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Pulmonary, Cardiovascular and Mechanical Complications of Implantable Cardioverter Defibrillators (ICDs)

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1. Introduction

Automatic implantable cardioverter defibrillators (ICDs) have been used widely for the prevention of recurrent sudden cardiac death and the treatment of life-threatening ventricular tachyarrhythmias in ambulatory patients, since 1980 (Mirowski M et al., 1982). The remarkable efficacy of the ICD has been demonstrated to be 95% at 3 years and this has led to its ever-increasing use (Linl G et al., 2009).

Despite this benefit, there are many potential complications associated with ICDs (Pfeiffer D et al., 1994; Linl G et al., 2009). Nevertheless, the rate of complications related to the ICDs has fallen markedly with the evolution from a large device that required an abdominal pocket and insertion of an epicardial lead system via thoracotomy to the current use of much smaller transvenous pectoral devices (Krohn J et al., 2001; DiMarco JP et al., 2003). The incidence of ICD complications is difficult to determine due to inconsistent definitions and the lack of mandatory reporting. Nevertheless, women are more likely than men to have in-hospital adverse events related to ICD implantation (Peterson PN et al., 2009).

2. Definitions, aims and search strategy

The European Community and the International Standards Organization have provided standard criteria for adverse events observed during trials with implantable medical devices, defining an adverse event as any undesirable clinical occurrence and taking into account the severity and relationship to the implanted device. This does not include any information regarding the underlying technical or clinical cause (Rosenqvist M et al., 1998). Thus, complications are defined as any undesirable clinical occurrence related to the ICD implantation and function including intraoperative mortality and 30 day post-operative mortality.

This chapter aims to perform a systematic review of the published literature to provide the reader with the best available evidence and most current medical knowledge regarding pulmonary, cardiovascular and mechanical complications that occur due to the implantation of ICDs. Moreover, this chapter will attempt to ameliorate the weaknesses inherent in the current medical literature and scientific published medical literature regarding the issue.

Therefore, English language and adult population published literature from 1980 to December 2010 was searched using PubMed, Current Contents, Cochrane Library, Embase, Cinahl, Google Scholar and supplemented by a manual review of bibliographies of all relevant papers. Proceedings from relevant conferences, reference lists, relevant clinical trials and research registers were also searched. English language published literature from 1980 to 2010 was sought utilizing the following search strategy: *implantable cardioverter defibrillators* [MeSH] *and/or pulmonary and/or cardiac and/or mechanical/ device-related complications.* Studies were required to present sole evidence justifying the presence of complications secondary to the ICD. Data from each study was extracted by one author and reviewed by the two others.

3. Pulmonary complications

The majority of pulmonary complications were associated with the placement technique of the ICDs (Kuck KH et al., 2000). Implantation of an ICD involves placement of both the ICD lead system and the pulse generator. Current ICD lead systems are typically placed transvenously via the axillary, subclavian, or cephalic vein. This approach has largely replaced the surgical epicardial lead placement of the ICD which was associated with considerable postoperative morbidity (Chevalier P et al., 1996; Kuck KH et al., 2000) and with pulmonary complications that are unique to thoracotomy such as atelectasis with pneumonia, symptomatic pleural effusions, ARDS (adult respiratory distress syndrome).

In 1985, Lurie AL et al reported complications in a series of 22 patients that underwent ICD placement by thoracotomy. Among them, 77% presented with atelectasis, infiltrates or pleural effusion in the chest radiograph and 13% with pneumothoraces resulting from transvenous lead placement. The authors (Lurie AL et al., 1985) reported these as transient postimplantation complications. Pneumothorax, infections, and bleeding tend to occur soon after implant (Krohn J et al., 2001; Krohn J et al., 2003). As subclavian vein puncture may be associated with pneumothorax in approximately 1% of patients, the cephalic vein should be preferred for nonthoracotomy lead placement (Krohn J et al., 2003).

Pneumothorax occurs uncommonly and is directly related to operator experience, the difficulty of the subclavian puncture, and is almost eliminated using the cephalic cut-down technique. However, these traditional comparisons may become obsolete as the axillary vein cannulation technique (Martin C et al. 1996) threatens to eliminate this controversy. Often the pneumothorax is asymptomatic and noted in routine follow up plain chest radiograph.

Recurrent hemoptysis has been reported as a delayed complication on the grounds of ICD placement (Kao N et al., 1991; Verheyden CN et al., 1994; Dasgupta A et al., 1998; Driscoll JA et al., 2005). Clinicians caring for patients with an ICD should be aware of this complication and consider patch erosion into the bronchus as a cause, albeit rare, in the differential diagnosis of recurrent hemoptysis. Rarely, the patch erosion can be visualised bronchoscopically as the underlying cause of hemoptysis (Dasgupta A et al., 1998). Common radiographical findings can be a pleural infusion and vague infiltrates (Dasgupta A et al., 1998). Patch removal may reveal destruction of a significant part of the underlying bronchus, thereby prohibiting bronchial reconstruction. In patients who have undergone placement of an automatic ICD using pericardial or epicardial defibrillator patches and present with hemoptysis, bronchopericardial fistula should be also suspected (Nolan RL et al., 1999). Air between a defibrillator patch and the heart on chest radiographs or CT is diagnostic.

Infections of the ICD electrodes causing recurrent pneumonias are common and could lead to ARDS if remain uncontrolled. In reported cases, the ICD electrodes grew Methicillin sensitive *Staphylococcus epidermidis/aureus,* Methycillin Resistant *Staphylococcus aureus, Haemophillus Paranfluenzae, Aspergillus fumigatus, Pseudomonas aeruginosa,* while clots on the ICD patch grew *Haemophillus Influenzae (*Dasgupta A et al., 1998; Cook RJ et al., 2004; Pai RK et al., 2004; Ioannides MA et al., 2006; Rusanov A, Spotnitz HM. 2010*);* these pathogens are quite commonly isolated in infections caused by ICD placement. Infection of the patch can ultimately lead to its dislodgement and migration to the lung. Regarding the culture findings, it is difficult to be sure whether there was primary device infection with the pathogens presumably introduced during the ICD placement, a primary pneumonia with subsequent seeding of the ICD patch, or an underlying hematoma formation at the time of patch placement which then secondarily became infected, leading to subsequent erosion and pulmonary complications. Concomitantly, infections have increased the frequency of lead extraction. The frequency of lead infections has also risen faster than expected based solely on the number of implanted leads (Voigt A et al., 2006)

Patients developing severe pneumonias could also present with septic pulmonary embolism secondary to the infection(Cook RJ et al., 2004). This typically produces abnormalities on chest radiography, but its appearance is not uniform (Ryu JH et al., 2003; Huang RM et al., 1989; Rossi SE et al.,2000); multiple bilateral cavitary nodules at the lung periphery are most typical (Wong KS et al., 2002). Characteristic CT findings are discrete nodules in various stages of cavitation with visible feeding vessels (Rossi SE et al., 2000). However, multifocal cavitary lesions in the lung can also be associated with neoplasms, pulmonary infarctions, abscesses, vasculitides, congenital abnormalities, rheumatoid nodules, and pneumoconioses (Ryu JH et al., 2003). Consequently, correlation with the clinical context is important to narrow the broad differential diagnosis.

However, in this context, in 1997 Lick SD et al considered a cavitary lesion in a patient bearing an ICD and presenting with cough, malaise and weight loss commonly suspicious for a cavitary malignant neoplasm. Fiberoptic bronchoscopy was performed and no evidence of a malignant tumour was seen. Surprisingly, a defect was found in the lingular bronchus through which the bronchoscope could pass, opening into a large air-filled cavity, in part bordered by the patch itself. Cultures from the cavity debris grew *Aspergillus* and *Staphylococcus aureus*.

Hemothorax has also been reported as a late pulmonary complication of an ICD placement usually after the onset of pleuritic pain (Kremmers MS et al., 1995; Quigley RL et al., 1996). This complication results from trauma to the great vessels rather than the lung. The risk can be minimised by direct inward and outward passes of the puncture needle rather than a side-to-side potentially lacerating movement (Pavia S, Wilkoff B. 2001).

Air embolism has been reported during deep inspiration at the time of central venous access causing significant air to be drawn into the venous system due to the physiological negative pressure developed (Pavia S, Wilkoff B. 2001). It can be prevented through operator care and using introducers with hemostatic valves. The diagnosis is obvious because it is heralded by a hissing sound as the air is sucked in and with the fluoroscopic confirmation that follows. Patients are surprisingly tolerant of this occurrence. However, respiratory distress, hypotension, and arterial oxygen desaturation may occur depending on the size of the embolus and 100% oxygen should be administered alone with ionotropic support in some cases (Ellenbogen KA, Wood MA. 2005). Aspiration of the embolus from the right

heart has also been successful. However, usually no therapy is required, as the air is filtered and consequently absorbed by the lungs.

4. Cardiac complications

Endocarditis secondary to leads' infection is one of the most common cardiac complications of ICDs. The majority of infections are caused by coagulase negative *Staphylococcus* and *Staphylococcus aureus* and rarely *Staphylococcus lugdunensis* (Anguera et al 2005; Liu PY et al., 2010; Chopra A et al., 2010). Although the risk of infection of intracardiac devices is well known, the clinical presentation of this complication can be insidious, delayed in onset and difficult to diagnose. The onset of symptoms can be in the first 6 months (Cacoub P et al., 1998) or in the first few years. Right sided endocarditis on the grounds of *Aspergillus fumigatus* infection has been reported (Cook RJ et al., 2004) as a delayed complication. The infection presented as persistent pulmonary infiltrates and anemia more than 2 years after the implantation of the device. Endocarditis caused by *Staphylococcus capitis* has also been reported and presented with a subacute course (Cone LA et al., 2005). Infrequently, gram negative bacilli can also cause lead infection and endocarditis. *Klebsiella pneumoniae* is a pathogenic gram negative bacillus which has been reported to cause ICD associated endocarditis (Pai RK et al., 2006).

Acute pericardial effusion and tamponade can occur secondary to lead perforation of the heart. This is an infrequent complication of device implantation which may also present as a subacute process days later, or even as a delayed process (Mahapatra S et al., 2005; Khan MN et al., 2005; Henrikson CA et al., 2006).

Some series have suggested an increase in ventricular arrhythmias after the implantation of epicardial patch electrodes (Bocker D et al., 1993). However, one study which randomized 900 patients undergoing coronary artery bypass grafting to an ICD or no ICD found no difference in the incidence of postoperative ventricular or supraventricular arrhythmias between the two groups (Curtis AB et al., 1998).

Many patients who receive an ICD have left ventricular dysfunction. An unresolved question is whether worsening myocardial function would affect the defibrillation threshold. In an animal model, the development of congestive heart failure did not alter defibrillation energy requirements (Friedman PA et al., 1998).

Multiple low energy defibrillation shocks via an endocardial right ventricular electrode cause significant myocardial damage in dogs, manifested as mitochondrial injury and dysfunction (Schirmer U et al., 1997). Although these changes are more apparent in the right ventricle, they are also seen in the left ventricle.

Myocardial necrosis is another cardiac ICD complication. Rapid consecutive shocks from the ICD results in elevation in serum troponin I, reflecting subtle injury to the heart. In one series of 12 patients who received a mean of 6 shocks with a mean cumulative energy of 112 J during ICD implantation, 5 had elevated troponin I levels which peaked within the first 12 hours after the shocks and were normal or near normal by 24 hours (Joglar JA et al., 1999). Only 1 of these patients had an increase in CK-MB and no patient had associated ECG changes.

5. Mechanical complications

Mechanical complications can be divided into lead/device/pocket related and inappropriate defibrillator shocks.

Lead-related problems occur in approximately 5% of patients, including lead dislodgement, fracture, and insulation defects, which can lead to either over- or undersensing. Lead-related problems can occur at any time during long-term follow-up (Kron J et al., 2001; Yap SC et al., 2007) with the vast majority of lead dislodgments occurring within the first post-operative months. With increasing age of the transvenous lead systems, a growing number of lead fractures and insulation defects have to be expected (Mewis C., 1997; Kron J., 2003). Necessity for operative revision is reported for 6% within 1 year of initial implant and up to 15% during 4 years (Kron J et al., 2001). A relation between the incidence of lead-related complications and the number of leads used in ICD systems has been reported (Takahashi T et al.,2002). Lead dislodgments occur significantly more frequently in patients with dual chamber ICDs (12%) and in patients with biventricular ICDs (19%) when compared with single chamber ICDs (3%).

One study evaluated 171 patients who received an epicardial lead system and were followed for 4 years: lead malfunction occurred in 11% of patients and in up to 28% with some systems (Brady PA et al., 1998). The majority of lead malfunctions occurred more than 2 years after implantation; most patients were asymptomatic (58%). Another report evaluated 132 patients who received a transvenous lead system and pectoral implantation and were followed for 30 months (Mehta D et al., 1998). A 13% incidence of erosion of the lead insulation was noted when systems using long transvenous leads and relatively larger generators were used. This problem is caused by pressure of the generator against the lead.

Pocket-related complications including skin erosion, hematoma and seroma, wound infection, or device migration usually occur within the first 6 months after implantation (Gold MR et al., 1996).

Device-related complications include migration, skin erosion, and necrosis (due to the size and weight of the generator) and premature battery depletion. Fortunately these problems are uncommon, occurring in less than 2% of patients. In addition, hematomas or seromas can form in the pulse generator pocket.

Twiddler's syndrome can also be included in the device-related complications, in which twisting or rotating the device in its pocket results in lead dislodgement and device malfunction, can occur in patients with an ICD. It is most likely to develop when the device is implanted in the abdomen of an obese patient who is able to rotate it within the abdominal pocket (Boyle NG et al. 1998). Patients most often present with an increase in bradycardic pacing threshold or lead impedance; however, there is a possibility that the device will fail to sense and treat an arrhythmia. Careful suturing of the device to the fascia and matching pocket and device size is important to avoid this complication.

Defibrillation-related problems represent a serious technical entity. A high defibrillation energy requirement (over 24 Joules) provides little margin for safety. However, in the absence of any changes in the clinical status of the patient, defibrillation energy requirements with a transvenous lead system are generally stable over a three month period (Newman D et al., 1997).

Inappropriate shocks most often occur due to supraventricular tachycardia, self-terminating VT, and sensing artifacts, e.g., myopotentials or T wave oversensing. It has been reported that inappropriate shocks for supraventricular tachyarrhythmias are more often in younger patients and in patients who have nonischemic dilated cardiomyopathy compared to patients with coronary artery disease (Alter P et al., 2005; Lin G et al., 2009; Lee DS et al., 2010). Potential induction of fatal ventricular fibrillation by inappropriate shocks is known from anecdotal reports (Messali A et al., 2004). Use of leads with true bipolar sensing can reduce

sensing artifacts. The most important precipitating factor is myocardial ischemia, but other causes are electrolyte disturbances, and episodes of congestive heart failure resulting in an increase in sympathetic tone. However, newer atrial/dual chamber devices can effectively detect specific atrial and ventricular arrhythmias and can accurately discriminate between atrial tachycardia/atrial flutter and atrial fibrillation (Swerdlow CD et al., 2000). These devices can be programmed for mode switching to prevent inappropriate tracking of an atrial arrhythmia; to withhold inappropriate ventricular therapy; and to deliver appropriate therapy for the atrial tachyarrhythmia, such as pace termination of atrial flutter. Treatment with a sufficient dose of β-blockers may help to decrease the number of inappropriate shocks due to atrial flutter of fibrillation by slowing the ventricular rate (Pacifico A et al., 1999). In addition, prognostic benefits of β-blockers are well known in patients with coronary artery disease as well as in patients with heart failure (Packer M et al., 2001). Therefore, all patients with coronary heart disease and with non-ischemic dilated cardiomyopathy should receive β-blockers as standard therapy, unless there is a contraindication. Furthermore, it has been found a significantly lower incidence of inappropriate shocks due to supraventricular tachyarrhythmias in patients with versus without amiodarone therapy (Alter P et al., 2005). In addition, the rate cut-off for detecting ventricular tachyarrhythmias should not be programmed too low in order to decrease the overlap with supraventricular tachyarrhythmias. Since the latter is not possible in many patients with symptomatic slow ventricular tachyarrhythmias, state-of-the-art ICD discrimination algorithms should be used in order to distinguish supraventricular arrhythmias from VT.

Moreover, ICD related shocks to rescuers during CPR have been reported (Clements PA., 2003, Siniorakis E et al., 2009). In the case presented by Siniorakis E et al., CPR was performed in a patient bearing an ICD and presenting cardiac arrest with an initial rhythm of pulseless electrical activity. Ten minutes after starting CPR, the rescuer received an electric shock. This first shock of 21.9 J affecting the rescuer was triggered by chest compression-related muscular noise. This was mistaken by the ICD as ventricular fibrillation. In this context, there should be a warning from ICD manufacturers about the risks of shocks from ICDs during CPR.

An increase in the chronic defibrillation threshold may also occur (Martin DT et al., 1995). This problem may result from intense fibrosis and the cumulative acute damage produced by defibrillation discharges at the ICD electrode-myocardial interface (Epstein AE 1998 et al., 1998). However, the increase in defibrillation threshold may not be clinically significant with modern devices. In addition, changing the polarity of the leads may result in a reduction in the defibrillation threshold (Schauerte P et al., 1997).

6. Conclusions

The potential complications of ICDs are significant in terms of diversity and patient impact. For this reason, the decision to implant a device should be based on sound guidelines with definite expected patient benefit. Although it is a relatively simple procedure the potential complications may, at times, be life-threatening. Early recognition of these complications is the prerequisite for advances in ICD technology, in management strategies to avoid their recurrence and in improved patient quality of life. With a clear understanding of the accepted implant indications and possible complications and a meticulous approach to the implant and post-implant follow-up, the incidence of complications can be minimised.

7. References

[1] Anguera I, Del Río A, Miró JM, Matínez-Lacasa X, Marco F, Gumá JR, Quaglio G, Claramonte X, Moreno A, Mestres CA, Mauri E, Azqueta M, Benito N, García-de la María C, Almela M, Jiménez-Expósito MJ, Sued O, De Lazzari E, Gatell JM; Hospital Clinic Endocarditis Study Group. Staphylococcus lugdunensis infective endocarditis: description of 10 cases and analysis of native valve, prosthetic valve, and pacemaker lead endocarditis clinical profiles. Heart. 2005 Feb;91(2):e10.

[2] Bocker, D, Block, M, Isbruch, F, et al. Comparison of frequency of aggravation of ventricular tachyarrhythmias after implantation of automatic defibrillators using epicardial versus nonthoracotomy lead systems. Am J Cardiol 1993; 71:1064.

[3] Boyle, NG, Anselme, F, Monahan, KM, et al. Twiddler's syndrome variants in ICD patients. Pacing Clin Electrophysiol 1998; 21:2685.

[4] Brady, PA, Friedman, PA, Trusty, JM, et al. High failure rate for an epicardial implantable cardioverter-defibrillator lead: Implications for long-term follow-up of patients with an implantable cardioverter-defibrillator. J Am Coll Cardiol 1998; 31:616.

[5] Cacoub P, Leprince P, Nataf P, Hausfater P, Dorent R, Wechsler B, Bors V, Pavie A, Piette JC, Gandjbakhch I. Pacemaker infective endocarditis. Am J Cardiol. 1998 Aug 15;82(4):480-4.

[6] Clements PA. Hazards of performing chest compressions in collapsed patients with internal cardioverter defibrillators. Emerg Med J. 2003 Jul;20(4):379-80.

[7] Chevalier, P, Moncada, E, Canu, G, et al. Symptomatic pericardial disease associated with patch electrodes of the automatic implantable cardioverter defibrillator: An underestimated complication? Pacing Clin Electrophysiol 1996; 19:2150.

[8] Chopra A, Gulati D, Woldenberg N, Singh M. Intracardiac lead endocarditis due to Staphylococcus lugdunensis.Int J Infect Dis. 2010 Sep;14 Suppl 3:e291-3. Review.

[9] Cone LA, Sontz EM, Wilson JW, Mitruka SN. Staphylococcus capitis endocarditis due to a transvenous endocardial pacemaker infection: case report and review of Staphylococcus capitis endocarditis. Int J Infect Dis. 2005 Nov;9(6):335-9.

[10] Cook RJ, Orszulak TA, Nkomo VT, Shuford JA, Edwards WD, Ryu JH.Aspergillus infection of implantable cardioverter-defibrillator. Mayo Clin Proc. 2004 Apr;79(4):549-52.

[11] Curtis, AB, Bigger, JT, DiMarco, JP, et al, for the CABG Patch Trial Investigators. Epicardial; cardioverter-defibrillators do not cause postoperative arrhythmias. Am J Cardiol 1998; 82:1114.

[12] Epstein, AE, Kay, N, Plumb, VJ, et al. Gross and microscopic pathologic changes associated with nonthoracotomy implantable defibrillator leads. Circulation 1998; 98:1517.

[13] Friedman, PA, Foley, DA, Christian, TF. Stability of the defibrillation probability curve with the development of ventricular dysfunction in the canine rapid paced model. Pacing Clin Electrophysiol 1998; 21:339.

[14] Gold MR, Peters RW, Johnson JW, Shorofsky SR. Complications associated with pectoral cardioverter-defibrillator implantation: Comparison of subcutaneous and submuscular approaches. Worldwide Jewel Investigators. J Am Coll Cardiol 1996; 28: 12781282.

[15] Henrikson CA, Leng CT, Yuh DD, Brinker JAComputed tomography to assess possible cardiac lead perforation. Pacing Clin Electrophysiol. 2006 May;29(5):509-11.
[16] Huang RM, Naidich DP, Lubat E, Schinella R, Garay SM, McCauley DI. Septic pulmonary emboli: CT-radiographic correlation. AJR Am J Roentgenol. 1989;153:41-45.
[17] Ioannides MA, Christodoulides TA, Georgiou GM, Konis PA, Nicolaides EP. Recurrent Pneumonia Related To Pacemaker Lead Infection. The Internet Journal of Cardiology. 2006; 3(2)
[18] Joglar, JA, Kessler, DJ, Welch, PJ, et al. Effects of repeated electrical defibrillations on cardiac troponin I levels. Am J Cardiol 1999; 83:270.
[19] Kao N, Messersmith RN, Klich J.Hemoptysis complicating AICD patch placement controlled by temporary selective bronchial balloon occlusion.Chest. 1991 May;99(5):1301-3.
[20] Kenneth A. Ellenbogen MD, Mark A. Wood MD (2005). Cardiac pacing and ICDs. BlackWell Publishing
[21] Khan MN, Joseph G, Khaykin Y, Ziada KM, Wilkoff BL. Delayed lead perforation: A disturbing trend. Pacing Clin Electrophysiol 2005; 28:251–253.
[22] Kremers MS, Hastings JC 3rd, Herndon WM, Howe H. Delayed ICD patch migration after thoracoscopic placement with resultant hemothorax. Pacing Clin Electrophysiol. 1995 Jun;18(6):1335-7.
[23] Kron J, Herre J, Renfroe EG, et al. Lead- and device-related complications in the antiarrhythmics versus implantable defibrillators trial. Am Heart J 2001; 141: 9298.
[24] Kron J. Clinical significance of device-related complications in clinical trials and implications for future trials: insights from the Antiarrhytmics Versus Implantable Defibrillators (AVID) trial. Card Electrophysiol Rev 2003; 7: 473478
[25] Lee DS, Krahn AD, Healey JS, Birnie D, Crystal E, Dorian P, Simpson CS, Khaykin Y, Cameron D, Janmohamed A, Yee R, Austin PC, Chen Z, Hardy J, Tu JV; Investigators of the Ontario ICD Database. Evaluation of early complications related to De Novo cardioverter defibrillator implantation insights from the Ontario ICD database. J Am Coll Cardiol. 2010 Feb 23;55(8):774-82.
[26] Lick SD, Conti VR.Automatic internal cardioverter-defibrillator patch erosion into the upper airway presenting as a cavitary lesion. Chest. 1997 Oct;112(4):1144-6.
[27] Lin G, Nishimura RA, Gersh BJ, Phil D, Ommen SR, Ackerman MJ, Brady PA. Device complications and inappropriate implantable cardioverter defibrillator shocks in patients with hypertrophic cardiomyopathy. Heart 2009;95:709-714
[28] Liu PY, Huang YF, Tang CW, Chen YY, Hsieh KS, Ger LP, Chen YS, Liu YC. Staphylococcus lugdunensis infective endocarditis: a literature review and analysis of risk factors. J Microbiol Immunol Infect. 2010 Dec;43(6):478-84.
[29] Lurie AL, Udoff EJ, Reid PJ. Automatic implantable cardioverter-defibrillator: appearance and complications. AJR Am J Roentgenol. 1985 Oct;145(4):723-5.
[30] Mahapatra S, Bybee KA, Bunch TJ, et al. Incidence and predictors of cardiac perforation after permanent pacemaker placement. Heart Rhythm 2005; 2:907–911.
[31] Martin C, Auffray JP, Saux P, Albanese J, Gouin F. The axillary vein: an alternative approach for percutaneous pulmonary artery catheterization. Chest. 1986 Nov;90(5):694-7.

[32] Martin DT, John R, Venditti FJ. Increase in defibrillation threshold in non-thoracotomy implanted defibrillators using a biphasic waveform. Am J Cardiol 1995; 76:263.

[33] Mehta D, Nayak HM, Singson M, et al. Late complications in patients with pectoral defibrillator implants with transvenous defibrillator lead systems: High incidence of insulation breakdown. Pacing Clin Electrophysiol 1998; 21:1893.

[34] Messali A, Thomas O, Chauvin M, Coumel P, Leenhardt A. Death due to an implantable cardioverter defibrillator. J Cardiovasc Electrophysiol 2004; 15: 953956.

[35] Mewis C, Kuhlkamp V, Dornberger V, Mermi J, Seipel L. [High incidence of isolator fractures in transvenous implantation of cardioverter defibrillators]. Z Kardiol 1997; 86: 8594

[36] Mirowski M, Mower MM, Reid PR, Watkins L, Langer A. The automatic implantable defibrillator. New Modality for treatment of life-threatening ventricular arrhythmias. Pacing Clin Electrophysiol. 1982 May;5(3):384-401.

[37] Newman, D, Barr, A, Greene, M, et al. A population-based method for the estimation of defibrillation energy requirements in humans. Assessment of time-dependent effects with a transvenous defibrillation system. Circulation 1997; 96:267.

[38] Nolan RL, McAdams HP Bronchopericardial fistula after placement of an automatic implantable cardioverter defibrillator: radiographic and CT findings. AJR Am J Roentgenol. 1999 Feb;172(2):365-8.

[39] Pai RK, Pergam SA, Kedia A, Cadman CS, Osborn LA. Pacemaker lead infection secondary to Haemophilus parainfluenzae. Pacing Clin Electrophysiol. 2004 Jul;27(7):1008-10.

[40] Pacifico A, Hohnloser SH, Williams JH, et al. Prevention of implantable-defibrillator shocks by treatment with sotalol. d,l-Sotalol Implantable Cardioverter-Defibrillator Study Group. N Engl J Med 1999; 340: 18551862.

[41] Packer M, Coats AJ, Fowler MB, et al. Effect of carvedilol on survival in severe chronic heart failure. N Engl J Med 2001; 344: 16511658.

[42] Pai RK, Wall TS, Macgregor JF, Abedin M, Freedman RA. Klebsiella pneumoniae: a rare cause of device-associated endocarditis. Pacing Clin Electrophysiol. 2006 May;29(5):540-2.

[43] Pavia S, Wilkoff B.The management of surgical complications of pacemaker and implantable cardioverter-defibrillators. Curr Opin Cardiol. 2001 Jan;16(1):66-71. Review.

[44] Peterson PN, Daugherty SL, Wang Y, Vidaillet HJ, Heidenreich PA, Curtis JP, Masoudi FA; National Cardiovascular Data Registry. Gender differences in procedure-related adverse events in patients receiving implantable cardioverter-defibrillator therapy. Circulation. 2009 Mar 3;119(8):1078-84.

[45] Pfeiffer, D, Jung, W, Fehske, W, et al. Complications of pacemaker-defibrillator devices: Diagnosis and management. Am Heart J 1994; 127:1073.

[46] Quigley RL, Hamer ME, Swiryn S. Migration of an automatic implantable cardioverter-defibrillator patch causing massive hemothorax. Tex Heart Inst J. 1996; 23(1): 54–57

[47] Rosenqvist M, Beyer T, Block M, den Dulk K, Minten J, Lindemans F. Adverse events with transvenous implantable cardioverter-defibrillators: A prospective multicenter study. European 7219 Jewel ICD investigators. Circulation 1998; 98: 663670.

[48] Rossi SE, Goodman PC, Franquet T. Nonthrombotic pulmonary emboli. AJR Am J Roentgenol. 2000;174:1499-1508.

[49] Rusanov A, Spotnitz HM. A 15-year experience with permanent pacemaker and defibrillator lead and patch extractions. Ann Thorac Surg. 2010 Jan;89(1):44-50.

[50] Ryu JH, Swensen SJ. Cystic and cavitary lung diseases: focal and diffuse. Mayo Clin Proc. 2003;78:744-752.

[51] Schauerte, P, Stellbrink, C, Schondube, FA, et al. Polarity reversal improves defibrillation efficacy in patients undergoing transvenous cardioverter defibrillator implantation with biphasic shocks. Pacing Clin Electrophysiol 1997; 20:301.

[52] Siniorakis E, Hardavella G, Arvanitakis S, et al. Accidental shock to rescuer from an implantable cardioverter defibrillator. Resuscitation 2009 Mar;80(3):293-4.

[53] Schirmer, U, Hemmer, W, Lindner, KH, et al. Ultrastructural alterations in the right and left ventricular myocardium following multiple low energy endocardial countershocks in anesthetized dogs. Pacing Clin Electrophysiol 1997; 20:79.

[54] Swerdlow, CD, Schsls, W, Dijkman, B, et al, for the Worldwide Jewel AF Investigators. Detection of atrial fibrillation and flutter by dual-chamber implantable cardioverter-defibrillator. Circulation 2000; 101:878.

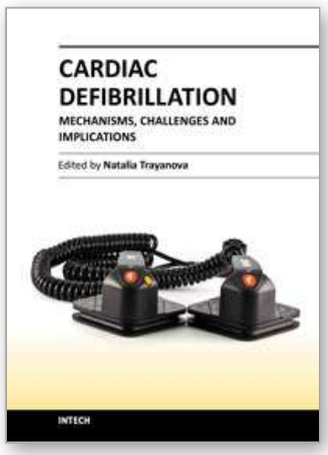
[55] Takahashi T, Bhandari AK, Watanuki M, Cannom DS, Sakurada H, Hiraoka M. High incidence of device-related and lead-related complications in the dual-chamber implantable cardioverter defibrillator compared with the single-chamber version. Circ J 2002; 66: 746750.

[56] Verheyden CN, Price L, Lynch DJ, Knight WL. Implantable cardioverter defibrillator patch erosion presenting as hemoptysis. J Cardiovasc Electrophysiol. 1994 Nov;5(11):961-3.

[57] Voigt A, Shalaby A, Saba S. Rising rates of cardiac rhythm management device infections in the United States: 1996 through 2003 J Am Coll Cardiol 2006;48: 590-591

[58] Wong KS, Lin TY, Huang YC, Hsia SH, Yang PH, Chu SM. Clinical and radiographic spectrum of septic pulmonary embolism. Arch Dis Child. 2002;87:312-315.

[59] Yap SC, Roos-Hesselink JW, Hoendermis ES, Budts W, Vliegen HW, Mulder BJ, van Dijk AP, Schalij MJ, Drenthen W. Outcome of implantable cardioverter defibrillators in adults with congenital heart disease: a multi-centre study. Eur Heart J. 2007 Aug;28(15):1854-61.



Cardiac Defibrillation - Mechanisms, Challenges and Implications
Edited by Prof. Natalia Trayanova

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The only known effective therapy for lethal disturbances in cardiac rhythm is deï¬brillation, the delivery of a strong electric shock to the heart. This technique constitutes the most important means for prevention of sudden cardiac death. The efficacy of defibrillation has led to an exponential growth in the number of patients receiving implantable devices. The objective of this book is to present contemporary views on the basic mechanisms by which the heart responds to an electric shock, as well as on the challenges and implications of clinical defibrillation. Basic science chapters elucidate questions such as lead configurations and the reasons by which a defibrillation shock fails. Chapters devoted to the challenges in the clinical procedure of defibrillation address issues related to inappropriate and unnecessary shocks, complications associated with the implantation of cardioverter/defibrillator devices, and the application of the therapy in pediatric patients and young adults. The book also examines the implications of defibrillation therapy, such as patient risk stratification, cardiac rehabilitation, and remote monitoring of patient with implantable devices.

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