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Complications of Pacemaker Implantation

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1. Introduction

The use of permanent pacemakers (PM) and implantable cardioverter–defibrillators (ICD) continues to grow worldwide (Birnie et al., 2006; Goldberger & Lampert, 2006) with over 3 million PM and 250000 ICD in use by the end of last century (Chua et al., 2000). The rate of device implantations is increasing with aging of the general population and expanding indications. Despite the relative ease of device implantation, the complication risk is still present and sometimes underestimated. The proposal for this chapter is at addressing the most common intra-operative and delayed complications with a special eye to the surgical complications. We will also address electromagnetic interference and psychological problems.

2. Complications related to the location type

Depending on the location type (venous access, pocket, lead and generator) it is possible to dissect several different clinical presentations of complications related to PM implantation which occur, more frequently, in the immediate post-operative course.

2.1 Venous access-related complications

Pneumothorax. This complication 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 et al., 1996) threatens to eliminate this controversy. Pneumothorax is often asymptomatic and noted on routine follow-up plain chest radiograph, but occasionally it requires active medical treatment including intercostal chest drain and aspiration. Aggarwal et al. (1995, 1996) reported a large series of 1088 consecutive patients; pneumotorax represented an overall rate of 1.9% of subclavian insertions. There was no significant difference in the pneumothorax rate between dual chamber (n = 12, 2.1%) and single chamber (n=7, 1.4%). Pneumothorax required active medical treatment in 8 patients (0 8%); 5 patients had an intercostal chest drain inserted and 3 were treated by aspiration. A further 11 patients (1.0%) developed an insignificant pneumothorax (< 10% of pulmonary field in chest x-ray film with no symptoms or progression in subsequent chest radiograph). More recently, Zhan et al. (2008) collected over 67000 patients and presented similar rates. Finally, Pakarinen et al. (2010) also concluded, in a retrospective 1-year single-centre survey, that short-term implantation-related

complications of contemporary device therapy are still frequent, and occur much more frequently when trainees rather than experienced cardiologists are the operators.

Hemothorax. This complication results from trauma to the great vessels rather than the lung. The risk can be minimized by direct inward and outward passes of the puncture needle rather than a side-to-side, potentially "lacerating" movement. If an arterial puncture is performed, recognition, withdrawal, and digital pressure are important. It is important to remember that the artery needs not be cannulated with the introducer. It is essential to always check the fluoroscopic path of the guidewire into the inferior vena cava before introducer insertion. In the Aggarwal et al. (1995) series, the inadvertent arterial puncture was the most common intra-operative complication which occurred in 27 patients (2.7% of subclavian insertions): no serious sequelae ensued.

Air embolism. Deep inspiration at the time of central venous access may cause significant air to be drawn into the venous system due to the physiological negative pressure developed. It can be prevented through operator care and using introducers with haemostatic 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. Usually no therapy is required, as the air is filtered and consequently absorbed in the lungs. The real incidence of this occurrence is not clearly defined in the Literature. However, several cases are reported (Ninio & Hii, 2006; Ostovan & Aslani, 2007; Turgeman et al., 2004): respiratory distress, hypotension, and arterial oxygen desaturation were the most frequent consequences, which was related to the size of the embolus. It is then appropriate to administer 100% oxygen along with inotropic support in some cases. Aspiration of the embolus from the right heart has also been successfully reported (Ostovan & Aslani, 2007).

2.2 Pocket-related complications

Hematoma. Pocket hematoma is an acute, relatively common complication after PM or ICD implantation. It was estimated that 14 to 17% of early reoperations were due to this complication (Aggarwal et al., 1995; Chauhan et al., 1994). The site of bleeding may be the pocket or back-bleeding around the lead venous entry site. The use of electro-cautery is useful to minimize pocket related bleeding. Bleeding from the venous entry site usually subsides during the procedure but ongoing bleeding is controlled by a firm suture placed through and around the lead entry/pectoral muscle interface (Pavia & Wilkoff, 2001). Usually, hematomas are managed conservatively unless expanding in size, tense or painful. In these occasions, reoperation to evacuate the hematoma and identify and arrest the site of bleeding is required. Evacuation was required in 1 to 2% of implant cases in a recent series (Kiviniemi et al., 1999). The risk is increased in anticoagulated patients. The large number of patients receiving coumarin for atrial fibrillation or valve replacement management of anticoagulation might be a major determinant of hematoma development. On the other hand, the complete avoidance of perioperative anticoagulation therapy might promote thromboembolic events and, in particular, cerebral stroke. Intravenous heparinization has been shown to be associated with an increased risk of hematoma development (Michaud et al., 2000) while oral anticoagulation therapy with warfarin did not increase the rate of pocket hematoma in two small series (al-Khadra, 2003; Goldstein et al., 1998). The increasing use of low-molecular-weight heparin and more effective inhibition of platelet aggregation

with ticlopidine/clopidogrel also may affect the susceptibility to intra-operative bleeding and pocket hematoma. It has been shown that long-term warfarin with an international normalized ratio of about 2.0 is safe over a 15-year experience (Belott & Reynolds, 2000), although anticoagulation is in general discontinued for at least the duration of the procedure. Wiegand et al. (2004) investigated the influence of patient comorbidity, implantation strategy, operator experience, antiplatelet therapy, and anticoagulation therapy on hematoma rate on a large series of 3164 devices (2792 pectoral PM and 372 ICD). Predictors of hematoma occurrence were determined prospectively and were analyzed by multivariate regression analysis. According to the results of this important investigation (Wiegand et al., 2004) the following recommendations were given: a) implantation, with the patient receiving combined ASA and clopidogrel treatment, should be performed only by very experienced implanters soon after coronary stenting has been performed (i.e. less than 1 month after) in patients who are highly symptomatic or in whom PM or ICD implantation is vitally indicated; b) in all remaining patients, either therapy with thienopyridine should be discontinued or implantation should be performed when drug treatment can be safely stopped; c) perioperative high-dose heparinization should be reserved for patients with artificial valves (particularly those in the mitral position) and those who have recently experienced arterial embolism, cardioversion, and deep venous thrombosis or pulmonary embolism. Continued oral anticoagulation therapy might be a suitable alternative in the latter patients. All remaining patients who are in need of anticoagulation therapy should receive low-dose heparinization post-operatively until oral anticoagulation therapy is re-established.

Erosion and wound dehiscence. It is a sub-acute complication after device implantation caused by a progressive skin erosion. If the subcutaneous pocket fashioned at the time of initial implantation is too small for the device, undue tension on the overlying skin may cause gradual subcutaneous tissue, and possible eventual skin erosion. Care should also be taken when fashioning the pocket to create the pocket plane on the surface of the muscle. If the pocket is too superficial, erosion may also occur. In the event of erosion, the associated potential for infection is high and therefore extraction of the total device-lead system is usually advised.

Wound pain. Minor wound pain is expected after device implantation, almost always controlled with simple analgesia. In general, the pre-pectoral site is extremely well-tolerated. Continuing pain will usually improve or manifest an obvious infection eventually. However, pain that initially improves then recurs or occurs temporarily remote from the implant may suggest infection even in the absence of any outward localizing signs, and consequently may necessitate surgical exploration or even empirical removal and reimplant at another site. Alternatively, mechanical trauma from the device location adjacent to anterior chest wall structures may be the culprit. In this situation, device relocation or pocket revision may be indicated.

Infection. Similar to other prosthetic materials, infections complicate a small proportion of patients with these devices. Along with the increase in device implantation, the incidence of device infections has also been increasing, but at a faster rate. (Voight et al., 2006). In the USA, data from Medicare recipients from 1990 to 1999 showed an increase in the number of device infections from 0.94 per 1000 recipients to 2.11 per 1000 recipients, an increase of 124%; the estimated rate of infection of endocardial leads being between 1 and 2%, but

varying from 0.13 to 12.6%. (Cabell et al., 2004; Voight et al., 2006). Grimm et al. (1993) reported an incidence of ICD system infection ranging from 2 to 8%. The exact reason for a time-related increase of infections remains unexplained, but it is hypothesized to be due to increasing co-morbidities in device recipients, improved surveillance and detection of cardiac device infection, and improving survival of patients with devices (Uslan & Baddour, 2006). The mortality of persistent infection when infected leads are not removed can be as high as 66% (Rettig et al., 1979). The physical manifestations range from mild systemic symptoms with no local reaction to fulminant life-threatening sepsis. Early infections (no more than 60 days from implant) tend to be more clinically evident as opposed to the more indolent course of late onset infections. These infections can present with only pain at the implant site. When infection is present, complete device removal with transvenous lead extraction is followed by antimicrobial therapy with the device reimplanted at a later date. In the Chua et al. (2000) experience including both PM and ICD leads, the median time for device reimplantation was 5 days with no subsequent evidence of recurrent or new infection at a mean follow-up period of 46 weeks. Partial system removal is associated with high recurrence rate and should be reserved for very selected cases. Once a strong clinical suspicion for infection is established, the whole system should be considered contaminated. The time of onset of the infection is extremely variable. Margey et al. (2010) reported a median duration from device implant or revision to presentation with confirmed cardiac device infection of 150 days (range 5–2920 days, with an interquartile 25% of 35 days and an interquartile 75% of 731 days). Fever, chills and malaise were the most common presenting symptoms. Evidence of generator site inflammation was present in 90%. Frank erosion or purulent discharge could be identified in 66%. A third of cases met diagnostic criteria for cardiac device related endocarditis. The majority had non-specific laboratory abnormalities, including elevation in leukocyte count, anaemia, elevated erythrocyte sedimentation rate, or C-reactive protein level. Therefore, the clinical diagnosis of device infection is very easy.

Microbiological findings. It is not always possible to define the agent of infection: in a very recent report (Margey et al., 2010) out of 39 cardiac device infection cases, a causative organism was identified only in 62%. The most frequent causative organism was methicillin sensitive *Staphyloccoccus aureus* (30.8%), followed by coagulase negative *Staphylococcus* (20.5%), and *Streptococcus* species (7.7%). Blood cultures were performed in 84% of the cardiac device infection group, and were positive in 54% of these cases. Cultures of generator site tissue and lead tips were performed in those undergoing device extractions (82%) and were positive in 38 and 18%, respectively. In cases where all 3 swabs were positive, the same causative organism was identified in each case. Of those patients in whom blood cultures were negative, all had already received antibiotic therapy by the time cultures were drawn.

Echocardiographic findings. Cardiac ultrasound does not seem to have an high diagnostic sensitivity. In the Margey et al. (2010) series 87% of the patients underwent transthoracic echocardiography during their admission and 36% also underwent transoesophageal echocardiography. In those in whom echocardiography was performed, vegetations were identified on the lead in 18%, and involving the heart valves in 5%. The tricuspid valve was the only valve involved. On the other hand, patients with intracardiac vegetations identified on transesophageal echocardiogram can safely undergo complete device extraction using standard percutaneous lead extraction techniques. Permanent devices can safely be reimplanted provided blood cultures remain sterile. The presence of intracardiac

vegetations identifies a subset of patients at increased risk for complications and early mortality from systemic infection despite device extraction and appropriate antimicrobial therapy (Grammes et al., 2010).

2.3 Lead-related complications

Cardiac perforation. Permanent PM implantation may be complicated by cardiac perforation, which can lead to longer hospital stays, tamponade, or even death (Aizawa et al., 2001; Ellenbogen et al., 2002; Garcia-Bolao et al., 2001; Gershon et al. 2000). The incidence of perforation after permanent PM reportedly is between 0.5% and 2%, but the predictors of perforation have not been defined (Hill, 1987; Sivakumaran et al., 2002). Lead perforation is a less-recognized delayed complication of device implantation. Delay in recognition may prove fatal. Careful evaluation of pacing and sensing thresholds and follow-up echocardiographic evaluation is mandatory to remain vigilant for this potentially fatal complication. The clinical manifestations of significant perforations are variable and include chest pain, dyspnoea, and hypotension. These signs, in conjunction with a new pericardial effusion immediately following permanent PM implantation, suggest PM-related cardiac perforation. Predictors of post implantation pericardial effusion, which serves as a marker of perforation, include concomitant use of trans-venous PM, steroid use within 7 days, and older age. Perforation of the right ventricle as a late complication of device implantation is rare and requires a high suspicion for prompt recognition and intervention. Routine chest radiography may not be diagnostic, and further testing such as CT scan and echocardiography are essential, combined with PM interrogation, for evaluation of high thresholds. A higher clinical suspicion should be maintained in the elderly, in whom perforation occurs more frequently (Mahapatra et al., 2005). In addition, consideration should be given in the elderly to implant the lead in sites other than the right ventricular apex, such as the right ventricular septum or outflow tract, in an attempt to minimize the risk of this complication later during the follow-up. Acute perforation of the right atrium or right ventricle has been reported in up to 1% of patients (Chauhan et al., 2005).

Malposition. There have been several reports of inadvertently lead malposition during PM implantation. Some of those are related to cardiac structural abnormalities. In patients with an atrial septal defect lead could be erroneously implanted in the left ventricle. The presence of a right bundle branch block configuration during ventricular pacing should induce the suspect of a malposition that can be confirmed by a lateral chest x-ray or by ultrasound. The malposition could be discovered years after the implantation and pacing (Vanhercke et al., 2008). There are not recommendations about the removal of lead if there are not concomitant complications such thrombus, embolism or the posterior mitral leaflet perforation causing an acute mitral incompetence (Seki et al., 2009; Van Gelder et al., 2000). In any case, if timely removal of a malpositioned lead in the left ventricle, through a patent foramen ovale or atrial septal defect is not performed, life-long anticoagulation with warfarin should be recommended. Cases are reported too of a malpositioned lead, which had inadvertently been inserted into the left subclavian artery and passed through the aortic valve into the left ventricular apex (Reising et al., 2007), despite the finding of an apparently elevated "venous" pressure at the time of insertion. The error was not detected despite the anterior–posterior chest radiography (no lateral view), electrocardiograms, and a computed tomography scan of the chest with contrast during which the lead was said to be in the right ventricle. Careful analysis of post-procedure electrocardiograms and lateral chest x-ray film

images can minimize the chance that such an error will go undetected There is no consensus as to how to proceed in cases such as this because few are reported in the Literature, which may also be related to publication bias. Recently, it was suggested that transcatheter lead removal should not be performed because of an inability to detect the presence of thrombi on the lead wires (Van Gelder et al., 2000). To our knowledge, the risk of trauma to the valve leaflets has not yet clarified. However, cases of endocarditis have been reported, including one with a large aortic valve vegetation (Schulze et al., 2005).

Lead dislodgement. Lead dislodgement is a clinically relevant and possibly dangerous occurrence. It typically occurs either early (within the first 6 weeks of implantation) or late (beyond the first 6 weeks of implantation), occasionally very late dislodgments were also described (Tokano et al., 2004). Early dislodgements are more common then late dislodgements. Atrial lead dislodgment occurs more commonly than ventricular dislodgements in the early period (Chauhan et al., 2005). The incidence of early dislodgement of a ventricular lead in a DDD PM was 0.5 to 2.5% (Aggarwal et al., 1995; Fortescue et al., 2004). Lead dislodgment occurs less commonly as a consequence of advancements in pacing lead placement and lead design. After dislodgement, the lead usually remains intracardiac but cases are described where leads were completely pulled out (Von Bergen et al., 2006), as in the lead dislodgement secondary to a "ratchet-like" mechanism through the sewing sleeve. Fluoroscopy is occasionally needed to identify the location of the lead tip. However, some mechanisms of PM lead dislodgement involve retraction of the lead toward the generator. This includes Twiddler's syndrome, which refers to lead dislodgement due to conscious or unconscious manipulation of the pulse generator causing it to rotate around its long axis (Abrams & Peart, 1995; Bracke et al., 2005; Chauhan et al., 1994; Higgins et al., 1998; Newland & Janz, 1994; Solti et al., 1989). Typically, placement beneath the pectoral muscle and suturing the generator to the underlying fascia is thought to preclude the patient from manipulating the device. Additionally, "Reel syndrome" (Carnero-Varo et al., 1999; Vural et al., 2004) has been described, which entails the generator rotating around its transverse axis "reeling" in the lead. In certain adult populations, such as in patients with a history of significant weight loss, the "Sagging Heart syndrome" may represent a previously unrecognized cause of acute lead dislodgment (Iskos et al., 1999). It is a rare form of lead dislodgment that may occur due to an unexpected and marked postural descent of the heart after permanent PM implantation. Iskos et al. (1999) described this, in 2 patients, which was related to a history of morbid obesity followed by weight loss of over 40 kilograms. Lead replacement with active fixation leads was required in both cases.

2.4 Generator-related complications

Set screw loose. Set screw could be a cause of complication: although PM infections that were apparently localized at set screw site were reported (Henrikson & Brinker, 2006), the most important complications are related to the loose of set screw and of electrical contact. Inappropriate mode switching in a dual chamber PM due to oversensing of a high frequency signal from a conductor/ring discontinuity (loose set screw) was reported by Kuruvilla et al. (2002). A retrospective review of complications with connectors and lead-to-header interfaces was performed (Tyers et al., 1992) years following 649 pacing procedures between 1980 and 1990. There were 88 lead revisions (13.6%), 81 device replacements or modifications (12.5%), and 480 new implants (74%) using devices of 5 manufacturers. Two

basic connector types were studied, one utilizing a set screw and the other using a side-lock compression fitting. The set screw makes electrical contact and mechanically secures the lead connector pin with a set screw insulated by a self-sealing grommet or an integral or separate set screw cover. The side-lock makes electrical contact with an automatic spring mechanism while the plastic lead terminal is secured in the connector block of the pacemaker by a Delrin side-lock compression fitting. The follow-up during at most 12 years of 459 set screw connector devices gave 14 complications (3.1%) whereas 82 side-lock connector devices were followed for up to 5 years with 1 complication (1.2%). The set screw and side-lock connectors were reliable over the period of follow-up. Although the complication rate appeared lower with the side-lock, follow-up was shorter and the number of implants smaller. With the leads used in this study (Tyers et al., 1992), the side-lock proved to be a desirable feature due to simplicity, speed, safety, and ease of use. One limitation is the requirement for a precise IS-1 connector terminal diameter.

3. Complications unrelated to the location site

There are a series of complications after PM or ICD implantation, more frequently occurring late after insertion, which do not recognize a clear-cut relation with the location site and rather present as a more generalized syndrome.

3.1 Superior vena cava syndrome

Superior vena cava syndrome (SVCS) is characterized by gradual, insidious compression/obstruction of the superior vena cava. Although the syndrome can be life-threatening, its presentation is often associated with a gradual increase in symptomatology. For this reason, diagnosis is often delayed until significant compression of the superior vena cava has occurred. Initially, there are few symptoms, however, over time, symptoms of superior vena cava compression/obstruction gradually develop. As the compression becomes more severe, the patient may develop shortness of breath and swelling of the arms and face. SVCS is chiefly associated with malignancy. Currently, more than 90% of patients with SVCS have an associated malignancy as the cause. Of the nonmalignant causes of SVCS, thrombosis from central venous instrumentation (catheter, PM, guidewire) is an increasingly common event, especially as these procedures become more common. PM-induced SVCS is a rare complication of permanent PM insertion, with an estimated prevalence ranging from 1:40000 to 1:250, and usually occurring more than 1 month after implantation (Bolad et al., 2005; Gilard et al., 2002; Spittel & Hayes, 1992). The obstruction is internal and composed of thrombus, fibrosis, or a combination of the two, typically involving the pacing wires within the superior vena cava (Spittel & Hayes, 1992). Although the mortality associated with benign causes of SVCS is low, those patients who become symptomatic are often debilitated by it, necessitating intervention. Various therapies have been used to treat PM-related SVCS. The various treatment modalities used to relieve SVCS symptoms may be summarized under the following categories: a) anticoagulation (heparin, warfarin, acenocoumarol, phenprocoumon, dicumarol); b) thrombolysis, either systemic or catheter-directed (tissue plasminogen activator, urokinase, alteplase, or streptokinase); c) surgical, intervention on superior vena cava using a spiral saphenous vein conduit, or reconstruction using a pericardial patch, or a thrombectomy; d) balloon venoplasty alone; and e) stenting of the lesions (usually preceded by venoplasty) using wallstents, SMART

stents, Palmaz stents, or Z stents. There is no current consensus about their relative efficacy and merits. Riley et al. (2010) recently reviewed and summarized all of the reported cases of this complication that were treated in order to aid clinical decision making and spur future research in this area. It was recognized from the outset of this venture that any attempt at a pooled analysis of results could be significantly hampered by the small number of cases in each treatment group and publication bias, particularly the underreporting of treatment failures and of SVCS recurrences after initially successful interventions. The poor efficacy of anticoagulation and thrombolysis is not surprising given that histological studies in cases of lead-induced SVCS have shown that only a minority of cases are due to thrombosis without concomitant fibrosis; these types of lesions usually present relatively soon after device implantation. The majority are due to fibrotic narrowing and superimposed thrombosis, particularly if symptoms develop after more than a year (Gilard et al., 2002); among patients reported in this survey, the median interval between implantation and onset of SVCS symptoms was 48 months. Therefore, residual superior vena cava stenosis after successful thrombolytic therapy is likely a factor in re-thrombosis and short symptom-free periods (Leonelli et al., 2000). The disappointing results of venoplasty alone may also reflect high rates of restenosis. For example, patency rates as low as 30% have been reported at 1-year following balloon dilations of benign axillary and subclavian vein stenoses (Bolad et al., 2005; Frances et al., 1995; Gilard et al., 2002; Spittel & Hayes, 1992). The short-term results of surgery and stenting are more encouraging but the paucity of data on long-term efficacy and outcomes is a matter of concern. Even though surgical reconstruction has been practiced for over three decades, adequate follow-up data were only available for 17 patients over a median period of 16 months. Given that surgical reconstruction is a highly invasive and expensive undertaking, it is not surprising that percutaneous intervention employing venoplasty plus stenting has become the most commonly reported treatment for SVCS.

3.2 Pericarditis

The first reported case of pericarditis following permanent PM implantation was published in 1975 (Kaye et al., 1975). Pericarditis should be distinguished from acute or delayed lead perforation in which a migration of the electrode tip is observable. In pericarditis there are no changes in the tip position at x-ray examination and no changes in pacing or sensing thresholds (Ellenbogen et al., 2002). To date only a few cases of pericarditis related to PM implantation have been published (Ellenbogen et al., 2002; Kaye et al., 1975; Kono et al., 2008; Levy et al., 2004; Snow et al., 1987; Vinit et al., 2007;). Clinical manifestation of pericarditis resembled the classic post-pericardiotomy syndrome with pleuritic chest pain, dispnoea, and the presence of pericardial and pleurical effusion, raised erythrocyte sedimentation rate without polyarthopathy. Usually, effusion does not require pericardio-centesis because of the small size of effusion that responds to non steroid drugs or to steroids. Large effusion volumes could requires pericardiocentesis when tamponade is present. In the Greene et al. (1994) and Levy et al. (2004) series a higher incidence of pericarditis was found when active fixation leads were used overall for atrial stimulation. Several reported cases were related with an atrial active-fixation lead (Schiariti et al., 2009; Sivakumaran et al., 2002). Generally, pericarditis after permanent PM implantation has a benign and self-limited course. However, replacement of an active-fixation lead with a passive one has also been reported in order to prevent recurrence of pericarditis (Schiariti et al., 2009).

3.3 Undersensing

Loss of ventricular sensing could be caused by dislodgment or mechanical damage to the lead connected to the system, as perforation of the insulating sheath. Sometimes undersensing could be resolved after repositioning the lead, restoring its integrity with silicone adhesive as described by Costeas & Schoenfeld (1991), or reprogramming the device. In the past the ability to attach an unprotected unipolar lead to a bipolar connector, shared by the Voluntary (VS-1) and International (IS-1) designs, invited the possibility of injury to the insulating sheath by accidental tightening of the proximal screw. Sensing in PM and ICD is crucial to normal device behaviour. Since both devices treat different arrhythmias, the technical approach to signal detection is also completely different. A PM has a fixed threshold of sensing, above which events are sensed and therapy of the device withheld. On the other hand, the defibrillator has a variable threshold of sensing to detect tachyarrhythmias, with sometimes very small and changing electrogram amplitudes. Van Casteren et al. (2009) described undersensing of ventricular fibrillation due to interference between a PM and defibrillator in the same patient. The R-on-T phenomenon is a well-known entity that predisposes to dangerous arrhythmias. Typically, a premature ventricular complex occurring at the critical time during the T wave of the preceding beat precipitates ventricular tachycardia and fibrillation. This phenomenon can occur not only in asynchronous ventricular PM, but also in synchronous PM, if loss of sensing of the intrinsic rhythm becomes evident (Chemello et al., 2010).

3.4 Oversensing

Oversensing is a phenomenon potentially leading to ICD malfunctioning by interfering with intracardiac signals and usually is related to myopotentials, T-waves, electrical spikes of an additionally implanted PM or other technical devices i.e. microwaves, stimulation therapy in physiotherapy, and electric coagulation (Lin et al., 2004; Schulte et al., 2001; Secemsky et al., 1982; Weretka et al., 2003). The ICD system often misinterprets these cardiac/non-cardiac potentials as a malignant arrhythmia and delivers inappropriate shock therapy, which in many patients is an extremely uncomfortable event and greatly affects quality of life. In some cases, inappropriate therapy delivery is described as potentially life-threatening (Kiviniemi et al., 1999), especially when the shock falls in the vulnerable phase of QRS complex, triggering ventricular tachycardia or ventricular fibrillation (VF). Rauwolf et al. (2007) evaluated in a large cohort of 518 ICD patients the incidence and various types of ventricular oversensing (VO), the occurrence of inappropriate shock deliveries as well as the frequency of complications requiring invasive procedures to solve VO during a long-term follow-up. The most frequent oversensing mechanism was observed as T-wave oversensing in 10 patients, 8 (1.5%) patients were noticed with VO due to myopotentials; 5 patients suffered from VO due to electrode failure and consecutive noise sensing with inadequate therapy delivery. Double-counting was recorded in 4 patients, leading to 8 inadequate shocks in 2 patients and short episodes without therapy delivery; 3 patients experienced VO according to electromagnetic field interference with the ICD device by inducing an alternating current in the sensing electrode or to alternating current. In 1 patient, microwave energy was applied for pain relief at the skin close to the ICD pocket. The signals from electromagnetic field transmitter were detected from the atrial and ventricular channel of the ICD, misinterpreted as VF inducing an inappropriate shock delivery. The second patient had VO while swimming through an electrically opening pool door. In another case, the

inadequate shock was due to an alternating current application during physiotherapy session using electric stimulation of the skin. There were 6 patients who had an ICD and an additionally implanted PM. There were different reasons for these double device implantations: a) first ICD generation with large volume were implanted abdominally; b) first ICD generation were only available as single-chamber devices; c) pre-existing biventricular pacemaker for CHF therapy; and d) insurance requirements (Geiger et al., 1997; Shivkumar et al., 2000).

3.5 Crosstalk

The oversensing of an atrial pacing pulse in the ventricular channel, has been described primarily in patients with unipolar PM and high atrial outputs (Helguera & Pinski, 2005). Although their high operating ventricular sensitivity could make ICD prone to cross-talk inhibition, this complication has been very uncommon. Crosstalk is one of the serious complications occurring with dual-chamber PM and ICD and it is defined as the inappropriate detection of a spontaneous or PM-generated event in one channel by the other channel, which can cause the inhibition of the second channel's output. There are two functions designed to prevent the crosstalk inhibition. One is the ventricular blanking period that coincides with the atrial stimulus. This prevents the detection of the atrial spike or activation that could cause the inhibition of the ventricular channel output. An additional backup system is the ventricular-safety-pace, which prevents asystole if crosstalk occurs due to atrial far-field activity sensed after the blanking period of the ventricular electrode.

3.6 Pacemaker syndrome

The PM syndrome consists of the cardiovascular signs and symptoms of heart failure and hypotension induced by the loss of both A-V and V-V synchrony that is associated with right ventricular apical pacing. It was first described by Mitsui et al. (1969) and occurs with single o dual chamber pacing mode. The clinical adverse effect are most severe when intact ventriculo-atrial conduction is present. Atrial contraction can coincide with ventricular systole against closed atrioventricular valve. This situation may lead to raised mean pressures in both atria, to atrial dilatation and retrograde blood flow into vena cava and pulmonary veins and contributes to decreased cardiac output. Nishimura et al. (1982) showed a great decrease in blood pressure more than 20 mmHg is suggestive of PM syndrome. When ventricular function is normal, estimates of atrial contribution to cardiac output vary from 15-20%. In elderly patients, in hypertensive cardiomyopathy, hypertrophic cardiomyopathy, and restrictive cardiomyopathy, the atrial kick contributes as much as 50%. The reduction in cardiac output associated with non physiologic pacing and loss of atrioventricular synchrony triggers changes in vascular tone. The autonomic nervous system enhances sympathetic activity modulated by arterial baroceptors which are triggered by low blood pressure (Alicandri et al., 1978; Ellenbogen et al., 1990; Pehrsson et al., 1988). The increase in left atrial, pulmonary and left ventricular filling pressures result in inhibitory reflexes mediated by vagal nerve and in increased production of atrial natriuretic peptide, a potent arterial and venous vasodilator (Theodorakis et al., 1992). The opposing reflexes may cause an inadequate vasoconstrictor response and decrease vascular tone. The dual chamber pacing does not ensure that pacemaker syndrome will not develop (Travill & Sutton, 1992). When the leads stimulate the myocardium to depolarize, paced depolarization of both atria and ventricles occurs cell to cell fashion rather than via usual conduction pathways. This

depolarization is slower and less efficient and can result in dyssyncrony of atria and ventricles and of the right and left sides of the heart. It is necessary to program an appropriate atrioventricular delay between 120 and 200 ms. Too much short a delay reduces filling of the ventricles in diastole and of the atria during systole; too much a long one may produce asynchrony. In presence of atrio-ventricular block a rapid atrial pacing (AAI or AAIR) prevents the regular atrial filling and reduce cardiac output. Many patients cannot tolerate the rapid pacing associated with activity mode (Ellenbogen et al., 1997; Ellenbogen et al., 2007). An inappropriate mode switching of DDDR pacing in response to interference noise or dying battery, then rhythm change, can also cause PM syndrome. The retrograde depolarization of the atria which occurs with dual- o single–chamber devices can cause endless-loop tachycardia or PM-mediated tachycardia (Pioger et al., 2007).

The majority of symptoms of PM syndrome are likely attributable to the reduction in cardiac output that is associated with right ventricular pacing. It induces a contraction similar to that caused by left bundle branch block, asynchronous ventricular contraction leading to altered diastolic filling time, increase in mitral regurgitation and reduction in left ventricular ejection fraction (Lamas & Ellenbogen, 2004). The cardiac output is greater during AAI pacing than with dual-pacing DDD. Wigger (1925) showed that ventricular pacing results in reduced ventricular-pump function in mammals. Ventricular desyncronization imposed by pacing results in chronic left ventricular remodeling, including asymmetric hypertrophy and dilatation. Ventricular pacing increases atrial pressure and size, as well as to favor the development of electrophysiological proprieties that could facilitate the development of atrial fibrillation. In patients with an ICD, dual-chamber pacing paradoxically led to increased risks of heart failure, hospitalization and death by a factor of 1.6 by inducing ventricular pacing (Wilkoff et al., 2002). Bordachar et al. (2004) showed an increase in cardiac output from 2.2 l/min at baseline to 3.8 l/min with institution of biventricular pacing in patients with heart failure.

Symptoms of PM syndrome are nonspecific and diagnosis depends heavily on correlation between onset of symptoms and onset of pacing or change in pacing mode. The PM interrogation plays a crucial role in determining PM mode contribution to symtoms: dyspnoea on exertion, paroxysmal nocturnal dyspnoea, orthopnoea, hypotension, pre-syncope and syncope (Furman, 1994). Physical examination can often reveal elevated neck veins, rales, and pedal oedema. Syncope is uncommon and usually associated with systolic blood pressure declines of greater than 20 mmHg. The other symptoms attributed to PM syndrome include easy fatigability, malaise, headache, and sensation of fullness and pulsations in the head and neck. A diagnosis can be confirmed by placing a magnet over the PM, converting the system to VOO mode at predetermined rate to induce ventriculo-atrial conduction and symptoms (upright) (Ross & Kenny, 2000). ECG, Holter monitor or event recorder may reveal a prolonged PR interval, ventriculo-atrial conduction, or atrio-ventricular dissociation. Echocardiogram may show decreased cardiac output with ventricular pacing versus conduct sinus activity or atrio-ventricular synchronous pacing. Symptoms usually resolve after reprogramming PM parameters, such atrio-ventricular delay, postventricular atrial refractory period, sensing level, and pacing threshold voltage or using hysteresis to help maintain atrio-ventricular synchrony in patients with VVI PM and intact sinus node function or addition of an atrial lead.

The incidence of PM syndrome in various studies ranges from 25% (Travill & Sutton, 1992) to 83% (Heldman et al., 1990), depending to large degree on diagnostic criteria and to the

therapy used for diagnosis. When surgical revision is required to upgrade a patient from VVIR pacing, the incidence has been low, 2.7% in the Canadian trial of Physiolgic Pacing (Connolly et al., 2000). In the PASE (Lamas et al., 1998) and MOST (Lamas et al., 2002) studies in which devices could be easily upgraded to DDDR mode by simple reprogramming, the incidence of PM syndrome was higher. The PM syndrome may also be underestimated or it may be subclinic. The symptoms often have been ascribed to the aging process, to the worsening of heart failure or to coronary artery disease. Memory deficit in elderly patients complicate reporting of symptoms. Therefore, up to 75% of patients who have felt generally well for many years with single-chamber PM have noted improvement in their quality of life after upgrading to dual–chamber PM at the time of a pulse-generator change (Sulke et al., 1992).

Atrio-ventricular dyssynchrony is associated with atrial fibrillation and, therefore, thromboembolic complications, and also heart failure. The investigators have assumed the same complications for PM syndrome. The results of completed randomized clinical trials of PM mode selection have been somewhat conflicting. Andersen et al., (1994) compared AAI with VVI pacing in 225 patients with sinus node dysfunction and demonstrated persistent reduction in primary endpoints of atrial fibrillation, thromboembolic events, chronic atrial fibrillation , and all cause mortality in AAI paced group. In the PASE study 407 patients in sinus rhythm and bradycardia were randomized to VVIR or DDDR. There was a 26% crossover rate due to the development of PM syndrome but no difference in quality of life, death, stroke, first hospitalization for heart failure, development of atrial fibrillation were observed. Also larger trials, the Canadian Trail of Physiologic Pacing and MOST performed to randomizing 2568 and 2020 patients to atrial-based or ventricular pacing failed to show differences in the endpoints, stroke or death. Only the incidence of atrial fibrillation was lower with dual-chamber pacing. In subsequent analyses of MOST data the percentage of ventricular pacing correlated with risk of congestive failure. This percentage was greater in DDR versus VVIR mode (90% versus 58%). The ventricular dyssyncrony induced by pacing even when atrio-ventricular synchrony is preserved increases the risk of PM syndrome, heart failure hospitalization and atrial fibrillation. The benefit derived from atrio-ventricular sequential pacing is likely counterbalanced by detrimental effects of frequent and unnecessary right ventricular pacing. These observations have led to renewed interest in single-chamber atrial pacing (Pioger et al., 2007) for sinus node dysfunction and the development of new dual-chamber PM algorithms designed to minimize right ventricular pacing (Sweeney et al., 2007a) and additional research will determine if different forms of ventricular pacing, such as biventricular or ventricular-septal pacing, will improve outcome in patients who require ventricular stimulation.

3.7 ICD-specific complications

After the first clinical implantation (Mirowski et al., 1980), numerous primary and secondary prevention trials resulted in rapid expansion of indication for use of ICD. In the successive 15 years, the annual ICD insertion has increased by 20-fold (Maisel et al., 2006). Consequently, the number of implanted ICD system and the number of patients with longer follow-up has continuously increased. Despite advances in system design and manufacturing to answer to any different clinical situation, device malfunctions and software glitches will continue to occur. The increasing complexity in ICD hardware with significant proportion of biventricular resynchronization devices may result in higher

complications rates. The surgical complications, infection or erosion, hematoma, pneumothorax, are similar in type and frequency to those seen with routine PM implantation (DiMarco, 2003). The other generator–related complications are defined as early generator depletion (less than 4 years after implantation), recalls, power reset and lead connector problems (an average of 1.4% per patient-year) (Ezekowitz et al., 2007). The lead is a life-line whose purpose is to convey critical information about heart's rhythm to the ICD generator and, in turn, to deliver life-sustaining therapy when needed. The long-term reliability of the leads is the main problem. ICD leads are significantly more complex than PM leads and must allow high voltage energy delivery for defibrillation when necessary and, may be inherently more susceptible to failure. The mechanical stress can cause fracture in a lead or failure in insulation by chronic excessive pressure on the body of the lead by the ligature used for fixation (Dorwarth et al., 2003) or the subclavian crush syndrome (Antonelli et al., 1998) or the higher activity patients or the multiple leads or the anatomic reasons in females. The insulation degradation by breakdown of polyurethane polymers due, in most cases, to metal ion oxidation like in the Medtronic models 6936 and 6966. This lead failure can produce oversensing following appropriate shocks (Ellelbogen et al., 2003). It is likely that this problem remains clinically silent until a high-voltage shock is delivered. Detection of a ring-to-coil impedance drop and a high short interval counter may be a useful indicator of middle insulation breach. The newer lead models have multi-lumen design with steroid elution: each conductor is individually insulated by silicon rubber, which should prevent the lead injury. Silicone leads also seem to be prone to insulation failure (Mewis et al., 1997). More than half of lead defects (56%) results from insulation failure (Kleemann et al., 2007). The lead defect depends mainly on follow-up time after implantation. The lead survival rates at 5 and 8 years are 85% and 60%, respectively (Kleemann et al., 2007). The annual lead failure rate, defined as lead-related problem requiring surgical revision, reaches 2.5% in 5 years (Eckstein et al., 2008) and 20% in 10 years old leads (Kleemann et al., 2007).

The great variation observed in ICD lead survival is due to variety of factors including variable study definitions of lead malfunction, variable performance of models and patient characteristics and implantation techniques (Maisel & Kramer, 2008). The lead dislocation and exit block tend to occur early, while the other problems are more evenly distributed over time (Duray et al., 2009). The left ventricular lead dislocation reaches rate varied between 0%, 4.7% and 7.5% for increased complexity to implant procedure in the coronary sinus (Duray et al., 2009). The dislocation can be observed on x-ray combined with significant changes in sensing/pacing performance. The exit block is a failure to capture at reasonable device output without change in impedance and lead position. The lead fracture results in changes in impedance (more than 2000 Ohm), in changes in sensing/pacing performance (intermittent o permanent) and is confirmed by fluoroscopy. The oversensing is sensing of artifacts (chaotic, far field, T-wave, myopotential, noise from contact with another lead) without significant change in lead impedance o position, similar to what seen with PM. The other recorded problems are: unstable impedance measurements, R-wave reduction and loss of capture (Eckstein et al., 2008).

The tools available to detect impending ICD lead failure are limited. At implantation, all ICD systems are tested including determination of sensing, lead and shock impedances, pacing thresholds, and defibrillation thresholds after repetitive induction of ventricular fibrillation. Follow-up, every 3-6 months or early as needed, consisted of interrogation and retrieval of all stored data since last visit, as well determination of sensing, impedance

measurements, and pacing thresholds. The need for increased surveillance of patients with older leads should include test for oversensing during provocative maneuvers and measurement of high voltage coil and pace/sense impedances and defibrillation underthreshold testing. A big help could have come from wireless home monitoring of device than can immediately detect any abnormality thus reducing the risk for patients (Corrado et al., 2009). Anyway electrical testing, x-ray, fluoroscopy, or direct visualization may be used to detect lead abnormalities but are imprecise.

ICD dysfunction may result in failure to deliver therapy for ventricular tachycardia and thus result in syncope or sudden death. Pooled data of randomized clinical trials that involve around 3500 patients indicate an ICD-unresponsive sudden-death rate of nearly 5% (Anderson, 2005). Primary ventricular arrhythmia detection is based on the tachycardia detection rate which is usually programmed with a safety margin of 20 beats/min below the clinical arrhythmia. But there is high (30%) slow ventricular tachycardia incidence (less than 150 beats/min) in ICD recipients without prior history of slow ventricular tachycardia (Sadoul et al., 2005). The patients could exhibit symptoms such as syncope, palpitations, and congestive heart failure, leading to hospital admission and sometimes death. A low detection rate may lead to inappropriate therapy during supraventricular tachycardia. Most ICD can be programmed to enhance the discrimination between supraventricular and ventricular arrhythmias by additional criteria such sudden onset, stability, ventricular electrogram morphology or vector timing and correlation, and relationship between the atrium and ventricle. For terminating monomorphic tachycardias, antitachycardia pacing is standard technique and is painless for patient. But is not always effective and can accelerate ventricular tachycardia or, if applied during a supraventricular rhythm, induce a ventricular arrthymia and following shock. Trappe et al (1995) reported an acceleration rate of only 3% in selected patients with recurrent inducible ventricular tachycardia that had been successfully terminated during predischarge test. Inappropriate delivery of ICD therapy, triggered by artefacts due to lead dysfunction, extraneous noise interference, or rapid atrial rates, mainly during atrial fibrillation or sinus tachycardia, remains a major clinical challenge.

Clinical trial experience has revealed that up to 25% of patients receive inappropriate shocks (Germano et al., 2006). ICD shocks reduce the physical functioning and mental well-being and increase anxiety of patients while obligating strict driving restrictions. The risk of death with inappropriate shocks in SCD-HeFT was increased by a factor of 2, while for an appropriate shock the risk was increased by a factor of more than 5. The arrhythmia much likely signaling a meaningful change in patient's clinical status, including a worsening of heart failure and myocardial ischemia with abnormalities in the levels of electrolytes. An examination of randomized trials for primary and secondary prevention has shown that the number of appropriate shocks exceed the sudden death and overall mortality rate in the control group (Germano et al., 2006). Many episodes may have been nonsustianed nonfatal events. Therefore, there are appropriate shocks and necessary shocks. Alternatively the ICD proarrhytmic effect may be considered (Tung et al., 2008). The pacing–associated short-long sequences were found at the onset of 21% to 35% of all episodes of ventricular tachycardia and fibrillation (Sweeney et al., 2007b). Device malfunction or local lead effect with mechanical irritation and late fibrosis may be potential mechanism for ventricular arrhythmias. The reversal activation wavefronts from epicardial resynchronization increases dispersion of refractoriness with demonstrated improvement of ventricular arrhythmias and

sudden death after implantation (Basu et al., 2007). Nevertheless, ICD is clinically proven to improve survival in selected patients at risk for sudden cardiac death, but monitoring of this device remains critical to inform physicians and patients about device performance and to identify underperforming products as early as possible. The last highly publicized recall of Sprint Fidelis (Medtronic Inc., Minneapolis, Minnesota) lead was issued October 2007 (Food and drug Administration, 2007). The higher rates of lead fracture (2.3% to 6.7% at 30 months) led to more media-provoked mass hysteria as about 268000 patients were at risk in the world. The lead failure produced inappropriate shocks and ICD storm due to sensing of electrical noise: 5 deaths were reported in initial advisory (Catanchin et al., 2008). To overcame the problems of lead insertion and of lead failure an entirely subcutaneous implantable cardioverter-defibrillator was designed and tested. A report of the initial evaluation was recently published (Bardy et al., 2010).

Any effort to development ICD technology should be mainly directed to reliability of the system and to patient safety. Electrical storm is defined as a state of cardiac electrical instability manifested by several episodes of ventricular tachyarrythmias over a short period of time, there or more appropriate ventricular detection in 24 hours, usually requiring antitachycardia pacing or the delivery of multiple electrical shocks. It occurs in approximately 25% of ICD patients within 3 years, with typically 5-55 individual ventricular tachyarrythmias within one storm (Israel & Barold, 2007). The majority of episodes are ventricular tachycardias. Electrical storm consists of monomorphic ventricular tachycardia by reentry but ventricular fibrillation indicating acute ischemia is rare. The aetiology of electric storm is not clearly understood. The factors contributing to storm onset are: worsening of cardiac function, electrolyte disturbance, autonomic imbalance, drug proarrhythmia, a context with other illness, psychological stress, excess ethanol consumption and cardiac resynchronization therapy (Kantharia et al., 2006). In 35% of the patients storms represent their first event. The majority of storms occurred during winter (Greene et al., 2000). The immediate mortality is low, but the re-admission to hospital are frequent (50-80%). The prognosis is relatively good with an overall survival at 2 years of 95% and at 6 years of 77.5%. These patients could be treated with amiodarone i.v., and with reprogramming high rate pacing. The key intervention is reduction of elevated sympathetic tone by beta blockers and frequently benzodiazepines. Substrate mapping and radio frequency ablation may be useful in treatment and prevention.

3.8 Removal of devices

The value of extraction of infected or hazardous epicardial and endocardial PM or ICD leads is well established (Rusanow & Spotnitz, 2010) by open or percutaneous techniques including all lead types and indications. PM and ICD infections generally respond to antibiotics, complete hardware removal, and a hardware free interval. However, these principles cannot always be invoked, and the risk of complications is likely to increase when hardware cannot be completely removed or when a hardware-free interval is unsafe or inadvisable. Percutaneous lead extraction is superior to open extraction in terms of safety and comfort, but epicardial extraction techniques remain critically important in selected patients. Lead extraction is performed in the operating room under general anesthesia, with pump standby. Temporary transvenous pacing wires are placed when indicated despite presence of vegetations until a permanent system can safely be implanted.

Recommendations for extraction of chronically implanted transvenous pacing and defibrillator lead were published by Love et al. (2000). The percutaneous techniques should be attempted first in all patients except those presenting with large vegetations, atrial thrombus, epicardial leads, or ICD patches. After endocardial leads are dissected free at their venous entry site, anchoring collars are removed. A gentle traction is applied and if failed, a locking stylet could be inserted into the lead, and traction should be again attempted. If this too failed, telescoping sheaths could be advanced over the lead, maintaining countertraction with the locking stylet. Techniques described by Byrd et al. (1985; 1990) and electrosurgical dissection by means of an equipment manufactured by Cook Medical Inc (Bloomington, IN) or Excimer laser (Spectranetics Corp., Colorado Springs, CL) are used to extract heavily fibrotic leads. More recently, a new extraction tool, the Evolution mechanical dilator sheath (Cook Medical, Bloomington, IN), was introduced. It uses a rotational mechanism with a stainless-steel bladed tip to overcome fibrosis and cut adherences. Its rotational mechanism allows the sheath to advance down the lead body while cutting fibrotic attachments. The outer sheath covers the cutting edge when cutting activity is not desired so that venous walls are protected from damage. In addition, a shorter Evolution dilator sheath (Shortie) has been designed with a sharper and tougher blade to facilitate venous access in cases with extensive calcification under the clavicle. Initial experience with the Evolution mechanical dilator sheath for lead extraction has been reported recently (Hussein et al., 2010). Extensive surgical debridement of the pocket is performed using electrocautery and blunt dissection. Timing of device reimplantation is based primarily on sterility of blood cultures, but also on resolution of vegetations. Reimplantation is usually performed on the contralateral side. In select cases, an ipsilateral lateral sub-pectoral location may be used depending on the extent of pocket infection. Persistent inability to extract the lead prompted conversion to an open technique, when indicated. The commonest open technique after failed endocardial extraction was pursestring or snare-controlled right atriotomy through a median sternotomy (Byrd et al., 1990). Median sternotomy with cardiopulmonary bypass are used in the presence of atrial or superior vena cava thrombi, or large vegetations on the leads or on the tricuspid valve. Epicardial leads and patches were removed through a median sternotomy or left, right, or subxiphoid thoracotomy. The surgical approach is based on lead and patch location according to the the North American Society of Pacing and Electrophysiology Guidelines (Love et al., 2000).

4. Electromagnetic interference (EMI)

Electrical devices generate electromagnetic fields that may interfere with PM and ICD. The incidence of that interference is still controversial and only partially explored because the plenty of new electronic devices that are continuously introduced. These facts forced physicians to adopt very restrictive rules for use of electronic devices by patients who had cardiac implantable devices, on the other hand industries paid more attention to project safe devices. Nowadays third-generation mobile phones, Universal Mobile Telecommunication System (UMTS), were recently introduced in Europe. The safety of these devices with regard to their interference with implanted devices was studied among 100 patients by Ismail et al. (2010) who concluded that third-generation mobile phones are safe for patients with permanent PM regardless of atrial and ventricular sensitivity settings. This is due to the high-frequency band for this system (1800-2200 MHz) and the low power output between

0.01 W and 0.25 W. Also media players cause telemetry interference with PM, but it is not known whether they cause direct interference with them. Thaker et al. (2009) studied the interaction between PM and 3 different media players. PM interference was categorized as type I, II, or III. Types I and II interferences described telemetry interference and type III interference was defined as any direct interference with PM function or programmed parameters. It was concluded that media players cause telemetry interference with PM, but they do not directly interfere with PM function. Recently Lee et al. (2009) published the evidence that interferences could be generated by magnetic field of portable headphones. PM or ICD function was assessed in 100 patients during exposure to 8 different models of portable headphones to determine the incidence of clinically relevant magnetic interference. The magnetic field strength of the headphones was also measured in vitro. They concluded that clinically relevant magnetic interference from portable headphones occurred in 30 (30%) of 100 patients and more commonly affected ICD than PM patients (21/55, 38.2% versus 9/45, 20.0%; P = .048). All patients affected by magnetic interference experienced a magnet response, characterized by asynchronous pacing in PM patients and by inhibition of tachyarrhythmia detection in ICD patients. In all but one of the 30 cases of magnetic interference, removal of the headphones from the patient's chest immediately restored normal device function. Headphones with a measured magnetic field strength more or equal to 10 Gauss at 2 cm were much more likely to cause magnetic interference than were those with lower magnetic field strength (30/100 patients versus 0/100 patients; p <0.0001). Magnetic interference was not observed when headphones were placed more than 3 cm apart from the skin surface. It was concluded that clinically significant magnetic interference can occur when portable headphones are placed in close proximity to implanted PM and ICD. Patients with such a device should be advised to keep portable headphones at least 3 cm distant from their device.

5. Psychological problems

All studies concerning the quality of life in patients instrumented with PM or ICD are limited by the inability to mask therapy. The recorded effects can reflect the beliefs and expectations of patients. The patients may perceive the device as an electronic security or as a source of physical and emotional discomfort. Introducing a foreign body into the heart may cause a change in body image, cause problems in psychosocial adaptation and contribute to development of affective disorders. There are differences in intrusiveness of the two devices: ICD shocks are often painful and are delivered at unpredictable times, while pacing is unlikely felt by the patients. The interval between follow-up visit is closer for ICD patients who are encouraged to contact the clinic when they experience some problems and they can feel more dependent. Over 70% of the PM recipients are at least 70 years old (Ross & Kenny, 2000). It is difficult to examine the quality of life in the elderly because of age–related cardiovascular and cerebrovascular physiological changes, including reduced cardiac output, blunted autonomic compensatory responses and altered cerebral autoregulation. The aging and the development of the other conditions may overwhelm the moderate improvements in cardiovascular functional class and minimize the long-term effect on general quality of life. Depression and anxiety are more common in patients with permanent PM than in the general population. In one trial the quality of life was found to be similar to that of patients who require long-term hemodialysis (Lamas et al., 2002). Aydemir et al. (1997) reported that 19.1% of PM patients warranted a psychiatric diagnosis, and 10.7%

were clinically depressed. Pycha et al. (1990) identified depression of moderate severity in 35% of ICD patients, Heller et al. (1998) in 20-58%. In other studies the prevalence of affective disturbance is relatively low (Duru et al., 2001). There are important methodological differences that may account for divergent results: some studies have used short-term design and have measured quality of life with non standard instruments. In PASE study, in which the primary end-point was quality of life, no overall benefit from dual-chamber pacing as compared with single-chamber ventricular pacing was found (Lamas et al., 1998). Only patients with sinus-node dysfunction did appear to benefit from dual-chamber pacing, but those with PM implanted because of heart block did not. In individual patient the quality of life appeared to improve after upgrading to dual-chamber (Sulke et al., 1992), while it was lower at time of PM syndrome than at time of implantation. PM implantation improved health-related quality of life. The mode select was associated with much smaller, but significant, improvements in several domains, particularly physical function (Fleischmann et al., 2006). Atrioventricular synchronous pacing has a beneficial effect on most domains of quality of life in patients with hypertrophic obstructive cardiomyopathy refractory to drug treatment (Gadler et al., 1999). In a large primary-prevention population with moderately symptomatic heart failure ICD therapy was not associated with adverse quality effects during 30 months of follow-up (Mark et al., 2008). The occurrence of ICD shocks was associated with increased psychological distress in both patients and their families. The quality of life of patients in the month after a shock was significantly decreased in perceived general health, physical and emotional functioning, social functioning, and self-rated health. This result did not persist however, because the shock experience remembers the patients have a device with a life preserving function. They report limitations in their activities and admit anxiety about battery depletion and technical problems. The most distressing aspects of receiving a shock are the lack of warning, multiple shocks, nervousness, fear of sudden death, dizziness, weakness and chest soreness. Educational interventions and support group might incorporate knowledge about the effects of devices, to facilitate anticipatory guidance and preparation of patient and family members for ICD shocks.

6. Conclusion

Strict adherence to the widely accepted guidelines and recommendations, awareness of potential complications, and a meticulous approach to the implant and post implant techniques and follow-up may certainly reduce the incidence of complications after implantation of PM and ICD, more than often life-saving devices. To be aware of PM and ICD complications is an essential first step for good clinical practice in this area.

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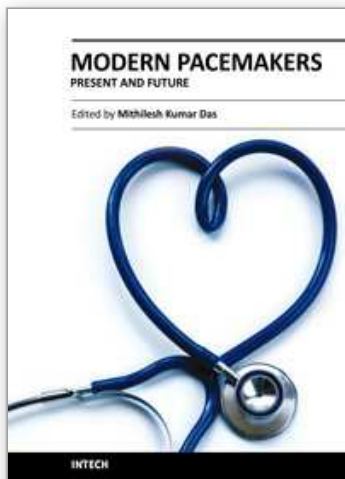
Modern Pacemakers - Present and Future

Edited by Prof. Mithilesh R Das

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

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