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Chapter 6

Complications of Pacemaker Implantation

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Additional information is available at the end of the chapter

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1. Introduction

Approximately 180,000 patients undergo pacemaker implantation in the U.S. each year [1]. In addition, the extreme elderly are the most rapidly growing segment of the U.S. [2,3] and pacemakers are commonly implanted in this population. There are reports of pacemaker implant complications (generally clinical trials reporting outcomes and incident complication rates) and fewer reports of complication rates in the extreme elderly (with a persistent exclusion of elderly patients from ongoing clinical trials [4]). A comprehensive review of pacemaker implant complications can help improve informed consent in preoperative patients. Major and minor complications are defined based upon prior reports of device-related complications. [5,6,7,8] Major complications have been defined as death, cardiac arrest, cardiac perforation, cardiac valve injury, coronary venous dissection, hemothorax, pneumothorax, transient ischemic attack, stroke, myocardial infarction, pericardial tamponade, and arterial-venous fistula. Minor complications have been defined as drug reaction, conduction block, hematoma or lead dislodgement requiring reoperation, peripheral embolus, phlebitis, peripheral nerve injury, and device-related infection. This chapter will include discussion of common and uncommon complications of pacemaker implantation including associated incidence as well as the associated radiographs and common clinical signs of these complications.

2. Demographics of pacemaker implantation

From 1993 to 2006, 2.4 million patients received a primary pacemaker and 69,000 pacemaker generator changes; women comprised 49% of pacemakers. [1] A review of studies involving pacemaker implantation in adults reveals an average age range of 69-86 years with approximately 30-40% of patients aged > 80 years [9,10]. There is a tendency for higher percentage of female patients as age increases; a prior study from our Heart Rhythm Center [11] examined pacemaker implant outcomes of extremely elderly patients (>80 years) with an average implant age of 86 and revealed 61% of implants were performed in females.
3. Preprocedural issues

3.1. Preoperative risk assessment

As with all surgical interventions requiring anesthesia, recognizing and managing comorbid conditions preoperatively helps to mitigate the risks during and immediately after pacemaker implantation. The association of heart failure and other structural heart disease with cardiac conduction system disease as well as the expanding role of biventricular pacemakers specifically indicated for patients with symptomatic congestive heart failure means that there is an inherently high risk population of patients frequently served in the EP lab. Indeed, CHF increases the risk of all surgery. A decreased LVEF has been found to be a predictor of perioperative mortality and morbidity, with the highest risk group being those with an EF < 35%; the very patients brought to the lab for resynchronization therapy. Pre-procedural management of CHF is not only integral to our practice but vitally important to the safety of the procedure. Patients certainly should not be in a state of decompensated heart failure.

3.2. Infection

Patients who present with systemic infection and positive blood cultures carry the highest risk of infection. Infection of implantable devices is one of the most feared complications due to the dismal prognosis of untreated infections and risk of device removal. Often we are asked to evaluate patients for bradyarrhythmias when they happen to be identified at the time of hospitalization for infectious etiologies such as pneumonia and urosepsis as well as post cardiac surgery. Pre-implant evaluation for potential sources of infection is critical. The estimated rate of infection of permanent endocardial pacing leads is between 1 and 2%, however the range is from under 1 percent to greater than 10%. In the PEOPLE study [12], device infection requiring removal was correlated to fever within 24 hours of device implant, temporary pacing prior to implant, and early reintervention for lead revision or hematoma evacuation. The likelihood of infection was nearly doubled by the presence of a temporary system. The association with temporary intravenous pacing wires certainly implies an association with any indwelling lines including central lines and PICC lines. The duration of hospitalization prior to implant was not correlated to higher risk of infection. Infections were negatively correlated with de-novo implantation and perioperative antibiotic prophylaxis. The latter intervention is considered controversial. Of 28,860 Danish patients [13], 3.6% had a lead related complication by 3 months. Risk factors for lead related complications included operator inexperience (<25 implants).

3.3. Procedural management of iodinated contrast agents

3.3.1. Contrast Induced Nephropathy (CIN)

Contrast-induced nephropathy is a surprisingly common complication of radio-contrast procedures occurring in 15% of cases. It is defined as an absolute increase in creatinine of 0.5mg/dl (in patients starting under 2) or an increase of 25% of baseline and typically peaks
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Creatinine may remain above baseline for 7-14 days. Naturally, the best way avoid this complication is to abstain from its use. Single and dual lead pacing systems can safely be implanted without the use of contrast at all; Prior data from our Heart Rhythm Center [11] revealed that contrast was used in 55 of 92 (59.8%) of the pacemaker implantations with a mean intravenous contrast usage of 9.6cc. We often use contrast to ensure that the subclavian/axillary venous system is patent and to help guide venous access. No contrast was used for generator changes and there were no contrast reactions. CRT implantation may require contrast agents to define the coronary (CS) anatomy. The vast majority of patients undergoing CRT implants are patients with heart failure and its associated comorbidities which very frequently include diabetes and chronic kidney disease, therefore a working knowledge and respect for these agents is a necessity.

We typically use 5cc of iso-osmolar contrast in a 1:1 ratio with NSS and limit CS venography to 1 cine if possible. For patients with GFR 30-60ml/min, hydration can usually be achieved with 4-6 glasses of water the evening before the procedure. ACE inhibitors, ARBs, and NSAIDS can be held the day prior and day of exposure and be resumed 24 hours after exposure. For patients with a GFR <30ml/min, the above recommendations should be followed with consideration of one or both of the following: 1.) IV hydration using 1L of NaCl 0.45% with 50mEq NaHCO3 (1/2 normal saline with 1 amp of sodium bicarbonate) run at 1ml/kg/hr for 12 hours. For same day procedures, this can be administered at 3ml/kg/hr for 1 hour. 2.) N-acetylcysteine 600mg PO BID the day prior and day of the procedure, totaling 4 doses. It must be noted that besides preprocedural hydration, the evidence is not conclusive that bicarbonate and acetylcysteine offer additional benefit. Treatment of CIN after evident is largely supportive and infrequently requires short-term dialysis.

CS lead placement can be performed successfully without the use of contrast in patients at risk of CIN. [8] CS lead placement at our Heart Rhythm Center without the use of contrast begins with CS access by engagement of the CS with a 5French octapolar deflectable electrophysiology (EP) catheter or hydrophilic coated 0.035” guidewire that the CS sheath is advanced over. Next, a 0.014” guidewire is advanced out distally and the entire CS is probed for LV branch vessels in 360 degree fashion; we then work proximally and if no branches are found (including very proximal posterolateral “bailout” vessels) then we repeat the process. We have demonstrated a 97.3% success rate in LV lead placement with these techniques. [8]

3.3.2. Contrast allergies

Immediate anaphylactic reactions including angioedema, bronchospasm, arterial hypotension, and shock can occur within minutes of and up to 60 minutes after injection. [14] The reported incidence of severe immediate reactions to ionic contrast material (CM) is 0.1-0.4% and with newer non-ionic, low osmolar or iso-osmolar contrast is 0.02-0.04% but death rates from the two materials do not differ. [15] Although incompletely understood, direct release of histamine from circulating basophils and eosinophils is probably the primary mechanism with IgE mediated mast cell activation (i.e. true allergy) being a much less frequent secondary mechanism. A skin testing study showed that only 4% of patients with anaphylaxis symptoms had an IgE-mediated mechanism [16]. Delayed reactions from 1 hour
to 7 days after injection of CM are T-cell mediated and most typically are skin reactions. [15] Patients with even mild anaphylactoid (immediate) reactions should be considered high risk in future CM administration. Indeed, a distinction between immediate anaphylactoid and non-immediate anaphylactoid reactions may be more clinically relevant than a history of mild, moderate, and severe reactions. [17] For purposes of reporting, CM reactions are graded 1-3. Grade 1 reactions include one episode of nausea/vomiting or sneezing, grade 2 reactions being fever/chills, hives, and more severe nausea/vomiting, and grade 3 reactions are potentially life-threatening and include angioedema, bronchospasm, laryngospasm, pulmonary edema, hypotension, and shock. [18] It is common practice to pre-medicate with corticosteroids with or without H1 blockers in patients with a history of moderate or severe immediate reactions, despite the fact that randomized trials comparing pretreatment strategies are severely lacking. No trials of pre-treatment have tested a steroid-antihistamine combination which is the most commonly utilized. [18] In a consensus document published by Marcos et al in 2001, 91% of survey respondents administered corticosteroids at least 11 hours prior to CM administration (though dose frequency varied from 1-3x) and 55% used an H1 blocker (diphenhydramine 25-50mg) typically administered once. [14] H2 blockers are used, but rarely. In a meta-analysis including 10,011 patients with no history of contrast allergy, routine pre-treatment with steroids alone reduced respiratory symptoms after contrast injection (frequently ionic, high osmolar) from 1.4% to 0.4% and Grade III symptoms from 0.9% to 0.2%. Only a “double dose” steroid regimen reduced Grade III symptoms. [18] In this same cohort, there were no “disastrous” consequences. Cutaneous manifestations were more often prevented by H1 antagonists and respiratory symptoms show improvement with steroid pretreatment. [18] Essential information to be sought from the patient prior to administration of contrast is history of previous CM reaction, asthma, renal insufficiency, diabetes, and metformin therapy. [19] Routine pre-medication of all patients to receive CM is probably not warranted given the overall low incidence of a reaction; in fact some have advocated abandoning this procedure all together. [18] Patients with a history of severe CM allergy who will likely need IV CM injection should probably receive pre-exposure prophylaxis with corticosteroids as well as H1 antagonists although strong evidence of benefit is lacking. [16] The likely mechanism for the benefit of corticosteroids is a reduction in circulating basophils and eosinophils available for direct activation. If CM administration cannot be delayed for 4-6 hours after steroid injection, some would omit use and administer only H1 blockers. [19] One can also consider the addition of H2 antagonists such as ranitidine but evidence is also lacking. Low or iso-osmolar contrast such as ioxaglate, iohexol, or ioversol should be used due to the lower overall incidence of reactions in patients with a history of asthma or a CM allergy. The specific CM causing the prior reaction should be sought and avoided if possible. Cutaneous allergic testing can be performed on patients with a history of anaphylactic reactions and a “skin test negative” CM should be used but it should be noted that only a fraction of those patients will have a positive skin test. [15, 18] The ESUR guidelines on prevention of CM reactions recommend Prednisone 50mg PO at 13, 7, and 1 hours prior or methylprednisolone 32mg orally 12 and 2 hours prior to exposure to CM in addition to
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3.4. Thyroid

Hypothyroidism has been found in 0.5-0.8% of the population demonstrated by elevated serum levels of thyroid-stimulating hormone (TSH) or decreased serum thyroxine levels. [20] Undiagnosed (and hence untreated) hypothyroidism can lead to major perioperative complications including severe hypotension or cardiac arrest following induction of anesthesia, extreme sensitivity to narcotics and anesthetics with prolonged unconsciousness and hypothyroid coma following anesthesia and surgery. [20] Ideally, hypothyroidism is caught early and thyroxine administered until the patient is euthyroid (generally, 4-6 weeks).

Hyperthyroidism affects approximately 0.2% of men and 2% of women and may cause atrial fibrillation, congestive heart failure and thrombocytopenia. [21] In addition, anesthetic drugs may be affected by the hypermetabolic state of hyperthyroidism. When total intravenous anesthesia is used (often at our center when high frequency jet ventilation is used to minimize respiratory motion), propofol infusion rates should be increased to reach anesthetic blood concentrations because the clearance of propofol is increased in hyperthyroid patients. [21] Supportive management of thyroid crisis includes hydration, cooling, inotropes and steroids. Beta-blockade and antithyroid drugs are used as the first line of treatment.

Generally, more thyroid related perioperative complications stem from hypothyroidism as opposed to hyperthyroidism however, recognition of either prior to implantation is important. Our Heart Rhythm Center generally obtains TSH prior to all device implantation and allows 4-6 weeks for the patient to become euthyroid prior to proceeding with surgery. Emergent cases with thyroid abnormalities require close coordination with anesthesiology and will generally be undertaken with general anesthesia.

4. In-hospital complications

4.1. Sedation / airway:

Less than 20% of electrophysiology (EP) programs in the United States exclusively use anesthesia professionals for procedural sedation. [22] Minor complications (e.g., atelectasis, fever, vascular congestion) may simply be reflective of common postoperative pulmonary complications (PPC’s) seen after general anesthesia. Atelectasis can be seen on CT scan in up to 90% of patients who are anesthetized [23] and PPC’s have been found to occur in 9.6% of patients. [24] There are data to suggest that patients undergoing invasive EP procedures may require deep conscious sedation that often is converted to general anesthesia; [25] thus, the use of general anesthesia (including HFJV) during EP procedures may enhance patient safety. [26,27] The advantage of general anesthesia was mostly studied in patients undergoing complex procedures, such as AF ablation, where precise electroanatomical mapping is required. There is not much data supporting the use of general anesthesia
(which may increase costs) versus conscious sedation in routine PM implantations. In our center, we often use general anesthesia with a laryngeal mask airway to minimize patient movement during LV lead implantation.

4.2. Pneumothorax

Pneumothorax may occur in as many as 3-4% [28,29] and as few as 0-1% [8,10,11,30,31,32] but generally ranges from 1-3% [5,9,33,34,35,36,37] of patients undergoing pacemaker implantation. Routine chest radiographs are often performed immediately after pacemaker implantation though clinical signs of pneumothorax include hypoxia, shortness of breath, pleuritic pain, and hypotension. Figure 1 depicts the radiographic appearance of small, medium, and large pneumothoraces. Figure 2 depicts a pseudopneumothorax that resulted from external artifact; close review of radiographs by radiologists can limit false-positive pneumothorax interpretations. Emergent treatment of pneumothorax includes decompression of the pressure tension by thoracentesis or chest tube. Oftentimes, high concentrations of inspired oxygen can lead to a resolution of a pneumothorax that comprises less than 30% lung volume. (38) This conservative treatment of pneumothorax can reduce morbidity and duration of hospitalization and avoid invasive drainage procedures. The traditional treatment of patients with traumatic hemo- or pneumo-thoraces has been an insertion of a chest tube (CT). CT have larger caliber than pigtail catheters and can cause significant trauma during insertion, cause pain, prevent full lung expansion, and worsen pulmonary outcomes. [39] Pigtail catheters, smaller and less invasive than chest tubes, have been used successfully in patients with nontraumatic pneumothorax. Pigtail catheters have demonstrated a non-significant increase (11% vs 4% for CT) in the tube failure rate (defined by a requirement for an additional tube or by recurrence requiring intervention) [39].

![Figure 1. Examples of Pneumothoraces, Small (A), Medium (B), and Large (C). The edge of the pneumothorax is indicated by the arrows. A small left apical pneumothorax is shown in A. A moderate-sized apical and basilar pneumothorax is shown in B. An almost complete collapse of the left lung is shown in C. Please note that examples shown in B and C are from defibrillator implantations.](image-url)
4.3. Vascular access and hemothorax

The axillary venous approach has been associated with less frequent pneumothorax and subclavian crush syndrome. [40,41] The axillary vein is the continuation of the basilica vein that terminates immediately beneath the clavicle at the outer border of the first rib, at which point it becomes the subclavian vein. [42] Direct subclavian venous punctures are associated with increased rate of pneumothorax [5] while cephalic vein cutdown has been associated with the lowest rate of pneumothorax and lead damage. [33,31] Fluoroscopic-guided, first rib approach to axillary vein access is the most effective means to access the vessel while minimizing the risk of pneumothorax. [42] A prior study did examine pacemaker implantation complication rates of 632 consecutive implants at a single non-community institution.[33] They found a 0.6% rate of hemothorax with a substantially large incidence of complications experienced by low-volume (<12 implants per year) implanters.

Hemothorax can be caused by pacemaker lead placement (more frequently atrial lead perforation) as well as vascular access damage to the subclavian and axillary veins as well as the vena cava. Figure 3 depicts a post-implant CXR of a hemothorax from damage to the superior vena cava during upgrade of dual chamber pacemaker to biventricular defibrillator. This patient experienced pain during passage of the introducer placed over a guidewire seen in inferior vena cava with subsequent development of right pleural effusion. The injury was believed to have occurred during passage of CS guiding catheter introducer over the wire. Recognition of new effusions should be treated as possible procedural related hemothorax and surgical consultation is warranted.
4.4. Perforation / tamponade

Perforation (both acute and subacute) has been reported to occur in up to 1% of pacemaker implantations. [5,8,10,31,32,35,36,37] In addition, asymptomatic subclinical perforation may occur in 15% of patients after device implantation. [43] Symptoms of perforation include pleuritic chest pain from pericarditis, diaphragmatic or intercostal muscle stimulation and, in the presence of pericardial effusion, patients may develop shortness of breath and hypotension as tamponade develops. [44] Other signs/symptoms of perforation include right bundle-oid paced QRS morphology (though we have seen RBBB configuration and diaphragmatic stimulation in RV apical lead position) or friction rub after implant. If perforation is suspected, urgent evaluation of the patient and device function is warranted though lead parameters are often within normal limits. [44] Figure 4 shows examples of coronary sinus damage that can occur during LV lead implantation. Figure 5 depicts right ventricular lead perforations. Cardiac surgery is typically not required for a majority of patients diagnosed with cardiac perforation from a pacemaker implantation. Rather, most cases can be managed with pericardiocentesis for symptomatic effusions and repositioning of the lead in the EP laboratory with close cardiothoracic surgical collaboration. [45,46,44] Figure 6 shows a large cardiac silhouette developing after pacemaker implantation that was due to large pericardial effusion. The effusion was treated with pericardiocentesis (with no evidence of reaccumulation) and did not require lead repositioning. Though perforation and subsequent tamponade are infrequent complication of pacemaker implantation, they can be responsible for significant patient morbidity and mortality. The risks of perforation cannot be underestimated; death from tamponade with subsequent cardiac arrest was responsible for 21.8% of the mortality in a worldwide study of perforation after ablation for atrial fibrillation. [47]
Figure 4. Damage to the coronary sinus during left ventricular lead implantation. Image A depicts a dissection/perforation flap and the resulting pericardial staining from engaging the coronary sinus with a deflectable electrophysiology recording catheter. Image B shows a similar instance of pericardial staining with no focal dissection flap or perforation. Both patients underwent successful LV lead implantation at the time.

Figure 5. Examples of Right Ventricular Pacemaker Lead perforation. Images A and B depict an RV lead perforation that exits the right ventricular base in A (arrow) and reenters near the right ventricular apex in B (arrow). Images C and D depict an right ventricular apical perforation. The lead is seen exiting the cardiac silhouette in C (arrow); the lateral view (D) depicts an abrupt change in lead course (arrow) that is often seen in right ventricular apical perforations as the lead courses posteriorly in the pericardial space.
4.5. Complications of Left Ventricular (LV) lead placement via the coronary sinus

The emergence of resynchronization therapy has led to an increase in attempts at left ventricular lead placement via the coronary sinus. The MIRACLE study program [48] reported a 91.6% success rate for LV lead placement, while COMPANION [49] revealed an 89% success rate for LV lead placement. Another report indicated a similar 92% success rate with LV lead placement. [50] Though we counsel our patients on a LV lead placement success rate at 88-92%, our center demonstrated a 97% success rate (64 of 66 patients) with LV lead placement within the range from 2:30 to 5:30 o’clock in the left anterior oblique (LAO) view. [8]

Complications of biventricular pacing, specifically LV lead placement, include cardiac perforation, coronary sinus dissection, electrical trauma (damage to the native conduction system), failure to place the lead, dislodgement of the lead, and diaphragmatic stimulation. [51] CS dissections or perforations, cardiac perforations, or cardiac vein dissection or perforation was reported in 45 of 2078 (2%) in the MIRACLE study program. [48] Figure 4 depicts damage done to the coronary sinus during LV lead implantation. Loss of LV capture and diaphragmatic stimulation leading to interruption of resynchronization therapy has been found to occur in 10% and 2% of patients, respectively. [52] The development of new LV leads with up to 4 electrodes offer the possibility of numerous pacing vectors that can minimize loss of capture and diaphragmatic stimulation.
4.6. Arrhythmias (SVT, VT, VF)

The incidence of sustained atrial, pacemaker-mediated and ventricular rhythm disturbances after pacemaker implantation is low. [53]. In patients without prior atrial arrhythmias, Jordaens et al found early atrial fibrillation (during the first week) in 2 of 112 patients and late atrial fibrillation was seen in seven patients, flutter in one, yielding a total incidence of 8.9% for 22 months. There were no significant differences with respect to age, etiology, electrocardiographic diagnosis, pacing history, or the measured intracardiac P wave between the group with and the group without atrial fibrillation. Ventricular fibrillation has been reported to occur in 0.1% of all patients undergoing pacemaker implantation and up to 0.6% of patients aged >90 years undergoing pacemaker implantation. [10]

It has been reported that ventricular tachyarrhythmias may be present in 12-31% of patients months to years after pacemaker implantation [54] but this may reflect underlying substrate issues. However, there are several situations where pacemaker implantation may cause the ventricular tachyarrhythmias. These include pacemaker lead irritation of the right ventricular inflow [55] and outflow tracts [56], pacemaker stimulus on T wave [57], reentrant circuit around endocardial pacemaker lead [58] and bradycardia-dependent VT facilitated by long pause caused by myopotential inhibition of a VVI pacemaker. [59]

4.7. Death

In-hospital death generally occurs in less than 1% of pacemaker implantations [5,8,10,11,37] however there is a concern that death is underreported as some studies do not specifically mention perioperative death. [9, 30,31,32, 33] The most common causes of confirmed device related in-hospital deaths are perforations (subclavian artery, brachiocephalic trunk, right atrium, and right ventricle). The most common cause of non-device related in-hospital deaths is myocardial infarction as well as less commonly pulmonary embolism, stroke, heart failure, and sepsis. [60]

4.8. Pocket hematoma

The incidence of pocket hematoma has been reported at 4.9% and leading to prolonged hospitalization in 2.0% of all patients. [61] Reoperation for pocket hematoma was required in 1.0% of patients. High-dose heparinization, combined acetylsalicylic acid (ASA)/thienopyridine treatment after coronary stenting, and low operator experience were independently predictive of hematoma development. [61] In addition, development of postoperative hematoma places the patient at elevated risk of device infection. [62] There is data to suggest that warfarin causes fewer pocket complications than heparin products. Specifically, temporarily interrupting anticoagulation is associated with increased thromboembolic events, whereas cessation of warfarin with bridging anticoagulation is associated with a higher rate of pocket hematoma and a longer hospital stay. [63]
4.9 Hospital lengths of stay

There is little data available on average length of stays post pacemaker implantation. An estimate of 2-3 days as an average length of stay post implant can be made from the available studies. [5,8,11,37] There is evidence that complications cause a substantial increase in the length of stay up to 16 days. [64] The mean complication costs are $4345 ± $1540 for pacemaker lead revision, $24,459 ± $14,585 for pacemaker infection, and $6187 ± $2631 for hematoma evacuation. [64]

5. Subacute post-implant complications (< 30 days)

5.1 Pacemaker and lead function / failures

Electrocardiographic signs of pacemaker malfunction can be grouped into four categories: failure to capture, failure to output, undersensing, and inappropriate pacemaker rate. Sensing abnormalities may occur in 3% of patients, failure to capture in 1%, and failure to capture and inappropriate pacemaker rate in another 1% of patients. [65]

5.1.1 Failure to capture

Loss of capture after pacemaker implantation has many causes: Dislodgement, Elevated Thresholds, Inappropriate Lead Placement, Fracture, Insulation Failure, Loose set screw, Exit Block (>4 weeks), Perforation, Battery/circuit Failure, Air in Pocket, and Metabolic/Drugs (Flecainide). Lead dislodgement, the most common cause of failure to capture, [65] has been reported to occur in up to 4-6% of pacemaker implantations [28,35] but is generally reported with a 1-3% incidence [5,10,11,30,32,33,34,36,37]. Figure 7A, B, and C depicts examples of right atrial, right ventricular, and left ventricular lead dislodgements that may result in loss of capture. Lead dislodgements are treated by repositioning in the EP laboratory.

5.1.2 Failure to output

Failure to pace with no obvious pacemaker output may be caused by battery or circuit failure, lead fracture, internal insulation failure, oversensing, loose set screw, or crosstalk. Random component failure is rare however, total battery depletion can occur if routine pacemaker followup is inadequate. [65] Once initial end-of-life indicators appear, there is usually a period of months before the battery reaches a critically low voltage and pacing fails. [65] The incidence of pacemaker lead fracture has been reported at 0.1% to 4.2% per patient-year and usually occurs adjacent to the generator or near the site of venous access. [66] Figure 8 shows a lead fracture discovered at reoperation when patient presented in complete heart block with no ventricular capture almost one year from pacemaker implant. Figure 9 depicts chest radiograph findings of lead fracture (failure to pace) versus a pseudofracture (normal pacing function). Finally, air in header may cause noise oversensing (as air released out of header) as shown in Figure 10. Other electrical signals that may cause...
oversensing include diaphragmatic myopotentials (especially with extending bipolar sensing), T waves, P waves, and environmental noise.

Figure 7. Right Atrial (A), Right Ventricular (B), and Left Ventricular (C) Leads Before (Pre) and After (Post) Dislodgements. Right atrial lead became dislodged after patient twiddled with device. Right ventricular lead dislodged by moving more basilar in position (arrow) one day after implant. Left ventricular lead dislodged and reseated itself in the body of coronary sinus 3 months after initial placement (arrow).

Figure 8. Right Ventricular Lead Fracture. Ventricular lead that was fractured one year after implantation resulting in failure to pace. Figure 8A shows appearance of right ventricular lead on CXR that was suspicious for fracture location (arrow). Figure 8B depicts the intraoperative appearance of lead that was likely site of fracture (arrow).
Figure 9. The radiograph in A shows a lead fracture that resulted in no capture. The radiograph in B depicts a “pseudofracture” where digital frame shift causes artifact to simulate a lead fracture in a properly functioning lead.

Figure 10. Noise caused by air in header. Atrial electrogram during device interrogation revealed high-frequency noise during sinus rhythm. This patient was not pacemaker dependent so there was no failure to pace.
5.1.3. Undersensing

Undersensing of intrinsic cardiac activity results in inappropriate pacing output that competes with intrinsic activity. Undersensing is most likely caused by lead dislodgement, poor lead position at time of implantation, or an interruption in the insulation of the pacing lead. [65]

5.2. Hospital readmission

The average rate of hospital readmission within 30 days of pacemaker implant is 4-6%. [8,11,28,35] We examined possible factors influencing readmission rates in extreme elderly undergoing pacemaker implantation. [11] Overall, increased age and Device Type (e.g., single-chamber, dual-chamber, biventricular, generator change) demonstrated a non-significant trend toward increased readmission rate. The order of decreasing significance in a multivariate analysis of readmissions was: Device Type > Age > Creatinine > Urgent/Emergent > EF > Sex > Weight.

5.3. Death

Early all-cause mortality 30-days after pacemaker implantation has been reported in 0.1-0.7% of patients. [30,5,36] Death rates may be increased (2%) in the extreme elderly aged > 80 years due to increased age-related mortality in this group. [11]

6. Late complications (> 30days)

6.1. Lead function / failures

6.1.1. Twiddling

Originally described in 1968 [67], twiddling refers to patient manipulation of pacemaker can or leads that may lead to malfunction. It has a reported incidence of 0.07% in a series of 17000 patients. [68] Figure 11 depicts lead orientation before and after patient twiddling resulted in lead dislodgement.

6.1.2. Exit block

Transient disruptions should be excluded first: metabolic and electrolyte abnormalities, drug effects, extreme hypothyroidism (myxedema), and cardiac ischemia. There is an expected rise in capture threshold in the 2-6 week period after lead placement attributed to local inflammation or foreign body reaction at the tip-tissue interface. In the era of epicardial and early endocardial leads, this was a much greater concern. Passive fixation endocardial leads have, on average, lower stimulation thresholds than active fixation leads due to lack of trauma at tissue interface. The degree that the capture threshold increases is markedly blunted with steroid eluting endocardial leads which have thus become generally preferred for their more favorable delivery characteristics having overcome the problem of
Figure 11. A and B. Lead orientation before and after patient twiddling resulted in lead dislodgement. Image A depicts the post-implant radiograph baseline lead positioning after biventricular defibrillator implant. Image B shows retracted right and left ventricular leads and leads tangled in the pocket superior to device can that is rotated.

higher stimulation thresholds. [69,70] The first randomized trial to compare a standard active fixation lead to a similar designed lead with a steroid eluting reservoir was reported in 1995. [71] Prior to that time, passive fixation leads were generally preferred because the capture threshold was relatively lower than standard active fixation leads. The disadvantage to passive fixation leads was an inability to perform atrial mapping, unreliable lateral wall stability, and requirement for placement in the atrial appendage which may be difficult in patient who have undergone bypass. The most dramatic difference in stimulation threshold between steroid eluting leads and standard endocardial leads was in the magnitude of increase in the acute phase as well as the duration of peak before returning to the chronic threshold. [71] The steroid lead returned to chronic capture threshold by week 2 whereas the non-steroid eluting lead remained above chronic threshold for 12 weeks. A non-significant increase in atrial lead dislodgements occurred with the steroid lead (0% vs 2%, p=0.58). Lower capture thresholds allow for lower programmed output to maintain 2x safety margin, ultimately improving generator longevity. A rise in capture threshold may occur beyond 6 weeks after implantation (chronic phase of lead maturation). As the threshold steadily rises, it may exceed the maximum output of the pulse generator, known as exit block. Exit block is recognized by high pacing thresholds without radiographic evidence of dislodgement. It may be related to inflammation or fibrosis at the electrode-myocardium interface and generally presents >4weeks after implantation. [65] Some patients and particularly pediatric patients are particularly prone to this phenomena and may lead to multiple lead revisions.
6.2. Device/lead advisories

A large multicenter Canadian observational study showed that the complication rate from device replacement for an advisory indication was an astounding 9.1%. [72] Of these, 5.9% required reoperation and there were 2 deaths. Naturally, the risk of an adverse outcome during replacement must be balanced by the risk of death due to device malfunction. Pacemakers and defibrillators have saved thousands of lives but as is true of all man-made devices, malfunctions have and will continue to occur. In response to a marked increase in device advisories in 2005, and to balance alarmism with protection of patients with a high risk situation, the Heart Rhythm Society (HRS), utilizing the HRS Task Force on Device Performance Policies and Guidelines published guidelines in 2006. [73] Recognizing that physicians and patients need timely and accurate information regarding device performance, arguably the most important outcome was a call for greater transparency of post market analysis and reporting of failures. Device performance is defined as the percentage of devices that are in service and functioning appropriately in living individuals over time and depends not only on the characteristics of the device but the skill of the implanting physician and caregivers following the device. [73] Data compiled from 1990-2002 from FDA annual reports showed that confirmed device malfunctions leading to device explantation were about 0.1-0.9% for pacemakers and 0.7-3.9% for ICDs. [73] Although failure rates are low, there is a negative psychological impact on patients who have a device which is under advisory, particularly if pacer dependent or if placed for a secondary indication.

To assist in communication from industry to physicians and patients it is proposed that terminology be standardized. The term “recall” was changed to “Class I Advisory” which is just short of a directive for device replacement because of a reasonable probability that malfunction could result in death or significant harm. Class II and class III recalls are subsequently referred to as advisory notices (non-life threatening malfunctions) and safety alerts (potential malfunctions). This information is disseminated from industry via standardized Physician Device Advisory Notifications and Patient Device Advisory Notifications which are also available on the manufacturer’s website. Prior experience tells us the advisory information should be disseminated to physicians just before patients. Advisories should include general information about the malfunction and potential clinical implications but should acknowledge that treatment decisions should ultimately be determined by patients in consultations with physicians. The situations where device replacement is recommended are 1) When mechanism of malfunction is known and likely to be recurrent or lead to patient death, 2) The patient is pacer dependant, 3) The device was placed for a secondary prevention indication or have received appropriate therapy, 4) The device is approaching EOL. Conservative management (enhanced non-invasive and remote monitoring) should be considered when 1) The rate of malfunction is very low in non-pacer dependant patients or primary prevention without history of appropriate therapy, 2) The patient has significant comorbidities or high operative risk even when the risk of device malfunction is substantial. 3) Remote monitoring and software reprogramming can minimize risk (i.e., non physiologic noise).
6.3. Infection

Up to 60% of patients present with localized infection involving the device pocket whereas the remaining patients may present with endovascular infection but no evidence of inflammation of the device pocket. [74] Approximately 10% of patients may have intracardiac vegetations identified by transesophageal echocardiogram, though can still undergo percutaneous lead extraction safely [75]. See Figure 12 for echocardiographic image of vegetation adherent to device lead. Generally, the most common pathogen isolated is aerobic gram-positive organisms, of which 90% are Staphylococcus species, with a high rate of methicillin resistance (~50%). [74] The risk of pacemaker infection is lower than that of implantable defibrillators. The presence of epicardial leads and postoperative complications at the generator pocket are significant risk factors for early-onset ICD infection, whereas longer duration of hospitalization at the time of implantation and chronic obstructive pulmonary disease were associated with late-onset ICD infections. [62] In one of the largest studies of pacemaker infections [76], repeated operative procedures after the first pacemaker implantation were associated with a substantial incremental risk of infection. Female sex, older age, and preoperative antibiotics given at the initial implant were associated with a lower risk of later infection. The pacing mode, indication for pacing, and complexity of the procedure were not independently associated with the risk of later infection. Sixty percent of infections have been found to occur within 90 days of implant [77] though a large number of infections occur during the late follow-up (>1 year post-implant) (76). Generator changes and cardiac resynchronization therapy/dual-chamber devices have also been implicated as independent predictors of infection. [77]

Figure 12. Transesophageal echocardiographic image of vegetation adherent to pacemaker lead. This is a short axis view showing a large, mobile vegetation (encircled) on a right ventricular lead (arrow) in a patient with persistent bacteremia.
The generally accepted means of device infection treatment is removal of the generator and all implanted leads. [78] In a large series of device extractions including 1838 leads [75], post-operative 30-day mortality was 10% though no deaths were related directly to the extraction procedure. Another series of device extractions reported a 0.5% rate of intraprocedural mortality, 4.6% rate of in-hospital mortality, and 2.6% rate of relapsing infections within 1 year of reimplantation. [74]

6.4. Pacemaker syndrome

Occurs most commonly with single chamber ventricular pacemakers (e.g., VVI or VVIR modes) and symptoms are due to loss of atrioventricular synchrony. It must be noted that pacemaker syndrome can occur with any pacing mode if AV synchrony is lost. Symptoms include malaise, weakness, cannon A waves, CP, cough, confusion, or syncope.

6.5. Venous thrombosis

Upper extremity deep venous thrombosis (or stenosis) is uncommon in the general population but venous stenosis has been seen in up to 33-64% of patients after implantation of pacing leads. [79,80] Statistically significant factors that have been associated with an increased risk include previous transvenous temporary leads [80], left ventricular ejection fraction <40% [80], systemic infection [81], absence of anticoagulation, use of hormone treatment, personal history of venous thrombosis, and presence of multiple leads. [82] Symptoms may include shoulder or neck discomfort, ipsilateral arm edema with cyanosis, dilated collateral cutaneous veins around the shoulder, or jugular vein distension. [83] Venography is considered the gold standard for diagnosis but compressive ultrasonography is an effective and economical means of confirming the clinical diagnosis. [83] Treatment may include anticoagulation (warfarin and/or heparin), extraction of old nonfunctioning lead to create a new venous channel, or venoplasty to reduce venous stenosis or allow the implantation of subsequent leads. [83,84]

7. Predicting risks for procedural complications

There is some evidence that elderly patients are at increased risk of complications following pacemaker implantation. [85] Armaganijan et al [85] found that any early complication occurred in 5.1% of patients ≥ 75 years of age compared to 3.4% of patients aged < 75 years. The concomitant use of temporary transvenous pacemakers or steroid use within 7 days of implant have been shown to increase rates of post implant pericardial effusion. [45] Weaker predictors of post implant effusions were the use of helical screw ventricular leads, body mass index <20, older age, and longer fluoroscopy times. [45] Pneumothorax has been found to more common in older, lighter females. [5] A prior study examining predictors of complications in extremely elderly patients undergoing pacemaker implantation [11] found overall rates of implant complications comparable to data from younger patient populations while experiencing a higher 30-day all-cause mortality (that may have been attributable to elevated all-cause mortality rates in this age-group). Multivariate analysis revealed that
female sex, device type, and urgent/emergent placement demonstrated a non-significant trend toward increased rates of complication; increased age and device type demonstrated a non-significant trend toward increased readmission rate.

Higher (>12 implants/year) versus lower volume operators (<12 implants/year) have also demonstrated diverging rates of complication. [33] Finally, more complex devices (dual-chamber vs single ventricular chamber pacemakers) have been associated with higher rates of complications. [10,30,31,32,35,37] however, there is data that does not demonstrate increased rates of complications in dual-chamber devices. [9,11,34] Finally, it has been suggested that physician training (specifically, board-certification or board-eligibility in clinical cardiac electrophysiology) may result in lower rates of lead dislodgement. [7,86]

8. Conclusion

Major and minor complications occur in approximately 4-7% of patients within 30d of pacemaker implantation. [5,10,11,32,36,35] Permanent pacemakers are commonly implanted in patients over the age of 65 and this is the most rapidly growing segment of the U.S. population. That being said, pacemaker therapy is also associated with an 11-40% risk of complications in the pediatric population; the most common complications in this segment are pneumothorax, hematoma, and infection. [87] Figure 13 depicts the incidence of the most common complications after pacemaker implantation. Prompt recognition and treatment of complications after pacemaker implantation is essential for all implanting physicians regardless of background.

![Most Common Complications after Pacemaker Implantation](image)

**Figure 13. Most Common Complications Seen After Pacemaker Implantation.** This figure depicts the most common complications seen after pacemaker implantation from available prior. The data are for complications seen within 30-42 days depending upon the study parameters as referenced.
9. References


[49] Bristow MR, Saxon LA, Boehmer J, et al., “Cardiac-Resynchronization Therapy with or without an Implantable Defibrillator in Advanced Chronic Heart Failure for the


vegetations defined by transesophageal echocardiogram,” J Am Coll Cardiol, V. 55, No. 9 (March 2010), pp. 886-94.


