations seem to have more benign courses because they may be sealed spontaneously.

Symptom, signs and pacing parameter changes would be dependent on the location of the lead tip. A perforated lead tip can be located in pericardial space, extracardiac free space, mediastinum, lung, and chest wall muscles. Therefore, the presenting symptom and signs are variable:

1. Chest pain
2. Dyspnea
3. Hypotension
4. Syncope
5. Capture failure
6. Inappropriate ICD shocks
7. Muscle or diaphragm stimulation
8. Abdominal pain
9. Pericardial effusion
10. Cardiac tamponade
11. Hiccup has been also reported as a result of phrenic nerve stimulation due to cardiac perforation (Celik et al., 2009).

The most common symptom described in the previous reports is pacing or sensing failure. If a lead perforates myocardium, capture threshold will be increased and sensing threshold will be reduced in general. However, impedance will be variable because it depends on the tissue components such as muscle, blood, and air. In some asymptomatic patients with delayed perforation, pacemaker function and electrophysiological parameters appear normal (Hirschl et al., 2007). Therefore, normal pacemaker function cannot exclude cardiac perforation.

Hemodynamic stability is mainly determined by the development of hemopericardium. Hemopericardium and cardiac tamponade needs emergent management and usually requires open-chest surgical correction.

4. Predictors

Cardiac perforation could be associated with any factors that induce myocardial injury or weakening. The predictors of lead perforation have been evaluated by several investigators. They are as follows:

1. Temporary pacemaker implantation
2. Corticosteroid use
3. Active-fixation leads
4. Low body-mass index
5. Older age
6. Longer fluoroscopy times

Mahapatra et al investigated these predictors and found that steroid use was the most powerful predictor by using a multivariate analysis : use of temporary pacemaker (HR 2.7, 95% CI 1.4-3.9, $p = 0.01$), helical screw leads (HR 2.5; 95% CI 1.4-3.8, $p = 0.04$), and steroids (HR 3.2, 95% CI 1.1-5.4, $p = 0.04$) (Mahapatra et al., 2005).

Long-term use of corticosteroid may induce not only skeletal muscle atrophy, but also myocardial atrophy. The effect of chronic steroid use on myocardium may be mediated by some molecules such as muscle ring finger-1, a muscle specific protein (Willis et al., 2009).

Placement of temporary pacemaker before permanent device implantation can affect perforation by several mechanisms. Usually temporary pacing leads are stiffer than permanent leads, thus it could result in more myocardial damage. Situation of temporary pacing such as myocardial infarction might be associated with the risk of cardiac perforation.

A lead type may also increase the risk of cardiac perforation. It may occur more frequently with the following lead types (Rydlewska et al., 2010):

1. Atrial lead
2. Active fixation system
3. ICD lead
4. When excessive length is left.
5. Small diameter
6. High-resistance lead (small tip surface)

However, these have not been evaluated in large clinical trials. An active-fixation lead has a helical screw in its tip to attach the lead tip on the endocardial surface. Mahapatra et al found that ventricular screw leads were associated with cardiac perforation, but Sivakumaran et al reported atrial screw leads were associated with perforation after pacemaker implantation (Sivakumaran et al., 2002). The difference is unclear, which still remains to be elucidated in the further investigations.

Lead location is also a contributing factor. Perforations occur more frequently in the RV apex. This may be associated with the thinner wall thickness of the apex than that of the septum or RV outflow tract (Haq et al., 2008; Khan et al., 2005; Laborderie et al., 2008).

Incidence of cardiac perforations of ICD leads is associated with the number of shocks delivered. Chest trauma may affect perforation, especially during the early period of implantation (Lau et al., 2008; Sassone et al., 2009).

Interestingly, right ventricular systolic pressure >35 mmHg was the only protective factor (HR 0.70, 95% CI 0.50-0.92, $P = .02$) in Mahapatra et al.'s study. It may be associated with right ventricular hypertrophy that can be resulted from increased right ventricular pressure. Muscle hypertrophy might be able to reduce cardiac perforation.

5. Diagnosis

A patient with pacemaker and symptom such as chest pain, dyspnea, hypotension, syncope, inappropriate ICD shock, and diaphragm stimulation should be investigated to evaluate the existence of pacemaker-related complications including cardiac perforation. Pacemaker system interrogation, chest radiography and echocardiography can be helpful to evaluate possible extracardiac migration of leads.

Capture and sensing threshold should be compared with the previous values. Usually capture threshold is increased and capture failure is frequently developed as a result. Therefore a highly pacemaker-dependent patient experiences bradycardia-related symptom such as syncope, dizziness and exertional dyspnea. In addition it is easy to develop decrease of sensing threshold. It produces sensing failure and results in inappropriate pacing or ICD shock. Impedance change is variable because it depends on the tissue components such as muscle, blood, and air. If the location of the migrated tip is mainly filled with air (e.g. lung, pericardial space), impedance will be increased. However, if the tip is placed in spaces filled with fluid or blood (e.g. hemothorax, hemopericardium), impedance will not be increased significantly because blood has lower impedance than air. Although inappropriate pacemaker/ICD function may indicate possible perforation, normal function cannot exclude the diagnosis.

Key component of perforation diagnosis is visualization of the lead tip. Therefore, chest radiography, echocardiography (transthoracic or transesophageal), and CT scan are important tests. Chest radiography is useful to compare the lead tip position and lead curvature with those of the initial or previous follow-up data. Lead tip migration may be too subtle to get definite diagnosis in some cases. Diagnosis would be easier if lead migration outside the cardiac silhouette was observed. In addition, it is helpful to evaluate complications associated with perforation: pleural effusion, pericardial effusion, pneumothorax, etc.

Echocardiography is one of the first-line tools to assess the lead location in most cases (Mahapatra et al., 2005). It may show lead tip in the pericardial space and/or pericardial effusion. However, it occasionally fails to demonstrate such findings.

CT scan, especially with multi-detector scanners using cardiac protocols, can be useful when other modalities are nondiagnostic (Henrikson et al., 2006). Thus CT scan is becoming the gold standard in diagnosis of cardiac perforation and lead tip visualization. According to the report on the cases of asymptomatic cardiac perforation in patients who underwent chest CT due to other medical reasons, perforation rates were 15% in atrial leads and 6% in ventricular leads (Hirschl et al., 2007). Sensitivity and specificity of each test modality are still not available due to paucity of systematized studies.

6. Management

Most reports showed that most migrated leads could be removed safely by simple traction under fluoroscopy and/or echocardiographic monitoring in the operating room, with surgical backup support. However, Heart Rhythm Society Expert Consensus classifies this approach as class III for following conditions: *Lead removal is not indicated in patients with known anomalous placement of leads through structures other than normal venous and cardiac structures, (e.g. subclavian artery, aorta, pleura, atrial or ventricular wall or mediastinum) or through a systemic venous atrium or systemic ventricle. Additional techniques including surgical backup may be used if the clinical scenario is compelling* (Wilkoff et al., 2009).

Surgery is generally recommended when cardiac tamponade is expected strongly during extraction, the initial presentation is cardiac tamponade, or the location of the migrated lead is atypical. Therefore management strategy would be dependent on the existence of pericardial effusion. If a case is presented with pericardial effusion, surgical management will be the optimal treatment. If a patient has no pericardial effusion, simple traction can be considered under the guidance of careful monitoring including echocardiography and surgical backups. In asymptomatic cases with normal pacemaker function and no adjacent organ damage, necessity of lead extraction is controversial (Hirschl et al., 2007).

After lead extraction, a new pacemaker lead should be placed at a different location. That is, if the lead was initially placed in the RV apex, RV outflow tract or the septum would be the optimal site. In cases of open-chest surgical correction, epicardial leads would be another option. During the postoperative period, close observation with echocardiography should be performed because delayed cardiac tamponade could develop (Laborderie et al., 2008).

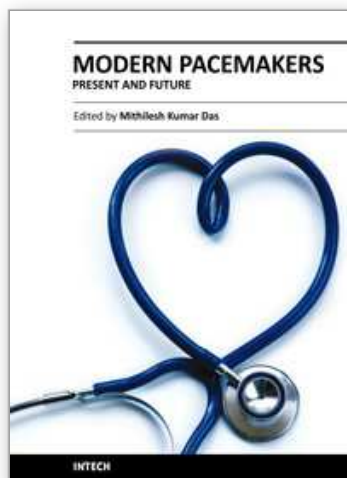
7. Conclusion

Cardiac perforation after pacemaker or ICD implantation is an infrequent complication. The clinical manifestations are variable. The predictors of lead perforation are temporary pacemaker implantation, corticosteroid use, active-fixation leads, low body-mass index, older age, and longer fluoroscopy times. Pacemaker system interrogation, chest radiography and echocardiography can be helpful to evaluate possible extracardiac migration of leads. Computed tomography, especially with multi-detector scanners using cardiac protocols, may be the gold standard. Most migrated leads can be removed safely by simple traction under fluoroscopy and/or echocardiographic monitoring in the operating room, with surgical backup support. Surgery is required when the initial presentation is cardiac tamponade or adjacent structures should be repaired.

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Modern Pacemakers - Present and Future

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

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