

Complications of Implantation of Cardiovascular Implantable Electronic Device

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Abstract

Introduction: Cardiovascular implantable electronic devices (CIEDs), including pacemakers, implantable cardioverter-defibrillators (ICDs), and cardiac resynchronization therapy (CRT) devices, are crucial for managing various cardiac conditions. However, their implantation is associated with a range of potential complications.

This article investigates the incidence, types, and risk factors of complications arising from CIED implantation.

Around 180.000 pacemakers are implanted every year in the USA [1]. Keeping in mind that pacemakers are implanted mainly in the elderly, the increasing proportion of this age group translates to a progressive increase in yearly implantations.

A review of pacemakers implanted in adults shows an age interval of 69-86 years old, with 30-40% of patients >80 years old [2,3].

In a case series article, 218 patients were included in those undergoing permanent pacemaker implantation in the UHC "Mother Theresa" Tirana.

Data were retrospectively collected from patients who underwent CIED implantation. The primary complications assessed included infection, lead dislodgement, device malfunction, hematoma, and vascular complications. Statistical analysis was performed to identify significant risk factors associated with these complications.

Conclusions: This article underscores the importance of meticulous procedural techniques, thorough patient assessment, and post-implantation monitoring to minimize the risks associated with CIED implantation. Enhanced understanding of these complications can lead to improved patient outcomes and the development of strategies to mitigate risks in clinical practice.

Keywords: permanent pacemaker implantation (ppm), cardiac implantable electronic device (cied), long-term care, postoperative complication

Introduction

Around 180.000 pacemakers are implanted every year in the USA [1]. Keeping in mind that pacemakers are implanted mainly in the elderly, the increasing proportion of this age group translates to a progressive increase in yearly implantations. A review on pacemakers implanted in adults shows an age interval of 69-86 years old, with 30-40% of patients >80 years old. [2,3].

Our study's case series included 218 patients undergoing permanent pacemaker implantation in the UHC "Mother Theresa" Tirana.

The mean age was 70.21 ± 12.3 years old, with 2.3% belonging to the 16–40-year-old group age, 15.6% belonging to the 41–60-year-old group age, 67.9% belonging to the 61–80-year-old group age, 11.9% belonging to the 81–90-year-old group, and 2.3% are older than 90 years old. [4].

Among them, being an invasive procedure performed in old patients, it also has complications, and knowing them can help improve pre-operative informed consent for the patient and family members.

Complications can be classified as primary (including death, cardiac arrest, cardiac perforation, valvular damage, coronary sinus dissection, hemothorax, pneumothorax, TIA, CVA, tamponade and arterio-venous fistula) and minor (including drug allergies, conduction disturbances,

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hematoma or lead dislodgement requiring re-operation, peripheral embolism, phlebitis, nerve damage, and device-related complications). This article will treat complications, their incidence, and diagnosis.

1 - Pre-procedural issues

Like in all invasive procedures, it is essential to thoroughly evaluate the risk factors, such as low Ejection Fraction (EF), increased creatinine, infection risk, and contrast medium allergy.

Patients should be clinically stable when they have Heart Failure.

Patients with systemic infection and positive blood cultures have the highest risk for infection. Infection of implantable devices is the most feared complication due to its terrible prognosis and the need to remove the device. It is often required to evaluate the patient for Brady-arrhythmia when they present to the hospital with an infectious disease such as pneumonia or urosepsis and after cardiac surgeries.

Contrast-induced nephropathy is not a rare complication, but it occurs mainly when a considerable amount of contrast

is used, such as when Cardiac Resynchronization Therapy (CRT) is implanted.

Allergy to contrast medium is a rare complication, but it can become life-threatening, and as such, it is necessary to pre-medicate patients at risk for contrast allergy.

2 - In-Hospital Complications

Pneumothorax;

Pneumothorax usually occurs in 1-3% of patients [2, 5, 6] undergoing PM implantation. The signs suggesting pneumothorax are hypoxia, dyspnea, pleuritic pain, and hypotension. Pneumothorax can be small, moderate, or large (Fig.1).

Chest X-ray and CT confirm the diagnosis. Due to artifacts, care must be taken not to confuse it with pseudo-pneumothorax (Fig. 2).

Emergency treatment of pneumothorax includes decompression of the tension pressure through thoracentesis or chest tube. In small pneumothoraxes <30%, high-pressure oxygen therapy often resolves the pneumothorax, avoiding invasive interventions.

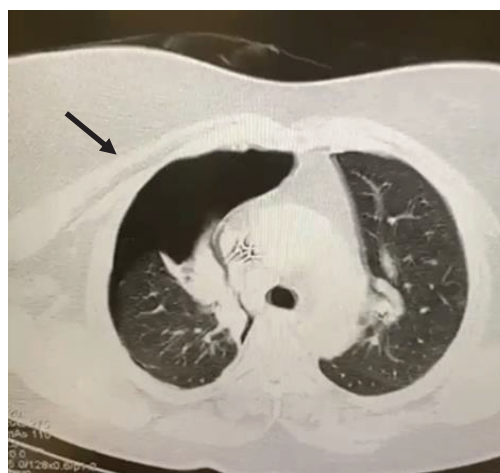
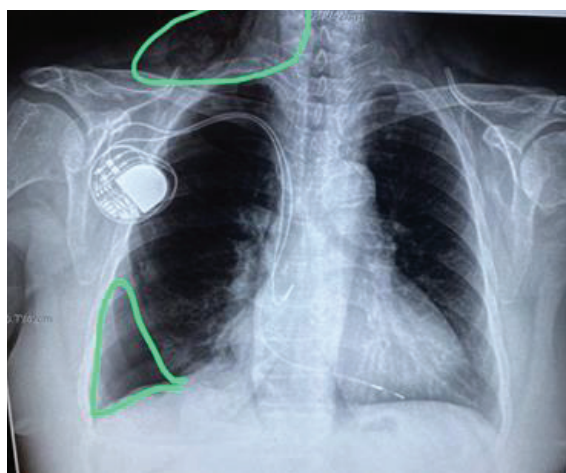


Figure. 1 Pneumothorax examples: (A) Small pneumothorax in X-ray, (B) Moderate pneumothorax in CT

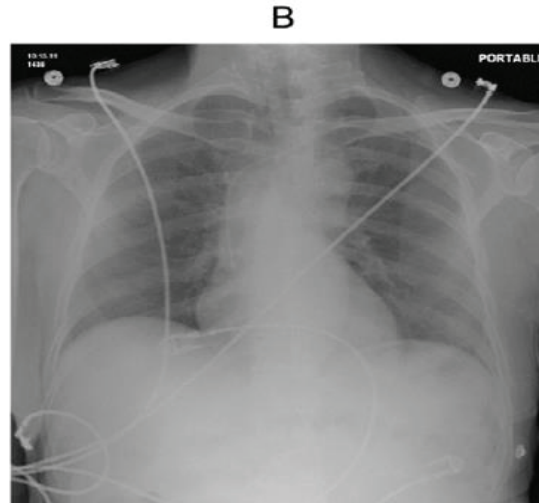
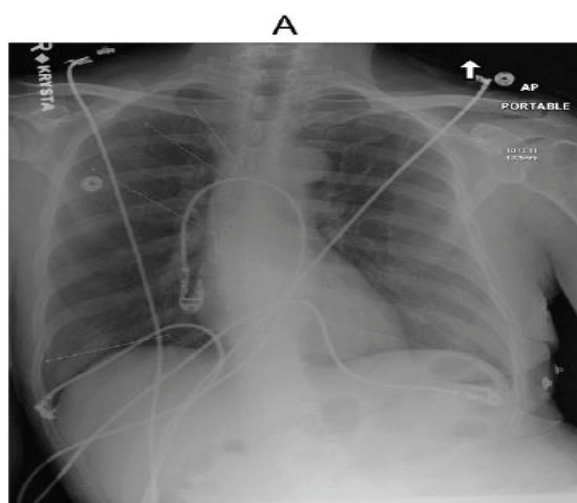


Figure 2. Pneumothorax after removal of a temporary pacemaker inserted through the internal jugular vein. (A) shows a line (arrows) suggesting pneumothorax in an asymptomatic patient. Repeating the X-ray in (B) shows their spontaneous disappearance, suggesting an external artifact from the white coat or skin.

Vascular access and hemothorax

Vascular access and hemothorax

The cephalic vein cut-down is the preferred access with the lowest risk for pneumothorax and lead damage. [5, 7] Direct subclavian vein puncture is associated with a higher risk for pneumothorax and hemothorax than the axillary vein. [4]. Fluoroscopic guided puncture of the axillary vein is more effective by minimizing the risk of pneumothorax. [8]

Hemothorax can be caused by PM lead implantation (more often atrial leads) and by damaging the subclavian or axillary vein during puncture. Figure 3 shows the chest X-ray of a hemothorax due to damage to the superior vena cava while upgrading from a dual-chamber Pacemaker to a CRT. The patient complained of pain, and we believe it happened while cannulating the coronary sinus. In case of a new pleural effusion should be treated as a procedural hemothorax, and a cardiac surgery consultation is needed.

Perforation/tamponade

Perforation (acute and subacute) has been reported in up to 1% of pacemaker implantations. [4, 9, 14] Moreover, asymptomatic subclinical perforation might happen in up to 15% of patients. [10]

Symptoms include pericardial chest pain and diaphragmatic or intercostal muscular stimulation, and in case of effusion, patients can develop shortness of breath and even hypotension up to tamponade. Other signs or symptoms include the appearance of RBBB (although it can also appear in diaphragmatic stimulations in RV apical lead positioning). When a perforation is suspected, you should urgently evaluate the patient and pacemaker parameters, although they are often normal. Figure 4 shows an example of Coronary Sinus damage during lead implantation in LV—figure 5 shows ventricular lead perforation. The majority of patients with PM lead perforation do not require surgery. In

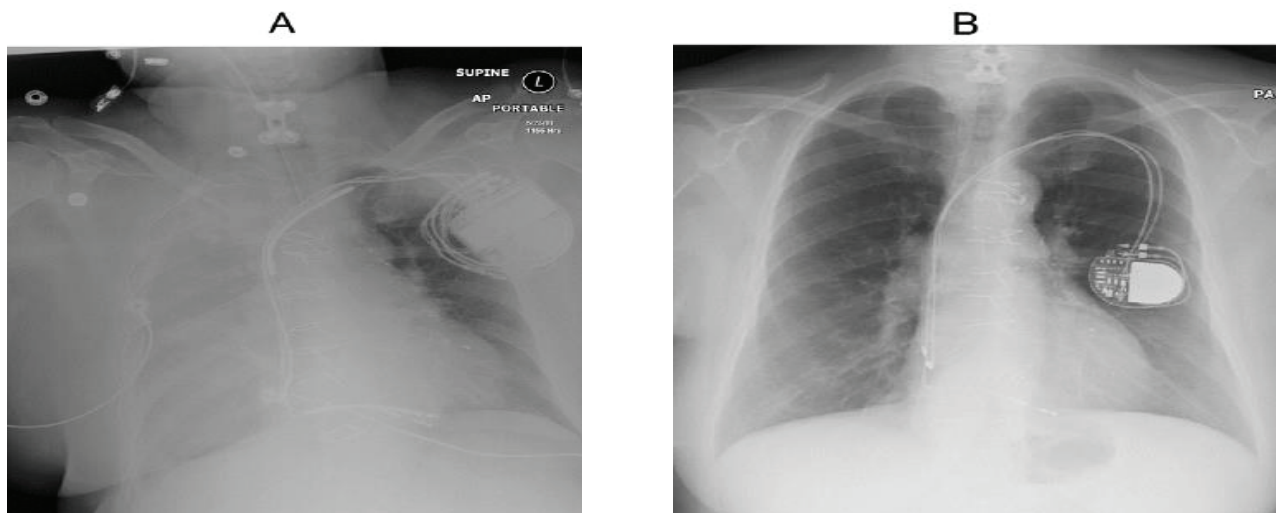


Figure 3 Hemothorax after PM implantation due to trauma of Superior Vena Cava from the Guidewire/Sheath

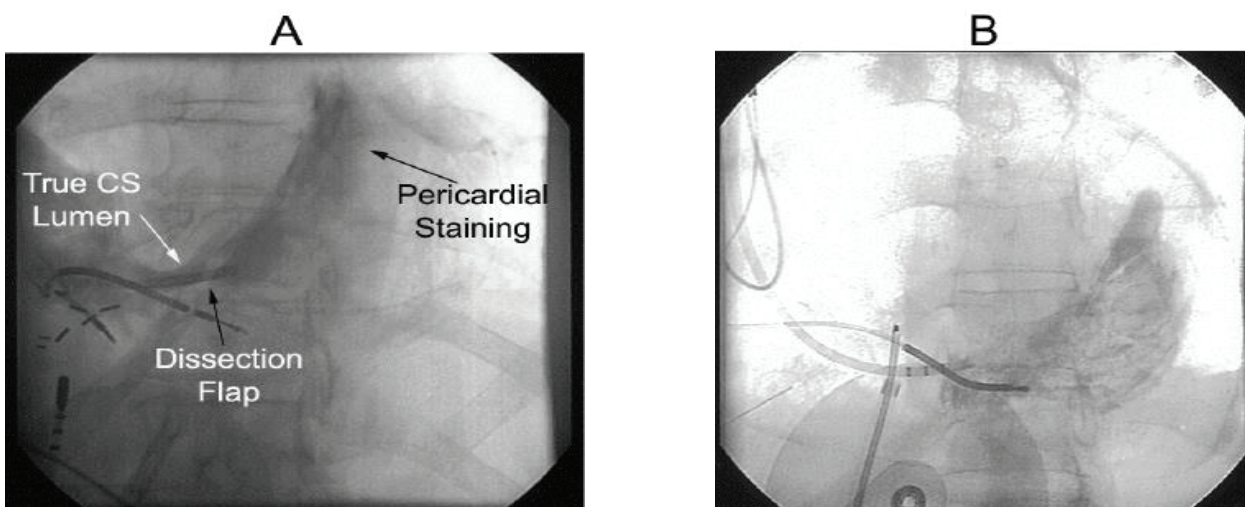


Figure 4. Sinus coronary damage during lead implantation in LV. (A) Coronary Sinus dissection/perforation and contrast extravasation in the pericardial space. (B) Similar contrast extravasation in pericardial space without signs of focal dissection/perforation. Both patients underwent successful lead implantation in LV.

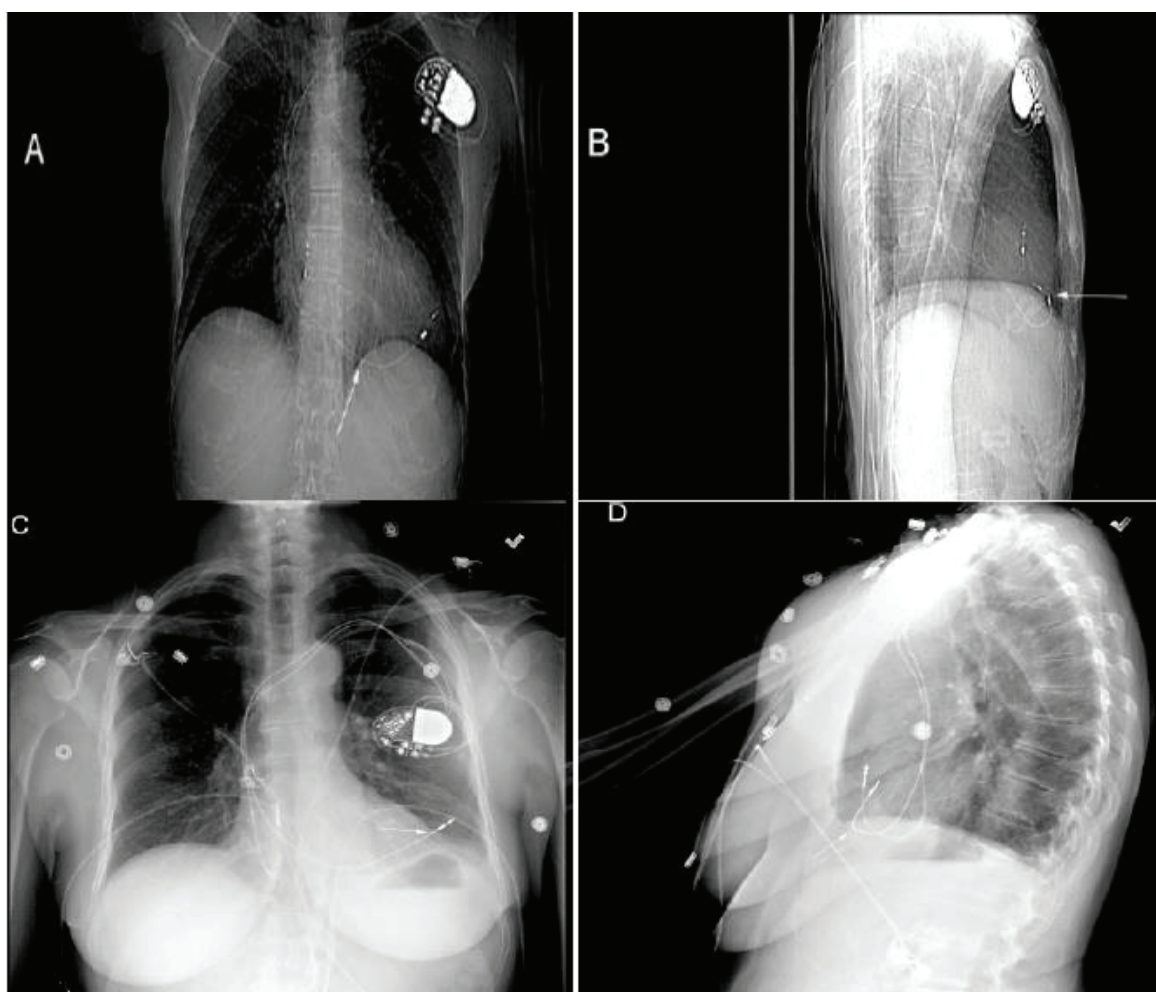


Figure 5. Examples of perforation from RV lead implantation. Images (A) and (B) show RV perforation from the leads perforating the base of the RV (arrow) and re-entering near the RV apex. Images (C) and (D) show RV apex perforations.

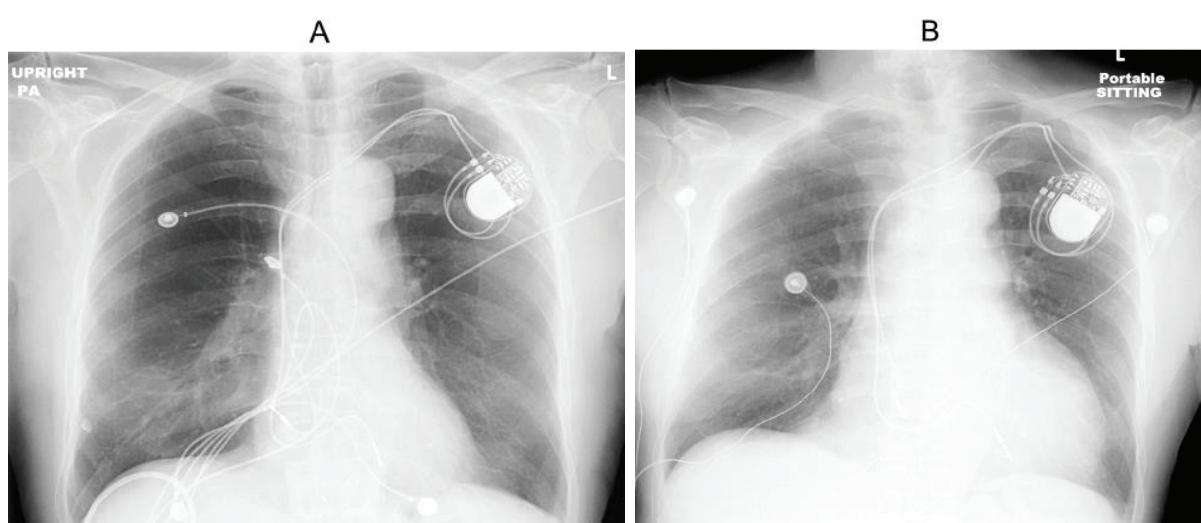


Figure 6. Chest X-ray in a patient with a large pericardial effusion after lead perforation. (A) X-ray immediately after PM implantation shows a regular cardiac silhouette. (B) X-ray of the same patient performed two weeks after implantation because the patient complained of chest pain and dyspnea, shows an enlarged cardiac silhouette. The patient responded well to pericardiocentesis with no need for lead repositioning.

close cooperation with cardiac surgeons, most cases can be treated with pericardiocentesis for symptomatic effusions and lead repositioning in the cath lab.

Figure 6 shows an oversized cardiac silhouette developing after PM implantation caused by pericardial effusion. The effusion was treated with pericardiocentesis (without data suggesting re-accumulation), and there was no need to reposition the lead. Although perforation and tamponade are rare complications, they should not be underestimated since the risk of death due to tamponade resulted in 21.8% in a worldwide study on perforation due to ablation for atrial fibrillation. [11]

Complications of lead implantation in the LV through the Coronary Sinus

These include cardiac perforation, coronary sinus dissection, electric trauma to the native conduction system, failure to implant the lead, lead dislodgement, as well as diaphragmatic stimulation. Coronary sinus dissection, perforations, or cardiac perforation were reported in approximately 2% of the MIRACLE study. [12] Loss of LV capture or diaphragmatic stimulation, leading to the interruption of resynchronization therapy, was seen in 10% and 2% of patients, respectively. [13]

In many cases, new quadripolar leads offer the possibility of various vectors resolving the problem..

Arrhythmias (SVT, VT, VF)

The occurrence of procedure-related stable arrhythmias is rare, with reports for VF up to 0.1% of all patients and up to 0.6% of patients >90 years old. [14] Arrhythmias are often related to the patients themselves. However, in some cases, they are procedure-related, such as irritation of the RVOT, PM stimulus on T wave, re-entry circuits around the lead, or bradycardia-dependent VT facilitated by long pause caused by myopotential inhibition of a VVI pacemaker.

Death

In-hospital death occurs in less than 1% of PM implantations [4,9,14]. The leading causes related to devices are perforations, whereas causes unrelated to the device are myocardial infarction and, more rarely, PE, CVA, HF, and sepsis. [15]

Hematoma

The incidence is approximately 4.9%, leading to extended hospitalization in 2% of the patients. [16] Re-operation occurs in 1% of the total group of patients. It is mainly related to the high doses of heparin, DAPT, triple therapy after stents, and low operator experience. On the other hand, hematoma creation increases the risk of infection. Data suggests that warfarin causes fewer complications than heparin products, and the temporary interruption in anticoagulation increases

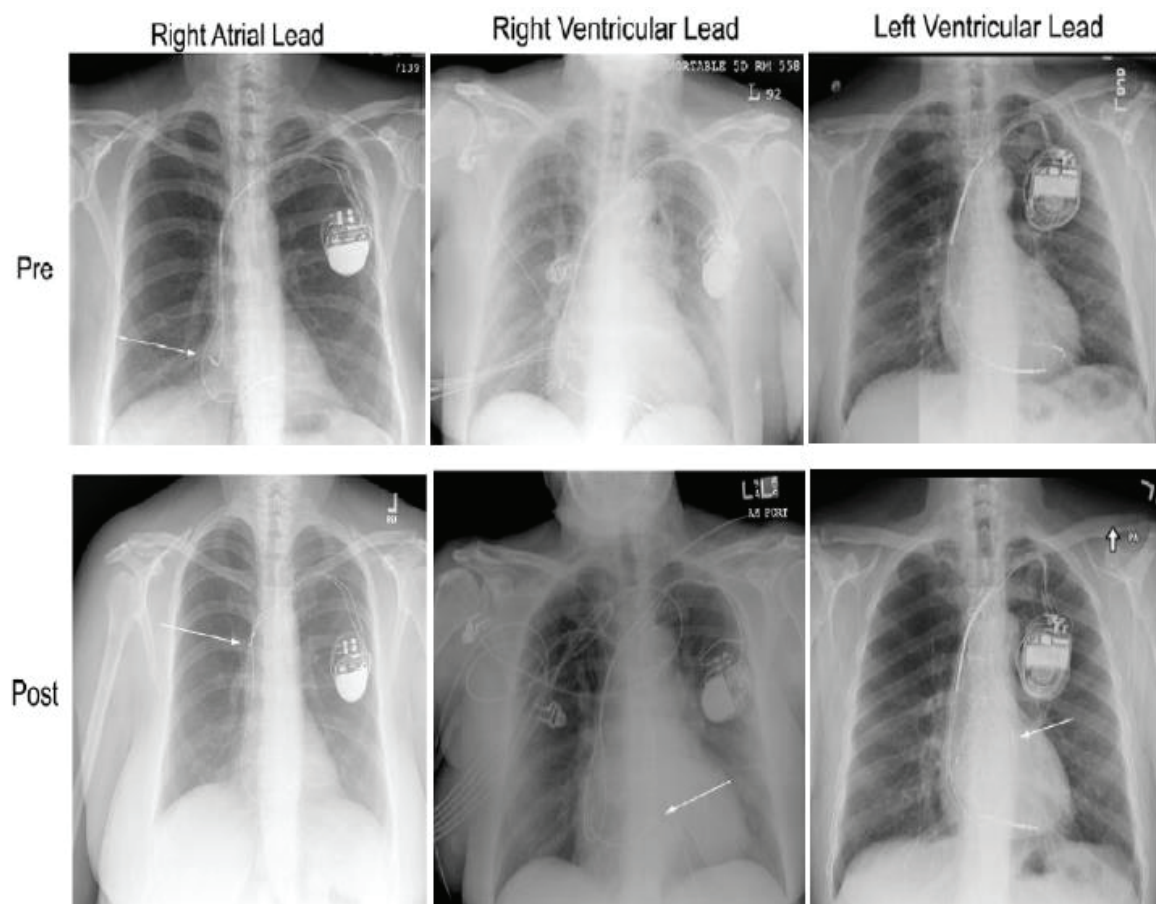


Figure 7. Lead in RA (A), RV (B) and LV (C) before (Pre) and after (Post) being dislodged due to patients' movements

the risk for thrombo-embolic events, whereas switching from warfarin to heparin products increases the risk for hematoma and extended hospitalization. [17]

4- Sub-acute post-implantation complications (<30 days)

Pacemaker and lead dysfunction

Electrocardiographic signs of pacemaker malfunction can be grouped into four categories: failure to capture, failure to output, undersensing, and inappropriate pacemaker rate.

Failure to capture

Various reasons can cause it: lead dislodgment, elevated threshold, inappropriate lead placement, lead fracture, Insulation Failure, Loose set screw, Exit Block (> 4 weeks), Perforation, Battery/circuit Failure, Air in Pocket, and Metabolic/Drugs (Flecainide). Lead dislodgment, the most frequent cause of failure to capture is generally reported to have an incidence of around 1-3%, but it can reach up to 4-6%. [18, 19].

Failure to output

Failure to output can be caused by problems in the battery

and circuit, lead fracture, insulation failure, oversensing, and loose set screws. Generally, several months are needed for complete battery depletion from the beginning of “end of life” signs. Lead fracture is reported in 0.1-4.2% of patients and usually happens near the generator or venous access. Figure 8 shows a lead fracture discovered during re-operation when the patient presented with complete AV block with no ventricular capture one year after pacemaker implantation. Figure 9 shows the chest X-ray of the lead (failure to pace) compared to a pseudo-fracture (usual pacing). Finally, air in the pocket can cause oversensing, as shown in Figure 10. Other electrical signals that can cause oversensing include diaphragmatic myopotentials (especially with extending bipolar sensing), T waves, P waves, and environmental noise

Undersensing

Intrinsic cardiac activity undersensing causes inappropriate pacing output which competes with intrinsic rhythm. Undersensing is mainly caused by lead dislodgment, inappropriate positioning at implantation, or a lead insulation defect

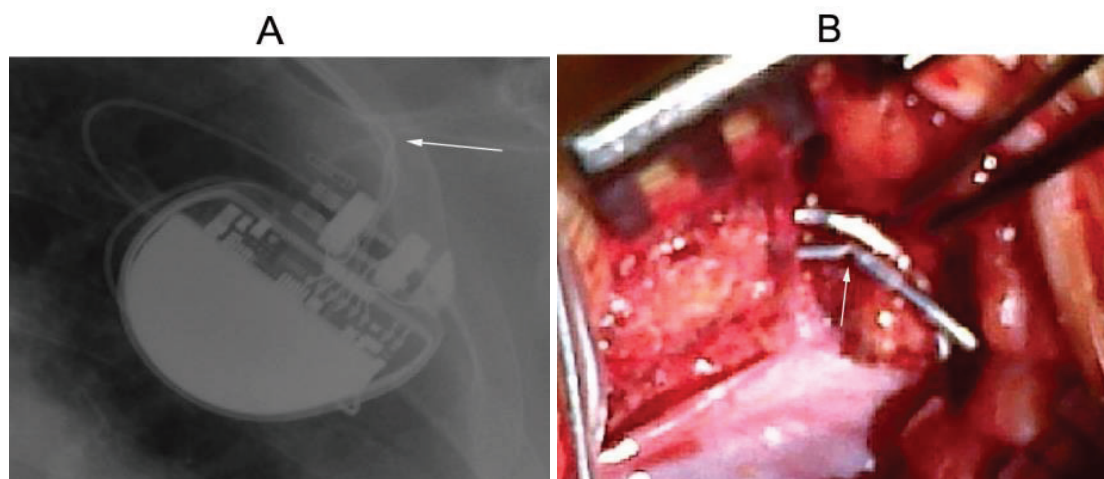


Figure 8. Fracture of lead in RV (A) X-ray and (B) intra-operative

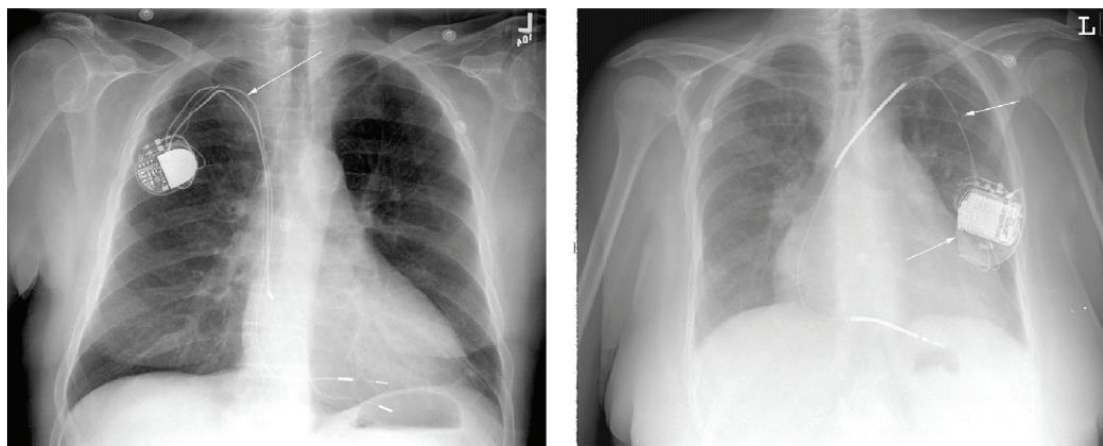
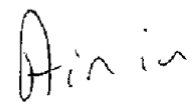


Figure 9. X-rays of (A) lead fractures cause failure to capture and (B) pseudo-fractures due to a digital processing problem, causing an artifact simulating a lead fracture in a usually working lead.



Readmissions

Death

5 - Late complications (> 30 days)

Lead dysfunction/failure - Twiddling

Exit block

Transient causes should be excluded first: metabolic and electrolytic disturbances, drug effects, extreme hypothyroidism (myxedema), and cardiac ischemia. There is an expected elevation of the threshold 2-6 weeks after lead implantation, which is related to local inflammation or reaction to a foreign body in the area of the lead tip. Passive-

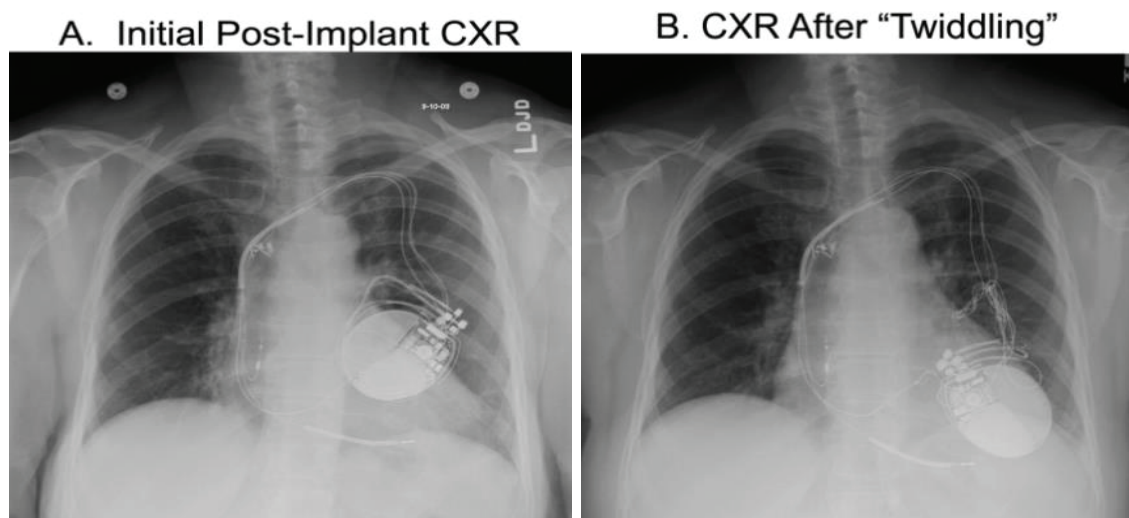


Figure 11. Leads orientation immediately after implantation (A) and after device twiddling (B).

fixation endocardial leads have generally lower thresholds compared to active-fixation leads due to lacking tissue trauma. The threshold elevation degree is markedly blunted by steroid-eluting endocardial leads, which have become preferred because they have overcome high thresholds, increasing device safety and duration. Threshold elevation can happen beyond six weeks after implantation in the chronic maturation phase. While there is a progressive increase in threshold, it can exceed the maximal generator output, known as exit block, and be recognized by a high threshold without radiographic evidence of lead dislodgment. It can be related to inflammation or fibrosis in the lead-myocardial interface and generally occurs over four weeks from implantation. Some patients, especially pediatric patients, are prone to this phenomenon and should undergo lead revision.

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. [22] About 10% of patients can have intra-cardiac vegetation identified on

trans-esophageal echocardiography. However, they can safely undergo percutaneous lead extraction.

Figure 12; A shows an ultrasound image of lead vegetation, B and C show pacemaker wound erosion and infection, whereas D shows a complete expulsion of the Pacemaker. The most common isolated pathogens are gram-positive aerobes, 90% of which are *Staphylococcus* species with a high percentage (50%) of methicillin resistance. The infection risk of pacemakers is lower than that of implantable defibrillators. The presence of epicardial leads and complications in the generator pocket are significant risk factors for ICD infection. In contrast, prolonged hospitalization at implantation and COPD were associated with late infections on the ICD. In one of the most extensive studies on pacemaker infections, repeated operations after the initial implantation were associated with an incremental increase in the risk of infection.[23]. Female gender, older age, and pre-operative use of antibiotics in the initial implantation were associated with a lower risk for later infection. Pacing modality, indication for pacing, and procedural complexity were not independently associated with a later infection. 60% of infections have been found to occur within 90 days of implant, although many infections

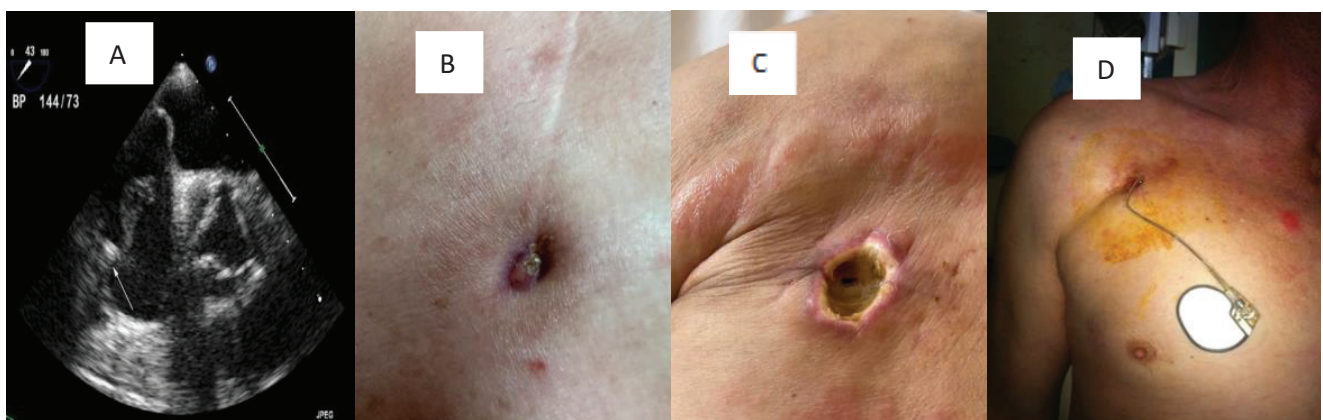


Figure 12. (A) TEE image of a mobile (arrow) vegetation attached to the PM lead in a patient with persistent bacteremia. (B) and (C) show PM wound erosion and infection. (D) shows complete expulsion of the Pacemaker

occur during the late follow-up (> 1 year post-implant). Generator replacement and biventricular resynchronization therapies have also been implicated as independent predictors of infection. [24]

The best-accepted method for treating a device infection is to remove the generator and all implanted leads. In a comprehensive series of device extractions, including 1838 leads, 30-day postoperative mortality was 10%, although there were no deaths directly related to the extraction procedure.[26] Another series with device extractions reported a rate of 0.5% intra-procedural mortality, 4.6% in-hospital mortality, and 2.6% infection relapse within one year of re-implantation.[22]

Pacemaker Syndrome

This syndrome occurs predominantly in single-chamber pacemakers (e.g., VVI or VVIR), and the loss of atrioventricular synchronization causes symptoms. Notably, pacemaker syndrome can occur with any pacing modality if AV synchrony is lost. Symptoms include weakness, malaise, cannon A waves, cough, confusion, or syncope.

Venous Thrombosis

Upper extremities vein thrombosis is rare in the general population, but venous stenosis has been encountered in 33-64% of patients after pacing leads implantations. [27] Statistically significant factors associated with an increase in risk include temporary pacemaker leads, EF<40%, systemic infections, lack of anticoagulation, hormone therapy, personal history of venous thrombosis, and presence of multiple leads. Symptoms include neck and shoulder discomfort, ipsilateral arm edema with cyanosis, cutaneous collateral veins around the shoulder, or jugular vein distension. Venography is considered the golden standard for diagnosis, but compressive ultrasonography is an effective and economical way to confirm the clinical diagnosis. Treatment might include anticoagulation (warfarin and heparin), extraction of the old non-functional lead to create a new venous channel, or venoplasty to reduce the degree of stenosis or to allow for implantation of new leads.

Predicting the risk for procedural complications

There is data that older patients are at higher risk for complications after pacemaker implantation. Armaganijan et al. [28] found that early complications occurred in 5.1% of patients >75 years old compared to 3.4% of those <75 years old. Using concomitant temporary leads or using steroids within seven days of implantation has been shown to increase the risk for pericardial effusion. Weak predictors for post-implantation pericardial effusion were active fixation leads, BMI<20, older age, and longer fluoroscopy time. Pneumothorax has been seen often in older and low-weight women. A previous study examining predictors of complications in very old patients [11] undergoing pacemaker implantation found similar complication percentage rates compared to the younger patients but a higher 30-day mortality rate (which can be attributed to a higher all-cause mortality rate in this age group). Multivariate analysis showed that female gender, type of device, and urgent/emergent implantation demonstrated a non-significant trend toward increased readmission rate.

High-volume operators (>12 implants/year) compared to low-volume operators (<12 implants/year) have also shown different complication rates. [5] More complex devices (dual-chamber devices compared to single-chamber devices) were associated with a higher rate of complications. However, other studies have found no difference between them. Lastly, training implanting physicians might lead to a lower rate of lead dislodgment.

Conclusions

Major and minor complications occur in approximately 4-7% of patients within 30 days from pacemaker implantation. [4, 14, 20] Permanent pacemakers are often implanted in patients older than 65, the most rapidly growing proportion of the population. At the same time, pacemaker implantation is associated with an 11-40% risk for complications in the pediatric population; the most common complications in this group of patients are pneumothorax, hematoma, and infection. [29] Figure 13 shows the incidence of the most

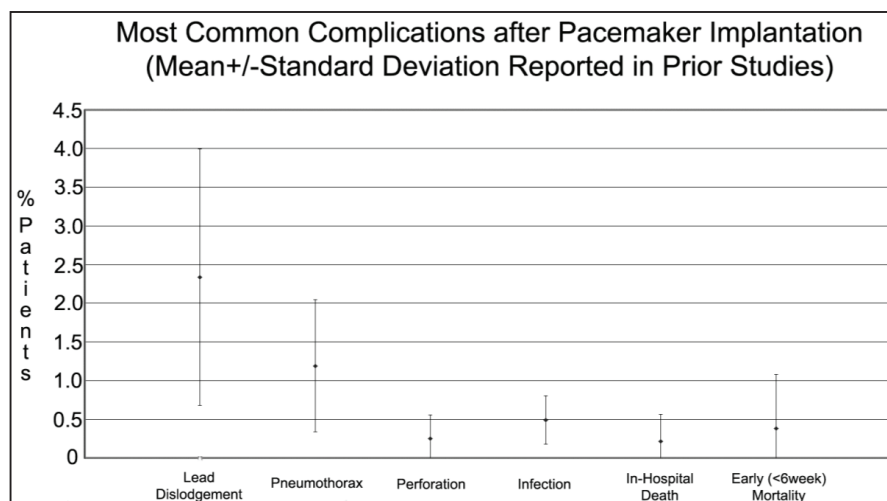


Figure 13. Most common complications after pacemaker implantation

common complications after pacemaker implantation. Prompt recognition and treatment of complications after pacemaker implantation is essential for all implanting physicians, regardless of their background.

COI Statement: This paper has yet to be submitted in parallel, presented fully or partially at a meeting, podium, or congress, published, or submitted for consideration beforehand.

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References

- Kurtz SM, Ochoa JA, Lau E, Shkolnikov Y, Pavri BB, Frisch D, Greenspon AJ, Implantation trends and patient profiles for pacemakers and implantable cardioverter defibrillators in the United States: 1993-2006," *Pacing Clin Electrophysiol*, V. 33, No. 6 (June 1, 2010), pp. 705-11.
- Rosenheck S, Geist M, Weiss A, Hasin Y, Weiss TA, Gotsman MS, "Permanent Cardiac Pacing in Octogenarians," *Amer J Ger Card*, V. 4, No. 6 (1995), pp. 42-47.
- Nowak B and Misselwitz B, "Effects of increasing age onto procedural parameters in pacemaker implantation: results of an obligatory external quality control program," *Europace*, V. 11 (November 2008), pp. 75-79.
- Ormir Shurdha , Endri Hasimi, Arkel Duka, Elona Çollaku, Ervina Shirka, Meriton Siqeca, Ela Petrela, Vilma Paparisto, Artan Goda, ""Profil i implantimit të pacemakerave në Qendrën Spitalore Universitare "Nënë Tereza, Tiranë", *Revista Shqiptare e Kardiologjise*, Vellimi XII, nr 2, faqe 3, Nentor 2014
- Parsonnet V, Bernstein AD, Lindsay B, "Pacemaker-Implantation Complication Rates: An Analysis of Some Contributing Factors," *JACC*, V. 13, No. 4 (March 15, 1989), pp. 917-921.
- Aggarwal RK, Connelly DT, Ray SG, Ball J, Charles RG, "Early complications of permanent pacemaker implantation: no difference between dual and single chamber systems," *Br Heart J*, V. 73 (1995), pp.571-575.
- Wiegand UKH, Bode F, Bonnemeier H, Eberhard F, Schlei M, Peters W, "Long-Term Complication Rates in Ventricular, Single Lead VDD, and Dual Chamber Pacing," *PACE*, V. 26 (October 2003), pp. 1961-1969
- Belott P, "How to access the axillary vein," *Heart Rhythm*, V. 3, No. 3 (March 2006), pp. 366-369.
- Williams JL, Lugg D, Gray R, Hollis D, Stoner M, Stevenson R, "Patient Demographics, Complications, and Hospital Utilization in 250 Consecutive Device Implants of a New Community Hospital Electrophysiology Program," *American Heart Hospital Journal*, V. 8, No. 1 (Summer, 2010), pp. 33-39
- Hirschl DA, Jain VR, Spindola-Franco H, Gross JN, Haramati LB, "Prevalence and Characterization of Asymptomatic Pacemaker and ICD Lead Perforation on CT," *PACE*, V. 30 (January 2007), pp. 28-32
- Cappato R, Calkins H, Chen S-A, Davies W, Iesaka Y, Kalman J, Kim Y-H, Klein G, Natale A, Packer D, Skanes A, "Prevalence and Causes of Fatal Outcome in Catheter Ablation of Atrial Fibrillation," *JACC*, Vol. 53, No. 19 (May 12, 2009), 1798-803.
- Leon AR, Abraham WT, Curtis AB, et al.; for the MIRACLE Study Program, "Safety of Transvenous Cardiac Resynchronization System Implantation in Patients with Chronic Heart Failure: Combined Results of Over 2000 Patients from a Multicenter Study Program," *J Am Coll Cardiol*, 2005;46(12):2348-56.
- Knight BP, Desai A, Coman J, Faddis M, Yong P, "Long-term retention of cardiac resynchronization therapy," *JACC*, V. 44, No. 1 (July 7, 2004), pp. 72-77
- Nowak B and Misselwitz B, "Effects of increasing age onto procedural parameters in pacemaker implantation: results of an obligatory external quality control program," *Europace*, V. 11 (November 2008), pp. 75-79
- Schulza N, Puschelb K, Turkc EE, "Fatal complications of pacemaker and implantable cardioverter defibrillator implantation: medical malpractice?" *Interactive CardioVascular and Thoracic Surgery*, V. 8 (2009), pp. 444-448
- Wiegand UKH, LeJeune D, Boguschewski F, Bonnemeier H, Eberhardt F, Schunkert H, Bode F, "Pocket Hematoma After Pacemaker or Implantable Cardioverter Defibrillator Surgery: Influence of Patient Morbidity, Operation Strategy, and Perioperative Antiplatelet/Anticoagulation Therapy," *CHEST*, V. 126, No. 4 (October 2004), pp. 1177-1186.
- Ahmed I, Gertner E, Nelson WB, House CM, Dahiya R, Anderson CP, Benditt DG, Zhu DWX, "Continuing warfarin therapy is superior to interrupting warfarin with or without bridging anticoagulation therapy in patients undergoing pacemaker and defibrillator implantation," *Heart Rhythm*, V. 7, No. 6 (June 2010), pp. 745-749.
- Hargreaves MR, Doulalas A, Ormerod OJM, "Early Complications Following Dual Chamber Pacemaker Implantation: 10-Year Experience of a Regional Pacing Centre," *Eur JCPE*, V. 5, No. 3 (1995), pp. 133-138
- Kiviniemi MS, Pirnes MA, Eranen HJK, Kettunen RVJ, Hartikainen JEK, "Complications Related to Permanent Pacemaker Therapy," *PACE*, V. 22 (May 1999), pp. 711-720.
- Stevenson R, Lugg D, Gray R, Hollis D, Stoner M, Williams JL, "Pacemaker Implantation in the Extreme Elderly," *Journal of Interventional Cardiac Electrophysiology*, V. 33, No. 1 (January 2012), pp. 51-58.
- T. Fahraeus and C. J. Hoijer, "Early pacemaker twiddler syndrome," *Europace*, Vol. 5 (July 2003), pp
- Tarakji KG, Chan EJ, Cantillon DJ, Doonan AL, Hu T, Schmitt S, Fraser TG, Kim A, Gordon SM, Wilkoff BL et al, "Cardiac implantable electronic device infections: Presentation, management, and patient outcomes," *Heart Rhythm* V. 7, No. 8 (August 2010), pp. 1043-104

23. Johansen JB, Jørgensen OD, Møller M, Arnsbo P, Mortensen PT, Nielsen JC, "Infection after pacemaker implantation: infection rates and risk factors associated with infection in a population-based cohort study of 46299 consecutive patients," *Eur Heart J*, V. 32, No. 8 (April 2011) pp. 991–998
24. Nery PB, Fernandes R, Nair GM, Sumner GL, Ribas CS, Menon SM, Wang X, Krahn AD, Morillo CA, Connolly SJ, Healey JS, "Device-related infection among patients with pacemakers and implantable defibrillators: incidence, risk factors, and consequences," *J Cardiovasc Electrophysiol*, V. 21, No. 7 (July 2010), pp. 786-90.
25. Wilkoff BL, "How to treat and identify device infections," *Heart Rhythm*, V. 4 (2007), pp. 1467–1470
26. Grammes JA, Schulze CM, Al-Bataineh M, Yesenosky GA, Saari CS, Vrabel MJ, Horrow J, Chowdhury M, Fontaine JM, Kutalek SP, "Percutaneous pacemaker and implantable cardioverter-defibrillator lead extraction in 100 patients with intracardiac vegetations defined by transesophageal echocardiogram," *J Am Coll Cardiol*, V. 55, No. 9 (March 2010), pp. 886-94.
27. Oginozawa Y, Abe H, Nakashima Y, "The incidence and risk factors for venous obstruction after implantation of transvenous pacing leads," *Pacing Clin Electrophysiol*, V. 25 (2002), pp. 1605-1611
28. Armaganijan LV, Toff WD, Nielsen JC, Andersen HR, Connolly SJ, Ellenbogen KA, Healey JS, "Are Elderly Patients at Increased Risk of Complications Following Pacemaker Implantation? A Meta-Analysis of Randomized Trials," *Pacing Clin Electrophysiol*, Epub ahead of Print, (2011 Oct 31).
29. Czosek RJ, Meganathan K, Anderson JB, Knilans TK, Marino BS, Heaton PC, "Cardiac rhythm devices in the pediatric population: Utilization and complications," *Heart Rhythm*, V. 9, No. 2 (Feb 2012), pp. 199-208