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Imaging in patients with cardiovascular implantable electronic devices: part 2—imaging after device implantation. A clinical consensus statement of the European Association of Cardiovascular Imaging (EACVI) and the European Heart Rhythm Association (EHRA) of the ESC

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Cardiac implantable electronic devices (CIEDs) improve quality of life and prolong survival, but there are additional considerations for cardiovascular imaging after implantation—both for standard indications and for diagnosing and guiding management of device-related complications. This clinical consensus statement (part 2) from the European Association of Cardiovascular Imaging, in collaboration with the European Heart Rhythm Association, provides comprehensive, up-to-date, and evidence-based guidance to cardiologists, cardiac imagers, and pacing specialists regarding the use of imaging in patients after implantation of conventional pacemakers, cardioverter defibrillators, and cardiac resynchronization therapy (CRT) devices. The document summarizes the existing evidence regarding the role and optimal use of various cardiac imaging modalities in patients with suspected CIED-related complications and also discusses CRT optimization, the safety of magnetic resonance imaging in CIED carriers, and describes the role of chest radiography in assessing CIED type, position, and complications. The role of imaging before and during CIED implantation is discussed in a companion document (part 1).

Keywords multimodality imaging • cardiovascular implantable electronic devices • pacemaker • cardiac resynchronization therapy • defibrillator • complications

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Introduction

While the implantation of cardiac implantable electronic devices (CIEDs), including permanent pacemakers (PMs), cardiac resynchronization therapy (CRT) devices, and implantable cardioverter defibrillators (ICDs), improves quality of life and prolongs survival, complications may occur both during and after implantation and these are often associated with unfavourable patient outcomes. In addition, even imaging for standard indications in patients with CIEDs may be more complex, with feasibility, safety, and image quality considerations.

Although perioperative and long-term complication rates have decreased with proper training in implantation technique, and procedure-related death is exceptionally rare (0–0.1%),[1] it is of paramount importance to prevent CIED-related complications and when they do occur to detect and treat them in a timely and efficient manner. In this document, we discuss the role and optimal use of different cardiac imaging techniques in patients with suspected CIED-related complications. We also discuss CRT optimization, the safety of magnetic resonance imaging (MRI) in CIED carriers, and describe the role of chest radiography in assessing CIED type, position, and complications. Clinical statements and a practical guide on cardiac imaging before and during CIED implantation are published in a companion document (part 1).

As in part 1, this clinical consensus statement document is based on a review of the literature performed by the members of the writing group. The clinical advice (key points) is based upon the evidence and/or consensus of the writing group and is classified into categories, as shown in *Table 1*.

Imaging of complications

Complications during and after CIED implantation may occur due to mechanical factors (e.g. cardiac perforation and tamponade, pneumothorax, and damage to the tricuspid valve or central veins), device-related infections, and pacing-induced dyssynchrony (pacemaker syndrome, cardiac remodelling, mitral regurgitation) (*Figure 1*). All patients with clinical worsening after CIED implantation should undergo appropriate clinical assessment and where necessary cardiac imaging tests. The selection of the most appropriate imaging modalities will depend on the clinical circumstances and the suspected complication. Chest X-ray (CXR) or ultrasound is the initial imaging modalities of choice for most complications, while computed tomography (CT), single-photon emission computed tomography (SPECT), or positron emission tomography (PET) is usually ordered in cases of diagnostic uncertainty. While MRI is often not the first line imaging modality to investigate complications of CIED, many CIED carriers may need MRI for other reasons during their lifetimes. Therefore, we will also outline the most important aspects when considering MRI in patients with conditional and non-MRI-conditional devices.

Table 1 Categories of clinical advice

	Definition	Symbol
Strength of advice	Clinical advice, based on robust published evidence	
	Clinical advice, based on uniform consensus of the writing group	
	May be appropriate, based on published evidence	
	May be appropriate, based on consensus within writing group	
	Area of uncertainty	

Infection

Infection can affect the pocket of the cardiac device and the intravascular segment of the leads, according to the 2023 ESC guidelines.[2] Localized infections are defined in the 2023 ESC guidelines and a recent EHRA consensus document as either superficial incisional infections (acute infection without involvement of the pocket or hardware) or isolated pocket infections (limited to the hardware in the pocket), while cardiovascular implanted electronic device-related infective endocarditis (CDRIE) is defined as evidence of CIED infection with clinical signs of pocket infection and/or imaging findings that fulfil the criteria for valvular IE.[2–4] Transthoracic (TTE) and transoesophageal (TOE) echocardiography are complementary methods and are both helpful in the diagnosis of lead vegetations and tricuspid valve (TV) involvement, sizing of vegetations, detection and quantification of tricuspid regurgitation (TR), and follow-up after lead extraction.[2,3] Of note, mobile intracardiac thrombi on transvenous leads can be frequently detected by TTE or TOE in asymptomatic CIED carriers[5–7] (*Figure 2*). It is advised to always include them in the report to allow comparisons with subsequent TTE/TOE examinations as this could later help to assess if lead masses are acute or chronic. However, in addition to the difficulties in distinguishing between vegetations and thrombi, echocardiography may be falsely negative in CDRIE that is why other imaging modalities, and nuclear techniques in particular, are essential in diagnostic approach to the patient with suspected CIED. According to the 2019 International CIED Infection Criteria,[3] major imaging criteria for diagnosis of CIED infections and/or infective endocarditis (IE) include echocardiogram positive for CIED infection or valve IE and 18-fluorodeoxyglucose (FDG) PET/CT or radiolabelled leucocyte SPECT/CT detection of abnormal activity at pocket/generator site, along leads, or at valve site. For 18-FDG PET/CT, caution is advised in case of recent implants (<6 weeks).[3,4] Nuclear modalities are particularly helpful in the subset of 'possible CIED infections', i.e. in patients presenting with systemic infection but without local findings at the generator pocket.[3,4]

In recent meta-analyses, the pooled sensitivity and specificity of PET/CT for diagnosis of CIED infection ranged from 83–87% and 89–94%, respectively, with a higher accuracy for detection of generator pocket infection than lead infection.[8,9] Pooled specificity and sensitivity were 93% and 98%, respectively, for pocket/generator infection, and 65% and 88%, respectively, for lead infection.[9] Data on accuracy of labelled leucocyte CT/SPECT scintigraphy for the diagnosis of CIED infection are limited, but available studies reported sensitivity above 90% and specificity of 100%.[9] Nuclear imaging modalities may also be considered to identify extracardiac foci of infection and related complications, such as pulmonary septic embolism (*Figure 3*).[2] Further details on the diagnosis and treatment of CDRIE can be found in the 2023 ESC guidelines and recent international consensus documents on IE and CIED.[2–4]

Clinical advice

TTE and TEE are advised initial imaging modalities in patients with suspected CDRIE	
Major imaging criteria for diagnosis of CIED infections and/or IE include echocardiogram positive for CIED infection or valve IE and 18-fluorodeoxyglucose (FDG) PET/CT or radiolabelled leucocyte SPECT/CT detection of abnormal activity at pocket/generator site, along leads or at valve site	

Cardiac perforation and tamponade

Cardiac perforation by atrial or ventricular leads is a rare but potentially life-threatening complication of CIED implantation. It usually happens acutely, at the time of lead insertion, but may also occur several months or years following implantation.[10–12] Clinical presentation is highly variable—from asymptomatic cases with loss of lead capture to chest pain, dyspnoea, and cardiac tamponade. The diagnosis can be made by chest radiography, TTE, and CT.[10,11]

On CXR, the diagnosis of perforation is certain if the tip of the lead is seen beyond the cardiac silhouette. Serial CXRs are useful for comparing post-operative lead position (*Figure 4*). Perforation is also suspected in the presence of a left-sided pleural effusion or lead displacement and also when a right-sided pneumothorax is noted after left-sided device implantation (due to right atrial perforation).[10] Echocardiography may be appropriate to identify the tip of the perforating pacing wire, and it also allows bedside detection of pericardial effusion and cardiac tamponade (*Figure 5*). However, chest radiography and TTE have a relatively low sensitivity and cannot be used to exclude the diagnosis of cardiac perforation if clinical suspicion is high.[10,11]

The accuracy of chest CT for the diagnosis of cardiac perforation exceeds 90%, and it should be performed in patients in whom perforation is highly suspected but not confirmed on chest radiography and/or echocardiography.[10] An ECG-gated contrast CT angiography protocol can provide the clearest assessment of lead position relative to the myocardium and also demonstrate concurrent complications, such as pleural effusion, pneumothorax, or unusual extracardiac migration of the lead.

Clinical advice

Chest radiography and TTE are advised as the initial imaging modalities in patients with CIED and suspected cardiac perforation	
If cardiac perforation is highly suspected but not confirmed on chest radiography and/or echocardiography, ECG-gated contrast CT angiography is advised	

Pneumothorax

Pneumothorax and haemothorax are potential immediate post-operative complications and are mostly seen on the ipsilateral side of implantation due to inadvertent puncture of the lung. The incidence of pneumothorax after device implantation is low and ranges between 0.8 and 2.8%.[1,13–15] The true incidence is probably higher because of clinically unrecognized cases and underdetection with routine chest radiography.[15]

A higher incidence of pneumothorax has been associated with subclavian vein punctures, older age, female gender, chronic obstructive pulmonary disease, and operator inexperience.[13–15] In a small randomized trial, the ultrasound-guided axillary approach was superior in terms of success rate, time to obtain venous access and procedural time, but with similar complication rate.[16] Depending on the urgency of the situation, the diagnosis is made using fluoroscopy (if a large pneumothorax is suspected during the implantation procedure) or by chest radiography after the implantation (*Figure 6*). On occasion, a CT scan may be needed to provide additional information, especially when concurrent complications or other chest pathologies are suspected. Also, CT is indicated if the CXR is negative but a pneumothorax remains clinically suspected.[1] Pneumothorax can also be diagnosed using lung ultrasound with a sensitivity higher than that of conventional anterior–posterior chest radiography.[17] However, the accuracy and utility of this approach has not been validated in CIED recipients.

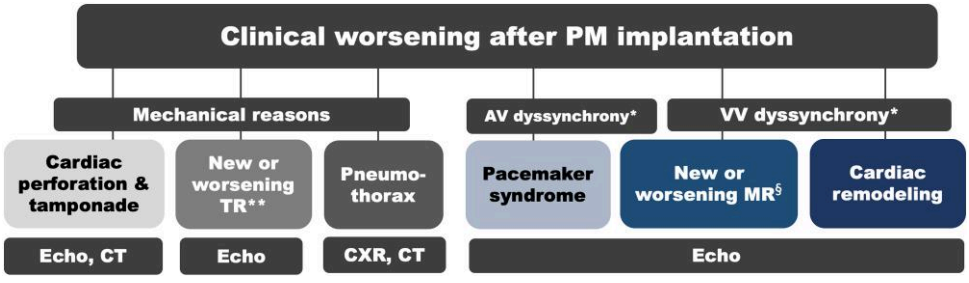

Figure 1 Imaging approach to patient with clinical worsening after device implantation. *Denotes conventional antibradycardia pacemaker; **tricuspid regurgitation (TR) may also worsen for other causes, i.e. right ventricular (RV) dyssynchrony from RV pacing or RV dysfunction from pulmonary hypertension secondary to LV dyssynchrony and dysfunction, and/or tricuspid annular dilation due to atrial fibrillation and right atrial remodelling; §new or worsening mitral regurgitation (MR) can occur in case of too long AV interval (diastolic MR), pacing-induced papillary muscle dyssynchrony, or cardiac remodelling (systolic MR). AV, atrioventricular; CIED, cardiac implantable electronic devices; CT, computed tomography; CXR, chest X-ray; Echo, echocardiography; VV, ventricular.

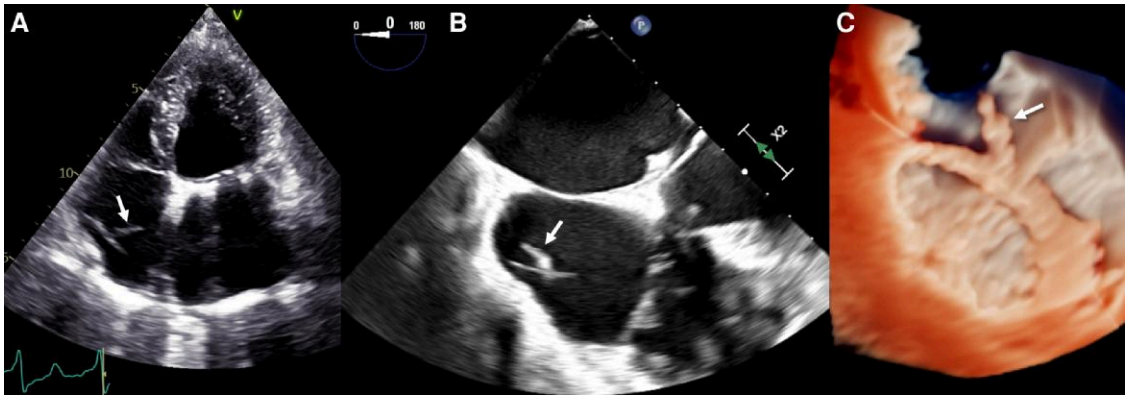

Figure 2 Incidentally detected thrombus (arrows) on a lead of a permanent pacemaker, as seen by transthoracic (*A*) and two- (*B*) and three-dimensional (*C*) transoesophageal echocardiography.

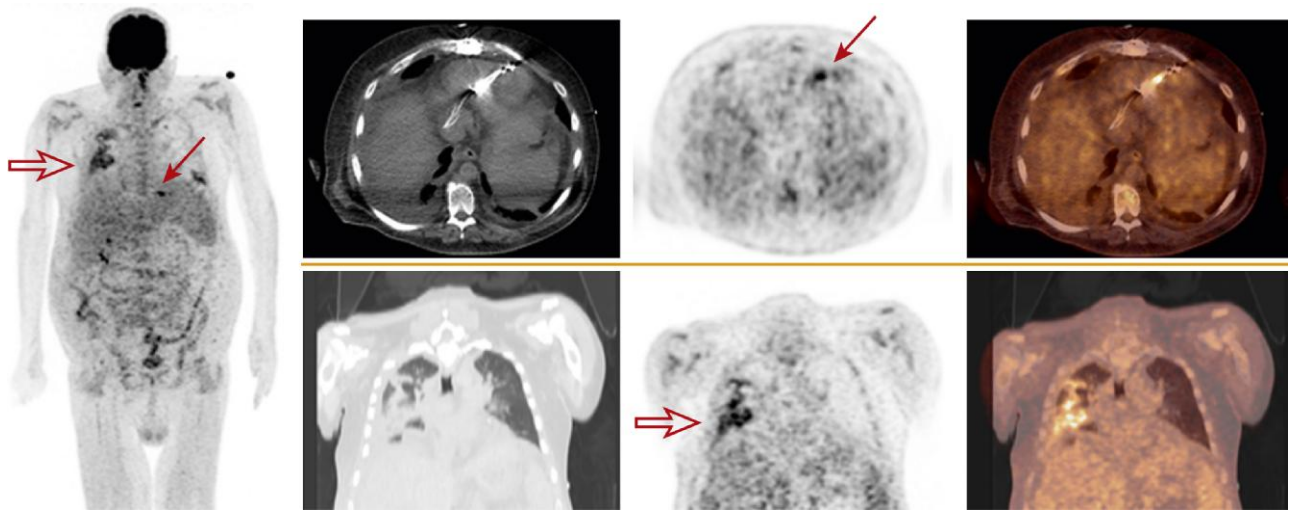

Figure 3 An example of defibrillator lead infection with septic lung emboli diagnosed by FDG PET/CT. There is a focus of abnormal fluorodeoxyglucose (FDG) uptake in the defibrillator lead entering the left ventricle compatible with a lead infection (red arrow) [right, maximum-intensity projection image; upper row represents short-axial views of computed tomography (CT) (right), FDG (middle), and fused positron emission tomography (PET/CT) (left)]. In addition, there is a patchy area of increased FDG uptake (open arrow) in the right lower lung corresponding to consolidation on lung window, representing infectious emboli [bottom, coronal views of computed tomography (CT), positron emission tomography (PET), and fused images]. Reproduced with permission from Chen W, Sajadi MM, Dilsizian V. Merits of FDG PET/CT and functional molecular imaging over anatomic imaging with echocardiography and CT angiography for the diagnosis of cardiac device Infections. *JACC Cardiovasc Imaging*. 2018;11(11):1679–1691.

Clinical advice

Chest radiography is advised in all patients following CIED implantation to look for pneumothorax

Lung ultrasound may be appropriate to screen for pneumothorax after CIED implantation if there is local expertise to perform and interpret this examination

CT is advised in patients with suspected pneumothorax when the CXR is inconclusive or concurrent complications and/or other chest pathologies are suspected

Lead-related tricuspid valve dysfunction

CIED may cause or worsen TV dysfunction. A lead or device placed in the right ventricle (RV) may interfere with the TV apparatus and contribute to or cause TR. This is observed in 7% to 45% of patients who receive a CIED.[18] The incidence of CIED-induced or mediated TR is expected to increase with the ageing population and the increasing the

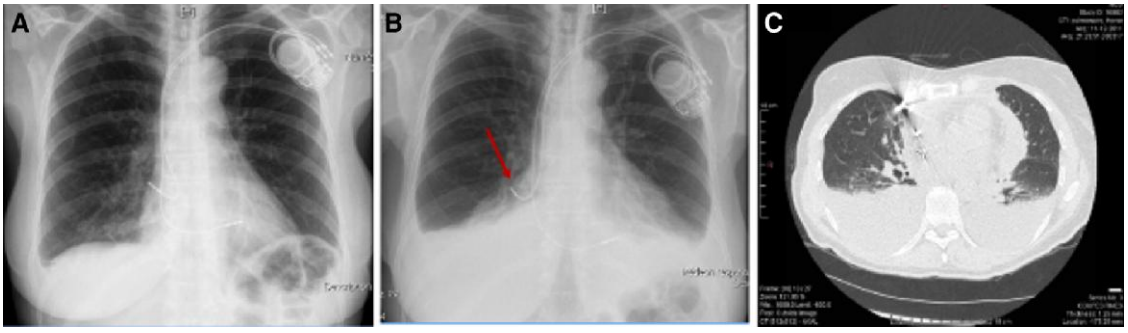

Figure 4 Delayed perforation of the right atrial lead. (*A*) Immediate post-operative chest X-ray (CXR). The atrial lead is positioned in the lateral right atrial (RA) appendage. (*B*) CXR after one month showing perforation of the atrial lead that projects outside the cardiac silhouette, and bilateral pleural effusion. (*C*) Computed tomography scan confirming RA lead perforation with pericardial effusion and bilateral pleural effusion. The atrial lead was repositioned under surgical standby.

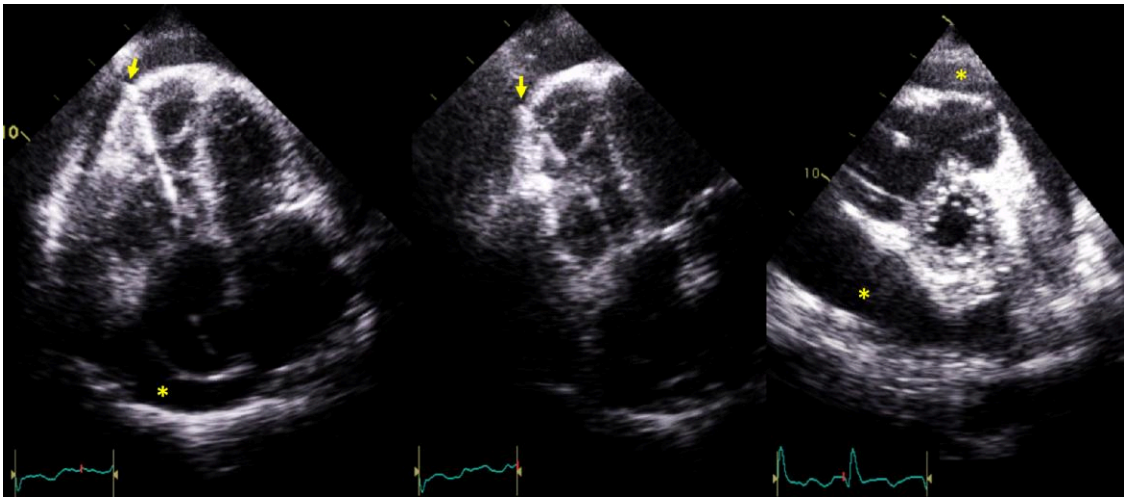

Figure 5 Right ventricular (RV) wall perforation by a pacemaker lead detected 8 days after device implantation. Echocardiography revealed the pacemaker lead outside the RV chamber (arrows), along with a large pericardial effusion (*).

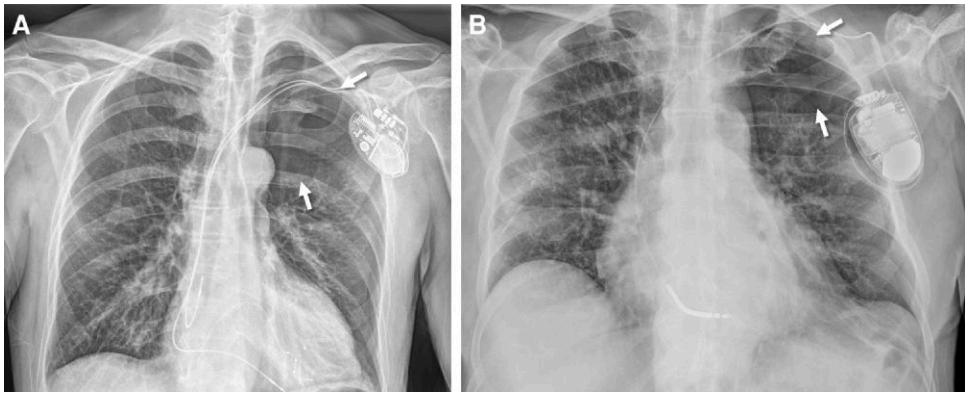

Figure 6 Pneumothorax complicating the implantation of cardiac resynchronization (*A*) and cardioverter defibrillator device (*B*). In both cases, there is a left-sided area with absent lung markings (arrows). Image courtesy Nikola Radovanovic, Pacemaker Center, University Clinical Centre of Serbia and Srdjan Raspopovic, Clinical Hospital Centre Zemun, Belgrade, Serbia.

number of CIED implantations.[19–21] In a recent prospective study, there was a 5% increase in the prevalence of clinically relevant (moderate or severe) TR, one year after endocardial lead insertion.[22]

Assessing the primary cause of TV dysfunction in a patient with CIED is challenging, since different mechanisms may be responsible for primary or secondary TR, depending on the time frame from implant. CIED-related TR can occur early due to leaflet mechanical impingement by the lead or direct damage of the TV apparatus during lead implantation and manipulation (e.g. leaflet perforation or laceration, lead entanglement in the subvalvular apparatus, or transection of the papillary muscles or chordae tendineae).[18] TR (or less often tricuspid stenosis) may also develop later due to lead adherence, encapsulation, or entrapment that occur because of fibrosis and adhesive interactions between the lead and endocardial surfaces. Yet, not all cases of CIED-induced TR relate to the lead. Often, the lead is a passive bystander (i.e. not interfering with TV apparatus), and TR is caused or aggravated via common 'functional' mechanisms. These include pacing-induced dyssynchrony and dysfunction in the left and right ventricles, elevated LV filling pressures, secondary mitral regurgitation (MR) and pulmonary hypertension, RV remodelling as well as the vicious cycle of TV annulus dilation due to TR and chronic volume loading leading to further regurgitation.[23] Finally, significant primary TR may be caused by endocarditis or by leaflet avulsion during lead extraction.

Leadless pacemakers may also cause or worsen TR, by these functional effects as well as mechanical interference with the TV subvalvular apparatus.[24] Septal implantation of these leadless devices has been shown to be 5 times more likely to worsen TR than apical implantation, due to entanglement of the leadless device with the chordae tendineae or direct interaction between the device and the leaflets.[24]

The data on the utility of echocardiography-guided CIED implantation to reduce device-related TR are inconsistent. In a small randomized study, the rate of lead-associated TR was low and a routine intraprocedural two-dimensional (2D) TTE did not have a significant role in reducing it.[25] In another pilot study, TEE-guided lead implantation was safe and feasible and was associated with less worsening of TR than standard lead implantation guided by fluoroscopy.[26]

Due to its multifactorial pathophysiology that shares features with both primary and secondary TRs,[23,27,28] as well as its different epidemiology, specific therapeutic options, and worse outcomes, it has been recently proposed to classify CIED-related TR as a distinct category from primary and secondary TRs.[29] Significant lead-induced TR (either newly developed or worsening moderate or severe TR) is associated with impaired RV performance and a higher incidence of long-term mortality and heart failure (HF) events.[30,31]

Confronted by a patient with a CIED presenting with signs of worsening right-sided HF, a high level of clinical suspicion must be maintained to rule out CIED-related TR. Imaging is essential for identifying and classifying the TV dysfunction, assessing the mechanism(s), and grading the severity of TR in CIED patients. In patients with severe CIED-related TR, lead extraction may further aggravate TR if irreversible damage to the TV leaflets (e.g. severe adherence, retraction, or perforation) has occurred.[32] Therefore, timely identification of the type of lead interference using imaging is important.

TTE is the imaging modality of choice for the diagnosis and severity grading of CIED-related TR. In this setting, TR may be underestimated due to the eccentricity of the regurgitant jet, and the acoustic shadowing and artefacts induced by the lead. Systolic hepatic vein flow reversal is generally considered specific of severe TR, but in patients with pacemakers and atrial fibrillation, this sign of severity is less reliable.[33] The sensitivity of 2D TTE for the identification of CIED-related TR is unacceptably low (12–17%), due to its inability to display routinely all the leaflets and the lead in a single short-axis image.[23] TOE may allow better spatial resolution in patients with suboptimal acoustic TTE windows, but standard 2D TOE images generally suffer from similar limitations to 2D TTE in the assessment of lead–leaflet interactions.

Three-dimensional (3D) echocardiography, either TTE or TOE, offers improved spatial definition of the actual position of the lead or leadless device,[34] and of its interactions with the components of TV apparatus (*Figure 7*). However, since the TV is located anteriorly in the mediastinum, it is often more easily accessed with the transthoracic approach than with the transoesophageal approach.[18] Visualization of a lead abutting a leaflet in a 3D dynamic *en face* view of the TV by 3D TTE has the highest sensitivity (94%) for the diagnosis of CIED-related TV dysfunction[35] (*Figure 8*). Careful positioning of the cropping plane just above the leaflet tips is key for properly visualizing the position of the lead (commissural, central, or impinging in the middle of a leaflet).[36] Cropping the data set too low (at the annulus level) or too high (far in the ventricle) might result in misleading conclusions about the lead position, as the electrodes might change their spatial orientation as they cross the TV (*Figure 9*). Excessive lead slack during the cardiac cycle can be tackled with careful frame-by-frame inspection of 3D rendered TV images. Acoustic shadowing from thicker ICD leads may occasionally obscure leaflet visualization by 3D TTE.

When 3D TTE image quality of the leaflets is not satisfactory, short-axis views of the TV by transgastric 2D or 3D TOE and cine cardiac CT may be appropriate. Cardiovascular MRI is affected by significant local artefact in proximity to CIED leads, which often impairs visualization of the lead, valve, and associated TR. Therefore, 3D echocardiography is the imaging modality of choice for assessment and for guiding interventional treatment of CIED-related TR.[27,29,36]

Clinical advice

TTE is the diagnostic imaging modality of choice for the diagnosis and severity grading of CIED-related TR	
3D TTE is the preferred imaging modality for assessing the mechanism(s) of TR in CIED recipients	
In patients with suboptimal transthoracic acoustic windows, 3D TOE is advised	

Pacemaker and pseudopacemaker syndrome

The hallmark of pacemaker and pseudopacemaker syndrome is suboptimal atrioventricular (AV) synchrony or AV dyssynchrony that may result in exercise intolerance, fatigue, dyspnoea, pulsations in the neck, dizziness, hypotension, presyncope, and syncope.[37] AV dyssynchrony can be observed in patients with AV blocks, including severe first-degree AV block, before pacemaker insertion (which is termed pseudopacemaker syndrome), but also after pacemaker implantation (pacemaker syndrome). Pacemaker syndrome is most commonly seen in the setting of a single-chamber RV lead and may require upgrading to dual-chamber pacing to restore AV synchrony.

The haemodynamic consequences of AV dyssynchrony in pacemaker and pseudopacemaker syndrome can be assessed by Doppler echocardiography. Regardless of aetiology, when atrial

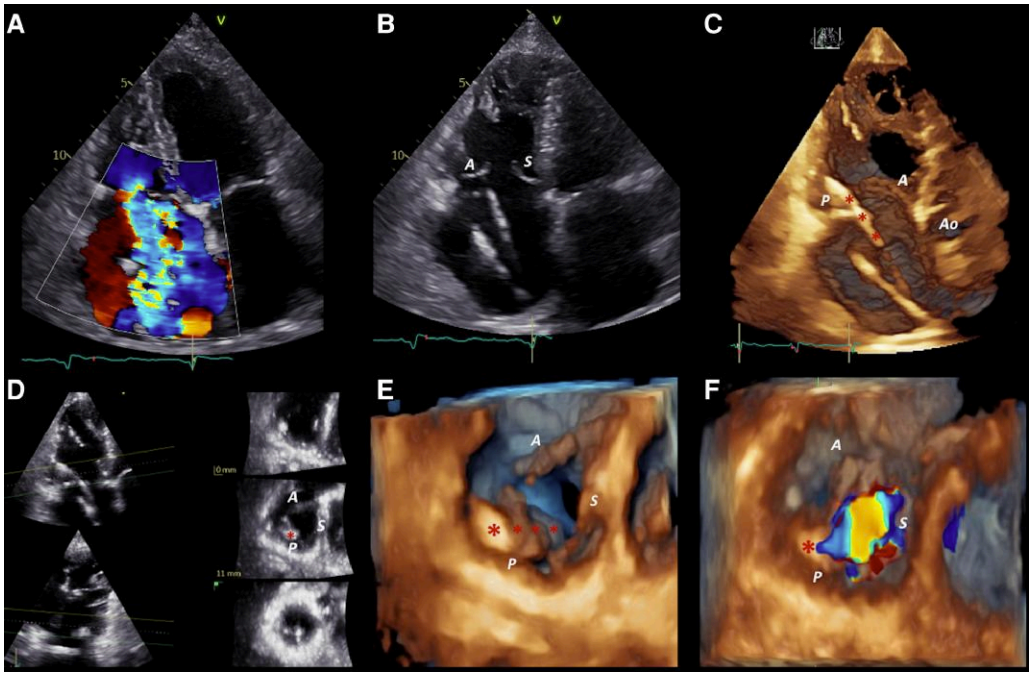

Figure 7 Added value of transthoracic three-dimensional echocardiography for visualizing the precise location of the pacemaker lead and identifying CIED-related tricuspid regurgitation. (*A*) Colour Doppler imaging showing massive TR in a patient with right-sided heart failure and pacemaker implanted 8 years ago. (*B*) Two-dimensional four-chamber view, showing large coaptation gap of the tricuspid leaflets and atrial and ventricular leads; the spatial relationship between the latter and the leaflets is difficult to appreciate in this view; (*C*) three-dimensional echocardiographic visualization of the right chambers, in which the view was oriented in order to display the posterior leaflet (not visible in standard apical four-chamber). Note that the depth perspective allows a much better appreciation of the trajectory of both atrial and ventricular (red asterisks) leads, and of the close contact between lead and posterior leaflet; (*D*) multi-slice display of three-dimensional data set, allowing to better appreciate that the lead was positioned in the middle of posterior leaflet, rather than in a commissure. (*E*) *En face* view of the tricuspid valve from the ventricular perspective that shows a large coaptation gap and a thickened pacemaker lead (red asterisks) with fibrotic adherences to the posterior leaflet that shows restricted mobility, confirming the CIED-rated TR diagnosis. (*F*) Colour 3D acquisition, showing a very large vena contracta area corresponding to massive TR. A, anterior; P, posterior; S, septal leaflet; Ao, aortic valve.

contraction is not followed by ventricular contraction, the increase in ventricular diastolic pressure after atrial contraction leads to diastolic AV pressure gradient inversion and diastolic tricuspid and mitral regurgitation (*Figure 10*). Furthermore, when the P-wave falls onto the preceding T-wave (as in severe first-degree AV block), the right atrium contracts simultaneously with the right ventricle against a closed TV, resulting in back pressure to the venous system that can be seen as cannon A-wave in the jugular venous pulse. Similarly, inappropriate timing of left atrial contraction in patients with pseudopacemaker syndrome causes a decrease in forward and an increase in reverse pulmonary venous flow.[38]

Clinical advice

Doppler echocardiography is the method of choice for assessing the haemodynamic consequences of AV dyssynchrony in patients with suspected pacemaker syndrome

Pacing-induced cardiac remodelling and mitral regurgitation

Right ventricular pacing induces QRS widening and asynchronous electrical activation of the LV. As discussed in part 1, only patients with hypertrophic cardiomyopathy and LV outflow tract obstruction may benefit from pacing-induced asynchronous septal contraction and LV remodelling. In all other patient populations, this abnormal electromechanical pattern is potentially detrimental, as it may cause less efficient LV contraction and result in LV remodelling, systolic dysfunction, functional MR, and HF.[39–41] Pacing-induced cardiomyopathy (PICM) has been defined as either ≥10% decrease in left ventricular ejection fraction (LVEF) resulting in LVEF < 50%, or a drop in LVEF to <40% following pacemaker implantation providing that alternative causes of cardiomyopathy are excluded (*Figure 11*).[39–42] The incidence of PICM may reach 25% and it is more likely to occur when RV pacing burden exceeds 20%.[39] Since CRT response rate in PICM is similar to that of *de novo* implantations,[43,44] clinical and echocardiographic surveillance of patients at risk of high RV pacing burden (e.g. chronic AV block) is worthwhile. Therefore, it seems prudent to reassess LVEF before a pacemaker generator change in patients with a significant proportion of RV pacing; according to guidelines, those with HF symptoms and LVEF ≤ 35% despite optimal medical therapy should be considered for upgrade to CRT.[45]

On the other hand, imaging predictors of PICM to justify up-front implantation of CRT in patients with advanced AV block and preserved LVEF have not been identified. Furthermore, conduction system pacing (His bundle and left bundle branch pacing) is being increasingly adopted to overcome PICM by providing a more physiological means of stimulation.[46,47]

Alternative imaging modalities may be required to exclude alternative causes of deterioration in LV function following pacemaker implantation, including cardiovascular MRI to detect new myocardial infarction or inflammation and CT coronary angiography (CTCA) for coronary assessment.

Functional MR after pacemaker implantation may occur despite preserved LV function due to papillary muscle dyssynchrony, and also as a late consequence of pacing-induced LV remodelling with papillary muscle displacement and distortion of the mitral apparatus.[48,49] Unfrequently, acute severe MR may develop as an immediate post-operative complication of pacemaker implantation (*Figure 12*). In a few published cases, patients with preserved LV systolic function and normal mitral valves experienced acute haemodynamic deterioration due to severe MR that subsided when the pacemaker was reprogrammed to allow restoration of intrinsic rhythm.[50,51]

The ability of chronic RV apical pacing to induce or worsen MR in the absence of LV remodelling has been observed in prospective and retrospective studies with short- and mid-term follow-ups.[48,49] MR may occur after implantation of either conventional or leadless pacemakers and is usually moderate.[24,49] While patients with mitral annular dilation and lengthening of the anterior leaflet might be more likely to develop post-implantation MR,[52] the relationship between MR and RV apical pacing is heterogenous and pre-implantation predictors of MR development are yet to be identified. In addition, in the setting of a prolonged AV interval, diastolic MR can occur and interfere with LV filling (*Figure 10*). Functional MR may also contribute to left atrial enlargement, fibrosis, and dysfunction providing a substrate for atrial fibrillation.[53] Finally, RV remodelling following PM implantation may occur due to volume overload from significant TR or secondary to LV dysfunction.

Clinical advice

- Echocardiography is the method of choice for assessing pacing-induced cardiac remodelling and mitral regurgitation
- In patients with a significant proportion of RV pacing, it is advised to reassess LVEF before a pacemaker generator change
- CMR and/or CTCA may help exclude alternative causes for a deterioration in LV function following pacemaker implantation

Central vein stenosis and obstruction

One of the major unwanted effects of CIED therapy is the lifelong occupation of the central veins by device leads. The reduction of functional room within the vein, the physical rubbing of the lead against the vein wall, and the possible turbulence of venous flow coupled with a patient's other co-morbidities and medication may promote vein thrombosis in the short-term after lead(s) implantation, as well as stenosis or complete occlusion in the long-term. Subclavian or axillary vein thrombosis soon after implantation is a rare event occurring in 0.5–1% of patients; it may manifest clinically as a swollen arm within weeks to months and is managed similarly to other causes of proximal vein thrombosis with anticoagulation.[54–56] The prevalence of long-term subclavian or brachiocephalic vein stenosis or occlusion is very high, although clinically asymptomatic and mostly unrecognized in the majority of cases. When looked for systematically, it is detected in ~40% of patients, a prevalence that has not changed over 30 years despite the evolution in lead manufacturing.[54,57] However, severe central venous stenosis due to fibrous tissue encapsulating the lead(s) occurs in 11–20% of patients.[54,57,58] In a systematic investigation on 184 consecutive asymptomatic CIED recipients, the brachiocephalic vein was the most frequently involved (20% of patients), followed by the subclavian and axillary veins.[57] The presence of multiple leads and an ICD lead seems to be the strongest predisposing factors to central vein stenosis/occlusion.[57,58] Although asymptomatic in the majority of cases, central vein stenosis/occlusion becomes a severe clinical issue in several scenarios: (i) superior vena cava (SVC) obstruction and SVC syndrome; (ii) need for device upgrading or for lead addition owing to malfunction; (iii) lead extraction procedures; (iv) inability to supply a high blood flow in the setting of dialysis via an ipsilateral arterio-venous fistula; and (v) loss of entry opportunity for central catheters.

The slow process of lead encapsulation and vein stenosis/occlusion allows the development of effective collateral flow over time that explains why many patients remain asymptomatic. However, patients frequently have clinical and imaging signs of a collateral circulation at ultrasound, venography, contrast CT scanning, and often also upon physical examination with the presence of engorged external jugular and subcutaneous veins mimicking the caput medusae appearance (*Figure 13*).

All imaging methods are sensitive in detecting a collateral circulation,[57–59] which is a highly specific sign of vein stenosis.[57] Peripheral venography and CT are superior in diagnosing brachiocephalic vein occlusion, as this site is not accessible to ultrasound. CT venography is particularly helpful to detect the site and extent of central vein occlusion, the coexistence of an occluded superior or inferior vena cava, and the roadmap of the collateral circulation (*Figures 13* and *14*).

A key issue in CIED recipients is the assessment of subclavian and brachiocephalic patency when an additional lead needs to be inserted. This is typically assessed pre- or peri-operatively by ultrasound with Doppler evaluation of venous flow, peripheral venography, CT venography, or direct venography via the axillary vein. The impact of these imaging assessments of vein patency is not negligible. If a vein is incorrectly thought to be occluded, patients will be denied a feasible and relatively simple procedure and instead undergo a technically demanding and rather riskier procedure (contralateral access and tunnelization, lead extraction to gain patency, epicardial lead placement, leadless system implantation).[60,61] On the other hand, assessment of residual vein patency in patients with severe brachiocephalic or subclavian vein stenosis is a very difficult task, owing to the low flow of blood across the stenosis and its preferential shift to the high-flow collateral circulation (see Supplementary data online). In this setting, ultrasound, contrast-enhanced CT venography, and peripheral venography all underestimate residual vein patency (see Supplementary data online). The preferred method to rule out complete occlusion and assess vein patency is therefore direct venography from the axillary or antecubital vein, which enables detection of contrast flow across nearly occluded veins and can help guide the placement of a guidewire into the right atrium (see Supplementary data online). This approach can also assess feasibility for balloon

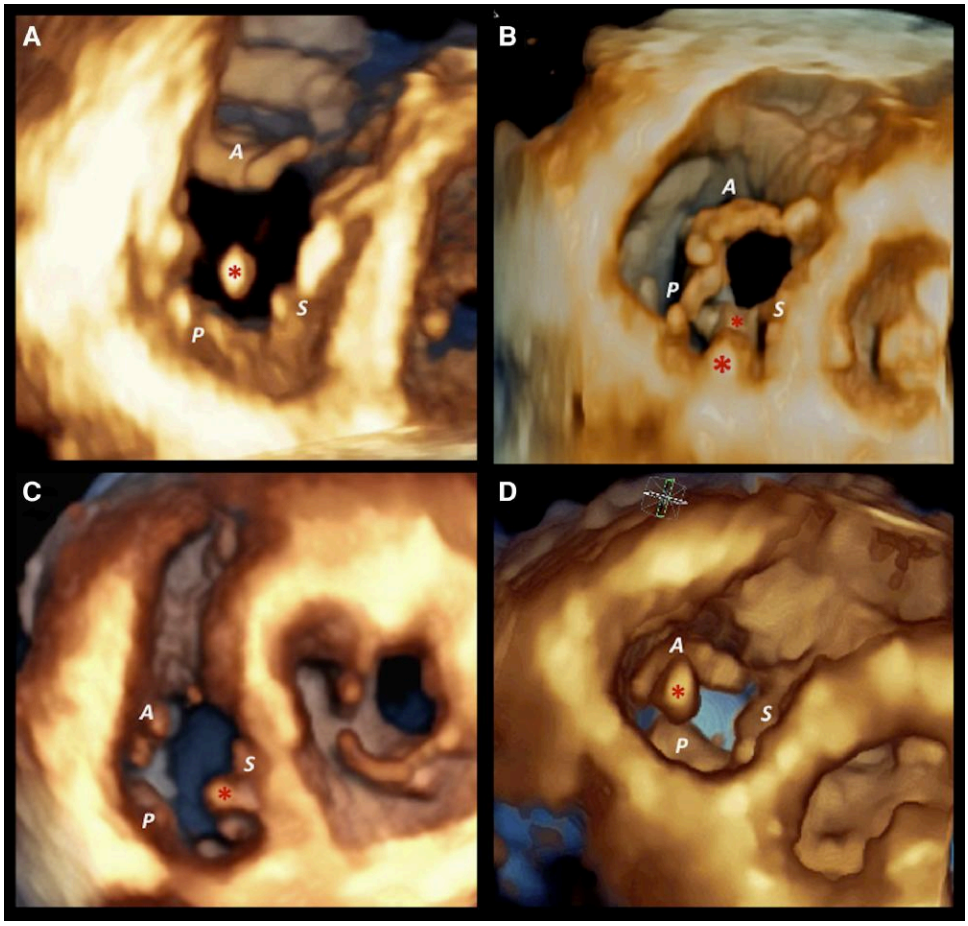

Figure 8 Different positions of the pacemaker leads at the level of the tricuspid valve, illustrated by transthoracic three-dimensional echocardiography (ventricular perspective). In the first two examples (*A*,*B*), the lead was not involved in the TR mechanism, while in the (*C*) and (*D*), the TR was lead-induced. (*A*) Central; (*B*) postero-septal commissure; (*C*) septal leaflet impingement; (*D*) anterior leaflet encapsulation.

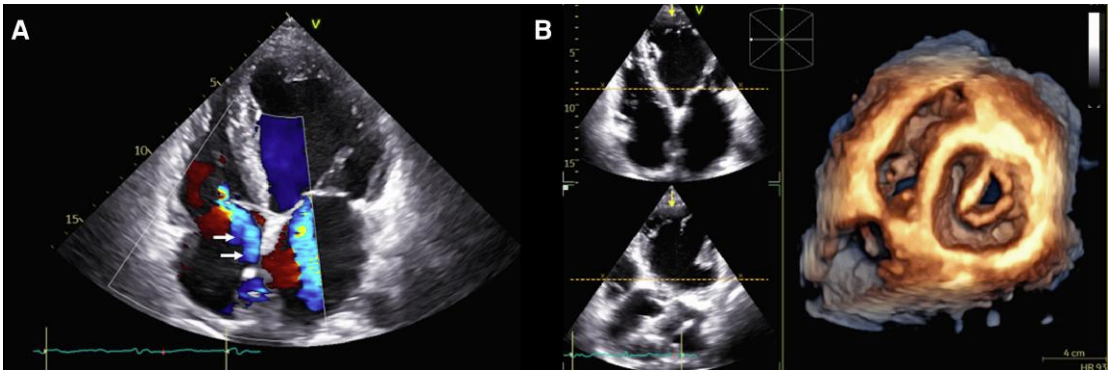

Figure 9 CIED-induced tricuspid regurgitation. (*A*) Eccentric TR jet oriented towards the interatrial septum (arrows), which in the presence of pacemaker lead crossing the tricuspid valve, is highly suggestive for CIED-induced mechanism of TR. (*B*) Three-dimensional transthoracic acquisition from the apical approach in order to confirm the CIED-induced mechanism. Note that in this example, the default cropping plane position (dashed lines) is not optimal, being too far in the ventricle and not parallel with tricuspid annulus, making the interpretation of the lead position challenging and potentially misleading. From this perspective, the lead seems rather centrally located, while after optimal cropping plane alignment the lead was seen abutting in the middle of the septal leaflet.

venoplasty[60] that can then lead to safe additional lead implantation (see Supplementary data online).

In summary, a haemodynamically significant vein stenosis is confirmed by imaging or clinical evidence of a collateral circulation. Residual lumen patency in a severely stenotic vessel needs to be assessed by direct venography via a proximal vein. Contrast-enhanced CT is helpful to detail the site and extent of vascular occlusion in patients with vena cava syndrome and for subsequent procedure planning.[60,61]

Clinical advice

Doppler ultrasound, peripheral venography, CT, or direct venography is advised to assess CIED recipients with suspected central vein obstruction or stenosis	
The preferred method to rule out complete occlusion and assess vein patency is direct venography from the axillary or antecubital vein	

CRT optimization

Echocardiography has historically been regarded as key technique to guide CRT optimization but it has fallen into disfavour due to the lack of evidence that it improves long-term patient outcomes compared with ECG-guided programming.[62] Although most current CRT devices have automatic optimization algorithms, they differ in their design and do not yield optimal settings in all patients. Most algorithms are based upon intracardiac electrograms, using either the right-sided intrinsic atrioventricular interval (AVI), which may not reflect left-sided delays, and/or an estimation of P-wave duration based upon the unipolar atrial signal, which may be imprecise. Therefore, it is useful to check the ECG after employing the programmed settings suggested by these algorithms, in order to verify a narrow paced QRS (ideally, with a 'physiological' rS or QS complex in V1)[62] that is associated with favourable outcomes.[62–64] Furthermore, it is advised that ventricular pacing is delivered ~40 ms after the end of the P-wave in order to avoid A-wave truncation[65] that can cause problems in patients with interatrial conduction delay (P-wave duration > 120 ms, see *Figure 15*).

While routine echocardiographic optimization is not necessary, post-operative echocardiography may be appropriate in selected cases to screen for A-wave truncation.[66,67] If a recent pre-implantation echocardiogram is available, it can be appropriate to compare transmitral

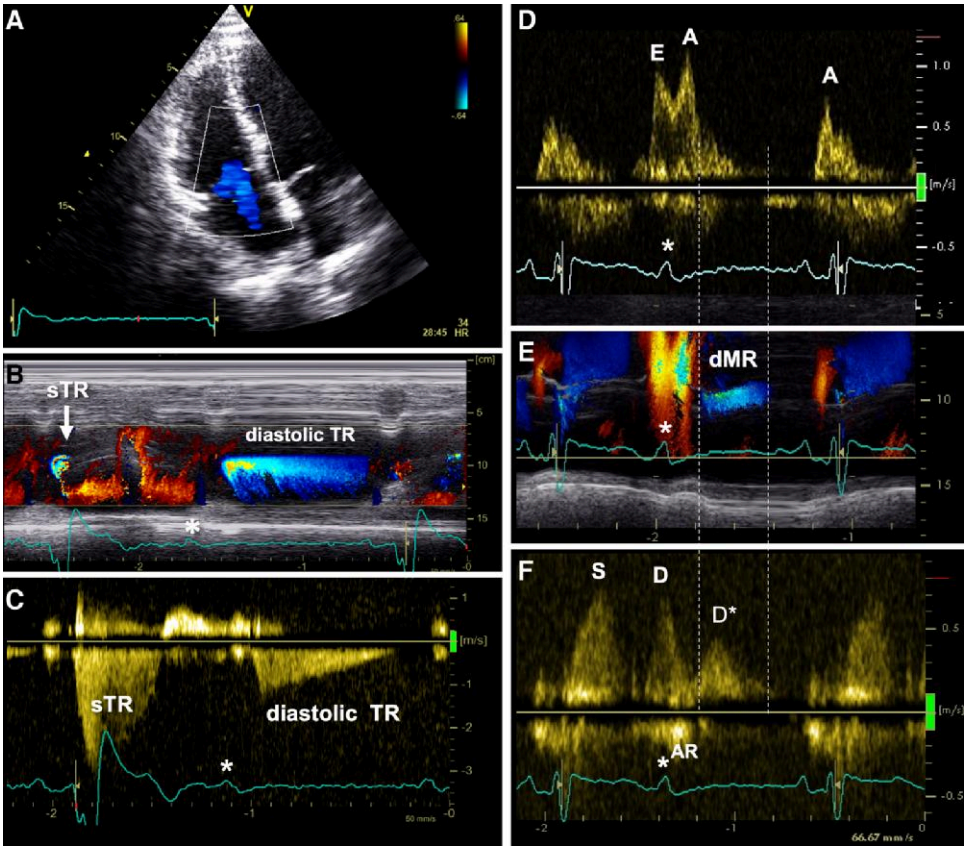

Figure 10 Loss of atrioventricular (AV) synchrony in a patient with second degree heart block resulting in diastolic AV pressure gradient inversion and diastolic mitral (MR) and tricuspid regurgitation (TR). Left: the appearance of systolic (sTR) and diastolic TR on colour Doppler (*A*), colour M-mode (*B*), and continuous-wave Doppler (*C*) echocardiography. Right: the pulsed wave Doppler (PWD) recording of mitral inflow showing partial fusion of E- and A-wave resulting from AV block (*D*). (*E*) Colour M-mode showing diastolic MR (dMR). (*F*) PWD recording of pulmonary venous flow showing systolic (S) and diastolic (D) forward flow, atrial reversal (AR), followed by a second diastolic pulmonary venous forward flow (D*) occurring in parallel with dMR (as indicated by vertical dashed lines). *Denotes the unconducted P-wave.

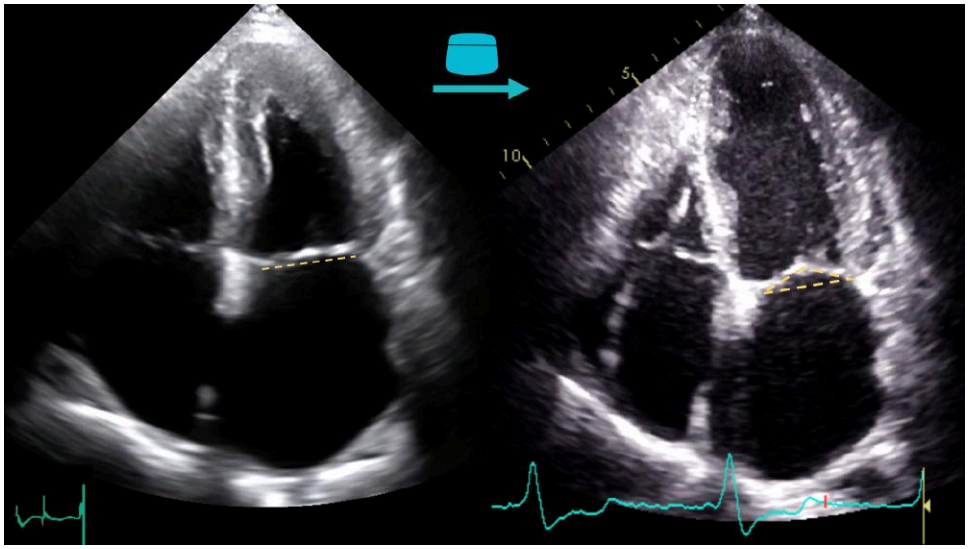

Figure 11 Pacing-induced cardiac remodelling. The apical four-chamber views (end-systolic frames) immediately before (left) and 8 months after permanent pacemaker implantation (right). Note the increase in end-systolic left ventricular volume and mitral leaflet tethering after device implantation.

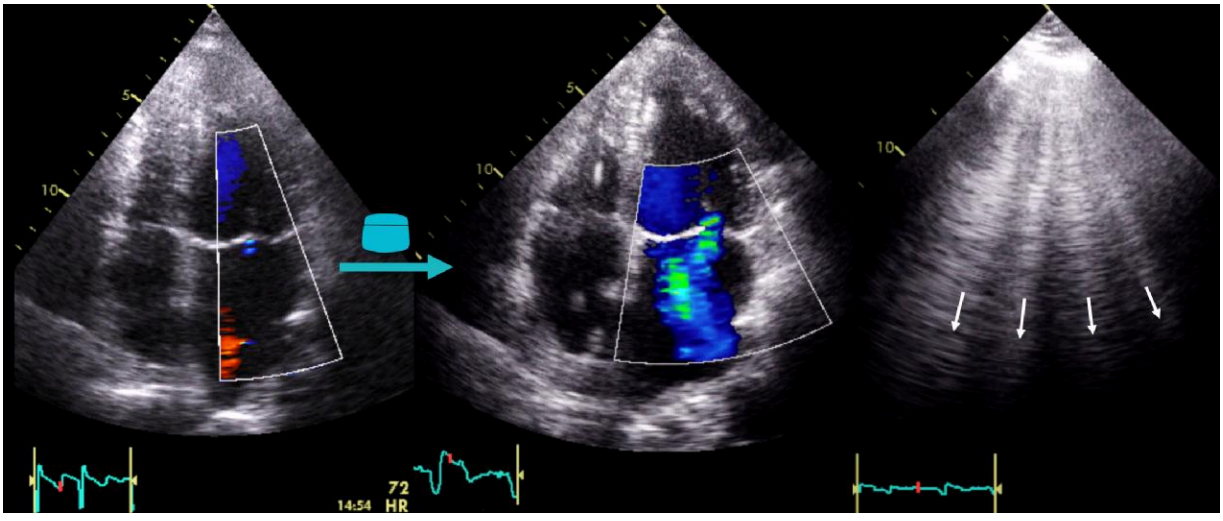

Figure 12 Pacemaker-induced mitral regurgitation (MR) and heart failure. A patient with sick sinus syndrome had trace MR prior to permanent pacemaker implantation (left). A few days after the implantation, the patient presented with signs of heart failure, while echocardiography revealed significant MR (middle) and ultrasound lung comets (arrows, right).

flow velocity patterns, which can facilitate identification of A-wave truncation, as this is not always apparent. A pragmatic strategy for post-operative CRT optimization is shown in *Figure 16*. There is general consensus that echocardiography is useful in CRT non-responders, as suboptimal AV delays are a frequent cause for poor outcome, which can be improved if optimization is feasible.[68]

Atrioventricular interval optimization

The iterative method is the simplest to use. This method aims at maximizing diastolic filling time, while at the same time avoiding A-wave truncation. First, the intrinsic conducted AVI is measured using the device electrograms. A long AV delay is then programmed (e.g. intrinsic AVI −40 ms), and the AVI is decremented in 20 ms steps until the A-wave becomes truncated. The AVI is then increased in 10 ms steps. This defines the shortest programmable AVI without A-wave truncation. Further adjustments may then be performed using the 12-lead ECG aiming to provide the narrowest QRS complex. This combined ECG-echocardiography approach is preferable to optimizing AVI by echocardiography alone, due to limited inter-observer agreement.[69] An example of the iterative method to define the minimum AVI is shown in *Figure 17*.

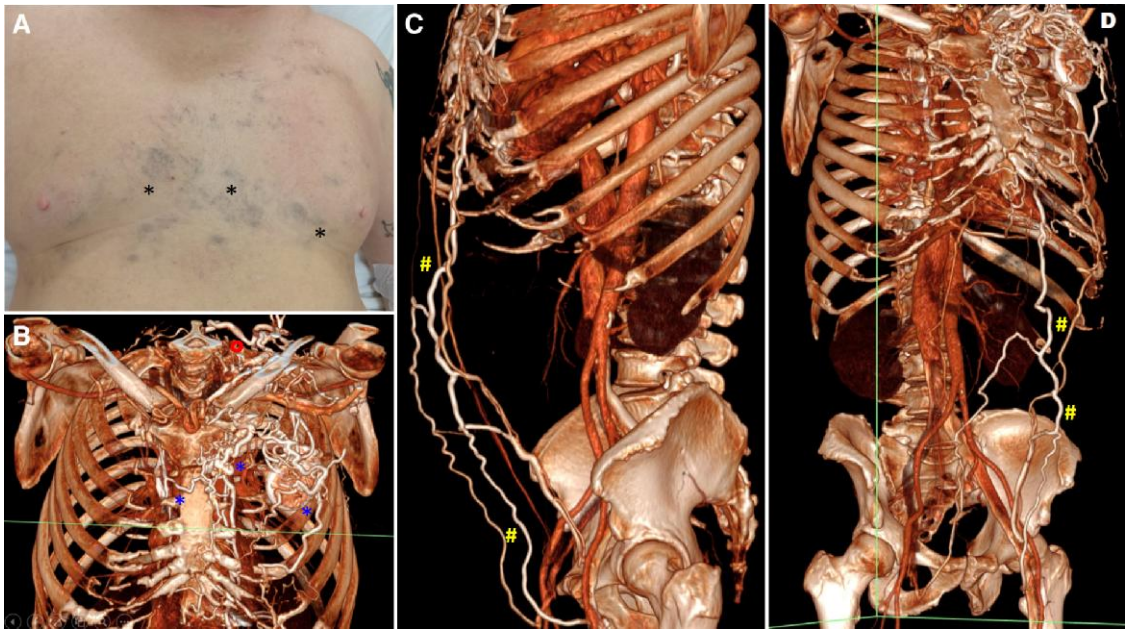

Figure 13 Superior vena cava syndrome in a patient with a non-functional dual-coil implantable cardioverter defibrillator lead. (*A*) Evidence of collateral circulation via subcutaneous and parietal engorged veins (***) from the superior to inferior vena cava at physical examination. (*B*) Absence of visible brachiocephalic veins and superior vena cava at computed tomography scan; (*) subcutaneous collateral circulation mimicking caput medusae; (°) posterior thoracic collaterals heading to the vertebral circulation. (*C,D*): evidence of parietal collateral circulation (#), both thoracic and abdominal, heading to the inferior vena cava.

Interventricular interval optimization

In general, changes in programmed AVI have a much greater haemodynamic impact than changes in VV delays.[70] Sequential biventricular pacing may, however, be useful in patients who display latency with LV pacing, which can be readily identified by analysing the ECG (presence of an isoelectric interval before the QRS with LV-only pacing, and QRS morphology with biventricular pacing resembling RV pacing).[64,71] Both aortic velocity time integral and dyssynchrony measurements have been used to optimize the VV delay, but their reproducibility is limited in the clinical setting[72] and randomized studies have not shown any benefit.[73,74] On the other hand, it has been shown that persistence of mechanical dyssynchrony after CRT implantation is strongly related to worse outcome.[75] It should therefore trigger a careful check of device function and, if appropriate, revision or optimization.

Radionuclide angiography has also been used and while it may be more reproducible than echocardiography, this technique has limited temporal resolution, limited feasibility regarding iterative optimizations, and utilizes ionizing radiation.[76]

Gaps in knowledge

As discussed in part 1 of this document, the 2021 ESC guidelines underlined several uncertainties regarding the use of imaging in CRT patient selection, the optimal choice of CIED for each patient, and also whether the use of any type of pre-implantation imaging in deciding about the placement of LV and RV electrodes in CRT may result in a better patient outcome.[45] It should also be noted that optimizing CRT programming using cardiac imaging has not been sufficiently explored. Furthermore, the vast majority of clinical studies investigating the acute and chronic effects of pacing delay optimization has only considered the effects on LV function and largely ignored the right ventricle. Recent pre-clinical research demonstrated that the left and right ventricles respond differently to changes of AV and VV delays.[77] LV pre-excitation improved LV contractility and decreased RV contractility, while RV pre-excitation had the opposite effects. Given the serial and mechanical coupling of the left and right hearts, LV filling is highly dependent on RV pump function. RV function has been shown to improve with CRT to a lesser extent than LV function.[78] To clarify the potential relevance of RV function in the context of pacing delay optimization, future clinical studies should follow a more integrative approach including imaging of both LV and RV functions.

Clinical advice

ECG analysis is advised to screen for suboptimal programming (ventricular pacing on or <40 ms after the end of the P-wave, and absence of QRS narrowing)

Post-operative echocardiography is useful to assess for A-wave truncation and to guide AV interval optimization in CRT non-responders

A combined ECG-echocardiography approach is preferable to optimizing AV interval by echocardiography alone

Safety of MRI in CIED carriers

Annually, the number of MRI examinations performed worldwide expands in parallel with the number of CIED recipients. Given the

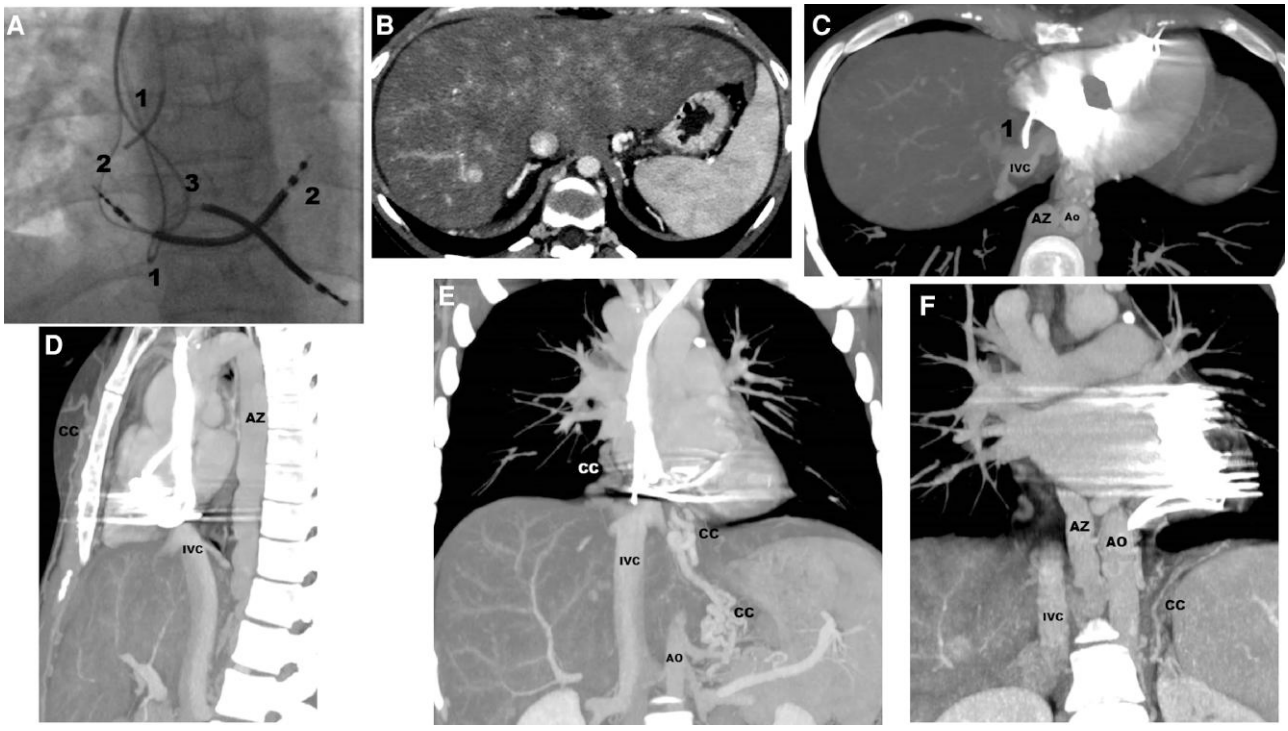

Figure 14 Inferior vena cava syndrome. A young patient with long QT syndrome, treated with a dual-chamber implantable cardioverter defibrillator (ICD) after ventricular fibrillation. The non-functional ICD lead is prolapsing in the inferior vena cava (*A*), and the patient was referred because of post-hepatic portal hypertension and liver abnormalities (*B*). A fibrotic ingrowth by the inferior vena cava encapsulated the lower ICD lead 1 loop causing vein obstruction (*C*), while ICD lead 2 and lead 3 crossed over the tissue ingrowth in the lower right atrium. Collateral circulation via paracardiac, phrenic, gastric, and vertebral veins developed heading to the superior vena cava via a massive enlargement of the azygos vein (*C–F*). Ao, aorta; AZ, azygos vein; CC, collateral circulation; IVC, inferior vena cava.

mean age of implanted patients, if medical recommendations were strictly applied, 50% of CIED carriers are likely to require at least one MRI examination during the device's life expectancy.[79] It is therefore of utmost importance that cardiologists and imaging specialists work to ensure that patients are not denied clinically warranted MRI scans for specious safety concerns or for logistical or reimbursement reasons. However, in reality provision of MRI to CIED patients remains poor, with barriers at many levels—from referrers failing to request scans, to many radiology departments declining to scan patients with devices.[80] This situation is partly the consequence of a longstanding contraindication due to historic concerns about the potential risks of MRI related to the generator (hardware or software damage), the leads (lead failure or lead-related tissue overheating), and induction of arrhythmias.[81] Other causes include limited or unavailable monitoring resources, the absence of specific reimbursement tariffs to reflect the required complex imaging protocols, and a lack of support/collaboration from cardiology to accommodate the device re-programming needed before and after scans.[82]

In this context, MRI-conditional devices have been specifically developed in order to improve the access of CIED carriers to MRI. According to the ESC guidelines, patients with MRI-conditional devices can undergo MRI safely provided that the manufacturer conditions are adhered to, including both those related to the device (hardware and programming considerations) and radiology (MRI strength, patient positioning, sequences, etc.).[45] In parallel, several large studies have more recently demonstrated that the risk of MRI in patients with non-MR-conditional (also termed 'legacy' or 'MR-Unlabelled') devices is low, provided that safety protocols are followed.[83–87] In current practice, the majority of patients with a CIED can therefore be imaged with MRI at 1.5 T, although scanning of patients with non-MR-conditional devices is generally performed in specialist centres.

Recent international guidelines and consensus documents[45,88–91] propose workflows in line with manufacturer recommendations and recent clinical data. Two main workflows have been established depending on the MR conditionality of the device (*Figure 18*). For both MRI-conditional and non-MRI-conditional devices, each institution should develop local protocols with involvement from cardiology, radiology, and medical physics. Any MRI request should highlight the presence of a CIED and provide the manufacturer and model of the generator and each of the leads. MR conditionality can then be assessed from manufacturer look-up tables, ensuring that all components are considered together and form part of an MR-conditional 'system'. On the day of the scan, according to the ESC guidelines, all patients require CIED interrogation and programming to 'MRI mode' for MR-conditional devices, while non-MR-conditional devices are programmed to pacing off (ODO/OVO) or asynchronous pacing (DOO/AOO/VOO) with ICD therapies also programmed off.[45] This requires support from cardiac physiologists and/or cardiologists. Some models of MRI-conditional CIEDs can automatically switch to a MRI mode in the MRI environment and return to initial settings after the MRI scan.[92]

During the scan, it is advised to monitor patients using an MR- conditional monitoring system, with at least continuous pulse oximetry waveform and ideally ECG monitoring. It is also advised that personnel able to provide advanced cardiac life support is available in the hospital

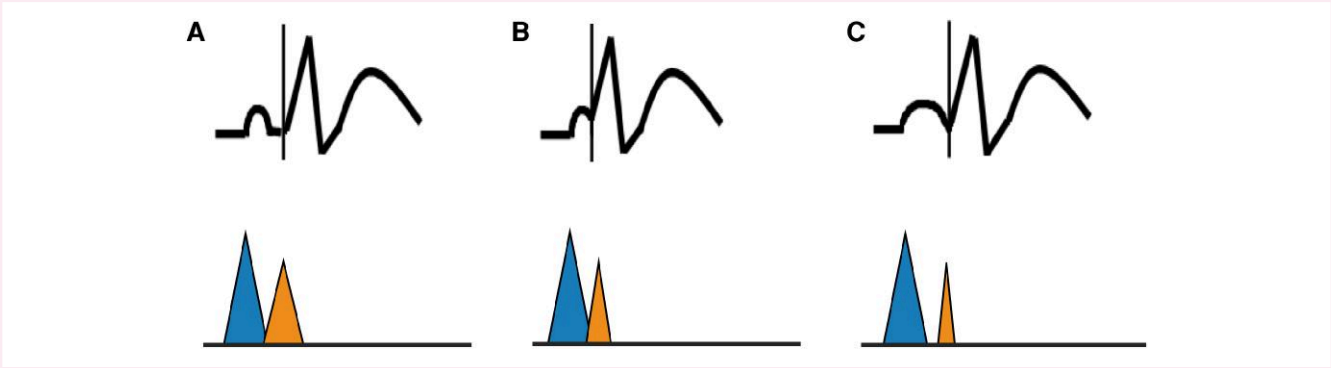

Figure 15 Effects of programmed atrioventricular (AV) delay and interatrial conduction delay on transmitral flow pattern. (*A*) Adequately timed biventricular pacing after the end of the P-wave with normal A-wave. (*B*) Excessively short programmed AV delay with truncation of the A-wave. (*C*) Interatrial conduction delay (P-wave > 120 ms), resulting in delayed left atrial contraction with truncation of the A-wave by delivery of ventricular pacing.

at the time of the scan, alongside a healthcare professional able to interrogate CIEDs. It is advised that the radiologist and MRI technicians ensure that manufacturer recommendations regarding patient positioning, scanner strength, and specific absorption rate are followed, prescribing the minimal number of pulse sequences as well as minimizing scan duration.

Similar to non-cardiac MRI, CMR is also possible after device implantation and can be helpful in various clinical scenarios (e.g. in clarifying the aetiology of LV dysfunction after implantation of a pacemaker). In cases of cardiac or chest MRI examinations, specific MRI techniques may be required to mitigate the impact of metallic artefacts arising from the generator and leads (more problematic with ICDs).[93] These include the use of gradient echo cine imaging and late gadolinium enhancement imaging using sequences with a wideband inversion pulse.[93,94] Following the scan, patients are reprogrammed back to their initial settings, and followed up as usual in the CIED or cardiology clinic. Experience shows that a dedicated form that documents the patient journey during this workflow is useful in providing caregivers with the appropriate information at all steps.

For non-MR-conditional devices, there are additional precautions advised, including obtaining confirmation from the referrer that an alternative imaging modality could not answer the clinical question. Once this possibility has been excluded, patients should be informed of the risks and benefits of undergoing MRI, including formal written consent. The clinical indication for the CIED, pacing dependence, and any history of ventricular arrhythmias should all be considered as well as the device type. There is clearly a spectrum of risk of MRI with non-MR-conditional CIEDs,[91] and advice may be required from the patient's cardiologist regarding individualized risk. For patients with MR-conditional generators but non-MR-conditional leads (such as following generator change following battery depletion for older devices), or with manufacturer mismatch for MR-conditional generator and leads, the risk has been shown to be negligible.[87] There is also emerging safety data for MRI in patients with abandoned/fractured/epicardial leads,[95–97] where, according to guidelines, decisions should be made on an individual basis after weighing the risks and benefits of MRI against the utility and availability of alternative imaging modalities.[45] CXRs are advised in doubtful cases as the presence of an abandoned or fractured lead is frequently underappreciated and not clearly documented.

It is advised that all patients with non-MR-conditional CIEDs are scanned at the lowest static magnetic field, typically 1.5 T, and personnel capable of re-programming the CIED should remain within the MRI department throughout the study in case of pacemaker-dependent patients, or otherwise on site.

The key to performing MRI in patients with CIEDs is effective communication between the physician in charge of the patient, the MRI team, the pacing team, and the cardiologist taking care of the patient before, during and after the examination.

Clinical advice

Local standard operating procedures should be in place for MRI in patients with CIEDs including guidance for pre-scan checks and device re-programming, monitoring, and supervision	
Manufacturer guidance should be followed when performing MRI in patients with MR-conditional CIEDs	
For patients with non-MR-conditional CIEDs, written informed consent should be obtained before MRI	
All patients with CIEDs require continuous ECG and pulse oximetry waveform monitoring throughout MRI scans, and personnel able to perform advanced life support should be available on site	
CMR in patients with CIEDs (especially ICD/CRTD devices) may require sequence adaptation including use of gradient echo cine imaging and wide band inversion pulse for late enhancement imaging	

Chest radiographs of cardiac devices

CXR plays a central role in assessing CIED type, their position, and associated complications.

Pulse generator

Position

The generator is usually positioned in the left or right infraclavicular region (*Figure 19A* and *B*). Other locations such as the abdomen, especially

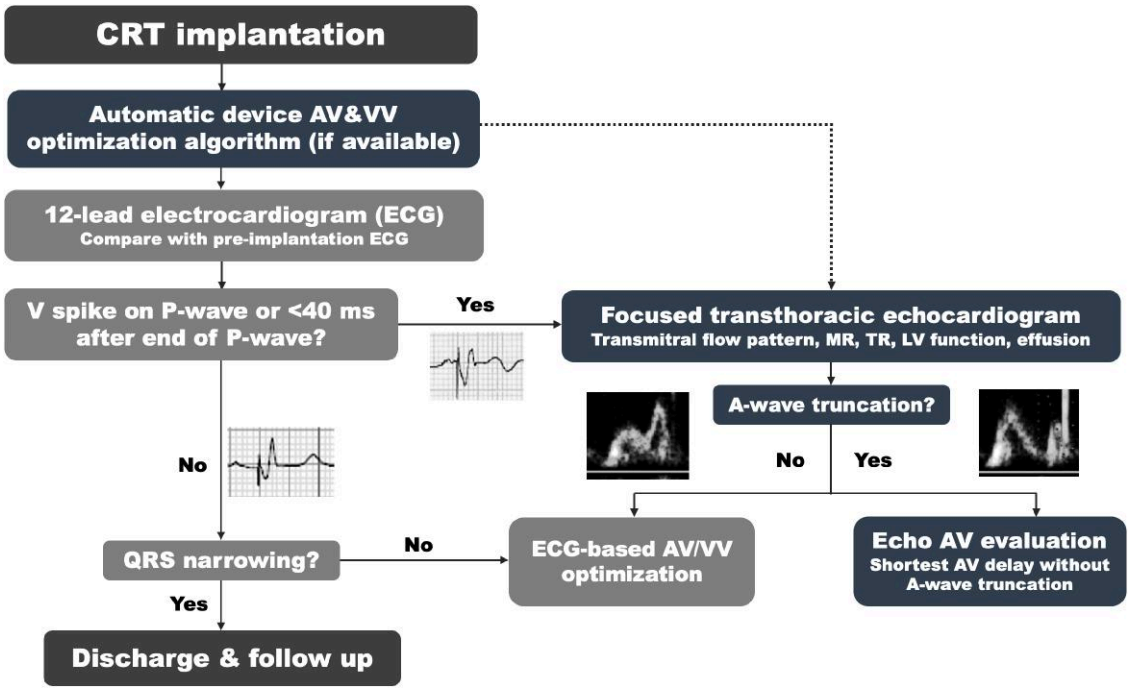

Figure 16 Algorithm for atrioventricular (AV) and interventricular (VV) optimization following cardiac resynchronization therapy (CRT) implantation. A routine focused echocardiogram may be appropriate after CRT implantation to screen for A-wave truncation, evaluate tricuspid regurgitation (TR), mitral regurgitation (MR), left ventricular (LV) systolic function with biventricular pacing, and presence of pericardial effusion.

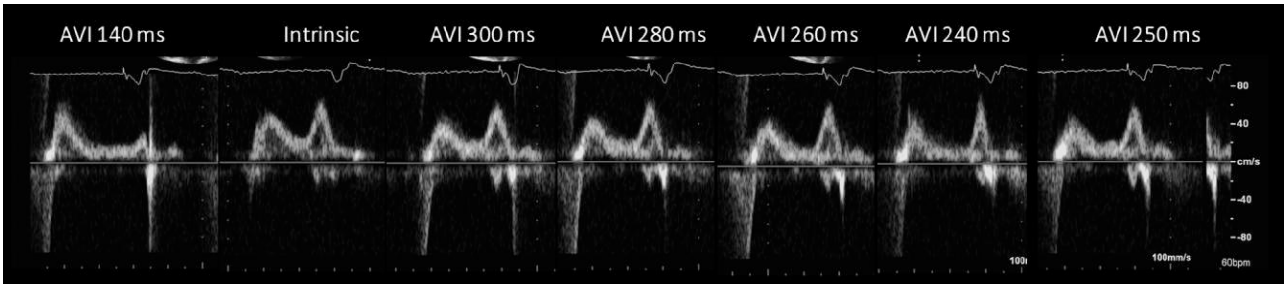

Figure 17 Iterative method for evaluating atrioventricular intervals (AVIs) in a non-responder to cardiac resynchronization therapy. From left to right: transmitral flow at 140 ms (the initially programmed AVI) showing A-wave truncation. Intrinsic rhythm with conducted AVI measured at 340 ms (note the large A-wave). Progressive shortening of the AVI from 300 to 240 ms in 20 ms decrements, showing narrowing (truncation) of the A-wave at 240 ms. Increase of the AVI to 250 ms, showing absence of A-wave truncation, thereby defining the shortest AV delay that may be programmed. Note the changes in QRS morphology (the 12-lead electrocardiogram showed the narrowest QRS at 280 ms, which was finally programmed, with clinical improvement of the patient).

in paediatric patients, are also possible. Generators of subcutaneous implantable cardioverter defibrillators (S-ICD) are positioned in the left mid-axillary line (*Figure 19C*), with more anterior positions being associated with high defibrillation thresholds (an additional reason being air in the pocket).

Pulse generators consist of a titanium casing that houses the electrical components and an epoxy connector block with set screws. It is essential that the connector pin of the CIED lead is properly advanced in the connector block as improper connection can cause sensing artefacts or loss of capture (*Figure 20A*).

Leadless pacemakers can be recognized as bullet-shaped devices positioned in the RV (*Figure 19E*). Atrial leadless pacemakers are being tested in pre-market studies at the time of writing of this document.

Identification of the manufacturer

There are different CIED manufacturers, each requiring a specific device programmer for interrogation. Although patients are provided with a CIED-identification card at the time of device implantation, they may not have it with them. A stepwise CIED-identification

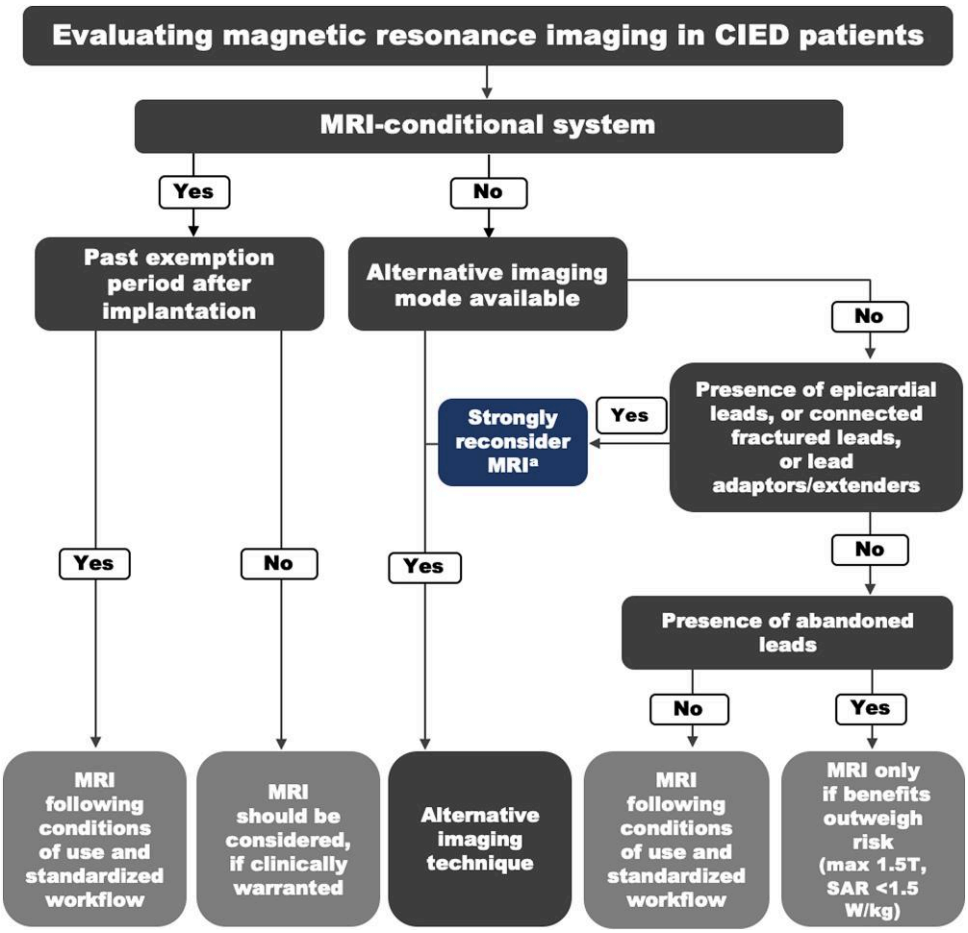

Figure 18 Flowchart for evaluating magnetic resonance imaging in CIED patients. CIED, cardiovascular implantable electronic devices; MRI, magnetic resonance imaging; SAR, specific absorption rate. [a]Consider only if there is no imaging alternative and the result of the test is crucial for applying life-saving therapies for the patient. Adapted from Glikson M, *et al.* 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. *Eur Heart J.* 2021;42(35):3427–3520 with permission.

algorithm (CaRDIA-X) has been developed to allow device identification based on chest radiography.[98] Artificial intelligence algorithms using CXRs have facilitated manufacturer identification with accuracies of 71–89%.[99] These algorithms are available as a mobile phone application (pacemakerID) or via a web platform (ppm.jph.am).

CIED leads

Lead position

To evaluate correct lead position, a good understanding of normal cardiac anatomy is required as well as an awareness of possible variants that may affect lead position (e.g. persistent vena cava superior and congenital heart disease—see *Figure 21G* and *H*). The preferred implantation site for the right atrial lead is the right atrial appendage. The distal part of the lead should have a J-shape to avoid tension at the lead tip during deep inspiration and arm movement (*Figure 21A* and *B*). The usual position of an RV lead is at the septum or apex. Alternative lead positions are the RV outflow tract or at the level of the His bundle or left bundle branch area (*Figure 22A*). LV pacing leads are inserted through the ostium of the coronary sinus (CS) into a suitable tributary vein. These leads do not cross the tricuspid valve and have a posterior orientation; because of their epicardial trajectory, they can be seen overlying the cardiac silhouette on lateral views (*Figure 21E* and *F*).

An anteroposterior, and, if possible, lateral chest X-ray is advised in all patients after lead implantation to evaluate presence of pneumothorax and lead position.[1] The RV lead may appear to be in a correct position on the AP view, but inadvertent placement through a patent foramen ovale into the LV (*Figure 21C* and *D*) or in the CS (*Figure 21E* and *F*) may only be apparent in the lateral view. Another route for inadvertent LV lead positioning is via unsuspected arterial puncture. The post-operative CXR should be carefully inspected for possible lead dislocation, reported in 1.2–3.3% of implantations[1] (*Figure 22A–C*).

For patients with an S-ICD, the lead is positioned in the left (or more rarely right) parasternal subcutaneous tissue (*Figure 19C*). The PRAETORIAN score identified three critical determinants that cause an increase in defibrillation threshold: (i) adipose tissue between the coil and the sternum (visible on the lateral chest X-ray); (ii) generator

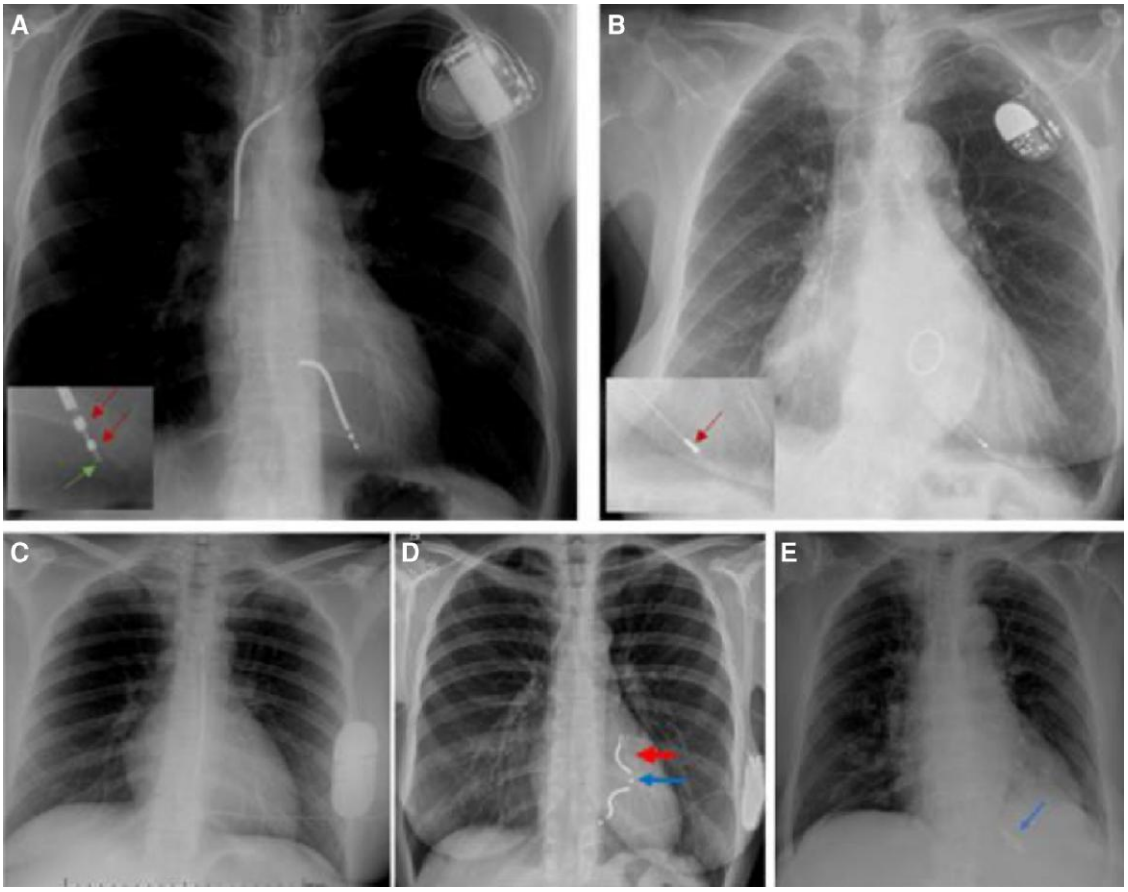

Figure 19 Examples of types of cardiac implantable electronic devices. (*A*) Single-chamber defibrillator in the left prepectoral region with a dual-coil, true bipolar, active-fixation implantable cardioverter defibrillator (ICD)-lead implanted at the right ventricular (RV) apex. Inlay: fixation screw (green arrow) and separate electrodes serving as anode and cathode (red arrows). (*B*) Single-chamber pacemaker in the left prepectoral region with a pacing lead in the RV apex. The inlay shows that the lead is unipolar with a single tip electrode (arrow) and has a passive fixation mechanism without a tip helix. (*C*) Subcutaneous ICD. (*D*) Extravascular ICD with a shocking coil (red arrow) and a recording electrode (blue arrow). (*E*) Leadless pacemaker in the RV apex (arrow).

malposition anterior to the mid-axillary line; and (iii) adipose tissue between the generator and the thorax.[100] Differently from the S-ICD, the extravascular ICD has a lead placed in the anterior mediastinum, substernally or at some distance from the left border of the sternum to ensure the optimal recording of the right ventricular signal. The lead is sigma-shaped, with two coils for defibrillation and two electrodes to detect the right ventricular signal and deliver antitachycardia pacing and pause prevention pacing (*Figure 19D*).

Lead design

Unipolar pacing leads (no longer in production) have the simplest design with a single distal tip electrode serving as cathode (and the generator as the anode, see *Figure 19B*). Bipolar pacing leads have a tip and an additional ring electrode (serving as anode, see *Figure 19A*).

An ICD lead consists of an RV shock coil and an optional proximal superior vena cava coil (*Figure 19A*). ICD leads are either true bipolar or integrated bipolar. True bipolar leads have a ring electrode similar to a pacing lead, used for sensing and pacing (*Figure 20B*). With integrated bipolar leads, the distal coil serves as the anode and there is no separate ring electrode (*Figure 20C*).

Passive fixation leads have radiolucent tines at their tip that anchor the lead (*Figure 19B*). An active-fixation lead has a helix at the lead tip (*Figures 19A* and *20B* and *C*).

LV pacing leads can be uni-, bi-, or quadripolar in design (*Figure 20D–F*). Most CS leads are simply wedged in one of the CS tributaries. The Medtronic Attain Stability™ lead is equipped with a fixation screw on the lead body, allowing for more options in lead placement reducing risk for lead dislodgement (*Figures 20D* and *21A* and *B*).

Lead integrity

Leads undergo substantial mechanical stress due to movement or direct pressure, with potential for metal fatigue and fracture (which is rarely visible radiologically). Direct mechanical stress can also cause insulation damage. It is imperative to be meticulous in examining the integrity of each lead along its entire course, since signs of lead damage are frequently subtle and may be seen only with image magnification.

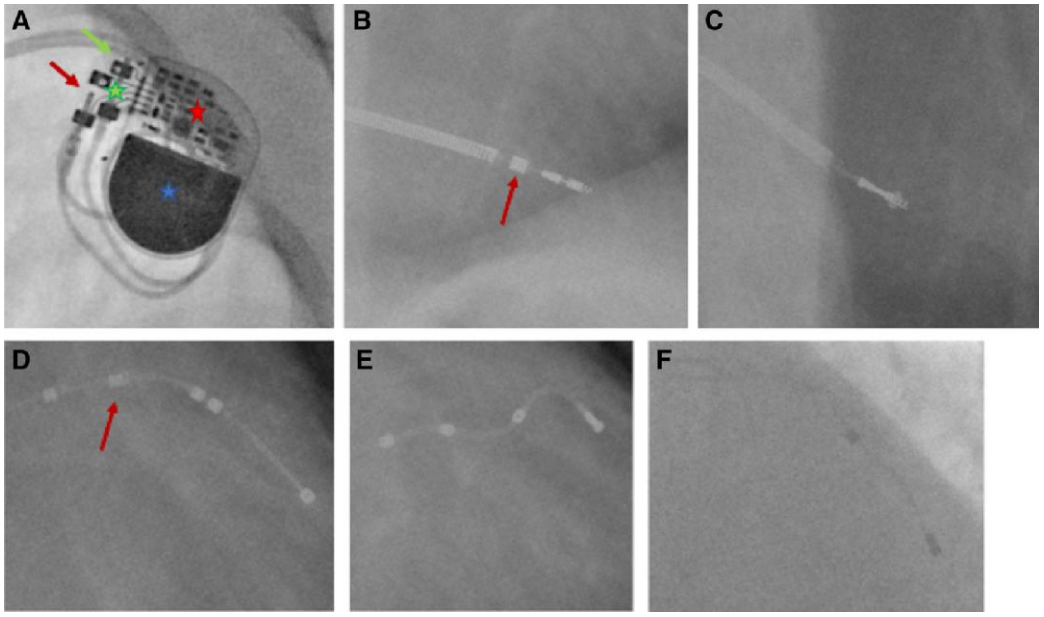

Figure 20 Detailed views of cardiac implantable electronic device components. Close-up of a dual-chamber pacemaker. The pacemaker battery (blue star), circuitry (red star), and connector block (green star) can easily be identified. The atrial lead is not properly inserted into the connector block (red arrow). The right ventricular lead is fully inserted in the header (green arrow). (*B*) True (or 'dedicated') bipolar defibrillation lead with a separate anode for detection (arrow) and an active-fixation mechanism (distal helix). (*C*) Integrated bipolar defibrillation lead with active-fixation mechanism (note absence of a proximal ring electrode). (*D*) Quadripolar left ventricular (LV) lead with active-fixation mechanism (screw, arrow). (*E*) Quadripolar LV lead, passive fixation (absence of a screw). (*F*) Bipolar LV lead with passive fixation.

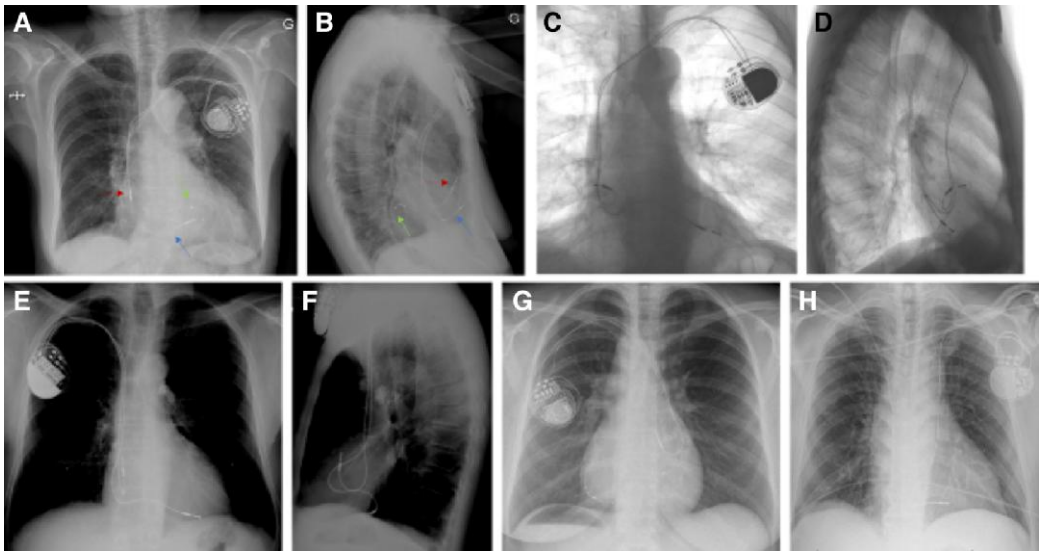

Figure 21 Evaluation of lead position. (*A,B*) Biventricular pacemaker. Bipolar, active-fixation atrial lead in the right atrial (RA) appendage with typical J-shaped curve, noticed best on lateral view (red arrow). Bipolar, active-fixation right ventricular (RV) lead implanted on the RV septum (blue arrow). Quadripolar, active-fixation left ventricular (LV) lead in a lateral branch of the coronary sinus (green arrow). Note the lead's epicardial trajectory and posterior orientation in the lateral view. (*C,D*) Dual-chamber pacemaker connected to two active-fixation, bipolar leads. The RV lead seems to be at the RV apex in the anteroposterior (AP) view, however on the lateral view, its posterior trajectory points towards an LV position through a patent foramen ovale. (*E,F*) Dual-chamber pacemaker in the right prepectoral region connected to two active-fixation bipolar leads. The RV lead seems positioned at the RV apex on the AP view but is in fact positioned in a coronary sinus tributary as can be seen by its epicardial and posterior course on the lateral view. (*G*) CXR of a patient with a congenital cardiopathy with situs inversus and L-transposition of the great arteries implanted with RA, RV, and LV leads in the coronary sinus. (*H*) RV lead positioned at the RV apex through a persistent left superior vena cava.

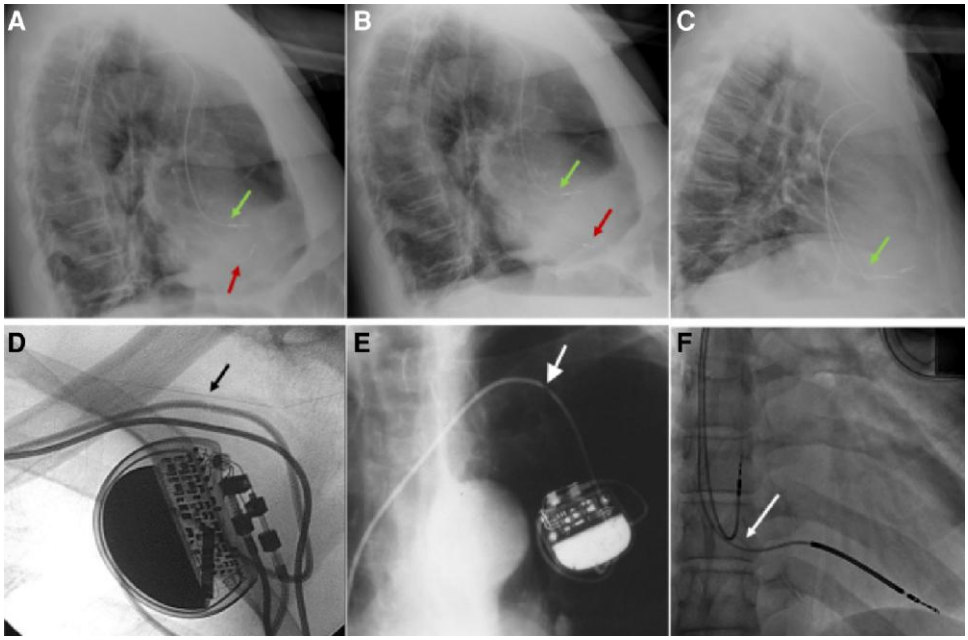

Figure 22 Lead complications. (*A,B*) Chest X-ray (CXR) after implantation of a dual-chamber pacemaker with a right atrial (RA) lead (green arrow) and a right ventricular (RV) lead for left bundle branch pacing (LBBBP, red arrow). Because of dizziness and bradycardia, a new CXR was taken a few hours later confirming dislodgement of the LBBP lead to the RV apex. The lead was successfully repositioned. (*C*) Dual-chamber pacemaker with RA lead dislodgement (green arrow). (*D*) Anchoring sleeve sutured too firmly causing lead damage (arrow). (*E*) Subclavian crush syndrome: lead fracture caused by entrapment of the lead between the first rib and the clavicle. (*F*) Inside-out abrasion with the St. Jude Medical Riata defibrillation lead. Note the externalization of the conductor (arrow).

Table 2 Assessment of cardiac implantable electronic devices on chest radiographs

Immediate post-operative assessment
• Device recognition
◦ Identify device type (pacemaker, ICD, or CRT)
◦ Lead recognition (unipolar, bipolar, quadripolar; integrated vs. true bipolar ICD lead; active vs. passive fixation)
• Pocket inspection
◦ Check for full lead pin insertion in the generator
◦ Exclude excessive kinking of the lead in the pocket
• Check for proper lead position and exclude possible lead dislodgement
• Exclude a pneumo- or haemothorax
• Evaluate signs of lead perforation
Device evaluation on follow-up CXR
• Evaluate the correct position of the pacemaker casing inside the pocket and look for complications (kinking of the lead, Twiddler's syndrome)
• Look for lead damage or breakage by tracing their entire course (pay particular attention to the subclavian region to evaluate signs of lead crush)
• Confirm the correct positioning of the lead tip and compare its position to previous CXRs

An anchoring sleeve is used to tie a suture to secure the lead to the underlying muscle at the insertion site. If sutures are secured too tightly, they may cause lead damage, which is sometime visible on the X-ray (*Figure 22D*). Kinking of the leads in the pocket may also cause fracture. Lead crush under the clavicle is associated with subclavian vein puncture (*Figure 22E*). Some ICD lead models are prone to externalization of the conductors through the insulation (*Figure 22F*) and may result in dysfunction.

Patient-related complications

Pneumothorax and cardiac perforation were discussed above. Twiddler's syndrome is a rare complication of CIED implantation. On CXR, the leads can be seen twisted in the pocket due to rotation of the generator along its long axis by the patient. *Table 2* gives a summary of the assessment of CIEDs on CXRs.

Clinical advice

Anteroposterior and lateral chest radiography plays an important role in the immediate post-operative assessment and for device evaluation on follow-up visits

Conclusions

Cardiac imaging is crucial for the detection of early and late complications associated with CIED use. Although many potential complications are detectable with routine CXR and conventional Doppler echocardiography, other imaging techniques, such as vascular ultrasound, 3D echocardiography, CT, or PET, are frequently needed for making a definite diagnosis. There is a growing body of evidence that both conditional and non-MR-conditional CIED carriers may safely undergo MRI when following protocols.

Supplementary data

Supplementary data are available at *European Heart Journal - Cardiovascular Imaging* online.

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Data availability

No new data were generated or analysed in support of this research.

References

1. Burri H, Starck C, Auricchio A, Biffi M, Burri M, D'Avila A *et al.* EHRA expert consensus statement and practical guide on optimal implantation technique for conventional pacemakers and implantable cardioverter-defibrillators: endorsed by the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), and the Latin-American Heart Rhythm Society (LAHRS). *Europace* 2021;**23**:983–1008.
2. Delgado V, Ajmone Marsan N, de Waha S, Bonaros N, Brida M, Burri H *et al.* 2023 ESC guidelines for the management of endocarditis. *Eur Heart J* 2023;**44**:3948–4042.
3. Blomström-Lundqvist C, Traykov V, Erba PA, Burri H, Nielsen JC, Bongiorni MG *et al.* European Heart Rhythm Association (EHRA) international consensus document on how to prevent, diagnose, and treat cardiac implantable electronic device infections—endorsed by the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), the Latin American Heart Rhythm Society (LAHRS), International Society for Cardiovascular Infectious Diseases (ISCVID) and the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). *Europace* 2020;**22**:515–49.
4. Erba PA, Lancellotti P, Vilacosta I, Gaemperli O, Rouzet F, Hacker M *et al.* Recommendations on nuclear and multimodality imaging in IE and CIED infections. *Eur J Nucl Med Mol Imaging* 2018;**45**:1795–815.
5. Korkeila PJ, Saraste MK, Nyman KM, Koistinen J, Lund J, Juhani Airaksinen KE. Transesophageal echocardiography in the diagnosis of thrombosis associated with permanent transvenous pacemaker electrodes. *Pacing Clin Electrophysiol* 2006;**29**:1245–50.
6. Golzio PG, Errigo D, Peyracchia M, Gallo E, Frea S, Castagno D *et al.* Prevalence and prognosis of lead masses in patients with cardiac implantable electronic devices without infection. *J Cardiovasc Med (Hagerstown)* 2019;**20**:372–8.
7. Ho G, Bhatia P, Mehta I, Maus T, Khoche S, Pollema T *et al.* Prevalence and short-term clinical outcome of mobile thrombi detected on transvenous leads in patients undergoing lead extraction. *JACC Clin Electrophysiol* 2019;**5**:657–64.
8. Mahmood M, Kendi AT, Farid S, Ajmal S, Johnson GB, Baddour LM *et al.* Role of 18F-FDG PET/CT in the diagnosis of cardiovascular implantable electronic device infections: a meta-analysis. *J Nucl Cardiol* 2019;**26**:958–70.
9. Juneau D, Golfam M, Hazra S, Zuckier LS, Garas S, Redpath C *et al.* Positron emission tomography and single-photon emission computed tomography imaging in the diagnosis of cardiac implantable electronic device infection: a systematic review and meta-analysis. *Circ Cardiovasc Imaging* 2017;**10**:e005772.
10. Rajkumar CA, Claridge S, Jackson T, Behar J, Johnson J, Sohal M *et al.* Diagnosis and management of iatrogenic cardiac perforation caused by pacemaker and defibrillator leads. *Europace* 2017;**19**:1031–7.
11. 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.
12. Laborderie J, Barandon L, Ploux S, Deplagne A, Mokrani B, Reuter S *et al.* Management of subacute and delayed right ventricular perforation with a pacing or an implantable cardioverter-defibrillator lead. *Am J Cardiol* 2008;**102**:1352–5.
13. Kirkfeldt RE, Johansen JB, Nohr EA, Moller M, Arnsbo P, Nielsen JC. Pneumothorax in cardiac pacing: a population-based cohort study of 28,860 Danish patients. *Europace* 2012;**14**:1132–8.
14. van Eck JW, van Hemel NM, Zuithof P, van Asseldonk JP, Voskuil TL, Grobbee DE *et al.* Incidence and predictors of in-hospital events after first implantation of pacemakers. *Europace* 2007;**9**:884–9.
15. Res JC, de Priester JA, van Lier AA, van Engelen CL, Bronzwaer PN, Tan PH *et al.* Pneumothorax resulting from subclavian puncture: a complication of permanent pacemaker lead implantation. *Neth Heart J* 2004;**12**:101–5.
16. Tagliari AP, Kochi AN, Mastella B, Saadi RP, di Leoni Ferrari A, Saadi EK *et al.* Axillary vein puncture guided by ultrasound vs cephalic vein dissection in pacemaker and defibrillator implant: a multicenter randomized clinical trial. *Heart Rhythm* 2020;**17**:1554–60.
17. Volpicelli G. Sonographic diagnosis of pneumothorax. *Intensive Care Med* 2011;**37**:224–32.
18. Addetia K, Harb SC, Hahn RT, Kapadia S, Lang RM. Cardiac implantable electronic device lead-induced tricuspid regurgitation. *JACC Cardiovascular imaging* 2019;**12**:622–36.
19. Anvardeen K, Rao R, Hazra S, Hay K, Dai H, Stoyanov N *et al.* Prevalence and significance of tricuspid regurgitation post-endocardial lead placement. *JACC Cardiovasc Imaging* 2019;**12**:562–4.
20. Prihadi EA, van der Bijl P, Gursoy E, Abou R, Mara Vollema E, Hahn RT *et al.* Development of significant tricuspid regurgitation over time and prognostic implications: new insights into natural history. *Eur Heart J* 2018;**39**:3574–81.
21. Vahanian A, Beyersdorf F, Praz F, Milojevic M, Baldus S, Bauersachs J *et al.* 2021 ESC/EACTS guidelines for the management of valvular heart disease. *Eur Heart J* 2022;**43**:561–632.
22. Van De Heyning CM, Elbarasi E, Masiero S, Brambatti M, Ghazal S, Al-Maashani S *et al.* Prospective study of tricuspid regurgitation associated with permanent leads after cardiac rhythm device implantation. *Can J Cardiol* 2019;**35**:389–95.
23. Chang JD, Manning WJ, Ebrille E, Zimetbaum PJ. Tricuspid valve dysfunction following pacemaker or cardioverter-defibrillator implantation. *J Am Coll Cardiol* 2017;**69**:2331–41.
24. Beurskens NEG, Tjong FVY, de Bruin-Bon RHA, Dasselaar KJ, Kuijt WJ, Wilde AAM *et al.* Impact of leadless pacemaker therapy on cardiac and atrioventricular valve function through 12 months of follow-up. *Circ Arrhythm Electrophysiol* 2019;**12**:e007124.
25. Marincheva G, Levi T, Perelshtein Brezinov O, Valdman A, Rahkovich M, Kogan Y *et al.* Echocardiography-guided cardiac implantable electronic device implantation to reduce device related tricuspid regurgitation: a prospective controlled study. *Isr Med Assoc J* 2022;**24**:25–32.
26. Gmeiner J, Sadoni S, Orban M, Fichtner S, Estner H, Massberg S *et al.* Prevention of pacemaker lead-induced tricuspid regurgitation by transesophageal echocardiography guided implantation. *JACC Cardiovasc Interv* 2021;**14**:2636–8.
27. Polewczyk A, Kutarski A, Tomaszewski A, Brzozowski W, Czajkowski M, Polewczyk M *et al.* Lead dependent tricuspid dysfunction: analysis of the mechanism and management in patients referred for transvenous lead extraction. *Cardiol J* 2013;**20**:402–10.
28. Vaturi M, Kusniec J, Shapira Y, Nevzorov R, Yedidya I, Weisenberg D *et al.* Right ventricular pacing increases tricuspid regurgitation grade regardless of the mechanical interference to the valve by the electrode. *Eur J Echocardiogr* 2010;**11**:550–3.
29. Praz F, Muraru D, Kreidel F, Lurz P, Hahn RT, Delgado V *et al.* Transcatheter treatment for tricuspid valve disease. *EuroIntervention* 2021;**17**:791–808.
30. Höke U, Auger D, Thijssen J, Wolterbeek R, van der Velde ET, Holman ER *et al.* Significant lead-induced tricuspid regurgitation is associated with poor prognosis at long-term follow-up. *Heart* 2014;**100**:960–8.
31. Al-Bawardy R, Krishnaswamy A, Rajeswaran J, Bhargava M, Wazni O, Wilkoff B *et al.* Tricuspid regurgitation and implantable devices. *Pacing Clin Electrophysiol* 2015;**38**:259–66.
32. Fortuni F, van der Kley F, Delgado V, Ajmone Marsan N. Pacemaker lead-induced tricuspid regurgitation: consider leaflet remodeling. *Int J Cardiovasc Imaging* 2021;**37**:1563–5.
33. Hahn RT, Thomas JD, Khalique OK, Cavalcante JL, Praz F, Zoghbi WA. Imaging assessment of tricuspid regurgitation severity. *JACC Cardiovasc Imaging* 2019;**12**:469–90.
34. Muraru D, Migliore F, Cipriani A, Iliceto S, Bertaglia E. Transthoracic 3D echocardiography imaging of transcatheter pacing system. *Eur Heart J Cardiovasc Imaging* 2017;**18**:937.

35. Seo Y, Ishizu T, Nakajima H, Sekiguchi Y, Watanabe S, Aonuma K. Clinical utility of 3-dimensional echocardiography in the evaluation of tricuspid regurgitation caused by pacemaker leads. *Circ J* 2008;**72**:1465–70.
36. Muraru D, Hahn RT, Soliman OI, Faletra FF, Basso C, Badano LP. 3-Dimensional echocardiography in imaging the tricuspid valve. *JACC Cardiovascular Imaging* 2019;**12**: 500–15.
37. Ellenbogen KA, Gilligan DM, Wood MA, Morillo C, Barold SS. The pacemaker syndrome—a matter of definition. *Am J Cardiol* 1997;**79**:1226–9.
38. Amici E, Neri R, Donati R, Gambelli G. Transesophageal echocardiographic color Doppler evaluation of pulmonary vein flow during ventricular pacing. *Am J Card Imaging* 1996;**10**:23–8.
39. Kiehl EL, Makki T, Kumar R, Gumber D, Kwon DH, Rickard JW *et al.* Incidence and predictors of right ventricular pacing-induced cardiomyopathy in patients with complete atrioventricular block and preserved left ventricular systolic function. *Heart Rhythm* 2016;**13**:2272–8.
40. Gillis AM. Optimal pacing for right ventricular and biventricular devices: minimizing, maximizing, and right ventricular/left ventricular site considerations. *Circ Arrhythm Electrophysiol* 2014;**7**:968–77.
41. Naqvi TZ, Chao CJ. Adverse effects of right ventricular pacing on cardiac function: prevalence, prevention and treatment with physiologic pacing. *Trends Cardiovasc Med* 2023;**33**:109–22.
42. Khurshid S, Epstein AE, Verdino RJ, Lin D, Goldberg LR, Marchlinski FE *et al.* Incidence and predictors of right ventricular pacing-induced cardiomyopathy. *Heart Rhythm* 2014;**11**:1619–25.
43. Bogale N, Witte K, Priori S, Cleland J, Auricchio A, Gadler F *et al.* The European Cardiac Resynchronization Therapy Survey: comparison of outcomes between de novo cardiac resynchronization therapy implantations and upgrades. *Eur J Heart Fail* 2011;**13**:974–83.
44. Stankovic I, Prinz C, Ciarka A, Daraban AM, Mo Y, Aarones M *et al.* Long-term outcome after CRT in the presence of mechanical dyssynchrony seen with chronic RV pacing or intrinsic LBBB. *JACC Cardiovasc Imaging* 2017;**10**:1091–9.
45. Glikson M, Nielsen JC, Kronborg MB, Michowitz Y, Auricchio A, Barbash IM *et al.* 2021 ESC guidelines on cardiac pacing and cardiac resynchronization therapy. *Europace* 2022;**24**:71–164.
46. Burri H, Jastrzebski M, Cano Ó, Čurila K, de Pooter J, Huang W *et al.* EHRA clinical consensus statement on conduction system pacing implantation: executive summary. Endorsed by the Asia-Pacific Heart Rhythm Society (APHRS), Canadian Heart Rhythm Society (CHRS) and Latin-American Heart Rhythm Society (LAHRS). *Europace* 2023; **25**:1237–48.
47. Kircanski B, Boveda S, Prinzen F, Sorgente A, Anic A, Conte G *et al.* Conduction system pacing in everyday clinical practice: EHRA physician survey. *Europace* 2023;**25**:682–7.
48. Bartko PE, Arfsten H, Heitzinger G, Pavo N, Strunk G, Gwechenberger M *et al.* Papillary muscle dyssynchrony-mediated functional mitral regurgitation: mechanistic insights and modulation by cardiac resynchronization. *JACC Cardiovasc Imaging* 2019; **12**:1728–37.
49. Alizadeh A, Sanati HR, Haji-Karimi M, Yazdi AH, Rad MA, Haghjoo M *et al.* Induction and aggravation of atrioventricular valve regurgitation in the course of chronic right ventricular apical pacing. *Europace* 2011;**13**:1587–90.
50. Miranda R, Almeida S, Brandão L, Alvarenga C, Ribeiro L, Almeida AR *et al.* Acute severe mitral regurgitation as an early complication of pacemaker implantation. *Europace* 2010;**12**:1791–2.
51. Haider A, Banerjee S, Brilakis ES. Acute pulmonary edema due to pacemaker-induced mitral regurgitation. *J Invasive Cardiol* 2008;**20**:E84.
52. Sassone B, De Simone N, Parlangeli G, Tortorici R, Biancoli S, Di Pasquale G. Pacemaker-induced mitral regurgitation: prominent role of abnormal ventricular activation sequence versus altered atrioventricular synchrony. *Ital Heart J* 2001;**2**:441–8.
53. Kosmala W, Saito M, Kaye G, Negishi K, Linker N, Gammage M *et al.* Incremental value of left atrial structural and functional characteristics for prediction of atrial fibrillation in patients receiving cardiac pacing. *Circ Cardiovasc Imaging* 2015;**8**:e002942.
54. Spittell PC, Hayes DL. Venous complications after insertion of a transvenous pacemaker. *Mayo Clin Proc* 1992;**67**:258–65.
55. Rozmus G, Daubert JP, Huang DT, Rosero S, Hall B, Francis C. Venous thrombosis and stenosis after implantation of pacemakers and defibrillators. *J Interv Card Electrophysiol* 2005;**13**:9–19.
56. Mandal S, Pande A, Mandal D, Kumar A, Sarkar A, Kahali D *et al.* Permanent pacemaker-related upper extremity deep vein thrombosis: a series of 20 cases. *Pacing Clin Electrophysio* 2012;**35**:1194–8.
57. Santini M, Di Fusco SA, Santini A, Magris B, Pignalberi C, Aquilani S *et al.* Prevalence and predictor factors of severe venous obstruction after cardiovascular electronic device implantation. *Europace* 2016;**18**:1220–6.
58. Abu-El-Haija B, Bhave PD, Campbell DN, Mazur A, Hodgson-Zingman DM, Cotarlan V *et al.* Venous stenosis after transvenous lead placement: a study of outcomes and risk factors in 212 consecutive patients. *J Am Heart Assoc* 2015;**4**:e00187.
59. Winters SL, Curwin JH, Sussman JS, Coyne RF, Calhoun SK, Yablonsky TD *et al.* Utility and safety of axillo-subclavian venous imaging with carbon dioxide (CO) prior to chronic lead system revisions. *Pacing Clin Electrophysiol* 2010;**33**:790–4.
60. Kella DK, Isath A, Yasin O, Padmanabhan D, Webster T, Mulpuru S *et al.* Fibroplasty (venoplasty) to facilitate transvenous lead placement: a single-center experience. *J Cardiovasc Electrophysio* 2020;**31**:2425–30.
61. Arora Y, Carrillo RG. Lead-related superior vena cava syndrome: management and outcomes. *Heart Rhythm* 2021;**18**:207–14.
62. Sweeney MO, Hellkamp AS, van Bommel RJ, Schalij MJ, Borleffs CJ, Bax JJ. QRS fusion complex analysis using wave interference to predict reverse remodeling during cardiac resynchronization therapy. *Heart Rhythm* 2014;**11**:806–13.
63. Jastrzebski M, Baranchuk A, Fijorek K, Kisiel R, Kukla P, Sondej T *et al.* Cardiac resynchronization therapy-induced acute shortening of QRS duration predicts long-term mortality only in patients with left bundle branch block. *Europace* 2019;**21**:281–9.
64. Trucco E, Tolosana JM, Arbelo E, Doltra A, Castel MÁ, Benito E *et al.* Improvement of reverse remodeling using electrocardiogram fusion-optimized intervals in cardiac resynchronization therapy: a randomized study. *JACC Clin Electrophysiol* 2018;**4**:181–9.
65. Jones RC, Svinarich T, Rubin A, Levin V, Phang R, Murillo J *et al.* Optimal atrioventricular delay in CRT patients can be approximated using surface electrocardiography and device electrograms. *J Cardiovasc Electrophysiol* 2010;**21**:1226–32.
66. Daubert JC, Saxon L, Adamson PB, Auricchio A, Berger RD, Beshai JF *et al.* 2012 EHRA/HRS expert consensus statement on cardiac resynchronization therapy in heart failure: implant and follow-up recommendations and management. *Europace* 2012;**14**: 1236–86.
67. Mullens W, Auricchio A, Martens P, Witte K, Cowie MR, Delgado V *et al.* Optimized implementation of cardiac resynchronization therapy: a call for action for referral and optimization of care: a joint position statement from the Heart Failure Association (HFA), European Heart Rhythm Association (EHRA), and European Association of Cardiovascular Imaging (EACVI) of the European Society of Cardiology. *Eur J Heart Fail* 2020;**22**:2349–69.
68. Mullens W, Grimm RA, Verga T, Dresing T, Starling RC, Wilkoff BL *et al.* Insights from a cardiac resynchronization optimization clinic as part of a heart failure disease management program. *J Am Coll Cardiol* 2009;**53**:765–73.
69. Jones S, Shun-Shin MJ, Cole GD, Sau A, March K, Williams S *et al.* Applicability of the iterative technique for cardiac resynchronization therapy optimization: full-disclosure, 50-sequential-patient dataset of transmitral Doppler traces, with implications for future research design and guidelines. *Europace* 2014;**16**:541–50.
70. Whinnett ZI, Davies JE, Willson K, Manisty CH, Chow AW, Foale RA *et al.* Haemodynamic effects of changes in atrioventricular and interventricular delay in cardiac resynchronisation therapy show a consistent pattern: analysis of shape, magnitude and relative importance of atrioventricular and interventricular delay. *Heart* 2006;**92**: 1628–34.
71. Herweg B, Ali R, Ilercil A, Madramootoo C, Cutro R, Weston MW *et al.* Site-specific differences in latency intervals during biventricular pacing: impact on paced QRS morphology and echo-optimized V–V interval. *Pacing Clin Electrophysiol* 2010;**33**: 1382–91.
72. Burri H, Sunthorn H, Shah D, Lerch R. Optimization of device programming for cardiac resynchronization therapy. *Pacing Clin Electrophysiol* 2006;**29**:1416–25.
73. Boriani G, Müller CP, Seidl KH, Grove R, Vogt J, Danschel W *et al.* Randomized comparison of simultaneous biventricular stimulation versus optimized interventricular delay in cardiac resynchronization therapy. The Resynchronization for the HemodYnamic Treatment for Heart Failure Management II implantable cardioverter defibrillator (RHYTHM II ICD) study. *Am Heart J* 2006;**151**:1050–8.
74. Abraham WT, León AR, St John Sutton MG, Keteyian SJ, Fieberg AM, Chinchoy E *et al.* Randomized controlled trial comparing simultaneous versus optimized sequential interventricular stimulation during cardiac resynchronization therapy. *Am Heart J* 2012;**164**:735–41.
75. Stankovic I, Prinz C, Ciarka A, Daraban AM, Kotrc M, Aarones M *et al.* Relationship of visually assessed apical rocking and septal flash to response and long-term survival following cardiac resynchronization therapy (PREDICT-CRT). *Eur Heart J Cardiovasc Imaging* 2016;**17**:262–9.
76. Burri H, Sunthorn H, Somsen A, Zaza S, Fleury E, Shah D *et al.* Optimizing sequential biventricular pacing using radionuclide ventriculography. *Heart Rhythm* 2005;**2**:960–5.
77. Willemen E, Schreurs R, Huntjens PR, Strik M, Plank G, Vigmond E *et al.* The left and right ventricles respond differently to variation of pacing delays in cardiac resynchronization therapy: a combined experimental- computational approach. *Front Physiol* 2019;**10**:17.
78. Burri H, Domenichini G, Sunthorn H, Fleury E, Stettler C, Foulkes I *et al.* Right ventricular systolic function and cardiac resynchronization therapy. *Europace* 2010;**12**:389–94.
79. Roguin A, Schwitter J, Vahlhaus C, Lombardi M, Brugada J, Vardas P *et al.* Magnetic resonance imaging in individuals with cardiovascular implantable electronic devices. *Europace* 2008;**10**:336–46.
80. Pieri C, Bhuva A, Moralee R, Abiodun A, Gopalan D, Roditi GH *et al.* Access to MRI for patients with cardiac pacemakers and implantable cardioverter defibrillators. *Open Heart* 2021;**8**:e001598.

81. Nordbeck P, Ertl G, Ritter O. Magnetic resonance imaging safety in pacemaker and implantable cardioverter defibrillator patients: how far have we come? *Eur Heart J* 2015; **36**:1505–11.
82. Celentano E, Caccavo V, Santamaria M, Baiocchi C, Melissano D, Pisanò E *et al.* Access to magnetic resonance imaging of patients with magnetic resonance-conditional pacemaker and implantable cardioverter-defibrillator systems: results from the Really ProMRI study. *Europace* 2018;**20**:1001–9.
83. Nazarian S, Hansford R, Rahsepar AA, Weltin V, McVeigh D, Gucuk Ipek E *et al.* Safety of magnetic resonance imaging in patients with cardiac devices. *N Engl J Med* 2017;**377**: 2555–64.
84. Russo RJ, Costa HS, Silva PD, Anderson JL, Arshad A, Biederman RW *et al.* Assessing the risks associated with MRI in patients with a pacemaker or defibrillator. *N Engl J Med* 2017;**376**:755–64.
85. Munawar DA, Chan JEZ, Emami M, Kadhim K, Khokhar K, O'Shea C *et al.* Magnetic resonance imaging in non-conditional pacemakers and implantable cardioverter-defibrillators: a systematic review and meta-analysis. *Europace* 2020;**22**:288–98.
86. Gopalakrishnan PP, Gevenosky L, Biederman RWW. Feasibility of MRI in patients with non-pacemaker/defibrillator metallic devices and abandoned leads. *J Biomed Sci Eng* 2021;**14**:83–93.
87. Bhuva AN, Moralee R, Brunker T, Lascelles K, Cash L, Patel KP *et al.* Evidence to support magnetic resonance conditional labelling of all pacemaker and defibrillator leads in patients with cardiac implantable electronic devices. *Eur Heart J* 2022;**43**:2469–78.
88. Dacher JN, Gandjbakhch E, Taieb J, Chauvin M, Anselme F, Bartoli A *et al.* Joint Position Paper of the Working Group of Pacing and Electrophysiology of the French Society of Cardiology (SFC) and the Société française d'imagerie cardiaque et vasculaire diagnostique et interventionnelle (SFICV) on magnetic resonance imaging in patients with cardiac electronic implantable devices. *Diagn Interv Imaging* 2020;**101**:507–17.
89. Indik JH, Gimbel JR, Abe H, Alkmim-Teixeira R, Birgersdotter-Green U, Clarke GD *et al.* 2017 HRS expert consensus statement on magnetic resonance imaging and radiation exposure in patients with cardiovascular implantable electronic devices. *Heart Rhythm* 2017;**14**:e97–e153.
90. Stühlinger M, Burri H, Vernooy K, Garcia R, Lenarczyk R, Sultan A *et al.* EHRA consensus on prevention and management of interference due to medical procedures in patients with cardiac implantable electronic devices. *Europace* 2022;**24**:1512–37.
91. Bhuva A, Charles-Edwards G, Ashmore J, Lipton A, Benbow M, Grainger D *et al.* Joint British Society consensus recommendations for magnetic resonance imaging for patients with cardiac implantable electronic devices. *Heart* 2022:heartjnl-2022-320810.
92. Dacher JN, Langguth P, Adam D, Winkler WB, Martí-Almor J, Prenner G *et al.* Safety and performance of MR-conditional pacing systems with automated MRI mode at 1.5 and 3 tesla. *Eur Radiol* 2023;**33**:6948–58.
93. Rashid S, Rapacchi S, Vaseghi M, Tung R, Shivkumar K, Finn JP *et al.* Improved late gadolinium enhancement MR imaging for patients with implanted cardiac devices. *Radiology* 2014;**270**:269–74.
94. Bhuva AN, Kellman P, Graham A, Ramlall M, Boubertakh R, Feuchter P *et al.* Clinical impact of cardiovascular magnetic resonance with optimized myocardial scar detection in patients with cardiac implantable devices. *Int J Cardiol* 2019;**279**:72–8.
95. Vuorinen AM, Paakkanen R, Karvonen J, Sinisalo J, Holmström M, Kivistö S *et al.* Magnetic resonance imaging safety in patients with abandoned or functioning epicardial pacing leads. *Eur Radiol* 2022;**32**:3830–8.
96. Schaller RD, Brunker T, Riley MP, Marchlinski FE, Nazarian S, Litt H. Magnetic resonance imaging in patients with cardiac implantable electronic devices with abandoned leads. *JAMA Cardiol* 2021;**6**:549–56.
97. Padmanabhan D, Kella DK, Mehta R, Kapa S, Deshmukh A, Mulpuru S *et al.* Safety of magnetic resonance imaging in patients with legacy pacemakers and defibrillators and abandoned leads. *Heart Rhythm* 2018;**15**:228–33.
98. Jacob S, Shahzad MA, Maheshwari R, Panaich SS, Aravindhakshan R. Cardiac rhythm device identification algorithm using X-rays: CaRDIA-X. *Heart Rhythm* 2011;**8**:915–22.
99. Chudow JJ, Jones D, Weinreich M, Zaremski L, Lee S, Weinreich B *et al.* A head-to head comparison of machine learning algorithms for identification of implanted cardiac devices. *Am J Cardiol* 2021;**144**:77–82.
100. Quast ABE, Baalman SWE, Brouwer TF, Smeding L, Wilde AAM, Burke MC *et al.* A novel tool to evaluate the implant position and predict defibrillation success of the subcutaneous implantable cardioverter-defibrillator: the PRAETORIAN score. *Heart Rhythm* 2019;**16**:403–10.