Received:
10 August 2014

Revised:
11 November 2014

Accepted:
19 November 2014

doi: 10.1259/bjr.20140540

Cite this article as:

Dipoce J, Bernheim A, Spindola-Franco H. Radiology of cardiac devices and their complications. *Br J Radiol* 2015;88:20140540.

REVIEW ARTICLE

Radiology of cardiac devices and their complications

¹J DIPOCE, MD, ²A BERNHEIM, MD and ³H SPINDOLA-FRANCO, MD

¹Department of Radiology, Hadassah Medical Center, Jerusalem, Israel

²Department of Thoracic Imaging, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

³Department of Radiology, Montefiore Medical Center, Albert Einstein College of Medicine, New York, NY, USA

Address correspondence to: Dr Adam Bernheim

E-mail: abernheim@mgh.harvard.edu

ABSTRACT

This article familiarizes the reader with several different cardiac devices including pacemakers and implantable cardioverter defibrillators, intra-aortic balloon pumps, ventricular assist devices, valve replacements and repairs, shunt-occluding devices and passive constraint devices. Many cardiac devices are routinely encountered in clinical practice. Other devices are in the early stages of development, but circumstances suggest that they too will become commonly found. The radiologist must be familiar with these devices and their complications.

Cardiac devices such as pacemakers have now been improving patient morbidity and mortality for decades. Radiology has played an integral role in device pre-procedural planning, placement and continued evaluation. This article aims to review cardiac devices that have been in use for a long time, including pacemakers, implantable cardioverter defibrillators (ICDs), the intra-aortic balloon pump (IABP) and valve repairs/replacements. Decades of use have provided ample data regarding their normal radiographic appearances and complications. In addition, contemporary advances have impacted our understanding of newer devices and their complications. Because of the increasing use of these devices, it is of utmost importance to be aware of their complications. The ventricular assist devices (VADs) have benefitted greatly from recent technological advances, and they continue to evolve. Furthermore, new devices such as percutaneous valve replacements and passive constraint devices continue to emerge. Radiologists interpreting thoracic imaging studies should become familiar with their appearances and potential complications.

PACEMAKERS AND IMPLANTABLE CARDIOVERTER DEFIBRILLATORS

Pacemakers and ICDs are ubiquitous with millions of devices implanted worldwide. Device reliability has improved markedly after decades of use. Nevertheless, these devices are not perfect, and many types of complications have been described. Notably, device malfunction seems to be independent of the manufacturer.¹ Many of these malfunctions, such as battery depletion, are radiologically occult and come to attention without the need for imaging.

However, imaging clearly plays a role in the evaluation of pacemaker/ICD complications. Radiologically identifiable complications include dislodgment, lead perforation, local structural damage, lead failure, thrombosis and infection.

Dislodgment is one of the most common causes of device failure (“non-capture”) in the early time period after implantation.² In the pacemaker selection in the elderly trial, the dislodgment complication was estimated to be 2.2%.³ Several factors contribute to a lead’s susceptibility to dislodge, and multiple solutions have been implemented to improve stability. Common considerations include the location of the lead, use of “passive” vs “active” fixation and percutaneous formation of atrial “J-shaped” leads.

The lead may be imbedded in the myocardium in what is referred to as “active” or “passive” fixation. Passive fixation involves inserting a lead bearing tines into the myocardial tissue that becomes trapped in the myocardium. Active fixation involves inserting a screw-laden lead into the myocardium. The atrial passive fixation leads require less fluoroscopy time to insert and demonstrate a better pacing threshold at 1 year.⁴ Additionally, a lower incidence of cardiac tamponade has been reported with passive fixation, which has been hypothesized to be secondary to irritation from perforated or even minimally protruded electrodes.^{5,6} Atrial active fixation leads have been shown to have a higher level of pericardial complications than do ventricular passive fixation leads, which has been speculated to be the result of the thinner atrial wall.⁷ Despite these differences, the choice of active vs passive fixation still remains largely dependent upon personal preference.⁸

Figure 1. A 7-year-old female with a history of complete atrioventricular heart block who had a pacemaker with a single chamber right ventricular lead placed. Note the lead redundancy of the correctly placed lead (a), which allows for growth in the paediatric population. 10 days later (b), there is retraction of the lead and coiling around the generator. (c) Further retraction and coiling are seen 30 days later. These findings are consistent with Twiddler's syndrome.

Atrial leads may displace slightly more commonly than do ventricular leads. When considering atrial implantation, straight leads dislodge more often than do the pre-formed “J-shaped” leads. However, the “J-shaped” leads tend to fail at a higher rate than do the straight leads, such that there is approximate equal efficacy of each lead type.⁹ In patients with unusual anatomy and

in paediatric patients, the complication rate may be slightly higher. Practitioner experience is one of the major factors in predicting dislodgment.¹⁰

The dislodgment may be radiographically apparent and is referred to as macrodislodgment. Microdislodgment describes

Figure 2. A 65-year-old male with shortness of breath. (a, b) The tip of the defibrillator lead extends beyond the right ventricular wall and towards the left ventricular free wall (arrow). This is suggestive of lead perforation. The coronal CT reformation (c) confirms the lead tip (arrow) is in the region of left ventricular pericardium/left lateral rib. Lead perforation did not cause pericardial effusion.

radiographically occult dislodgement. Therefore, detecting macrodislodgement may require comparison with a baseline radiograph, when the lead was placed in the correct position. In this way, the radiologist may suggest dislodgement independently from clinicians. When the pacemaker pulse generator turns in its pocket, the leads may retract or even protrude depending upon which way the box is turned, resulting in a phenomenon referred to as “twiddler’s syndrome” (Figure 1).^{11,12}

Chamber perforation may result when the lead penetrates the right atrial wall, right ventricular free wall or the coronary sinus. Perforation is usually recognized during placement but may present later after implantation as either an asymptomatic subacute perforation or as a late perforation.^{13–16} Pacemaker insertion generally results in a lower rate of perforation than ICD insertion with many studies citing <1% perforation rate for pacemakers and up to 5% for ICDs.^{17,18} This difference has been attributed to the ICD lead’s larger diameter than that of a pacemaker lead. Atrial perforation is also generally more common than ventricular perforation, which has been attributed to the thinner right atrial wall. Chamber perforation may be apparent clinically and may manifest itself as an arrhythmia despite the presence of the pacemaker, diaphragmatic/intercostal muscle contraction or local structural damage. While a rare occurrence, death has been attributed to a delayed perforation emphasizing the importance of identifying a perforation after

implantation.¹⁴ Interestingly, even if the lead has perforated the myocardium, it may continue to function if it is close to the epicardium.¹⁴ Therefore, the management of an asymptomatic perforation discovered incidentally is unclear with some authors advocating their removal under transesophageal echocardiography and the supervision of cardiac surgery.¹⁸

Perforation may only first be apparent on imaging, reinforcing the importance of careful lead inspection on routine examinations, as the radiologist may be the first to identify perforation. Echocardiography may be suggestive of perforation, but definitive determination usually requires that the lead be completely perforated through the myocardium. Chest radiographs may also be suggestive of perforation, especially if the lead is clearly travelling beyond the confines of the cardiac silhouette. However, CT has demonstrated increased value over radiography for the evaluation of perforation notwithstanding the inherent streak artefact (Figure 2).^{17,19}

Occasionally, a lead is placed in the left ventricular cavity, which may occur when the lead passes through a patent atrial or ventricular septal defect,²⁰ a perforated interventricular septum²¹ or inadvertent cannulation of the subclavian artery.²² Left ventricular lead placement is particularly dangerous because it may serve as a nidus for thrombus formation with systemic embolization.

Figure 3. A 26-year-old female with a single-chamber, unipolar, lead tip in the left ventricle in a patient post Senning procedure for dextrotransposition of the great arteries (D-TGA) (a, b). The pulse generator overlies the left hemithorax. Its lead enters the left subclavian vein and courses through the left innominate vein and superior vena cava (SVC), then enters the left ventricle through the neoright atrium. This accounts for the lead tip (arrow) being in a posterior position on the lateral image. Diagram depicting the Senning procedure (c). The SVC and inferior vena cava drain into the neoright atrium, which in turn drains into the left ventricle. Flow from the left ventricle goes through the pulmonary arteries to the pulmonary veins, which drain into the neoleft atrium. This then connects with the right ventricle, which feeds into the systemic circulation. The parallel circulation present in D-TGA is thus converted into a circulation in series. Reproduced from Spindola-Franco and Fish²³ with kind permission from Springer Science + Business Media.

Figure 4. A 78-year-old male with a dual chamber pacemaker pulse generator. The leads enter the right atrium and the right ventricle via a persistent left superior vena cava (arrow) and coronary sinus.

Uncommon anatomy occasionally necessitates unusual placement of pacemaker wires. These situations include when the left ventricle is entered in patients with dextrotransposition of the great arteries post Senning/Mustard repair (in which case the lead passes through a morphological left ventricle that functions as the subpulmonary ventricle, with therefore no risk of systemic embolization) or pacing the right atrium/ventricle through a persistent left-sided superior vena cava and the coronary sinus (Figures 3 and 4).

Similar to any percutaneous angiographic procedure, a number of local structures may be damaged during or after pacemaker placement. Accordingly, vascular injury and haemorrhage are expected possible complications (Figure 5). The haemorrhage may manifest as haemopericardium, haemothorax or mediastinal haematoma.

Additionally, pneumothoraces may result from a subclavian approach at a rate of around 2%, with one study finding that they were more common in older and leaner patients.³ The pneumothorax may be immediately apparent or have a delayed appearance of up to 48 h. A radiograph after device placement to evaluate for complications, including pneumothoraces, is recommended.²⁴ Other less common complications include thoracic duct injury, brachial plexus injury and arteriovenous fistula formation.

Device failure may result from fracture in the lead or breakdown of the insulation surrounding the lead, which are both considered late complications after implantation. External compression may result in either lead fracture or insulation breakdown. Fracture sites tend to occur at points of stress, such as between the clavicle and the first rib or the soft-tissue structures in this region.^{25–27} For this reason, thoracic outlet syndrome has been anecdotally associated with lead fracture.²⁸ The tricuspid valve has also been described as a fracture site.²⁹ Additionally, anchoring sutures have been a cause of fracture.³⁰ Unfortunately, the lead insulating material is not radio-opaque, and only focal, subtle irregularities in the lead contour can suggest problems with the insulation integrity. A breakdown in lead insulation can result in current leak.³¹ Furthermore, apparent discontinuities in the lead, as seen on radiography, do not necessarily mean fracture but rather represent “pseudofracture.” Pseudofractures have been described as a result of external compression by a suture without causing fracture.³² Furthermore, the “pseudofracture” appearance

Figure 5. A 67-year-old male with a medical history of pacemaker placement and coronary artery bypass graft surgery. The right atrial lead eroded through the atrium into the native right coronary artery with the formation of a 7.0 × 5.0-cm pseudoaneurysm arising from the right coronary artery. The pacing lead was resected, and the right atrium was partially excised. CT angiography (a) demonstrating the large pseudoaneurysm (black arrow) and the atrial lead (arrowhead), which eroded into the right coronary artery. A right coronary artery angiogram (b) demonstrating a contrast jet (white arrow) into the pseudoaneurysm with a large collection of contrast surrounding the jet (black arrow). The arrowhead points to the atrial lead that eroded into the native right coronary artery.

Figure 6. A 53-year-old male post-implantable cardioverter-defibrillator placement with a fractured, abandoned lead (a, thick arrow) and a new defibrillator lead with a pseudofracture (thin arrow). Incidentally, noted is an endotracheal tube, feeding tube and a left internal jugular venous line with distal tip coiled in the left innominate vein. A right axillary arterial line is barely perceptible. A 45-year-old male with pulse generator overlying the left upper hemithorax (b). The antenna (arrows) of the pulse generator should not be mistaken for a lead fracture.

has been noted in some leads having a normal construction of outer and inner coils. The bifurcation of the inner and outer coils may simulate a fracture (Figure 6a).^{30,33} Some units are also equipped with a transmitting antenna, which can also mimic a fracture lead (Figure 6b). Familiarity with normal radiographic appearances of various devices prevents unnecessary alarm. Ultimately, a history of device malfunctioning is helpful in avoiding both false-positive and false-negative reports of potential lead problems.

Infection of pacemakers/ICDs may be divided into those that affect the pocket and those that affect the leads.³⁴ Variation in the incidence of pacemaker/ICD infection has been reported, ranging from 0.5% to 5.7%.³⁵ Infection is a dreaded complication that usually requires removal of the hardware, with a mortality rate that ranges from 31% to 66% if the infected device is not removed and 18% or less with complete device removal and antimicrobial therapy.³⁶ Pocket infections may result in erosion of the skin surface by the generator. Pocket infection is commonly a result of infection at the time of implantation, while infection of the leads is usually associated with bacteraemia from a different source. The deeper infection mostly affects the lead's intracardiac portion along the right atrium, tricuspid valve or right ventricular contact. Vegetations may be found at these areas and demonstrated by transthoracic echocardiography in 23% of patients and transoesophageal echocardiography in 94% of patients.³⁷

Radiography may show an increasing air or fluid collection at the generator site.¹¹ CT has been anecdotally useful in isolating the location of the infection to epicardial patches when fluid is present around the patches for greater than 1 month after placement.^{38–40}

Some studies have demonstrated the utility of nuclear medicine in evaluating difficult cases of pacemaker endocarditis. One study showed that ventilation perfusion scintigraphy was positive for embolization in 21% of patients with pacemaker endocarditis. The same study showed a possible use for white blood cell scintigraphy in patients with atypical presentations or normal ventilation/perfusion examinations.⁴¹ Gallium scintigraphy may be useful, but non-infected healing wounds may be

a source of false positives.³⁸ Positron emission tomography has shown a possible benefit in difficult cases of patients with suspected pacemaker infection.⁴²

Notably, the presence of a haematoma at the time of insertion has been shown to be an independent risk factor for the development of infection.³⁵

While implantable loop recorders are used solely for long-term cardiac monitoring and not pacing, radiologists should be familiar with their appearance so as not to confuse them with other electronic equipment. The loop recorders are rectangular in shape and placed in a subcutaneous location to predominantly monitor patients with palpitations or syncope (Figure 7).

Figure 7. A 65-year-old female with recurrent unexplained syncopal episodes who had an implantable loop recorder placed, which is seen overlying the heart.

INTRA-AORTIC BALLOON PUMP

First described in 1962, the IABP has become the most common circulatory assist device.^{43,44} The pump has several circulatory benefits including increased diastolic perfusion, increased diastolic blood pressure and increased mean arterial pressure. The IABP is a polyethylene balloon mounted on a catheter placed in the descending aorta. During diastole, the IABP inflates which increases flow to the coronary arteries, great vessels and renal arteries. Just prior to systole, the IABP deflates creating a vacuum effect that promotes forward flow to the aorta and its branches. Indications for the IABP include cardiogenic shock,^{45,46} unstable angina,^{47,48} acute infarction or recurrent ischaemia,⁴⁹ refractory ventricular tachycardia⁵⁰ and as a bridge to transplantation.⁵¹ In a large study of 17,540 IABP insertions, the major complication rate (severe bleeding, major limb ischaemia, balloon leak or in-hospital mortality related to IABP) was 2.6%.⁵²

Improper positioning of the IABP accounts for the main radiographically identifiable complications.⁵³ Therefore, many authors recommend daily chest radiographs to confirm that the IABP remains in optimal position.⁵⁴ The IABP should be inserted such that the balloon tip is 2.5 cm distal to the left subclavian artery origin. If the IABP is placed too proximally, then it may occlude the brachiocephalic, left carotid or left

subclavian arteries. If the IABP is placed too distally, then it may occlude the celiac, superior mesenteric or renal arteries. These improper placements may result in neurological sequelae, limb ischaemia, renal insufficiency or mesenteric ischaemia depending upon which vessel is occluded. Embolic phenomena are another source of ischaemia in patients with the IABP. Because the exact origin of the great vessels is not readily apparent on chest radiography, anatomical landmarks have been used to estimate the IABP position. Traditionally, the aortic knob has been used as a landmark for positioning.^{53,55} Recent studies have questioned the use of the aortic knob as a landmark, as there may be individual variations of the left subclavian origin and the aortic knob that are exacerbated by age, owing to uncoiling of the aorta. Instead, the carina has been suggested as an alternative landmark (Figures 8 and 9).⁵⁶

Aortic dissection is a complication of the IABP, which occurs in 1–3% of insertion.^{57,58} The balloon pump infrequently may continue to function from within a false lumen emphasizing the importance of recognizing this complication on imaging studies. On plain radiograph, the IABP may appear laterally positioned, which is suggestive of dissection.⁵³ Unfortunately, an IABP in an anterior or posterior position within a false lumen may not be

Figure 8. A 70-year-old male with intra-aortic balloon pump (IABP) in diastole. The balloon (arrowhead) is inflated during diastole and is deflated during systole. The IABP tip (arrow) is radio-opaque and should be positioned approximately at the level of the carina or 2.5 cm distal to the origin of the left subclavian artery. A position that is too high may compromise left subclavian arterial flow, whereas a position that is too low may compromise mesenteric or renal arterial flow.

Figure 9. A 50-year-old male with an intra-aortic balloon pump (IABP) in place. The IABP tip overlies the aortic knob (arrow) and is considered high, as it is at the level of the origin of the left subclavian artery. This position is of concern in this relatively young patient where the aortic arch maintains a normal coiled configuration, thus interfering with left subclavian arterial flow. In older patients, this position may still be acceptable because of uncoiling of the aorta and a subsequent anteriorly displaced position of the origin of the left subclavian artery. Also present is a Swan-Ganz catheter.

Figure 10. A 65-year-old male with cardiomegaly and congestive heart failure post placement of right ventricular and left ventricular Heartware® assist devices (Heartware, Framingham, MA).

discernible on a frontal radiograph. CT is much more sensitive and specific for this complication.⁵⁹

Gas embolism from balloon rupture is a rare complication, but particularly dangerous as helium is often the gas used inside the balloon and is insoluble in blood. The normal appearance of an IABP as seen on CT must not be confused with rupture or other pathology. The characteristic location and regularly spaced, periodic, air-fluid levels within the aorta representing an inflating/deflating IABP are expected findings of a working pump.⁶⁰

Infection is a rare complication of the IABP, which becomes more frequent as the days of its use increase.⁶¹

VENTRICULAR ASSIST DEVICES

Aimed to replace or support the function of a failing heart by improving cardiac output, VADs may be employed in the acute setting in cardiogenic shock from myocardial infarction or cardiac surgery. Alternatively, they may be utilized on a more long-term basis—either as a bridge to cardiac transplantation or as a destination therapy in those not eligible for transplantation—for patients with congestive heart failure. VADs have improved both survival and quality of life in patients with end-stage heart failure, with a 48% reduction in the risk of death in patients with the device as compared with those managed with medical therapy alone.⁶² There are several varieties of VADs available today and can be used to improve either right or left ventricular function or both with the implantation of two devices (Figure 10, Table 1).

Radiology plays a role in the pre-operative, perioperative and post-operative assessment of patients with VADs.⁶³ Echocardiography is utilized prior to the placement of a VAD to identify a patent foramen ovale and to assess valvular function. Placing a left VAD (LVAD) results in diminished left atrial pressures, so an otherwise asymptomatic patent foramen ovale may become clinically significant following installation of the device; this is the result of a left-to-right shunt becoming a right-to-left shunt. Moreover, the presence of aortic valvular disease and mitral stenosis may lead to the selection of a particular type of VAD over another. Also, since right-sided heart failure is a complication of LVAD placement⁶⁴ owing to increased pulmonary arterial pressure following placement, the pre-operative diagnosis of tricuspid regurgitation is important.

Table 1. Types and examples of available ventricular assist devices (VADs)

Short-term (temporary) VADs	Long-term pulsatile (first generation) VADs	Long-term non-pulsatile (second generation) VADs
Abiomed BVS 5000™	Thoratec HeartMate® XVE	Berlin Heart Incor®
Abiomed Impella®	Thoratec® IVAD	Jarvik 2000
Cardiac Assist, Inc.	Thoratec PVAD	MicroMed DeBakey HeartAssist 5 pump
Levitronix CentriMag LVAS	WorldHeart Levacor VAD	MicroMed DeBakey VAD pump unit
Medtronic Bio-Medicus Bio-Pump®	WorldHeart Novacor LVAS	MiTiHeart Corporation LVAD
St Jude Medical Lifestream centrifugal pump		Sunshine Heart C-Pulse
TandemHeart™		Thoratec HeartMate II VAD
Terumo Sarns™ Centrifugal System		Heartware® HVAD LVAD

IVAD, implantable ventricular assist device; LVAD, left ventricular assist device; PVAD, paracorporeal ventricular assist device.

This table summarises several different ventricular assist devices organized according to length of intended use and pulsatility.

The ventricular assist devices are manufactured by the following: Abiomed BVS 5000 (ABIOMED, Inc., Danvers, MA); Abiomed Impella (ABIOMED, Inc.); CardiacAssist, Inc. (CardiacAssist, Inc., Pittsburgh, PA); Levitronix CentriMag LVAS (Levitronix LLC, Waltham, MA); Medtronic Bio-Medicus Bio-Pump (Medtronic, Inc., Minneapolis, MN); St Jude Medical Lifestream centrifugal pump (St Jude Medical, Chelmsford, MA); TandemHeart (CardiacAssist, Inc., Pittsburgh, PA); Terumo Sarns Centrifugal System (Terumo Cardiovascular Systems Corporation, Ann Arbor, MI); Thoratec HeartMate XVE (Thoratec Corporation, Pleasanton, CA); Thoratec IVAD (Thoratec Corporation); Thoratec PVAD (Thoratec Corporation); WorldHeart Levacor VAD (World Heart Corporation, Oakland, CA); WorldHeart Novacor LVAS (World Heart Corporation); Berlin Heart Incor (Berlin Heart, Berlin, Germany); Jarvik 2000 (Jarvik Heart, New York, NY); MicroMed DeBakey HeartAssist 5 pump (MicroMed Cardiovascular Inc., Houston, TX); MicroMed DeBakey VAD pump unit (MicroMed Cardiovascular Inc., Houston, TX); MiTiHeart Corporation LVAD (MiTiHeart Corporation, Gaithersburg, MD); Sunshine Heart C-Pulse (Sunshine Heart, Eden Prairie, MN); Thoratec HeartMate II VAD (Thoratec Corporation); Heartware HVAD LVAD (HeartWare, Framingham, MA).

Figure 11. A 45-year-old female with heart failure and an appropriately positioned HeartMate® II left ventricular assist device (Thoratec Corporation, Pleasanton, CA) sealed inflow conduit (cannula) (a, arrow). Also present is a TandemHeart™ cannula, with its tip in the left atrium. The femoral artery cannula is not shown. A 36-year-old female with a medical history of Coxsackie myocarditis and non-ischaemic cardiomyopathy post placement of HeartMate II left ventricular assist device (b). The orientation of the sealed inflow conduit (arrow) is directed towards the left ventricular wall instead of the ventricular lumen. This is suggestive of device malfunction. Intraoperatively, the sealed inflow conduit and the sealed outflow graft were extensively clotted.

Radiographs are used to confirm proper positioning of the VAD, which is dependent on the particular device. For instance, the HeartMate® LVAD pump (Thoratec Corporation, Pleasanton, CA) is often placed in an intra-abdominal location and can be seen on an abdominal radiograph overlying the left upper quadrant.⁶⁵

Post-operative complications of VAD placement include thromboembolism, aortic dissection and intramural haematoma, bleeding secondary to anticoagulation, pneumothorax, pneumomediastinum, infection, mechanical failure and right-sided heart failure. A change in the expected orientation may be suggestive of thrombosis (Figure 11). The HeartMate device is associated with the lowest rate of thromboembolism owing to its unique blood-contacting surface, so only aspirin is recommended when this device is used.⁶⁶ However, most of the other devices

require long-term warfarin administration, and despite this as many as 16% of patients have experienced a cerebrovascular accident, with a mean time following placement of the LVAD of 222 days.⁶⁷ Iatrogenic aortic dissection and intramural haematoma may occur. Haemothorax, haemopericardium and haemoperitoneum can also occur in addition to bleeding at the operative site. Bleeding rates have been estimated to be as high as 50%.⁶³ Pneumothorax and pneumomediastinum are potential complications.

Sometimes owing to device malfunction the HeartMate II body is replaced without exchanging the outflow graft. Because of differences in construction between new and old designs of the HeartMate II, there may be detachment and outflow graft kinking when the exchange is made. Some have successfully addressed this problem by wiring the bend relief so that it cannot be detached (Figure 12).⁶⁷

Figure 12. A 43-year-old male with ischaemic cardiomyopathy post coronary artery bypass, ICD placement and HeartMate® II (Thoratec Corporation, Pleasanton, CA) left ventricular assist device (LVAD). 3 months after LVAD placement, the patient presented with shortness of breath. Chest radiograph demonstrated congestive heart failure and malposition of the outflow cannula (white arrow) with separation and angulation (a). Echocardiography confirmed malposition with intermittent opening of the aortic valve. Subsequently, the outflow cannula was repositioned and reinforced with sternal wires (b).

Figure 13. A 67-year-old male status post triple valve replacement. The location of the valves can be identified by tracing a line from the anterior sulcus to the hilum. The aortic valve is positioned above the line, whereas the mitral valve (which is posterior and inferior to the aortic valve) is positioned below the line. The tricuspid valve is anterior and inferior to the aortic valve, thus it transects the line.

Infection following VAD placement is one of the most common and serious complications, with nearly a third of patients developing infection in the first 3 months following surgery.^{62,68} Sites of infection include the superficial driveline, the pump pocket, the mediastinum or the internal components of the pump termed endovaditis.⁶⁶ Contrast-enhanced chest CT may demonstrate peri-graft infection along the drivelines related to VAD placement as mixed-density collections with peripheral wall enhancement tracking along subcutaneous drivelines leading to entry and exit sites.⁶³

Mechanical device failure is an inevitable limitation of VADs and has been reported to occur 35% of the time within 2 years.⁶² Pulsatile devices fail sooner than continuous-acting devices. Controlling hypertension is a suggested therapy for reducing the likelihood of mechanical failure. Longitudinal surveillance with

echocardiography and evaluation of haemodynamics through cardiac catheterization are considered the mainstay approaches for discerning mechanical device failure.

Valve repairs and replacement

Radiologists should not only be familiar with the advantages and disadvantages of various surgical options in treating cardiac valvular disease but they also should be familiar with their post-operative appearance and be able to recognize potential complications (Figure 13). Diseased cardiac valves may be managed with either surgical reconstruction (valvuloplasty) or surgical replacement.⁵⁵ During valvuloplasty, radio-opaque annuloplasty rings (which usually have a gap to provide flexibility) may be utilized and can be recognized on radiological studies.

With regard to valve replacements, they are either mechanical or bioprosthetic, with each having advantages and disadvantages. Mechanical valves last longer than the bioprosthetic valves but require long-term anticoagulation, whereas bioprosthetic valves do not require anticoagulation.^{69,70} The radiologist should additionally be aware that all current valve devices are MRI compatible, as only the original Starr-Edwards metal ball and cage devices were incompatible, and these are no longer left *in vivo*.

Mechanical valve replacements are metallic and therefore radio-opaque, but bioprosthetic valves may only feature a ring base or strut that is radio-opaque. The radiographic appearance of prosthetic valves is dependent on the specific valve model used, the valve's position relative to the X-ray beam, whether the valve is open or closed at the time of exposure and whether or not one or more valve leaflets are blurred by motion.⁷¹ The identification of prosthetic cardiac valves may be particularly challenging on portable chest radiography examinations. Electrocardiography-gated cardiac CT is valuable for assessment of valve prostheses, as this modality can be useful for evaluation of valve dysfunction by assessing opening angles.

Prosthetic valve complications include stenosis, pannus formation, endocarditis, structural failure, periprosthetic leaks and thrombosis.⁵⁵ Rates of complications have fallen in recent years with improvements in prosthesis design and surgical technique. Conventional radiography and cross-sectional imaging are insensitive in their ability to detect many of these complications,

Figure 14. (a) A 73-year-old male post transapical transcatheter aortic valve implantation (TAVI) (arrow) with enlargement of the right-sided cardiac silhouette suggestive of pericardial effusion. Axial CT image (b) confirms the presence of mediastinal haematoma (thin arrows). Also note the TAVI device (thick arrow).

Figure 15. (a, b) A 78-year-old male status post mitral valve clip placement (arrows) for severe mitral valve insufficiency.

but echocardiography has traditionally played a great role in evaluating the integrity of prosthetic cardiac valves.⁷²

The past decade has seen a rapid increase in the utilization of percutaneous valve repairs.⁷³ Mitral regurgitation may be repaired with percutaneous annuloplasty or placement of the mitral valve clip, while severe aortic stenosis may be repaired with a new prosthetic valve via transcatheter aortic-valve implantation (TAVI).

TAVI has showed promise in treating aortic stenosis in patients who were not amenable to surgery. Two TAVI systems have gained popularity, which are the balloon-expandable bioprosthetic Edwards Sapien valve (Edwards Lifesciences, Irvine, CA) and the self-expandable bioprosthetic CoreValve® ReValving System (Medtronic Inc., Minneapolis, MN). The cobalt-chromium framed Edwards SAPIEN valve and the Nitinol framed CoreValve both have the radiographic appearance of a metallic stent. The native valve is accessed using either a retrograde transarterial approach or an anterograde transapical approach. Before inserting the new prosthetic valve, balloon aortic valvuloplasty expands the stenotic native valve. Then, the unexpanded valve is positioned within the native aortic valve. The Edwards SAPIEN valve is expanded by a balloon, while the CoreValve self-expands.⁷⁴ One study demonstrated 31% mortality for patients who underwent TAVI as compared with 51% mortality for patients for standard therapy, in patients who were not surgical candidates.⁷⁵ While a recent study has suggested that there are similar mortalities at 1 year for both the transapical and the transcatheter approach, the route choice carries separate procedure-related risks.⁷⁶ The retrograde route carries a significant risk of atherothrombotic embolic stroke, which multiple studies have demonstrated with diffusion-weighted MRI of the brain.^{77,78} Alternatively, the anterograde transapical route that requires a left ventricular puncture carries a risk of ventricular rupture. The precise positioning of the valve is critical to prevent complications. Unlike traditional valve replacement, TAVI placement relies entirely upon imaging for correct

placement using both echocardiography and fluoroscopy. Some advocate the use of CT for both pre-procedural implantation and long-term post-procedural assessment.⁷⁹ If the valve is placed too high, then potential complications include aortic injury, paravalvular regurgitation and distal embolization. If the valve is placed too low, then potential complications include mitral valve dysfunction, heart block, paravalvular regurgitation and embolization into the left ventricle (Figure 14).^{79–81}

Figure 16. Lateral chest radiograph in a 37-year-old female with a patent foramen ovale demonstrates an Amplatzer closure device in correct position overlying the interatrial septum.

Figure 17. (a, b) A 6-year-old male with a muscular ventricular septal defect. Before Amplatzer closure device placement, the patient was with cardiomegaly and pulmonary overcirculation (a). After Amplatzer closure device placement (arrow), the heart size normalized with return to normal pulmonary circulation (b). The Amplatzer occluder was inserted surgically through a transatrial approach.

Percutaneous repair of mitral valve regurgitation has emerged as an alternative to invasive mitral valve annuloplasty or leaflet repair. Percutaneous annuloplasty devices are currently in the early stages of development and therefore data are limited regarding their efficacy and complications.⁸² Leaflet repair was first accomplished via a direct surgical approach in the early 1990s by suturing the leaflets to make a double-orifice mitral valve.⁸³ This surgical edge-to-edge repair approach has demonstrated clinical benefit with 5 years freedom from reoperation and recurrent mitral regurgitation more severe than grade 2.⁸⁴ Because of the success, the surgical edge-to-edge repair, a less invasive percutaneous technique to perform the edge-to-edge repair has recently been pioneered called the MitraClip mitral valve repair system (Evalve, Menlo Park, CA) (Figure 15). The MitraClip is delivered to the left atrium through a transseptal puncture and positioned at the mitral valve orifice. The MitraClip is then deployed creating a mitral valve double orifice. Mixed results were encountered in the EVEREST II trial, which compared patients who were randomly assigned to undergo either percutaneous repair or mitral valve surgery who had chronic moderately severe or severe mitral regurgitation (grade 3+ or 4+).⁸⁵ The authors concluded that the MitraClip was less effective than was conventional surgery at reducing mitral regurgitation. However, the MitraClip was considered safer with similar improvements in clinical outcomes.

Cardiac occlusion devices

Deployment of cardiac occlusion devices is indicated when the closure of an abnormal intracardiac or great vessel communication is desired. Although traditionally conventional surgery was required to implant these devices, today this can be performed via a transcatheter approach.⁸⁶ The first cardiac occlusion device was the Rashkind “double umbrella” device used for closure of a patent ductus arteriosus, but today atrial septal defects, patent foramina ovale, ventricular septal defects and other abnormal communications are also routinely closed with

a variety of closure devices (Figures 16 and 17).⁸⁷ Most feature a filter, basket or umbrella that is situated within the defect with the goal of occluding it. Complications associated with the placement of closure devices include vascular trauma, cardiac perforation, device malposition, thrombus formation with resultant distal embolization, residual shunts, arrhythmias and infective endocarditis.⁸⁸ Both short-term and long-term complications can sometimes be appreciated on imaging examinations, including chest radiography.⁸⁹

In addition, left atrial appendage closure is now routinely performed as a prophylactic procedure for the prevention of thromboembolic disease in certain clinical settings, such as in patients with atrial fibrillation who have contraindications to anticoagulation therapy.⁹⁰ For example, the Watchman left atrial appendage closure device may be used for stroke prevention in

Figure 18. A 59-year-old male with dilated ischaemic cardiomyopathy and congestive status post placement of HeartNet (Paracor Medical, Sunnyvale, CA) (left ventricular wrap). A biventricular implantable cardioverter defibrillator is present as well.

patients with atrial fibrillation who are not candidates for anti-coagulation (although please note that general use of this device in patients who can otherwise undergo anticoagulation is not recommended). Aside from incomplete appendage closure and device dislodgement, the chief complications of placement of these devices include thromboembolism and pericardial effusion. Echocardiography is routinely utilized to search for these complications when they are suspected.

PASSIVE CONSTRAINT DEVICES

Patients with end-stage heart failure have been treated with multiple therapies, including medical therapy, biventricular pacing, VADs, surgical heart remodelling and heart transplant. Recently, a different class of devices has been used to passively constrain a dilated heart in patients with dilated cardiomyopathy, which is a common cause for congestive heart failure. Some examples CorCap Cardiac Support Device (Acorn Cardiovascular, Inc., St Paul, MN), MyoSplint® (Myocor, Maple Grove, MN) and Paracor HeartNet (Paracor Medical, Sunnyvale, CA) have encountered varying levels of success. The Myosplint

system is no longer in use because of a lack of therapeutic effect and increased mitral valve insufficiency.⁹¹ Emerging data from studies of the CorCap device show that the device is safe with long-term sustained benefits on ventricular remodelling, reduction in death and major cardiac procedure and improvement in heart failure.⁹² The HeartNet, which is an elastic ventricular restraint device, is implanted circumferentially around the heart and applies a low level of epicardial pressure (Figure 18). This prevents left ventricular remodelling. Preliminary results suggested a functional and clinical benefit and a trend towards reverse remodelling of the ventricle.⁹³

CONCLUSION

Cardiac devices are ubiquitous throughout medical practice. Familiarity with these devices is critical to evaluate for and prevent complications. Because devices such as pacemakers are encountered in routine practice, the radiologist may be the first to identify device malfunction. Other devices are seen much less frequently but require knowledge of possible complications, as failure to detect can result in fatal consequences.

REFERENCES

- Maisel WH. Pacemaker and ICD generator reliability: meta-analysis of device registries. *JAMA* 2006; **295**: 1929–34.
- Parsonnet V, Bilitch M, Furman S, Fisher JD, Escher DJ, Myers G, et al. Early malfunction of transvenous pacemaker electrodes. A three-center study. *Circulation* 1979; **60**: 590–6.
- Link MS, Estes NA 3rd, Griffin JJ, Wang PJ, Maloney JD, Kirchhoffer JB, et al. Complications of dual chamber pacemaker implantation in the elderly. Pacemaker Selection in the Elderly (PASE) Investigators. *J Interv Card Electrophysiol* 1998; **2**: 175–9.
- Luria DM, Feinberg MS, Gurevitz OT, Bar-Lev DS, Granit C, Tanami N, et al. Randomized comparison of J-shaped atrial leads with and without active fixation mechanism. *Pacing Clin Electrophysiol* 2007; **30**: 412–17.
- Greene TO, Portnow AS, Huang SK. Acute pericarditis resulting from an endocardial active fixation screw-in atrial lead. *Pacing Clin Electrophysiol* 1994; **17**: 21–5.
- Miller GL, Coccio EB, Sharma SC. Postpericardiotomy syndrome and cardiac tamponade following transvenous pacemaker placement. *Clin Cardiol* 1996; **19**: 255–6.
- Levy Y, Shovman O, Granit C, Luria D, Gurevitz O, Bar-Lev D, et al. Pericarditis following permanent pacemaker insertion. *Isr Med Assoc J* 2004; **6**: 599–602.
- Dodinet B MP, Galvao S. Tined or screw-in lead. In: Aubert. AE, ed. *Pacemaker leads*. Amsterdam, Netherlands: Elsevier Science; 1985. pp. 75–82.
- Glikson M, Yaacoby E, Feldman S, Bar-Lev DS, Yaroslavtzev S, Granit C, et al. Randomized comparison of J-shaped and straight atrial screw-in pacing leads. *Mayo Clin Proc* 2000; **75**: 1269–73.
- Parsonnet V, Bernstein AD, Lindsay B. Pacemaker-implantation complication rates: an analysis of some contributing factors. *J Am Coll Cardiol* 1989; **13**: 917–21.
- Sorkin RP, Schuurmann BJ, Simon AB. Radiographic aspects of permanent cardiac pacemakers. *Radiology* 1976; **119**: 281–6.
- Steiner RM, Tegtmeier CJ. The radiology of cardiac pacemakers. *Cardiovasc Clin* 1983; **14**: 63–95.
- Ellenbogen KA, Wood MA, Shepard RK. Delayed complications following pacemaker implantation. *Pacing Clin Electrophysiol* 2002; **25**: 1155–8.
- García-Bolao I, Teijeira R, Díaz-Dorransoro I. Late fatal right ventricular perforation as complication of permanent pacing leads. *Pacing Clin Electrophysiol* 2001; **24**: 1036–7.
- Irwin JM, Greer GS, Lowe JE, German LD, Gilbert MR. Atrial lead perforation: a case report. *Pacing Clin Electrophysiol* 1987; **10**: 1378–81.
- Kautzner J, Bytesník J. Recurrent pericardial chest pain: a case of late right ventricular perforation after implantation of a transvenous active-fixation ICD lead. *Pacing Clin Electrophysiol* 2001; **24**: 116–18.
- Hirschl DA, Jain VR, Spindola-Franco H, Gross JN, Haramati LB. Prevalence and characterization of asymptomatic pacemaker and ICD lead perforation on CT. *Pacing Clin Electrophysiol* 2007; **30**: 28–32.
- Khan MN, Joseph G, Khaykin Y, Ziada KM, Wilkoff BL. Delayed lead perforation: a disturbing trend. *Pacing Clin Electrophysiol* 2005; **28**: 251–3.
- Henrikson CA, Leng CT, Yuh DD, Brinker JA. Computed tomography to assess possible cardiac lead perforation. *Pacing Clin Electrophysiol* 2006; **29**: 509–11.
- Van Gelder BM, Bracke FA, Oto A, Yildirim A, Haas PC, Seger JJ, et al. Diagnosis and management of inadvertently placed pacing and ICD leads in the left ventricle: a multi-center experience and review of the literature. *Pacing Clin Electrophysiol* 2000; **23**: 877–83.
- Villanueva FS, Heinsimer JA, Burkman MH, Fananapazir L, Halvorsen RA Jr, Chen JT. Echocardiographic detection of perforation of the cardiac ventricular septum by a permanent pacemaker lead. *Am J Cardiol* 1987; **59**: 370–1.
- Mazzetti H, Dussaut A, Tentori C, Dussaut E, Lazzari JO. Transarterial permanent pacing of the left ventricle. *Pacing Clin Electrophysiol* 1990; **13**: 588–92.
- Spindola-Franco H, Fish BG. *Radiology of the heart*. Berlin, Germany: Springer; 1985.
- Grier D, Cook PG, Hartnell GG. Chest radiographs after permanent pacing. Are they really necessary? *Clin Radiol* 1990; **42**: 244–9.
- Fyke FE 3rd. Infraclavicular lead failure: tarnish on a golden route. *Pacing Clin Electrophysiol* 1993; **16**: 373–6.
- Jacobs DM, Fink AS, Miller RP, Anderson WR, McVenes RD, Lessar JE, et al.

- Anatomical and morphological evaluation of pacemaker lead compression. *Pacing Clin Electrophysiol* 1993; **16**: 434–44.
27. Magney JE, Flynn DM, Parsons JA, Staplin DH, Chin-Purcell MV, Milstein S, et al. Anatomical mechanisms explaining damage to pacemaker leads, defibrillator leads, and failure of central venous catheters adjacent to the sternoclavicular joint. *Pacing Clin Electrophysiol* 1993; **16**: 445–57.
 28. Suzuki Y, Fujimori S, Sakai M, Ohkawa S, Ueda K. A case of pacemaker lead fracture associated with thoracic outlet syndrome. *Pacing Clin Electrophysiol* 1988; **11**: 326–30.
 29. Clarke B, Jones S, Gray HH, Rowland E. The tricuspid valve: an unusual site of endocardial pacemaker lead fracture. *Pacing Clin Electrophysiol* 1989; **12**: 1077–9.
 30. Hecht S, Berdoff R, Van Tosh A, Goldberg E. Radiographic pseudofracture of bipolar pacemaker wire. *Chest* 1985; **88**: 302–4.
 31. Fyke FE 3rd. Simultaneous insulation deterioration associated with side-by-side subclavian placement of two polyurethane leads. *Pacing Clin Electrophysiol* 1988; **11**: 1571–4.
 32. Witte AA. Pseudo-fracture of pacemaker lead due to securing suture: a case report. *Pacing Clin Electrophysiol* 1981; **4**: 716–18.
 33. Dunlap TE, Popat KD, Sorkin RP. Radiographic pseudofracture of the medtronic bipolar polyurethane pacing lead. *Am Heart J* 1983; **106**: 167–8.
 34. Uslan DZ, Sohail MR, St Sauver JL, Friedman PA, Hayes DL, Stoner SM, et al. Permanent pacemaker and implantable cardioverter defibrillator infection: a population-based study. *Arch Intern Med* 2007; **167**: 669–75.
 35. Eggimann P, Waldvogel F. Pacemaker and defibrillator infections. In: Waldvogel F, Bisno AL, eds. *Infections associated with indwelling medical devices*. Washington, DC: American Society for Microbiology Press; 2000. pp. 247–64.
 36. Sohail MR, Uslan DZ, Khan AH, Friedman PA, Hayes DL, Wilson WR, et al. Management and outcome of permanent pacemaker and implantable cardioverter-defibrillator infections. *J Am Coll Cardiol* 2007; **49**: 1851–9.
 37. Klug D, Lacroix D, Savoye C, Goullard L, Grandmougin D, Hennequin JL, et al. Systemic infection related to endocarditis on pacemaker leads: clinical presentation and management. *Circulation* 1997; **95**: 2098–107.
 38. Bakker PF, Hauer RN, Wever EF. Infections involving implanted cardioverter defibrillator devices. *Pacing Clin Electrophysiol* 1992; **15**: 654–8.
 39. Gupta S, Prevel CD, Shaheen K, Wilkens E, Smith DJ Jr, Kirsh MM, et al. Wound complications and treatment of the infected implantable cardioverter defibrillator generator. *J Card Surg* 1993; **8**: 671–7.
 40. Molina JE. Undertreatment and overtreatment of patients with infected antiarrhythmic implantable devices. *Ann Thorac Surg* 1997; **63**: 504–9.
 41. Cacoub P, Leprince P, Nataf P, Hausfater P, Dorent R, Wechsler B, et al. Pacemaker infective endocarditis. *Am J Cardiol* 1998; **82**: 480–4.
 42. Ploux S, Riviere A, Amraoui S, Whinnett Z, Barandon L, Lafitte S, et al. Positron emission tomography in patients with suspected pacing system infections may play a critical role in difficult cases. *Heart Rhythm* 2011; **8**: 1478–81.
 43. Mouloupoulos SD, Topaz S, Kolff WJ. Diastolic balloon pumping (with carbon dioxide) in the aorta—a mechanical assistance to the failing circulation. *Am Heart J* 1962; **63**: 669–75.
 44. Mouloupoulos SD, Topaz SR, Kolff WJ. Extracorporeal assistance to the circulation and intraaortic balloon pumping. *Trans Am Soc Artif Intern Organs* 1962; **8**: 85–9.
 45. Chen EW, Canto JG, Parsons LS, Peterson ED, Littrell KA, Every NR, et al. Relation between hospital intra-aortic balloon counterpulsation volume and mortality in acute myocardial infarction complicated by cardiogenic shock. *Circulation* 2003; **108**: 951–7.
 46. Hochman JS, Sleeper LA, Webb JG, Sanborn TA, White HD, Talley JD, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. SHOCK Investigators. Should we emergently revascularize occluded coronaries for cardiogenic shock. *N Engl J Med* 1999; **341**: 625–34.
 47. Fuchs RM, Brin KP, Brinker JA, Guzman PA, Heuser RR, Yin FC. Augmentation of regional coronary blood flow by intra-aortic balloon counterpulsation in patients with unstable angina. *Circulation* 1983; **68**: 117–23.
 48. Gold HK, Leinbach RC, Sanders CA, Buckley MJ, Mundth ED, Austen WG. Intraaortic balloon pumping for control of recurrent myocardial ischemia. *Circulation* 1973; **47**: 1197–203.
 49. Ohman EM, George BS, White CJ, Kern MJ, Gurbel PA, Freedman RJ, et al. Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a randomized trial. The Randomized IABP Study Group. *Circulation* 1994; **90**: 792–9.
 50. Mahaffey KW, Ohman EM. Perspectives on the use of intra-aortic balloon counterpulsation in the 1990s. In: Topol EJ, ed. *Textbook of Interventional Cardiology*. Philadelphia, PA: WB Saunders; 1996. pp. 303–20.
 51. Cochran RP, Starkey TD, Panos AL, Kunzelman KS. Ambulatory intraaortic balloon pump use as bridge to heart transplant. *Ann Thorac Surg* 2002; **74**: 746–51; discussion 751–2.
 52. Ferguson JJ III, Cohen M, Freedman RJ Jr, Stone GW, Miller ME, Joseph DL, et al. The current practice of intra-aortic balloon counterpulsation: results from the benchmark registry. *J Am Coll Cardiol* 2001; **38**: 1456–62.
 53. Hyson EA, Ravin CE, Kelley MJ, Curtis AM. Intraaortic counterpulsation balloon: radiographic considerations. *AJR Am J Roentgenol* 1977; **128**: 915–18.
 54. Santa-Cruz RA, Cohen MG, Ohman EM. Aortic counterpulsation: a review of the hemodynamic effects and indications for use. *Catheter Cardiovasc Interv* 2006; **67**: 68–77.
 55. Attili A, Kazerooni EA. Postoperative cardiopulmonary thoracic imaging. *Radiol Clin North Am* 2004; **42**: 543–64, vi.
 56. Kim JT, Lee JR, Kim JK, Yoon SZ, Jeon Y, Bahk JH, et al. The carina as a useful radiographic landmark for positioning the intraaortic balloon pump. *Anesth Analg* 2007; **105**: 735–8.
 57. Jacobs LE, Fraifeld M, Kotler MN, Ioli AW. Aortic dissection following intraaortic balloon insertion: recognition by transesophageal echocardiography. *Am Heart J* 1992; **124**: 536–40.
 58. Meco M, Gramegna G, Yassini A, Bellisario A, Mazzaro E, Babbini M, et al. Mortality and morbidity from intra-aortic balloon pumps. Risk analysis. *J Cardiovasc Surg (Torino)* 2002; **43**: 17–23.
 59. Hurwitz LM, Goodman PC. Intraaortic balloon pump location and aortic dissection. *AJR Am J Roentgenol* 2005; **184**: 1245–6.
 60. Durand DJ, Weiss CR, Fishman EK. Computed tomography appearance of an intra-aortic balloon pump: a potential pitfall in diagnosis. *J Comput Assist Tomogr* 2005; **29**: 231–3.
 61. Aksnes J, Abdelnoor M, Berge V, Fjeld NB. Risk factors of septicemia and perioperative myocardial infarction in a cohort of patients supported with intra-aortic balloon pump (IABP) in the course of open heart surgery. *Eur J Cardiothorac Surg* 1993; **7**: 153–7.
 62. Rose EA, Gelijns AC, Moskowitz AJ, Heitjan DF, Stevenson LW, Dembitsky W, et al. Long-term use of a left ventricular assist device for end-stage heart failure. *N Engl J Med* 2001; **345**: 1435–43.
 63. Jonnala P, Deshmane S, Krishnam M. Postoperative imaging of ischemic cardiac disease. *Cardiovascular imaging*. St Louis, MO: Saunders; 2011.
 64. Goldstein DJ, Smego D, Michler RE. Surgical aspects of congestive heart failure. *Heart Fail Rev* 2006; **11**: 171–92.

65. Knisely BL, Collins J, Jahania SA, Kuhlman JE. Imaging of ventricular assist devices and their complications. *AJR Am J Roentgenol* 1997; **169**: 385–91.
66. Maisel WH, ed. *Device therapy in heart failure*. Boston, MA: Humana Press.
67. Ota T, Takayama H, Khalpey Z, Naka Y. Reinforcement of HeartMate II bend relief connection: champagne bottle technique. *Ann Thorac Surg* 2013; **95**: e107–8.
68. Deng MC, Edwards LB, Hertz MI, Rowe AW, Keck BM, Kormos R, et al. Mechanical circulatory support device database of the International Society for Heart and Lung Transplantation: third annual report–2005. *J Heart Lung Transplant* 2005; **24**: 1182–7.
69. Bloomfield P, Wheatley DJ, Prescott RJ, Miller HC. Twelve-year comparison of a Bjork-Shiley mechanical heart valve with porcine bioprostheses. *N Engl J Med* 1991; **324**: 573–9.
70. Hammermeister K, Sethi GK, Henderson WG, Grover FL, Oprian C, Rahimtoola SH. Outcomes 15 years after valve replacement with a mechanical versus a bioprosthetic valve: final report of the veterans affairs randomized trial. *J Am Coll Cardiol* 2000; **36**: 1152–8.
71. Landay MJ, Estrera AS, Bordlee RP. Cardiac valve reconstruction and replacement: a brief review. *Radiographics* 1992; **12**: 659–71.
72. Steiner RM, Mintz G, Morse D, Kotler M, Flicker S, Raichlen JS, et al. The radiology of cardiac valve prostheses. *Radiographics* 1988; **8**: 277–98.
73. Feldman T, Leon MB. Prospects for percutaneous valve therapies. *Circulation* 2007; **116**: 2866–77.
74. Augoustides JG, Wolfe Y, Walsh EK, Szeto WY. Recent advances in aortic valve disease: highlights from a bicuspid aortic valve to transcatheter aortic valve replacement. *J Cardiothorac Vasc Anesth* 2009; **23**: 569–76. doi: [10.1053/j.jvca.2009.03.020](https://doi.org/10.1053/j.jvca.2009.03.020)
75. Leon MB, Smith CR, Mack M, Miller DC, Moses JW, Svensson LG, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. *N Engl J Med* 2010; **363**: 1597–607. doi: [10.1056/NEJMoa1008232](https://doi.org/10.1056/NEJMoa1008232)
76. Smith CR, Leon MB, Mack MJ, Miller DC, Moses JW, Svensson LG, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. *N Engl J Med* 2011; **364**: 2187–98.
77. Ghanem A, Müller A, Nähle CP, Kocurek J, Werner N, Hammerstingl C, et al. Risk and fate of cerebral embolism after transfemoral aortic valve implantation: a prospective pilot study with diffusion-weighted magnetic resonance imaging. *J Am Coll Cardiol* 2010; **55**: 1427–32. doi: [10.1016/j.jacc.2009.12.026](https://doi.org/10.1016/j.jacc.2009.12.026)
78. Kahlert P, Knipp SC, Schlamann M, Thielmann M, Al-Rashid F, Weber M, et al. Silent and apparent cerebral ischemia after percutaneous transfemoral aortic valve implantation: a diffusion-weighted magnetic resonance imaging study. *Circulation* 2010; **121**: 870–8. doi: [10.1161/CIRCULATIONAHA.109.855866](https://doi.org/10.1161/CIRCULATIONAHA.109.855866)
79. Leipsic J, Gurvitch R, Labounty TM, Min JK, Wood D, Johnson M, et al. Multidetector computed tomography in transcatheter aortic valve implantation. *JACC Cardiovasc Imaging* 2011; **4**: 416–29. doi: [10.1016/j.jcmg.2011.01.014](https://doi.org/10.1016/j.jcmg.2011.01.014)
80. Al Ali AM, Altwegg L, Horlick EM, Feindel C, Thompson CR, Cheung A, et al. Prevention and management of transcatheter balloon-expandable aortic valve malposition. *Catheter Cardiovasc Interv* 2008; **72**: 573–8. doi: [10.1002/ccd.21667](https://doi.org/10.1002/ccd.21667)
81. Tuzcu EM. Transcatheter aortic valve replacement malposition and embolization: innovation brings solutions also new challenges. *Catheter Cardiovasc Interv* 2008; **72**: 579–80. doi: [10.1002/ccd.21788](https://doi.org/10.1002/ccd.21788)
82. Yuksel UC, Kapadia SR, Tuzcu EM. Percutaneous mitral repair: patient selection, results, and future directions. *Curr Cardiol Rep* 2011; **13**: 100–6. doi: [10.1007/s11886-010-0158-x](https://doi.org/10.1007/s11886-010-0158-x)
83. Alfieri O, Maisano F, De Bonis M, Stefano PL, Torracca L, Oppizzi M, et al. The double-orifice technique in mitral valve repair: a simple solution for complex problems. *J Thorac Cardiovasc Surg* 2001; **122**: 674–81.
84. Maisano F, Viganò G, Blasio A, Colombo A, Calabrese C, Alfieri O. Surgical isolated edge-to-edge mitral valve repair without annuloplasty: clinical proof of the principle for an endovascular approach. *EuroIntervention* 2006; **2**: 181–6.
85. Feldman T, Foster E, Glower DD, Kar S, Rinaldi MJ, Fail PS, et al. Percutaneous repair or surgery for mitral regurgitation. *N Engl J Med* 2011; **364**: 1395–406. doi: [10.1056/NEJMoa1009355](https://doi.org/10.1056/NEJMoa1009355)
86. Kim JH, Siegel MJ, Goldstein JA, Gutierrez FR, Lasala JM. Radiologic findings of 2 commonly used cardiac septal occluders with clinical correlation. *J Thorac Imaging* 2003; **18**: 183–9.
87. Hunter TB, Taljanovic MS, Tsau PH, Berger WG, Standen JR. Medical devices of the chest. *Radiographics* 2004; **24**: 1725–46.
88. Raghuram AR, Krishnan R, Kumar S, Balamurugan K. Complications in atrial septal defect device closure. *Interact Cardiovasc Thorac Surg* 2008; **7**: 167–9.
89. Schlesinger AE, Folz SJ, Beekman RH. Transcatheter atrial septal defect occlusion devices: normal radiographic appearances and complications. *J Vasc Interv Radiol* 1992; **3**: 527–33.
90. Obeyesekere MN, Lockwood S, Mottram P, Alison JF. Role for the left atrial appendage occlusion device in managing thromboembolic risk in atrial fibrillation. *Intern Med J* 2012; **42**: e15–9. doi: [10.1111/j.1445-5994.2011.02698.x](https://doi.org/10.1111/j.1445-5994.2011.02698.x)
91. Christiansen S, Klocke A, Autschbach R. Past, present, and future of long-term mechanical cardiac support in adults. *J Card Surg* 2008; **23**: 664–76. doi: [10.1111/j.1540-8191.2008.00696.x](https://doi.org/10.1111/j.1540-8191.2008.00696.x)
92. Mann DL, Kubo SH, Sabbah HN, Starling RC, Jessup M, Oh JK, et al. Beneficial effects of the CorCap cardiac support device: five-year results from the Acorn Trial. *J Thorac Cardiovasc Surg* 2012; **143**: 1036–42. doi: [10.1016/j.jtcvs.2011.06.014](https://doi.org/10.1016/j.jtcvs.2011.06.014)
93. Klodell CT Jr, Aranda JM Jr, McGiffin DC, Rayburn BK, Sun B, Abraham WT, et al. Worldwide surgical experience with the Paracor HeartNet cardiac restraint device. *J Thorac Cardiovasc Surg* 2008; **135**: 188–95. doi: [10.1016/j.jtcvs.2007.09.034](https://doi.org/10.1016/j.jtcvs.2007.09.034)