

## Injury to the coronary arteries and related structures by implantation of cardiac implantable electronic devices

### Benjamin J. Pang<sup>1,2</sup>, S. Serge Barold<sup>3</sup>, and Harry G. Mond<sup>1,2\*</sup>

<sup>1</sup>Department of Cardiology, Royal Melbourne Hospital, Grattan Street, Parkville 3050, Victoria, Australia; <sup>2</sup>Department of Medicine, University of Melbourne, Victoria, Australia; and <sup>3</sup>Florida Heart Rhythm Institute, Tampa, FL, USA

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Damage to the coronary arteries and related structures from pacemaker and implantable cardioverter-defibrillator lead implantation is a rarely reported complication that can lead to myocardial infarction and pericardial tamponade that may occur acutely or even years later. We summarize the reported cases of injury to coronary arteries and related structures and review the causes of troponin elevation in the setting of cardiac implantable electronic device implantation.

**Keywords** 

Pacemaker • ICD • Coronary artery • Perforation • Troponin

### Introduction

Major complications from implantation of permanent pacemakers and implantable cardioverter-defibrillators (ICDs) [cardiac implantable electronic devices (CIED)] such as pneumothorax, cardiac perforation, and infection can occur in 4–7% of patients.<sup>1–3</sup> While cardiac troponin is important in the diagnosis of myocardial infarction, in the setting of recent device implantation, raised levels may also indicate direct trauma to coronary arteries, inadvertent placement of leads into the arterial system, pulmonary embolism, and takotsubo cardiomyopathy.<sup>4</sup> The objectives of this manuscript are to: (i) characterize the presentation and treatment of lead injury to coronary arteries and related structures, and (ii) review the causes of troponin elevation in the setting of recent device implantation.

### **Methods**

A systematic search of the SCOPUS, MEDLINE, and Pubmed databases up to October 2014 was conducted for case reports of damage to coronary and bypass graft arteries as well as troponin elevations following insertion of a CIED. The MeSH search string was (artery [All Fields] and troponin [All Fields] and pacing [All Fields] and ICD [All Fields] and takotsubo cardiomyopathy [All Fields] and ('humans' [MeSH Terms] and English [Lang]). The literature search was limited to studies in humans and articles published in peer-reviewed journals in English. Reference lists from reviews and articles were also hand searched for relevant publications.

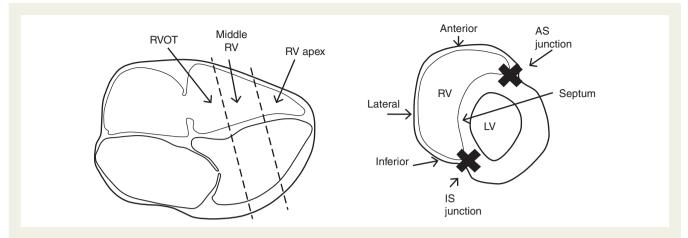
### Damage to coronary arteries and related structures

Acute perforation of the right atrium (RA) or right ventricle (RV) by pacemaker or ICD leads occurs in 0.5-2% of patients.<sup>5-7</sup> It can result in chest pain, pericardial tamponade, and potentially death. Delayed perforation months to years post-implantation may lead to pneumothorax, pericardial tamponade, and migration of leads to the lung or chest wall.<sup>7-12</sup> Damage to coronary arteries is a less well recognized sequelae of lead perforation. Cardiac implantable electronic devices leads are deployed using fluoroscopy, a two-dimensional imaging modality, where the proximity of lead tips to coronary arteries is not well appreciated unless simultaneous contrast angiography is performed.

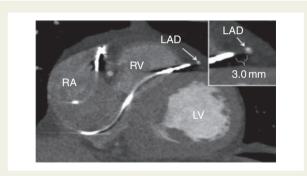
We recently characterized the position and proximity of CIED lead tips to coronary arteries using multiplanar cardiac computed tomography (CT)<sup>13</sup> and described a schema for defining lead tip position in the RV.<sup>14</sup> In this schema, the long axis of the RV is divided into thirds (RV outflow tract, middle RV, and RV apex), and the short-axis walls of the RV are defined as per *Figure 1*. In our analysis, a majority of RV 'septal' lead tips were not implanted on the 'septum', but instead on the RV antero-septal junction (*Figure 1*).<sup>14</sup> The left anterior descending (LAD) coronary artery runs within the interventricular groove superficial to the RV antero-septal junction. In our series, leads in the RV antero-septal junction were only a few millimetres (median 4.7 mm) away from the LAD (*Figure 2*).<sup>13</sup>

\* Corresponding author. Tel: +61 3 9342 7133; fax: +61 3 9347 2808. E-mail address: hmond@bigpond.net.au

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**Figure I** Schema for characterizing pacemaker lead location in the cardiac CT long and short axis. (Left) Computed tomography four-chamber long-axis view, classifying lead position into the RV outflow tract (RVOT), middle RV or RV apex. (Right) Computed tomography short-axis view RV pacing lead position: septum, anterior RV wall, antero-septal (AS) junction, lateral wall, inferior wall, and infero-septal (IS) junction.<sup>11</sup>



**Figure 2** Parasternal short-axis CT view. The RV lead is in the antero-septal junction in close proximity to the LAD coronary artery. (Inset) The measured distance between the RV lead tip and LAD coronary artery is 3.0 mm.<sup>13</sup>

There have been two reports of injury to the LAD and two cases of damage to the right coronary artery (*Table 1*)<sup>15–18</sup> from lead perforation. The three perforated active-fixation leads presented within 36 h of implantation,<sup>17,19,20</sup> and a passive-fixation lead was incidentally discovered 4 years later.<sup>17</sup>

Damage to the LAD resulted in acute ST elevation myocardial infarction. In both cases, fluoroscopic images showed lead placement in the RV antero-septal junction short-axis position and middle RV long-axis position. In the case by Parwani *et al.*,<sup>16</sup> the helix of the pacing lead was deployed into the LAD coronary artery. It was treated by simultaneous removal of the lead and insertion of a covered stent. Nishiyama *et al.*<sup>15</sup> described a case where an ICD lead caused myocardial infarction secondary to coronary artery spasm distal to the lead. Spasm resolved when the lead was removed percutaneously.

The two cases of lead injury to the right coronary artery required surgical repair. In the case by Nagakawa<sup>18</sup> implantation of an active-fixation RA lead was complicated 3 h post-procedure by pericardial tamponade and haemodynamic collapse. At surgery, only the helix was seen protruding out of the RA wall and there was active bleeding from a lacerated right coronary artery. Pericardium was used to

repair the coronary artery and to cover the helix protruding from the RA. Khoueiry et al.<sup>17</sup> described the incidental finding on chest X-ray of a RA mass 4 years post-pacemaker implantation. Cardiac CT showed a perforated passive-fixation lead that had resulted in a RA pseudoaneurysm with an arteriovenous fistula connected to the right coronary artery. Treatment consisted of surgical ligation of the artery, closure of the RA pseudoaneurysm, repair of the RA perforation, and replacement of the atrial lead.

In the previous cases, despite penetrating into coronary arteries, pacing characteristics were within the normal range and only one case was associated with a pericardial effusion. $^{18}$ 

### Damage to the aorta

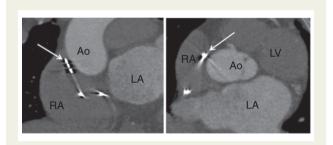
Using cardiac CT, we found RA pacing lead tips on the anterior RA medial wall in close proximity to the aorta (*Figure 3*).<sup>13</sup> Aortic root puncture and perforation are known rare complications of RA transseptal catheterization for left atrial access. A review of 5520 patients undergoing trans-septal catheterization reported five cases (0.09%) of aortic puncture and of these, three had aortic perforation.<sup>21</sup> There have been four reports of pericardial tamponade occurring secondary to active fixation pacing leads perforating through the RA wall and lacerating the adjacent aorta.<sup>22–25</sup> Clinical presentation ranged from hours<sup>25</sup> to 2 weeks<sup>22</sup> post-implantation. The aortic root<sup>25</sup> and the ascending aorta<sup>22–24</sup> were lacerated. All required surgical correction of the aorta. In one case the RA pacing lead was repositioned<sup>23</sup> and in the other three cases, the lead was left and the atrium surrounding the pacing lead was plicated.<sup>22–25</sup>

# Inadvertent implantation of leads into coronary arteries

Mistaken introduction of leads into the arterial rather than venous system is a rarely reported complication. There are reports <sup>19,20,26,27</sup> of implantation of leads into the lumen or adjacent to the ostium of coronary arteries: two into the right coronary artery, one into the

Study	Country	Age and sex	Lead fixation	Artery	Damage	Complication	Treatment
Parwani et al. <sup>16</sup>	Germany	49 M	VActive	LAD	100% occlusion	Ant STEMI	Covered stent
Nishiyama et al. <sup>15</sup>	Japan	68 M	VActive	LAD	75% occlusion	Coronary spasm and Ant STEMI	Percutaneous reposition
Nakagawa <sup>18</sup>	Japan	72 F	A Active	RCA	Laceration	Pericardial tamponade	Surgery
Khoueiry et al. <sup>17</sup>	USA	67 M	A Passive	RCA	AV fistula	RA pseudoaneurysm and AV fistula	Surgery

F, female; M, male; A, atrial lead; V, ventricular lead; STEMI, ST elevation myocardial infarction; LAD, left anterior descending coronary artery; RCA, right coronary artery; AV, arteriovenous.



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**Figure 3** (Left) CT coronal view. (Right) Computed tomography axial view. The RA pacing lead tip is on the superior part of the medial wall of the RA appendage in close proximity to the aorta (Ao). LA, left atrium.<sup>13</sup>

circumflex artery, and two attached to the wall of the aorta. These were accompanied by leads positioned across the aortic valve onto the endocardium of the left ventricle (LV).

Meier<sup>28</sup> and Heinroth *et al.*<sup>29</sup> have demonstrated that it is possible to acutely pace the myocardium from leads placed into coronary arteries with low thresholds. In the above cases, pacing from the circumflex artery,<sup>27</sup> right coronary artery,<sup>19,20</sup> and even in the aorta near the ostium of the right coronary artery<sup>20</sup> was associated with satisfactory RA pacing capture and sensing thresholds.

Two cases were complicated by ST elevation myocardial infarction: one at the time of implantation into the right coronary,<sup>27</sup> and the other 6 months post-implantation into the circumflex artery.<sup>20</sup> The delayed presentation likely represents gradual migration of the lead distally into the vessel. Both cases were treated with percutaneous lead extraction. In another case 4 weeks post-pacemaker lead implantation into the right coronary artery, the patient was treated with lead removal and bypass grafting of the residual occluded right coronary artery.<sup>19</sup> The authors did not explain why surgery was chosen over percutaneous coronary intervention.

There are two reported cases<sup>20,26</sup> of intended RA active-fixation leads being implanted into the endocardium of the aorta near the ostium of coronary arteries. In the case reported by Issa et  $al_{,,}^{26}$  5-year post-device implantation, an intended RA lead was found perforated through the wall of the aorta just below the ostium of the left main coronary artery and attached to the epicardium of the left atrium. The patient was treated conservatively due to the patient's poor clinical status and advanced age. Kosmidou et  $al_{,}^{20}$ 

presented a case where 12-month post-device implantation, the intended RA lead was found attached to the wall of the aorta, just below the ostium of the right coronary artery. The lead was removed percutaneously with no adverse clinical events. Despite leads being implanted into the arterial system in the above cases, there were no reported thromboembolic complications.

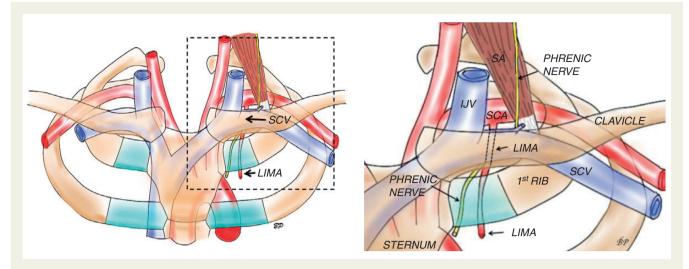
# Inadvertent injury to the internal mammary artery

The right and left internal mammary arteries (LIMA) arise from the medial end of the subclavian artery, traverse behind the subclavian vein, and then run along the sides of the sternum.<sup>30</sup> The proximal internal mammary artery is vulnerable to inadvertent damage during attempted subclavian venous puncture as it passes behind the subclavian vein in the space between the clavicle and first rib (*Figure 4*).

In patients without bypass grafts, damage to the internal mammary arteries secondary to pacemaker implantation can result in adverse events. Case reports have detailed pseudoaneurysms that either: (i) bled acutely with haemothorax and haemomediastinum,<sup>31</sup> (ii) caused compression of the adjacent phrenic nerve (*Figure 4*) with diaphragmatic paralysis,<sup>32,33</sup> or (iii) heart failure due to the development of an arteriovenous fistula between the LIMA and the innominate vein.<sup>34</sup> In one case a lead perforated the RV and lacerated the overlying LIMA, resulting a left haemothorax and hypotension.<sup>35</sup> A thoracotomy was performed with surgical ligation of the LIMA and extraction of the lead.

There are two reports of damage to LIMA bypass grafts during pacemaker implantation leading to acute myocardial infarction.<sup>36,37</sup> In one case,<sup>36</sup> injury to the LIMA was treated by coronary angioplasty and stenting. In the other,<sup>37</sup> the resultant large anterior myocardial infarct was complicated by a large ischaemic ventricular septal defect, cardiogenic shock, and eventually death of the patient. These complications could be avoided by cephalic or axillary venous access or attempting subclavian venous access on the contralateral side that has not been used for grafting.

Left internal mammary artery injury secondary to laser sheathassisted pacemaker lead extraction has been complicated by pseudoaneurysms and arteriovenous fistulas.<sup>38-41</sup> Chronic leads in contact with endothelium and endomyocardium provoke localized inflammatory changes that evolve to fibrous tissue that can extend and adhere to nearby vessels.<sup>42</sup> During the extraction of these



**Figure 4** (Left) Diagram showing the relationship of the LIMA to the subclavian vein (SCV). (Right—the insert in Left is expanded) The LIMA arises near the medial end of the subclavian artery (SCA). It passes posterior (dashed line) to the SCV and first rib. It then runs inferiorly (close to the phrenic nerve) lateral to the sternal border. The scalenus anterior (SA) muscle and phrenic nerve lie between the SCA and SCV.

leads, the cutting energy of laser sheaths increases the risk of pseudoaneurysm and arteriovenous fistula formation.<sup>38,43</sup> Arteriovenous fistulas are usually recognized at the time of procedure by chest or back pain and bright red blood flowing around the sheath,<sup>43</sup> but can also present late with symptoms of congestive heart failure.<sup>44</sup> In both implantation- and extraction-associated cases, pseudoaneurysms and arteriovenous fistulas were managed with percutaneous coil embolization<sup>31–34</sup> or covered stents.<sup>39,41</sup>

### Takotsubo cardiomyopathy

Takotsubo cardiomyopathy is a reversible cardiomyopathy with ECG changes, modest troponin elevation, and clinical symptoms that commonly occur following a physical or psychological stressor.<sup>45,46</sup> There have been 15 case reports of takotsubo cardiomyopathy following pacemaker implantation.<sup>47–58</sup> Similar to other published case series of takotsubo cardiomyopathy, the majority were women (79%), and their age ranged between 54 and 89 years old. Patients presented with chest pain and dyspnea and were in acute pulmonary oedema. There was either a modest or no troponin elevation and minor ST elevation in the precordial ECG leads. The time of presentation varied from a few minutes to 3 days post-device implantation.<sup>47</sup> Golzio et al.<sup>59</sup> reported a case where takotsubo cardiomyopathy occurred as a result of pacemaker syndrome secondary to a malfunctioning atrial lead. The majority (10 of 13) of patients had complete return to normal LV systolic function by a few days<sup>55</sup> to 2 months later.<sup>50</sup> Two patients did not recover normal systolic function,<sup>47</sup> and one patient died from intractable ventricular tachycardia and cardiogenic shock.<sup>57</sup> In all cases, coronary artery disease or damage to coronary arteries was excluded by coronary angiography.

### **Cardiac troponin elevation**

Cardiac troponin T and I are specific and sensitive biomarkers of cardiac necrosis that are important in the diagnosis of myocardial

infarction.<sup>60</sup> However, in the setting of recent CIED implantation, raised troponin levels can be secondary to myocardial trauma and inflammation from lead attachment to myocardium.<sup>61</sup> Other causes include: tachyarrhythmias, acute decompensated and chronic heart failure, sepsis, pulmonary embolism, and takotsubo cardiomyopathy.<sup>4</sup>

The majority of troponin is bound to actin and myosin and smaller amounts lie free in the cytoplasm. Myocardial damage causes an initial small short-lived peak followed by a slowly rising larger sustained elevation. The first peak is thought to represent troponin release from the cytosolic pool of troponin and the later larger peak is thought to be due to necrosis of myocardial tissue.<sup>62</sup>

Nikolaou *et al.*<sup>63</sup> studied the patterns of troponin release following CIED lead implantation. They demonstrated an early small short-lived peak of troponin I release<sup>63</sup> that contrasted with the larger sustained elevations seen with myocardial infarction.<sup>64</sup> In a subsequent larger trial,<sup>65</sup> 59% of 283 patients had elevated troponin levels (>0.1 ng/mL) at 6 h post-pacemaker implantation. Of these, five had 'markedly elevated' levels (>1.5 ng/mL). These five patients did not display ischaemic ECG changes and had normal coronary angiograms. The natural history of such patients is unknown.

Dworschak *et al.*<sup>66</sup> studied troponin T release in the setting of ICD lead implantation and defibrillation threshold testing. The level of troponin T correlated with the duration of the procedure, but had no relationship with the number of applied shocks or cumulative applied defibrillation energy from ICD threshold testing.

Non-coronary causes of elevated troponin levels are markers of poor prognosis. In pulmonary embolism, elevated levels correlated with clinical severity and 30-day mortality.<sup>67</sup> While symptomatic pulmonary embolism following device implantation is an uncommon complication (estimated to occur in 0-5% of patients<sup>68</sup>), the incidence of asymptomatic pulmonary embolism is thought to be much higher. Ventilation perfusion lung scans performed prior to and 14 days post-pacemaker implantation have found a 15% incidence of asymptomatic pulmonary embolism.<sup>69</sup>

### Conclusion

In line with the increasing number of CIED implantations worldwide, there have been sporadic reports of inadvertent damage to coronary arteries and related structures. Damage to coronary arteries can have a varied clinical presentation: chest pain, respiratory distress, symptoms of heart failure, and cardiogenic shock. Clinical presentation can occur at the time of implantation, or even years later. While raised troponin levels may indicate damage to coronary arteries, it may also represent alternative diagnoses such as takotsubo cardiomyopathy, pulmonary embolism, or simply direct mechanical trauma from lead implantation.

#### Conflict of interest: none declared.

#### References

- Poole JE, Gleva MJ, Mela T, Chung MK, Uslan DZ, Borge R et al. Complication rates associated with pacemaker or implantable cardioverter-defibrillator generator replacements and upgrade procedures: results from the REPLACE registry. *Circulation* 2010;**122**:1553–61.
- Kiviniemi MS, Pirnes MA, Eranen HJ, Kettunen RV, Hartikainen JE. Complications related to permanent pacemaker therapy. *Pacing Clin Electrophysiol* 1999;22:711–20.
- Ellenbogen KA, Hellkamp AS, Wilkoff BL, Camunas JL, Love JC, Hadjis TA et al. Complications arising after implantation of DDD pacemakers: the MOST experience. *Am J Cardiol* 2003;**92**:740–1.
- Agewall S, Giannitsis E, Jernberg T, Katus H. Troponin elevation in coronary vs. noncoronary disease. Eur Heart J 2011;32:404–11.
- Fahy GJ, Kleman JM, Wilkoff BL, Morant VA, Pinski SL. Low incidence of lead related complications associated with nonthoracotomy implantable cardioverter defibrillator systems. *Pacing Clin Electrophysiol* 1995;18:172–8.
- Mahapatra S, Bybee KA, Bunch TJ, Espinosa RE, Sinak LJ, McGoon MD et al. Incidence and predictors of cardiac perforation after permanent pacemaker placement. *Heart Rhythm* 2005;2:907–11.
- Ellenbogen KA, Wood MA, Shepard RK. Delayed complications following pacemaker implantation. *Pacing Clin Electrophysiol* 2002;25:1155–8.
- Akyol A, Aydin A, Erdinler I, Oguz E. Late perforation of the heart, pericardium, and diaphragm by an active-fixation ventricular lead. *Pacing Clin Electrophysiol* 2005;28: 350–1.
- Khan MN, Joseph G, Khaykin Y, Ziada KM, Wilkoff BL. Delayed lead perforation: a disturbing trend. *Pacing Clin Electrophysiol* 2005;28:251–3.
- Fuenmayor A AJ, Rodríguez S YA. Successful management of a late right ventricular and diaphragmatic perforation by a defibrillator lead. *Europace* 2014;16:886.
- Nakanishi H, Kashiwase K, Nishio M, Wada M, Hirata A, Ueda Y. Recurrent pericardial effusion caused by pacemaker lead perforation and warfarin therapy at seven years after implantation. *Europace* 2012;14:297.
- Tavernier R, Duytschaever M, Dossche K, Verleyen D, Van Den Brande F, De Greef Y et al. Subacute implantable cardioverter defibrillator lead perforation: a potentially life-threatening event. *Europace* 2009;**11**:966–7.
- Pang BJ, Joshi SB, Lui EH, Tacey MA, Alison J, Seneviratne SK et al. Proximity of pacemaker and implantable cardioverter-defibrillator leads to coronary arteries as assessed by cardiac computed tomography. *Pacing Clin Electrophysiol* 2014;**37**: 717–23.
- Pang BJ, Joshi SB, Lui EH, Tacey MA, Ling LH, Alison J et al. Validation of conventional fluoroscopic and ECG criteria for right ventricular pacemaker lead position using cardiac computed tomography. *Pacing Clin Electrophysiol* 2014;37:495–504.
- Nishiyama N, Takatsuki S, Kimura T, Aizawa Y, Fukumoto K, Hagiwara Y et al. Implantation of the right ventricular lead of an implantable cardioverter-defibrillator complicated by apical myocardial infarction. *Circulation* 2012;**126**:1314–5.
- Parwani AS, Rolf S, Haverkamp W. Coronary artery occlusion due to lead insertion into the right ventricular outflow tract. *Eur Heart J* 2009;**30**:425.
- Khoueiry G, Malpeso JM, Malpeso JV, Abi Rafeh N, Budoff MJ. A right coronary artery fistula communicating with a large right atrial pseudoaneurysm. *Eur Heart J Cardiovasc Imaging* 2012;**13**:278.
- Nakagawa E. Perforation of right coronary artery with an active-fixation atrial pacing lead resulting in life-threatening tamponade. Presented abstract at the Heart Rhythm Scientific Meeting 2013. Lecture ID 7268. Provocative Device Cases, 2013. http:// ondemand.hrsonline.org/common/presentation-detail.aspx/8/23/1006/6092.
- Overbeck M, Kolb C, Schmitt C, Schomig A, Lange R. Accidental transarterial implantation of dual chamber pacemaker leads in the left ventricle and the right coronary artery. *Pacing Clin Electrophysiol* 2005;28:469–71.

- Kosmidou I, Karmpaliotis D, Kandzari DE, Dan D. Inadvertent transarterial lead placement in the left ventricle and aortic cusp: percutaneous lead removal with carotid embolic protection and stent graft placement. *Indian Pacing Electrophysiol J* 2012;**12**:269–73.
- De Ponti R, Cappato R, Curnis A, Della Bella P, Padeletti L, Raviele A et al. Transseptal catheterization in the electrophysiology laboratory: data from a multicenter survey spanning 12 years. J Am Coll Cardiol 2006;47:1037–42.
- Kaljusto ML, Tonnessen T. Aortic perforation with cardiac tamponade two weeks after pacemaker implantation. J Thorac Cardiovasc Surg 2007;134:502–3.
- Kashani A, Mehdirad A, Fredman C, Barold SS. Aortic perforation by active-fixation atrial pacing lead. Pacing Clin Electrophysiol 2004;27:417–8.
- Sticco CC, Barrett LO. Delayed cardiac tamponade by iatrogenic aortic perforation with pacemaker implantation. *J Thorac Cardiovasc Surg* 2006;**131**:480–1.
- Di Marco A, Nunez E, Osorio K, Dallaglio P, Anguera I, Toscano J et al. Aortic perforation by active-fixation atrial pacing lead: an unusual but serious complication. *Tex Heart Inst J* 2014;**41**:327–8.
- Issa ZF, Rumman SS, Mullin JC. Inadvertent transarterial insertion of atrial and ventricular defibrillator leads. *J Interv Card Electrophysiol* 2009;24:63–6.
- Issa ZF, Gill JB. Transarterial pacemaker lead implantation results in acute myocardial infarction. *Europace* 2010;12:1654–5.
- 28. Meier B. Coronary or left ventricular pacing, the easy and obvious way out of asystole during cardiac catheterization. *J Invasive Cardiol* 2011;**23**:2.
- Heinroth KM, Stabenow I, Moldenhauer I, Unverzagt S, Buerke M, Werdan K et al. Temporary trans-coronary pacing by coated guidewires: a safe and reliable method during percutaneous coronary intervention. *Clin Res Cardiol* 2006;**95**: 206–11.
- Sinnatamby CS, Last RJ. Last's Anatomy: Regional and Applied. Edinburgh, NY: Churchill Livingstone/Elsevier, 2011.
- Chemelli AP, Chemelli-Steingruber IE, Bonaros N, Luckner G, Millonig G, Seppi K et al. Coil embolization of internal mammary artery injured during central vein catheter and cardiac pacemaker lead insertion. Eur J Radiol 2009;71:269–74.
- Ploux S, Bordachar P, Deplagne A, Mokrani B, Reuter S, Laborderie J et al. Electrocardiogram-based algorithm to predict the left ventricular lead position in recipients of cardiac resynchronization systems. *Pacing Clin Electrophysiol* 2009; 32(Suppl 1):S2-7.
- Pozo E, Gonzalez-Ferrer JJ, Perez Villacastin J, Macaya C. Diaphragm paralysis due to pseudoaneurysm of internal mammary artery after pacemaker implantation. *Europace* 2011;13:592–3.
- Anguera I, Real I, Morales M, Vazquez F, Montana X, Pare C. Left internal mammary artery to innominate vein fistula complicating pacemaker insertion. Treatment with endovascular transarterial coil embolization. J Cardiovasc Surg (Torino) 1999;40: 523–5.
- Kypson AP, Frazier DW, Moran JF. Internal thoracic artery injury after transvenous pacemaker implantation. J Thorac Cardiovasc Surg 2005;129:675–6.
- Chou TM, Chair KM, Jim MH, Boncutter A, Milechman G. Acute occlusion of left internal mammary artery graft during dual-chamber pacemaker implantation. *Catheter Cardiovasc Interv* 2000;51:65–8.
- Garcia-Bolao I, Macias A, Moreno J, Martin A. Fatal left internal mammary artery graft to subclavian vein fistula complicating dual-chamber pacemaker implantation. *Europace* 2008;10:890–1.
- Cronin EM, Brunner MP, Tan CD, Rene Rodriguez E, Rickard J, Martin DO et al. Incidence, management, and outcomes of the arteriovenous fistula complicating transvenous lead extraction. *Heart Rhythm* 2014;11:404–11.
- Azpurua FE, Dougherty KG, Massumi A, Strickman NE. Fistula from right internal mammary artery to superior vena cava after use of a laser sheath to extract a pacemaker lead. *Tex Heart Inst J* 2012;39:727–30.
- Kim HY, Heywood JT, Jacobson AK, Smith DC. Arteriovenous fistula: a complication of cardiac pacemaker lead placement and its management with percutaneous embolization. *Pacing Clin Electrophysiol* 1993;16:2310–2.
- Yamada T, Robertson PG, Kay GN. Innominate vein to left internal mammary artery bypass graft fistula during laser lead extraction: salvage with covered coronary artery stent. *Europace* 2013;15:717.
- Smith HJ, Fearnot NE, Byrd CL, Wilkoff BL, Love CJ, Sellers TD. Five-years experience with intravascular lead extraction. U.S. Lead Extraction Database. *Pacing Clin Electrophysiol* 1994;17:2016–20.
- Kumins NH, Tober JC, Love CJ, Culbertson TA, Gerhardt MA, Irwin RJ et al. Arteriovenous fistulae complicating cardiac pacemaker lead extraction: recognition, evaluation, and management. J Vasc Surg 2000;32:1225–8.
- Milla F, Mack CA, Girardi LN. Arteriovenous fistula after laser-assisted pacemaker lead extraction. Ann Thorac Surg 2006;81:2304–6.
- Akashi YJ, Goldstein DS, Barbaro G, Ueyama T. Takotsubo cardiomyopathy: a new form of acute, reversible heart failure. *Circulation* 2008;**118**:2754–62.
- Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. *Am Heart J* 2008;**155**: 408–17.

- Kurisu S, Inoue I, Kawagoe T, Ishihara M, Shimatani Y, Hata T *et al.* Persistent left ventricular dysfunction in takotsubo cardiomyopathy after pacemaker implantation. *Circ J* 2006;**70**:641–4.
- Chun SG, Kwok V, Pang DK, Lau TK. Transient left ventricular apical ballooning syndrome (takotsubo cardiomyopathy) as a complication of permanent pacemaker implantation. *Int J Cardiol* 2007;**117**:e27–30.
- Kimura K, Tanabe-Hayashi Y, Noma S, Fukuda K. Images in cardiovascular medicine. Rapid formation of left ventricular giant thrombus with takotsubo cardiomyopathy. *Circulation* 2007;**115**:e620–1.
- Kohnen RF, Baur LH. A Dutch case of a takotsubo cardiomyopathy after pacemaker implantation. Neth Heart J 2009;17:487–90.
- Abu Sham'a RA, Asher E, Luria D, Berger M, Glikson M. Apical ballooning syndrome: a complication of dual chamber pacemaker implantation. *Indian Pacing ElectrophysiolJ* 2009;9:229–32.
- Hsu C, Chen C-H, Liang H. Takotsubo cardiomyopathy after permanent pacemaker implantation. Acta Cardiol Sin 2010;26:264–7.
- Mazurek J, Gundewar S, Ji S, Grushko M, Krumerman A. Left ventricular apical ballooning syndrome after pacemaker implantation in a male. J Cardiol Cases 2011;3: e154–e8.
- 54. Brunetti ND, leva R, Correale M, De Gennaro L, Pellegrino PL, Dioguardi E et al. Combined exogenous and endogenous catecholamine release associated with Tako-Tsubo like syndrome in a patient with atrio-ventricular block undergoing pacemaker implantation. Acute Card Care 2011;**13**:112–4.
- Gardini A, Fracassi F, Boldi E, Albiero R. Apical ballooning syndrome (takotsubo cardiomyopathy) after permanent dual-chamber pacemaker implantation. *Case Rep Cardiol* 2012;2012:3.
- Zghal F, Trabelsi R, Mbarki S, Mourali M, Mechmeche R. Rapid reversibility of takotsubo cardiomyopathy. World J Cardiovasc Dis 2012;2:26–8.
- Kinbara T, Hayano T, Otani N, Furutani Y, Murakami T, Yano M. An autopsy case of tako-tsubo cardiomyopathy presenting ventricular tachycardia after pacemaker implantation. J Cardiol Cases 2013;8:134–7.
- Postema PG, Wiersma JJ, van der Bilt IA, Dekkers P, van Bergen PF. Takotsubo cardiomyopathy shortly following pacemaker implantation-case report and review of the literature. *Neth Heart J* 2014;22:456–9.
- Golzio PG, Anselmino M, Presutti D, Cerrato E, Bollati M, Gaita F. Takotsubo cardiomyopathy as a complication of pacemaker implantation. J Cardiovasc Med 2011;12: 754–60.

- Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, Joint ESCAAHAWHFTFfUDoMlet al. Third universal definition of myocardial infarction. J Am Coll Cardiol 2012;60:1581–98.
- Mond HG, Helland JR, Stokes K, Bornzin GA, McVenes R. The electrode-tissue interface: the revolutionary role of steroid-elution. *Pacing Clin Electrophysiol* 2014; 37:1232–49.
- White HD. Pathobiology of troponin elevations: do elevations occur with myocardial ischemia as well as necrosis? J Am Coll Cardiol 2011;57:2406–8.
- Nikoaou NI, Spanodimos SG, Tsaglis EP, Antonatos DG, Patsilinakos SP, Fournarakis GM et al. Biochemical evidence of cardiac damage following transvenous implantation of a permanent antibradycardia pacemaker lead. Pacing Clin Electrophysiol 2005;28:1174–81.
- 64. Mahajan VS, Jarolim P. How to interpret elevated cardiac troponin levels. *Circulation* 2011;**124**:2350–4.
- Nikolaou NI, Christou AH, Spanodimos SG, Antonatos DG, Korkonikitas PI, Patsilinakos SP. Marked troponin elevation after implantation of a permanent antibradycardia pacemaker. *Hellenic J Cardiol* 2011;52:489–92.
- Dworschak M, Franz M, Khazen C, Czerny M, Haisjackl M, Hiesmayr M. Mechanical trauma as the major cause of troponin T release after transvenous implantation of cardioverter/defibrillators. *Cardiology* 2001;95:212–4.
- Giannitsis E, Muller-Bardorff M, Kurowski V, Weidtmann B, Wiegand U, Kampmann M et al. Independent prognostic value of cardiac troponin T in patients with confirmed pulmonary embolism. *Circulation* 2000;**102**:211–7.
- Supple GE, Ren JF, Zado ES, Marchlinski FE. Mobile thrombus on device leads in patients undergoing ablation: identification, incidence, location, and association with increased pulmonary artery systolic pressure. *Circulation* 2011;**124**:772–8.
- Seeger W, Scherer K. Asymptomatic pulmonary embolism following pacemaker implantation. Pacing Clin Electrophysiol 1986;9:196–9.
- Lagi A, Meucci E, Cencetti S. Outcome of patients with elevated cardiac troponin I level after mild trauma. Am J Emerg Med 2008;26:248 e3–5.
- Novak M, Dvorak P, Kamaryt P, Slana B, Lipoldova J. Autopsy and clinical context in deceased patients with implanted pacemakers and defibrillators: intracardiac findings near their leads and electrodes. *Europace* 2009;11:1510–6.
- Barold SS, Herweg B. Electrocardiographic diagnosis of myocardial infarction and ischemia during right ventricular pacing. J Electrocardiol 2007;40:164–7.
- Sgarbossa EB, Pinski SL, Gates KB, Wagner GS. Early electrocardiographic diagnosis of acute myocardial infarction in the presence of ventricular paced rhythm. GUSTO-I investigators. Am J Cardiol 1996;**77**:423–4.