Chapter 8

Mechanism and Management of Pacing Lead Related Cardiac Perforation

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1. Introduction

1.1. Incidence and time course for cardiac perforation

Cardiac perforation due to a pacemaker or defibrillator lead occurs at a rate of 0.4–2.0%. Since first described [1], the overall incidence has decreased significantly. The reported incidence of this complication has been as low as < 1 % to as high as 15% [2-4]. Of 4,280 permanent pacemaker (PPM) implants only 50 (1.2%) patients developed a significant effusion and symptoms consistent with a perforation [5]. The incidence of this complication from the time of surgery decreases over time. By convention, a perforation detected within 24 hours, is classified as acute. If detected within 30 days of implantation (usually 5 days- 4 weeks), it is referred to as early (sub-acute); and, those detected after 30 days referred to as late, delayed or chronic perforation. The majority of perforations manifest within a year but rarely cases have been reported as late as five years following implantation. [6]

1.2. RV perforation

1.2.1. Modeling of RV perforation

In order to develop perforation resistant leads, several models of myocardium - lead interaction have been developed. Lead related factors that affect propensity for perforation include lead thickness as well as stiffness. Refinement of the current lead technology produce leads with both a reduced thickness and stiffness. In experimental model [7] bipolar leads with silicone rubber insulation had lower stiffness as compared with those coated with polyurethane. While a lower stiffness is a desirable property, it results in a

thinner lead with a higher penetration pressure (force per unit area). In a porcine perforation model [8] studying the effect of cylindrical punches, of variable diameter, on RV wall tissue the penetration pressure (force/cross sectional area) decreases as a function of an increasing punch diameter. The penetration pressure decreased by approximately 25 % as the punch diameter was doubled. **(Figure 1)**

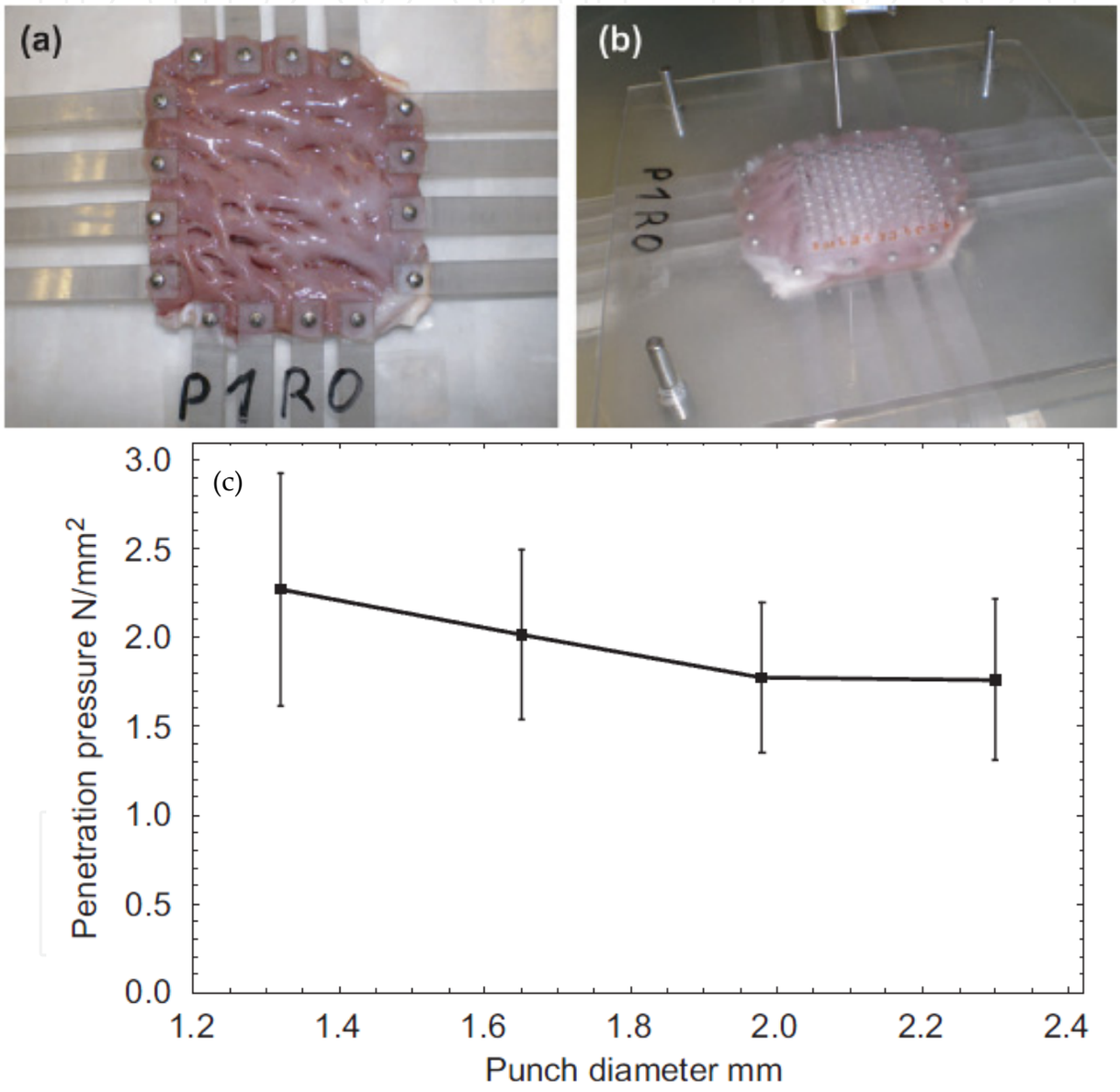

Figure 1. Figures show the porcine specimen of RV free wall fixed to the grips (a) and specimen bi-axially stretched to its testing conditions (b). Figure (c) shows penetration characteristics of the right ventricular myocardium. Penetration pressure (penetration force divided by punch cross section) with respect to the punch diameter. "(With permission from Gasser TC: Journal of Biomechanics. 42:629-633, 2009.)

Myocardium related factors which contribute to the perforation risk include the wall thickness and the viscoelastic properties of the tissue. Experimental data suggest that the myocardium is anisotropic (with the highest stiffness in the direction of the fiber's longitudinal orientation), non-linear, nearly elastic and exhibiting heterogeneity throughout a particular chamber (e.g. RV) [9].

The interaction between the lead tip and myocardium is complex, varies with the cardiac cycle, and changes over time. The lead tip penetrates the tissue, creating a fissure between local fibers. The lead wedges into this "crack", expanding the opening over time [7]. This is illustrated by the detection of a "crack" (fissure) both by light and electron microscopy, see **Figures 2, 3**. However, it should be noted that when a "crack" splits the plane of fibers, compared to a punch, there is higher likelihood of closure due to the opposing forces of the myocardium, which act to seal the fissure. This may explain why many perforations are subclinical. Despite advancements in the knowledge of the lead-tissue interaction and advances in lead technology, this complication has still not been eliminated.

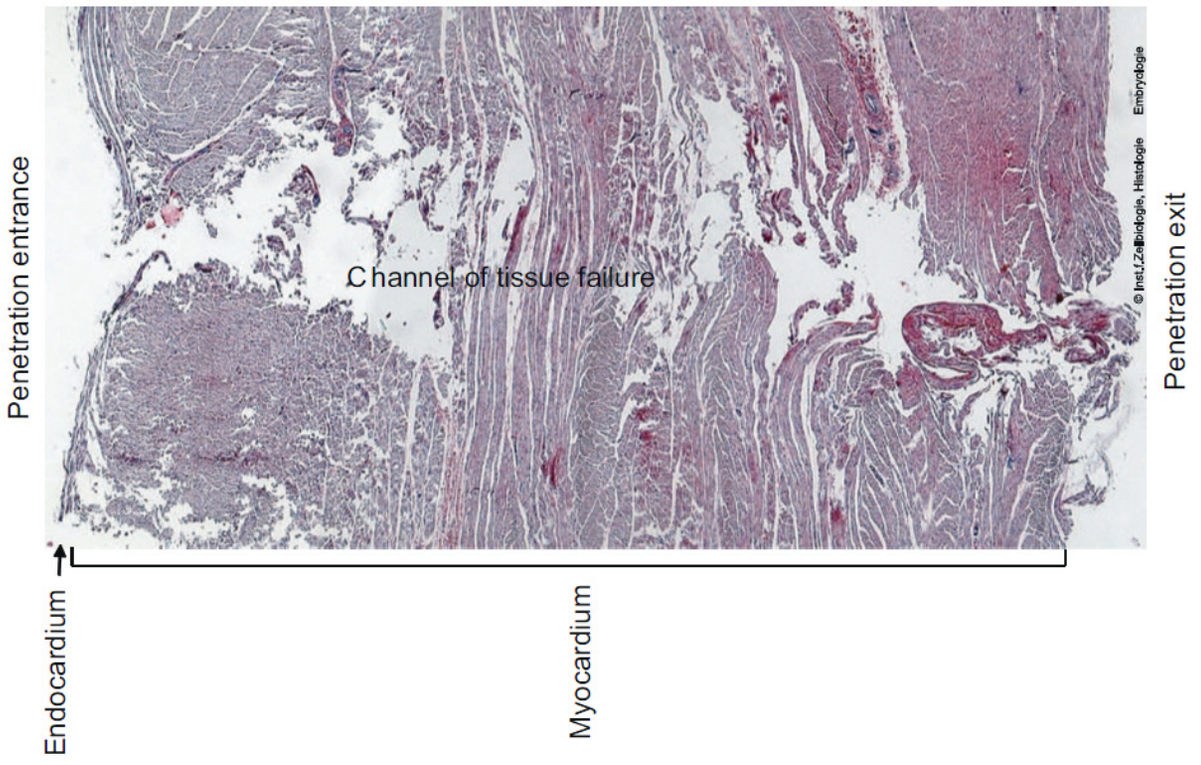

Figure 2. Microscopy of endocardium and myocardium of RV of a porcine specimen with a punch of 2.3 mm diameter. Picrosirius red stain illustrates tissue failure through the different layers of the ventricular tissue. "(With permission from Gasser TC: Journal of Biomechanics. 42:629-633, 2009.)

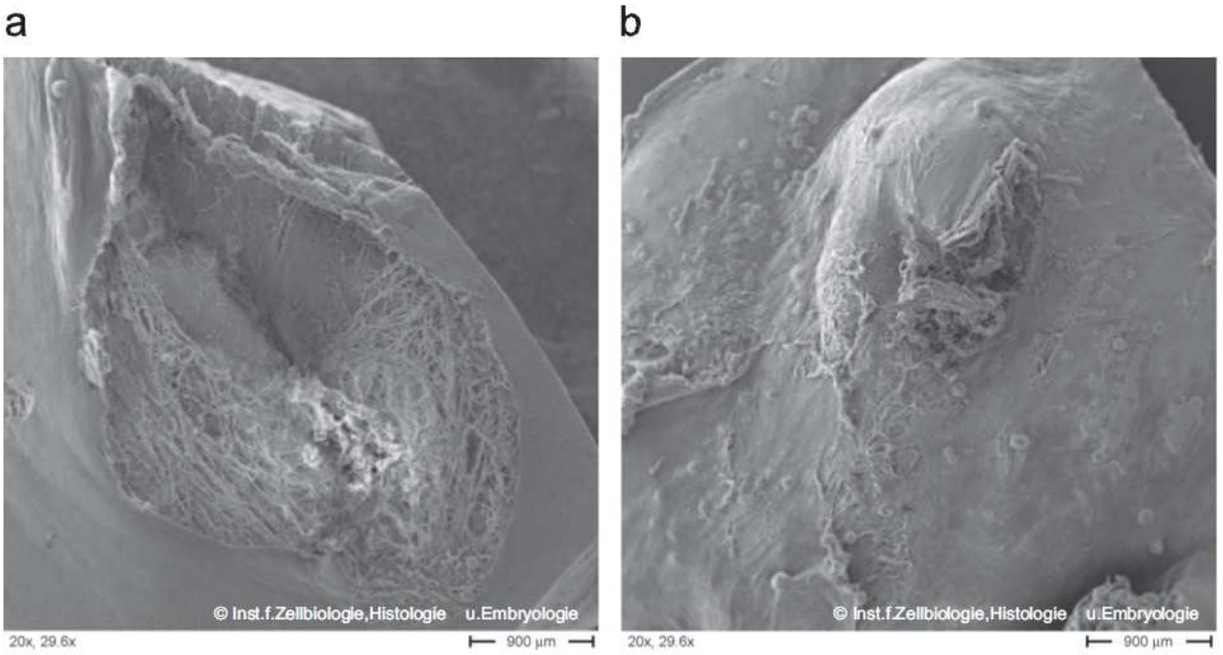

Figure 3. Electron microscopy images taken from the (a) entrance and (b) exit of the punch illustrating a splitting mode failure and the presence of remaining deformations respectively. "(With permission from Gasser TC: Journal of Biomechanics. 42:629-633, 2009.)

1.2.2. Free wall perforation

Hirschl et al studied the incidence of an asymptomatic lead perforation, by computed tomography and correlated with electrophysiologic data. Out of 100 consecutive PPM and intra-cardiac defibrillator (ICD) implantations, right ventricular (RV) perforation was less common (6% vs. 15%) than right atrial (RA) perforation, respectively [4]. However, in a recent study of 2,385 patients undergoing an electrophysiology study as well as pacemaker lead insertion, 7 (0.3%) patients experienced an RV perforation compared to only 1 (0.04%) RA perforation [10]. The predisposition to develop an RV perforation was a function of RV location; explained in part by the heterogeneous nature of the RV. The RV resembles a pyramidal structure with the interventricular septum (IVS) located posteriorly and the free wall located anteriorly. The RV free wall is thinner (<0.5 cm) than the septum (ranging from 0.6 to 0.9 cm) [11], accounting for the higher risk of perforation. Positioning leads in the RV IVS may not only reduce the risk of perforation but reduce the degree of QRS widening, possibly minimizing asynchrony. In the majority of cases the perforating lead is contained within the pericardium, however, in rare cases the lead can migrate along the pericardium traversing the diaphragm into the abdominal cavity. **Figure 4 and 5** (chest x-ray and CT scan) illustrate an example of a RV free wall perforation.

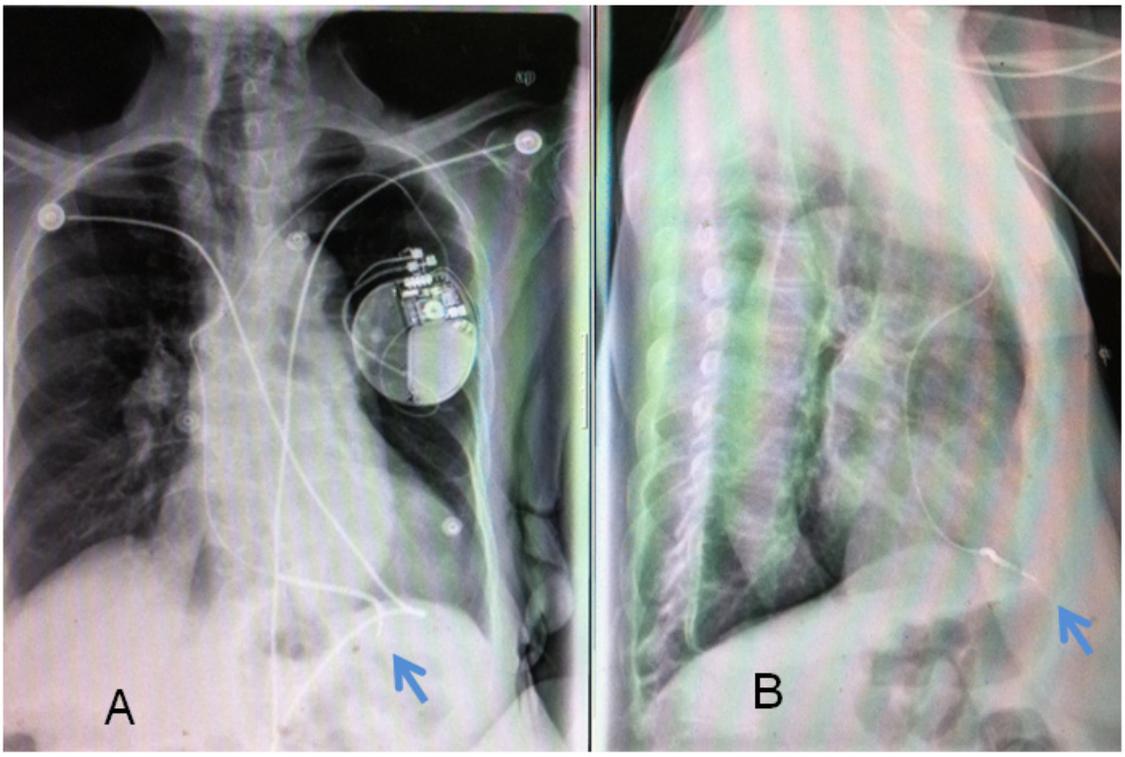

Figure 4. RV lead (arrows) perforating through the RV and sharply pointing below the diaphragm in PA view (A) and the lead is excessively close to the sternum and gastric air under the left hemidiaphragm in lateral view (B).

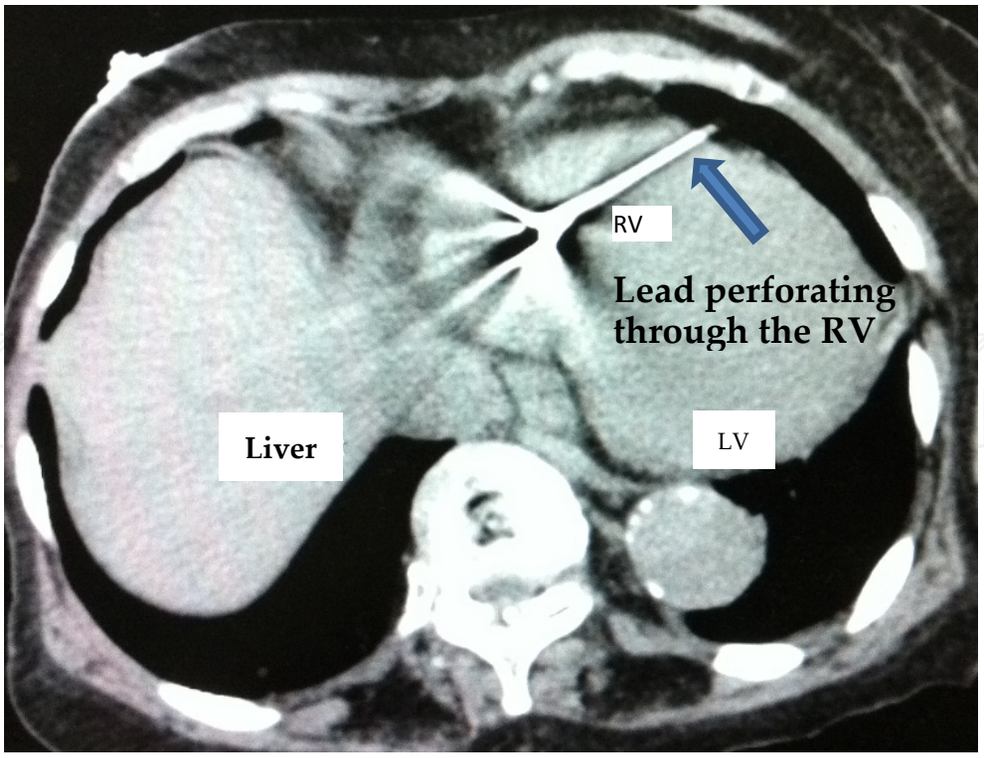

Figure 5. Axial view of CT scan of chest of the same patient in figure 4, showing the RV lead perforation through the RV apex. RV=right ventricle, LV=left ventricle

1.2.3. Migration to the LV cavity

As a consequence of the thicker IVS, direct perforation through the septum and migration of lead tip into the left ventricle (LV) is rare [12]. However, the RV PPM lead may end up in LV due to mal-positioning during implantation. A common route for the lead to access the LV is a patent foramen ovale [13] or atrial septal defect [14]. These patients may remain asymptomatic for a long time, and often lead malposition is detected incidentally. It can be detected by fluoroscopy or chest radiography, presence of right bundle branch block (RBBB) with pacing on electrocardiogram (ECG) or when the patient presents with symptoms of a peripheral embolism (e.g. cerebrovascular accident) [14]. LV epicardial pacing may result when the electrode perforates through the RV apex migrating and remaining along the LV epicardial surface [15].

1.3. RA perforation

The RA is more susceptible to perforation as compared to the RV, attributable to the thinner wall of the atrium [4]. Thicker electrodes and active fixation leads are more likely to cause this form of perforation. Most commonly, one observes RA appendageal perforation. Age, gender, device type and anticoagulation status appear to increase the risk for perforation [16]. The lead may traverse through the pericardium into the lung field. Although, surgical intervention may be needed most cases are treated with percutaneous drainage or simple lead repositioning.

1.4. Coronary Sinus (CS) dissection/perforation

CS perforation is a relatively rare complication during pacemaker implantation. The rate of CS perforation has seen an upturn with increased CS cannulation during LV lead insertion for cardiac resynchronization therapy (CRT). The incidence of CS dissection and CS perforation have been reported to vary from 2%-4%, 0.3%-2%, respectively [17]. Cardiac tamponade and pericarditis have been reported with a CS perforation [18].

In the early experience with PPM implantation the CS was selected for the purpose of RA pacing, affording a more stable catheter position. With a high perforation risk, tamponade and thrombosis, complicating lead insertion with CS manipulation, the RA became the preferred site. Enhanced stability with the RA appendage as the preferred site occurred once active fixation leads became mainstream. **Figure 6** shows a CS lead perforating through the body of coronary sinus. During CRT, LV pacing is achieved by placing an electrode via the os of the CS to one of its tributaries. This procedure may result in a CS dissection. The overwhelming majority of CS dissections are without incident, with the dissection flap sealed by blood flow within the CS. Even if contrast extravasates out of the CS, the low perfusion pressure within the CS prevents any significant bleeding. In the majority of cases the true lumen of the CS can be re-accessed. The lead serves to tamponade the dissection or perforation and the procedure can proceed with successful lead placement. Serial echocardiograms are needed to assure any significant pericardial blood accumulation. If

significant blood accumulation occurs within the pericardium this can be relieved via a percutaneous approach, very rarely patients require surgical drainage.

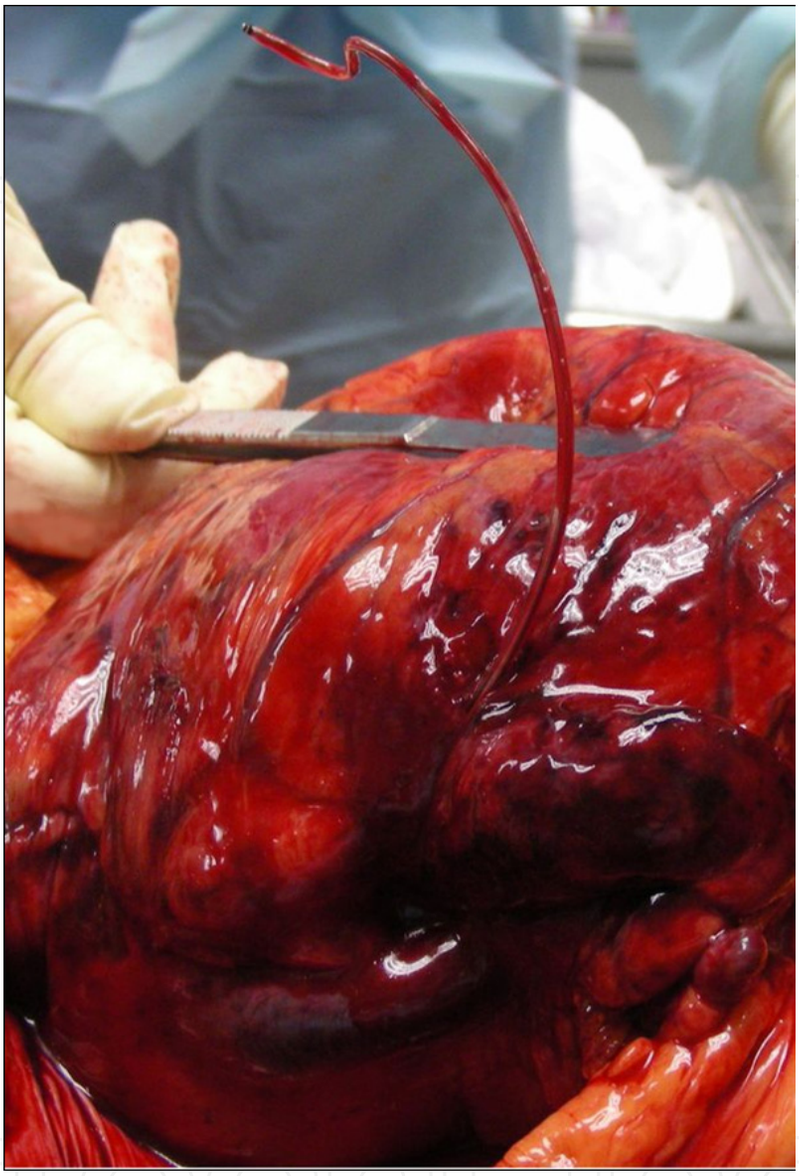

Figure 6. CS lead is perforating through coronary sinus as seen during autopsy. Patient was asymptomatic and died for unrelated reason. Courtesy of Jenny Libien, MD. Department of Pathology, SUNY Downstate. Brooklyn, NY.

1.5. Great vessel injury/dissection

Transvenous leads can be placed through various venous access sites, including, axillary, subclavian or cephalic veins. Innominate or internal jugular veins have been used as well. Lead placement via persistent left sided superior vena cava has been reported [19] and in such cases implantation could be challenging. These electrodes are designed to traverse the circulation from the point of vessel entry proximal to the generator to the targeted cardiac chamber. As one can imagine, injury can theoretical occur anywhere through this entire route.

Commonly, venous injury, hematoma or even inadvertent arterial puncture can occur during the time of implantation and usually respond to compression. Perforation of the innominate vein has been reported and is attributed to its curvaceous course. This tortuosity increases with age, with a higher predisposition in the elderly [20]. The use of the internal jugular vein was associated with complications in 3 out of 92 implantations, including one patient who suffered permanent recurrent laryngeal nerve injury and two with thrombophlebitis. [21]

2. Diagnostic approach

2.1. Presentation and symptoms

Manifestations of a cardiac perforation depend largely on the site, presence and rate of accumulation of pericardial fluid. Approximately 1%-7% of patients present acutely; 1% present early and 0.1% present as a late perforation. [2] Acute perforations often present with chest pain, dyspnea and signs of pericardial effusion or tamponade. Asymptomatic perforations are not infrequent and as a result the diagnosis is often delayed. The most common presentation of a delayed perforation is a hemopericardium (with or without cardiac tamponade), pericarditis, diaphragm or chest wall muscle stimulation, loss of capture and pneumothorax [22]. Penetration to the superior mediastinum may result in largyngeal nerve injury. Rarely patient's may present with a mediastinal bleed.

The presentation of patients with cardiac perforation can be variable; therefore, a high index of suspicion needs to be maintained for a rapid diagnosis. Patients present with chest pain, shortness of breath, presyncope or syncope. The physical examination may reveal signs of cardiac tamponade (hypotension, tachycardia, elevated jugular venous pressure, distant heart sounds, pulsus paradoxus etc.), friction rub, ascites or leg edema. In addition, signs of pneumothorax or pleural effusions may be noted. On ECG, a new RBBB may be noted, suggesting insertion or migration of the lead into the LV cavity, occasional penetration into the epicardium.

2.2. Analysis of interrogation data

Upon suspicion of a perforation the device should be interrogated immediately. Routine device checks are performed to ensure normal pacing and or sensing function. A thorough device evaluation can be helpful in increasing one's suspicion of lead migration. In many instances one will detect an alteration in pacing function. New onset diaphragmatic, pectoralis or intercostal muscle stimulation with intermittent or constant failure to sense and capture should increase ones suspicion of a lead migration and possible perforation. However, one should keep in mind that a normal interrogation does not exclude the presence of a cardiac perforation.

2.3. Use of remote monitoring for the early detection of a perforation

Most of the current defibrillators and some PPMs have the capacity for home monitoring. They routinely transmit data regarding rhythm and system integrity at set time intervals. In

the correct setting, abnormalities in the transmitted data should increase one's level of supposition for a perforation. A perforation often leads to a change in impedance and/or pacing threshold with a loss of capture. Several published reports have shown that early detection of a perforation was made via home monitoring. The perforations were detected 12 days and 4 weeks, respectively, following the insertion of the device [20, 21]. With growing support for the use of home monitoring the number of cases diagnosed via this modality is expected to grow.

2.4. Imaging studies

Multi-detector computed tomography (MDCT) is the imaging modality of choice in diagnosing a lead perforation, identifying the pericardial effusion while, assisting in the therapeutic approach. However, a simple chest x-ray (preferably postero-anterior and lateral view) should be the starting point in the diagnosis of a cardiac perforation. An enlarged cardiac silhouette, pneumothorax, pleural effusion can also be supportive in making the diagnosis. Usually the lead tip should be within 3 mm of the cardiac border; if the tip extends outside the cardiac border, a perforation should be suspected. Physicians should familiarize themselves with the expected position of the RA or RV leads. In this regard, a baseline x-ray is useful. If a lead deviates significantly from its original or expected position or has developed an unusual contour, one should suspect perforation. **Figure 7** shows the normal cardiac anatomy with respect to pacing leads. On a frontal radiograph, the tip of the right ventricular lead should be seen to the left of the spine. On the lateral view, it should traverse anteriorly towards the RV [23]. However, in some centers leads are preferentially positioned in RV outflow tract (free wall or septal aspect). In these cases a left lateral view (45 degree) x-ray would display the RV lead to be pointing to the left in the case of free wall and to the right in case of septal positioning of the lead. Similarly, a left lateral x-ray would show the RV lead projecting anteriorly in case of free wall or posteriorly in case of septal positioning of the lead. [24]

Fluoroscopy plays an important role during positioning of the leads. In this regard the implanter needs to have a thorough understanding of flouroscopic anatomy, in order to confirm proper positioning and detect a mal-positioned lead. In the case of a perforation, the lead electrodes can extend outside of the cardiac silhouette and not move with the contraction of the heart [22]. Further clues to the early detection of a cardiac perforation include: an unexplained decrease in blood pressure; a decreased pulsatility of the cardiac silhouette; an increase in the size of the cardiac silhouette; and, an abnormal position of lead relative to pericardial outline [23].

Echocardiography is widely available and easily performed to detect a cardiac perforation. Echocardiography can detect pericardial fluid, cardiac tamponade, malposition as well as extension through the myocardium but rarely is able to identify the extra-cardiac presence of the lead. [25] On occasion the perforation may not cause any detectable pericardial effusion or there may be poor data acquisition, hence the absence of an effusion does not rule out a cardiac perforation.

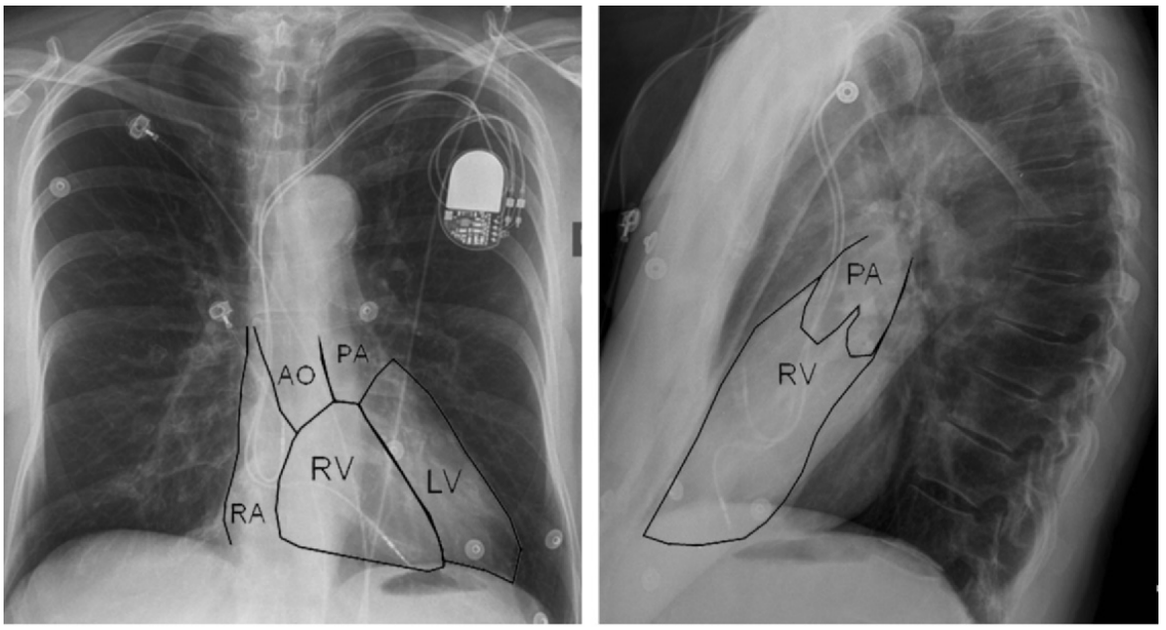

Figure 7. Normal cardiac anatomy. Frontal (a) and lateral (b) chest radiographs show the aorta (AO in a), left ventricle (LV in a), main pulmonary artery *(PA)*, right atrium (*RA* in a), and right ventricle (RV). "(With permission from Aguilera AL: Radiographics. 31:1669-1682,2011.)

3. Lead design and propensity for perforation

3.1. Active vs. passive fixation leads

The type of lead fixation mechanism contributes to the risk of perforation. Studies have shown that active fixation (screw-in) leads are associated with a higher risk of perforation, related to lead thickness and over-torquing. It has been speculated that the newer, thinner leads, exert a higher force per unit area on the endocardium, and exhibit a higher incidence of delayed perforations [25]. In addition, the tip force and the myocardial counterforce are variable, changing over time and with each cardiac cycle. When the lead tip force exceeds the counterforce a perforation can occur. On the other hand, passive fixation lead placed in vulnerable region of the RV (apex) may exhibit an even higher risk of perforation compared to the active fixation leads. It is speculated that successful insertion of a passive fixation lead uses the RV apex, whose trabeculations are used to anchor the lead; however, this represents the most vulnerable zone within the chamber. The higher incidence of perforation associated with active fixation leads may be attributed to the mere fact that we are actively "screwing" the lead into the RV wall, making sure we have adequate tissue contact by monitoring the current of injury.

3.2. Defibrillation lead size and risk of perforation

Recent studies have reported a surprisingly high incidence of cardiac perforation with small body (7F) active fixation defibrillator leads (Riata) as compared to Sprint Fidelis leads (3.8% vs. 0%) [23]. The reasons for higher perforation rate are largely unknown but may include

lead design and breeches in structural integrity [26]. Another important property in assessing the propensity of a lead to perforate is "thickness". Older manufactured leads were thicker than the newer models. Thicker leads are stiffer and exert a higher pressure on the cardiac tissue thus associated with higher cardiac perforations.

3.3. Preformed J shape lead design and risk for retained wire perforation

One of the first and most serious lead recalls involved the Accufix atrial pacing lead which was recalled due to a high risk of J retention wire fracture. The J wire fracture protruded with time through the insulation resulting in a high rate of atrial perforation [27], **(Figure 8).** Many of these leads needed to be extracted and were associated with a significant morbidity and mortality, due to the friability of the lead. Temporary atrial and ventricular epicardial pacing wires are routinely placed after cardiac surgery. These wires are routinely removed from patients prior to discharge. They are pulled and cut at skin level and allowed to retract into the pericardial sac. These wires can migrate to the RV extend into the pulmonary artery [28] or migrate to the left atrium and subsequently to the carotid arteries [29].

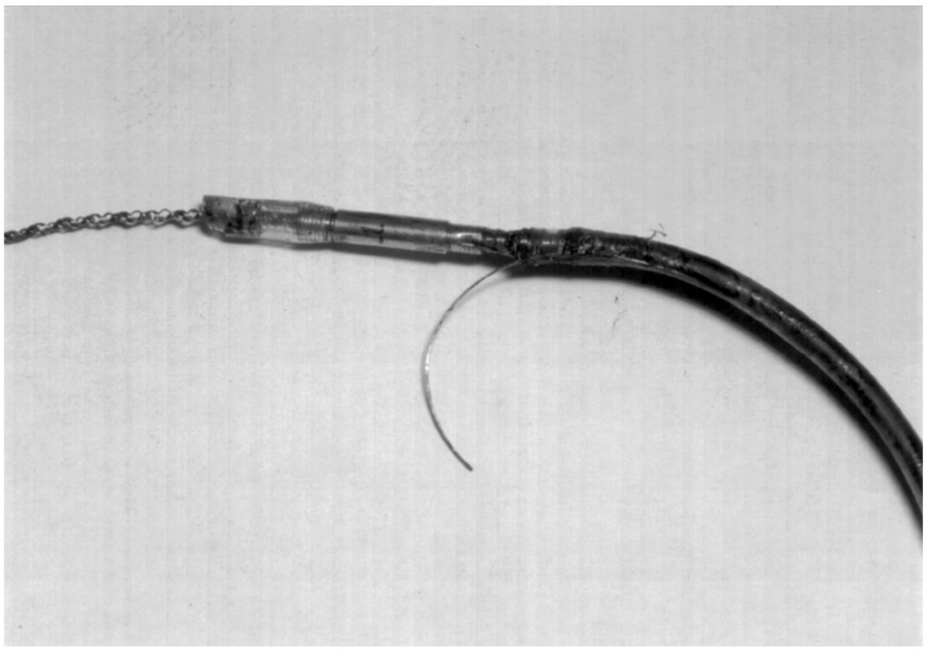

Figure 8. J-retention wire protruding through the insulation. (With Permission from Gorog DA: Heart. 83:563-563, 2000.)

4. Patient risk factors and risk for perforation

Cardiac perforation may be associated with intrinsic myocardial, as well as, patient-specific factors rendering this, rather uncommon complication more predictable. Mahapatra et al [5] have investigated the risk factors for the development of symptomatic pericardial effusion consistent with cardiac perforation in more than 4,000 patients. The multivariate analysis identified variables associated with perforation included: steroid use (HR 3.2); temporary

pacemaker wire insertion (HR 2.7); active-fixation leads (HR 2.5); low body-mass index (HR 2.5); advancing age (HR 1.4); and, longer fluoroscopy times (HR 1.3). Predictors with a lower perforation potential included, a pulmonary artery pressure >35 mm Hg (probable protective effect of hypertrophied RV) and BMI>30 (univariate predictor only). Interestingly, steroid use is associated with cardiac atrophy, mediated by a muscle specific protein called muscle ring finger-1 [30].

The specific lead type, site of implantation, and the occurrence of traumatizing factors may result in a higher incidence of RV perforation. Although factors, such as defibrillator leads, excessive slack during lead implantation, smaller diameter leads with higher resistance, chest wall trauma especially soon after implantation and multiple shocks delivered around the time of implantation are yet to be validated, they remain important considerations when one considers the predisposition to perforation. It has previously been suggested that RV free wall and/or apical insertions, especially in the elderly are associated with higher perforation rate attributed to the thinner wall in that region, which atrophies with age. In addition, the use of anticoagulants may increase the risk of perforation.

5. Implantation techniques to minimize the risk of perforation

Certain steps should be taken to reduce the incidence of lead related cardiac perforation. It is imperative that in obtaining an informed consent, patients should be made aware of the symptoms to watch for following a surgery for a more rapid recognition of complications. Only experienced implanters with highly trained personnel, in dedicated electrophysiology laboratories, should perform these procedures.

Careful patient selection and avoidance of temporary pacing wires would be helpful in reducing this complication. In addition, selection of more compliant (less stiff) leads, wider lead tip area and placement in the RV septum can further reduce this risk. It has also been suggested that excessive slack on the lead after placement can increase the tension on the free wall resulting in a late perforation. Post-procedure, a high index of suspicion needs to be maintained; while the clinical examination, routine chest x-ray, ECG and pacemaker interrogation including remote monitoring can help expedite the diagnosis.

6. Management of lead perforation

The management of cardiac perforation depends on the timing of this complication. Acutely, the pacing lead can be repositioned and the patient is monitored with serial echocardiography. The risk of bleeding during removal of a lead increases rapidly in non-acute perforations. Subacute or delayed perforation is dealt with on a case by case basis. In some cases, when the cardiac perforation is asymptomatic or not associated with pacing/sensing malfunction or mediastinal bleeding or if the risk of lead removal outweighs the non-removal, the lead can be left in place. Patients can be observed and watched for lead migration and at that point lead extraction could be performed. [16, 30, 31] Simple lead positioning can be utilized in some cases especially in acute or early stages when minimal fibrotic adhesions facilitate repositioning. [32] If there is bleeding within or outside of the mediastinum, accompanied by

lung or other vascular damage, especially if the lead was recently implanted, the lead should be explanted surgically to insure adequate surgical correction of the problem.

6.1. Non-thoracotomy approach

Active Fixation leads can be removed transvenously under direct fluoroscopic visualization. However, an Expert Consensus Statement published by the Heart Rhythm Society [33] recently classifies following conditions as class III indication for transvenous extraction of non-functional leads. They cited the following in their decision: lead removal is not indicated in patients with a known anomalous placement of leads through structures other than normal venous and cardiac structures, (e.g. subclavian artery, aorta, pleura, atrial or ventricular wall or mediastinum) or through a systemic venous atrium or systemic ventricle. Additional techniques including surgical backup should be deployed in the appropriate clinical scenario. Simple perforations can be managed with a percutaneous approach. In one series of 11 patients with an RV perforation, in 10 patients, the leads were removed by simple traction under fluoroscopic guidance in the operating room with surgical backup support. In one patient, surgical intervention was required after a pericardiocentesis, underscoring the need for close monitoring [34]. In the analysis of 33 consecutive patients who developed cardiac perforation with associated with tamponade, within 24 hours of procedure (electrophysiology or device implant), nineteen (58%) patients were managed conservatively with intravenous fluid and pressor support. Fourteen (42%) required a pericardiocentesis. The authors suggested that in the setting of acute tamponade associated with a large pericardial effusion (> 2 cm), associated with RV diastolic collapse, pericardiocentesis would likely be needed. As a less invasive measure, video assisted thoracoscopy surgery (VATS) with adjuvant anterior minimally invasive anterior thoracotomy has also been used [35].

6.2. Thoracotomy approach

A surgical approach is recommended when cardiac tamponade is the presenting feature, complicating a lead extraction or an anomalous approach to lead implantation is present. Passive fixation leads are bulky and have tines potentially worsening damage if the leads are “pulled” transvenously.

During the procedure the lead tip is freed up from the tissue, and then the body of the lead is removed transvenously. Surgeons can also place epicardial leads as an alternative approach. It is recommended that pericardium be cleansed after extracting the contents of the hemopericardium to reduce the risk of future constrictive pericarditis [36). A surgical approach results in an increased hospital stay and its concomitant risks. In this regard the use of VATS with adjuvant anterior minimally invasive anterior thoracotomy appears promising in reducing the hospital stay and its associated morbidities.

7. Summary

The overall incidence of a cardiac perforation as a result of a transvenous lead insertion is low. Perforations can exhibit a range of manifestations, from asymptomatic to life-threatening. In

order to minimize complications, temporary pacing lead placement should be avoided unless absolutely necessary. Extreme caution should be used when implanting a device in patients who are on steroids or anticoagulants. Once perforation is detected the management depends on the duration of implant (acute vs. chronic), accompanying findings (tamponade or pneumothorax) and the type of lead. In the case of an acute perforation, leads can be repositioned under fluoroscopy. Hemopericardium with tamponade can be managed via percutaneous pericardiocentesis and on occasion may require surgical drainage.

Abbreviations

BMI= body mass index
CRT= cardiac resynchronization therapy
CS= coronary sinus
CT= computerized tomography
ECG= electrocardiogram
FDA= federal drug administration
IVS= interventricular septum
HR= hazard risk
LV= left ventricle
PPM or ppms= pacemaker or pacemakers
RA= right atrium
RBBB= right bundle branch block
RV= right ventricle
VATS= video assisted thoracoscopy surgery

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