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CARDIAC DEVICE TROUBLESHOOTING IN THE INTENSIVE CARE UNIT

An educational review

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ABSTRACT

Numerous patients with a cardiac implantable electronic device are admitted to the cardiac intensive care unit. When taking care of these patients, it's essential to have basic knowledge of potential device problems and how they could be tackled. This review summarizes common issues with pacemakers, implantable cardioverter defibrillators and cardiac resynchronization devices and provides a framework for troubleshooting in the intensive care unit. In addition, specific aspects of intensive care that might interfere with cardiac devices are discussed.

Keywords: implantable cardiac electronic device – intensive care unit – pacemaker – implantable cardioverter defibrillator – cardiac resynchronization therapy - troubleshooting

INTRODUCTION

Cardiac pacemakers, implantable cardioverter defibrillators (ICD) and cardiac resynchronization therapy (CRT) are increasingly used in cardiovascular medicine. As prognosis of cardiovascular disease has improved in the last decades, the number of patients with cardiac implantable electronic devices (CIED) has also grown accordingly. A lot of patients with CIEDs may be admitted to the intensive care unit (ICU) for various reasons. Although a CIED problem can be a reason for admission, more often other conditions warrant ICU admission. Therefore, threating physician's should be aware of common CIED problems and their solutions, how CIEDs can affect patient care and how CIEDs can also sometimes be a part of the solution. This review focuses on CIED troubleshooting in the ICU and aims to guide general cardiologists and intensivists through common faced problems. In addition a framework for problem solving is provided (central figure).

BASIC PRINCIPLES OF CIEDS

When taking care of patients with CIEDs it is important to understand basic functioning of the CIED to be able to discriminate between normal and abnormal function (table 1). Besides the type of CIED, it's important to also consider the indication for implantation. If a pacemaker was implanted for 3rd degree AV block, there's a high chance that the patient will be 'pacemaker dependent' and failure of the device can result in asystole when there is insufficient escape rhythm. The percentage of atrial and ventricular pacing can also give a clue about how much the patient is in need of pacing. Of note, patients with a high degree of atrial pacing or patients with sick sinus syndrome, might have chronotropic incompetence, implicating they cannot increase their heart rate sufficiently if metabolic demands increase (e.g. shock). In patients with an ICD, it's important to know if the patient already experienced ventricular arrhythmias before, which increases the risk of recurrence. Transvenous ICDs can also function as a pacemaker, but subcutaneous ICDs cannot.

Second, the programming features of the CIED should be known for troubleshooting. In particular, the programmed lower rate and the pacing mode are of interest. A contemporary overview of different pacing modes was recently published [1]. The most common modes in clinical practice are DDD and VVI. In the DDD pacing mode, the pacemaker can pace both the atrium and the ventricle. In addition, the DDD pacing mode allows for 'tracking' of the atrial rate by ventricular pacing if there is no intrinsic conduction after a predefined AV interval. In VVI mode, the pacemaker is only able to sense and pace in the ventricle irrespective of the atrial rhythm. Both DDD and VVI mode can be combined with a rate response (R-mode), which means a sensor can adapt the heart rate to rates higher than the lower rate, when activity is sensed (DDDR and VVIR). Common sensors are accelerometers (measuring motion of the pectoralis region where the device is placed), minute ventilation sensors and cardiac contractility sensors. The latter two are more physiological sensors allowing to increase heart rate without moving of the patient (ie hyperventilation or increased sympathetic tone). Rate response is used in patients who have chronotropic incompetence to increase exercise capacity.

For ICDs the most important programming features are the tachycardia detection zones and the consequent therapy delivered. Transvenous ICDs can deliver either anti-tachycardia pacing (ATP) or defibrillator shocks to terminate ventricular arrhythmias, while subcutaneous ICDs can only deliver shocks. Classically a 'slower' ventricular tachycardia (VT) zone at rates 185-200 bpm is programmed, which is first treated with ATP, which can either be 'burst' (fixed timing interval) or 'ramp' (decreasing timing interval) pacing. ATP is typically programmed with a minimum of 8 stimuli and a cycle length of 84–88% of the tachycardia cycle length [2]. A shock is only delivered when ATP was not successful. In patients who have a secondary prevention indication with known VT cycle length, the detection zone can be individualized. For rates > 230-250 bpm, the success rate of ATP is lower and default therapy is shock [2]. Nevertheless, ATP will mostly be delivered during capacitor charging and can be successful. Most devices also allow to program a monitor zone in which ventricular arrhythmias are detected and registered, but not treated. The primary parameter that is used to detect a VT is the programmed rate. As a result, rates below the lower VT detection zone will never result in therapy by the device. Of note,

several discriminator programming features allow the device to discriminate between supraventricular tachycardia and ventricular tachycardia, but are discussed elsewhere [2].

A CRT device has the same features as a pacemaker with (CRT-D) or without (CRT-P) ICD features. The most important difference in programming is the intention to resynchronize the left ventricle by maximally providing biventricular pacing. More on how to optimize and program CRT devices can be found in a recent European position paper [3].

Last, there is a growing interest and experience with alternative pacing methods such as His bundle pacing [4] and left bundle branch area pacing [5] . In contrast to conventional right ventricular pacing, these methods can provide a more physiological ventricular activation through the native conduction system. Both His bundle pacing as well as left bundle branch area pacing have been proposed as an alternative for pacing and CRT indications, but evidence is limited to observational studies. Importantly, if these methods are used as alternative to CRT, maximal pacing should also be provided. Of note these alternative pacing methods have distinct electrocardiographic and programming features, warranting advise form a cardiac rhythm specialist.

PACEMAKER MALFUNCTIONING

Loss of capture

Loss of capture occurs when the delivered energy by the pacemaker no longer succeeds in inducing ventricular activation. This can be recognized on the surface ECG as pacing spikes, not followed by atrial or ventricular complexes (figure 1A). A systematic approach to loss of capture is provided in figure 2. The differential diagnosis for loss of capture on the ICU is broad. First, lead dysfunction/dislocation should be checked with a chest X-ray and interrogation of the device. Both acidosis and alkalosis increase pacing threshold and can contribute to loss of capture. Electrolyte disturbances such as hyperkalemia, hypomagnesemia and hypocalcemia increase the pacing threshold. In addition hypoxia

and myocardial ischemia can also raise the pacing threshold. Thus correcting acid-base, electrolytes, ischemia and hypoxia can reinstitute the pacing threshold. Of note, multiple medications used in the ICU can influence the pacing threshold either favourably or unfavourably (table 2). Especially class Ic anti-arrhythmics (e.g. flecainide and propafenone) can significantly increase pacing threshold and should be stopped if possible. Hypothyroidism should also be corrected as this might increase pacing thresholds. Finally, emphysema or pneumothorax might interfere with unipolar pacing and should be treated.

No output and oversensing

In case of no output, the pacemaker does not respond adequately to bradycardia. This can be recognized as absence of pacing spikes on the ECG in patients in whom the heart rate drops below the programmed lower rate of the pacemaker (figure 1B). No output can be the resultant of failure of the device but is more often caused by oversensing, meaning signals are wrongly sensed as ventricular depolarization and inhibit pacing (figure 3). Importantly, oversensing of ventricular signals in a pacemaker dependent patient can lead to death. An easy tool to differentiate between both is application of a magnet, which inhibits all sensing. If pacing fully resumes, oversensing is the most probable cause. Oversensing mostly results from non-cardiac signals that can either originate from the leads, due to problems with lead integrity or loose connection of the setscrew with the battery (which will most often be visible early after implant), or originate from electromagnetic interference of close-by electrical devices. In case unipolar sensing is programmed, myopotentials from the chest wall muscles can also cause oversensing. Atrioventricular (AV) crosstalk, where atrial activation is sensed in the ventricular channel, can also inhibit ventricular pacing but is rare with current pacemaker programming features. A chest X-ray should be obtained to check the leads and special attention should also be given to the connection of the setscrew to the battery. The device should be interrogated to confirm oversensing, check lead impedance, evaluate for ‘noise’ during manipulation of the pocket or moving of the arm and to check for AV crosstalk. On the ICU, electromagnetic

interference is rare, but could be due to close (< 1m) contact with (older) cellular phones, radiology equipment and electrocautery equipment [6]. Oversensing due to lead insulation defects or fracture can sometimes temporarily be resolved during ICU admission by decreasing sensitivity (thus increasing sensing threshold), if the amplitude of the oversensed signals is small and the ventricular signal amplitude is significantly larger. In case of an ICD lead, it might be prudent to temporarily program ICD therapies 'off' to avoid inappropriate therapies. In case of myopotential oversensing, reprogramming to bipolar sensing should be done whenever possible. Otherwise, sensitivity can be decreased, but adequate sensing of ventricular signals should be guaranteed. Leads should be replaced as indicated when the patient's condition allows it.

In case no pacing occurs after magnet application, the cause of no output is device failure, which could be due to battery depletion, pulse generator defects or interruption of the pacing circuit caused by lead failure or a loose setscrew. When no connection can be established between the programmer and the device, most probably the battery is depleted and should be replaced. Lead integrity and connections should be assessed as described above. Temporarily, the output can be programmed higher until pacing occurs, awaiting lead replacement. Otherwise transcutaneous or transvenous temporary pacing can be used, before definitive restoration of the circuit.

Undersensing

In case of undersensing, the device fails to adequately sense intrinsic atrial/ventricular activation, resulting in asynchronous pacing (figure 1C). This can happen either intermittently or continuously. Causes of undersensing are listed in table 3. If the cause of undersensing is not immediately reversible, sensitivity can be cautiously increased by lowering the sensing threshold, carefully inspecting not to induce oversensing. Alternatively, a unipolar sensing configuration can be tested. If unipolar sensing is better than bipolar sensing, there is a high possibility of an insulation defect. Last, pacemakers have different refractory and blanking periods, during which activity is ignored or not sensed at all. Conventionally, after every ventricular or atrial pace, all paced chambers will have a blanking period

during which any activity will not be sensed to avoid oversensing of the post-pacing activation and repolarization and to avoid AV crosstalk in dual chamber pacemakers. In contrast to a paced atrial activation, after intrinsic atrial activation, only an atrial blanking period will start but no ventricular blanking. In addition, after every ventricular beat a longer refractory period is programmed beyond the blanking period, which is called the post-ventricular atrial blanking period (PVARP) and the ventricular refractory period (VRP). During this refractory period, activity is sensed by the device, but will be ignored. The goal of the PVARP is to avoid sensing of retrograde P-waves, that of the VRP is to avoid oversensing of T-waves. As a consequence, any activity (e.g. premature ventricular or atrial complexes) that occurs during these blanking and refractory periods will not reset the timing of the device. This is normal pacemaker behaviour (sometimes called 'functional undersensing') and should not be considered as undersensing.

Loss of biventricular pacing

CRT devices are aimed at resynchronization of the LV through biventricular pacing. Loss of biventricular pacing can have deleterious effects on haemodynamics and outcomes and should be recognized on the ICU [7]. Ventricular pacing can either be absent or there can be capture of the LV or RV lead. Absent pacing spikes on the surface ECG, suggest intrinsic AV conduction for which reprogramming of the AV-interval and/or additional use of AV nodal slowing agents might be indicated. A supraventricular tachycardia might also induce loss of biventricular pacing and an attempt to restore sinus rhythm should be undertaken. Importantly, the presence of a pacing spike does not indicate biventricular pacing. When loss of capture of either the LV or RV lead occurs, the morphology of the ECG will no longer resemble a typical biventricular pacing pattern, which should be recognized (figure 4). The device should be interrogated and a chest X-ray performed to assess lead integrity, position and connection to the pulse generator. Further, the same principles as described for loss of capture of a pacemaker lead can be applied (see above). Finally, sometimes the CRT device is programmed by the treating physician or by an automated algorithm in a LV pacing modus only. Therefore, different ECG

morphologies might occur in patients with CRTs. The advice of a cardiac rhythm specialist should be considered.

ICD THERAPY

Often VT's can be stopped with ATP thereby avoiding a shock, which should be reserved for hemodynamically unstable sustained ventricular tachy-arrythmias. For the patient, an ICD shock is often a traumatic event to which a prompt response is necessary. After a shock has been delivered it is of utmost importance to distinguish between appropriate and inappropriate shocks. If the event happens during cardiac monitoring, looking at the monitoring tracings can already indicate if a ventricular arrhythmia preceded the shock and whether ATP was delivered before. If the event happened outside the ICU, the context can be helpful to distinguish between appropriate and inappropriate shocks. For example, repeated shocks during manipulation of an electrical device might indicate inappropriate shocks due to electromagnetic interference. In contrast, a patient that received an ICD in secondary prevention and already had appropriate shocks before, most probably experienced a new ventricular arrhythmia event. The device should be interrogated with following questions in mind: (i) Was the arrhythmia correctly detected? (ii) Was therapy appropriately delivered? (iii) Did the therapy successfully terminate the arrhythmia? (iv) Was the patient hemodynamically stable at the moment of the shock warranting potentially different ICD programming? (v) Does the device still function normally?

Discriminating VT vs. SVT

First the stored electrograms (EGM) of the event should be inspected. ICDs store 2-4 source channels with mostly both a near-field EGM between the tip and the adjacent ring or coil and a far-field EGM between two widely-spaced, high-voltage electrodes (most commonly between the RV coil and the pulse generator 'can'). A shock could be delivered because of a tachy-arrhythmia (either ventricular or supraventricular) or because of oversensing (figure 5). A true ventricular arrhythmia typically has a

different morphology than sinus rhythm and there should be AV dissociation. Of note, morphology can best be assessed in the far-field channel, as this provides a broader EGM incorporating a larger fraction of myocardium [8]. Further, ventricular arrhythmias have a sudden onset and, in case of a VT, have a stable cycle length. However, supraventricular tachycardias (SVT) can also present with sudden onset and stable cycle length. In addition, aberrant conduction during SVT might also change far-field EGM morphology. Irregularly irregular rhythms with stable morphology suggest atrial fibrillation with rapid ventricular conduction. If an atrial EGM is available, the aforementioned AV dissociation and the chamber of onset can aid in diagnosing VT.

Inappropriate shocks

Inappropriate shocks constitute around a third of delivered shocks and are associated with impaired survival [9,10]. SVT is the most common reason for inappropriate shocks (up to 80% of cases) [9,10], and should be aggressively treated with anti-arrhythmic drugs and/or ablation to avoid recurrence. Sometimes, reprogramming of the VT detection to higher rates can help to avoid recurrent inappropriate shocks. Oversensing is the cause of inappropriate shock in the remaining 20% of cases. Just as in pacemakers, oversensing can be caused by cardiac and non-cardiac signals. T-wave oversensing was a common problem in early ICDs (figure 6) - especially in the subcutaneous ICDs - but this has been tackled by most manufacturers by applying dynamic sensitivity and/or filters. Depending on the manufacturer, after a ventricular activation has been sensed, the device will lower its sensitivity temporarily and/or filter the lower frequency T-wave out [11]. If T-wave oversensing nevertheless occurs it can be resolved by: (i) decreasing sensitivity if R>>T, (ii) changing dynamic sensitivity or filter settings if programmable, (iii) repositioning the lead. R-wave double counting results from slow ventricular depolarization that reaches beyond the short ventricular blanking period. The long ventricular activation can be the resultant of conduction delay, metabolic derangements (especially hyperkalemia) or administration of class I anti-arrhythmic drugs. R-wave double counting can be resolved by prolonging the ventricular blanking period. P-wave oversensing is uncommon and is mostly

the resultant of dislodgment of the ventricular lead early after implantation. The non-cardiac sources of oversensing and their solutions in ICDs are similar as those in pacemakers and were already discussed. Often, lead dysfunction can induce noise oversensing (figure 7).

Unsuccessful shocks

It's important to recognize whether the shock succeeded in stopping the arrhythmia. A single shock can be unsuccessful, but with current high voltage ICDs it rarely occurs that repeated shocks are not successful. Defibrillation failure can be the consequence of device failure or elevation of the defibrillation threshold. Potential causes are listed in table 4. Of note, several drugs can raise the defibrillation threshold (table 1). In case of unsuccessful ICD shocks, external defibrillation might be indicated. In addition, the ICD therapy can be switched off with close monitoring on the ICU if a solution is not possible within an acceptable time window.

Recurrent shocks and VT storm

Recurrent shocks can be either appropriate or inappropriate and are experienced as emotionally very stressful and traumatic by patients. A magnet or program header should be applied to deactivate the tachy-arrhythmia therapy as soon as possible. While interrogating the device appropriateness of therapy should be checked. If therapy was inappropriate due to SVT or oversensing, these should be treated. ICD therapy is best left off until these causes of inappropriate shocks are resolved, while the patient is monitored closely.

If ≥ 3 sustained VT's occur within 24 hours, this is defined as a 'VT storm', irrespective of whether the VT ended spontaneously, after ATP or after defibrillation. Patients with a VT storm often have recurrent ICD shocks and should be admitted to a ICU for monitoring, anti-arrhythmic drug administration, anxiolytics and diagnosis and treatment of precipitating causes [12]. Sometimes sedation and intubation is necessary to maximally reduce adrenergic activity. In addition, reprogramming of the device can be helpful. First, increasing the lower rate of the ICD to perform overdrive pacing (preferentially by atrial pacing) can prevent VT recurrence. Second, detection time

can be prolonged so that VT's have more time to end spontaneously. Last, shock therapy can be switched off and arrhythmias treated with ATP or external defibrillation to preserve the battery and lead integrity.

No ICD therapy delivered

Some ventricular tachy-arrhythmias might not induce ICD therapy. First, VT's below the detection rate can never be detected . ICDs use the detection rate as first criterion to detect VT and then use detection time and different discriminators to confirm VT. If a VT occurs at a rate of 160 bpm and the first VT zone is programmed at 180 bpm, the ICD will never recognize the VT. Depending on the hemodynamic stability of the patient, a sustained VT below the detection rate can be treated with anti-arrhythmic drugs, ATP and/or internal/external defibrillation. Reprogramming of a lower detection rate at least 10 bpm below the undetected VT will be necessary to avoid future undetected VT's [2]. Further, discriminators might misclassify VT as SVT and incorrectly withhold therapy, for which reprogramming might also be necessary.

Due to the dynamic sensitivity of ICDs, undersensing of ventricular arrhythmias is very uncommon. However, it can occur if a VF has a very fast varying EGM amplitude, so that the sensitivity cannot adapt timely. Of course, pulse generator or lead defects can also cause undersensing, just as in pacemakers. Thus, lead impedance should be checked and a chest X-ray performed to evaluate lead integrity and connections. Lead replacement might be necessary.

PSEUDO MALFUNCTIONING

Several specific events might mimic malfunctioning of the device, but are inherent to its programming features. These 'pseudo malfunctions' are further discussed here.

Pacemaker mediated tachycardia

Pacemaker mediated tachycardia (PMT) can only occur in an atrial tracking mode and is the consequence of retrograde conduction of a ventricular beat. The retrogradely conducted atrial signal

is sensed and induces a ventricular pace after the pre-set AV interval. This typically leads to a sudden fixed ventricular paced tachycardia at the upper tracking rate (figure 8C). Most often, PMT is initiated by a premature ventricular beat or atrial non-capture. The tachycardia can be terminated by applying a magnet, which confers DDD to a non-sensing asynchronous DOO mode. PMT can also spontaneously terminate if retrograde conduction stops or because of PMT recognition algorithms of the device. In case of recurrent PMT, reprogramming of the pacemaker might be indicated.

Supraventricular tachycardia with ventricular tracking

The differential diagnosis of tachycardia in a pacemaker patient does not only include pacemaker related problems, but also all SVT's and ventricular arrhythmias. Of specific importance is SVT in a ventricular pacing dependent patient in DDD mode. This will inevitably result in a ventricular paced tachycardia, and might mistakenly be considered as a pacemaker problem. Careful inspection of the surface ECG will allow to discriminate intrinsic atrial activity that precedes the ventricular pacing spike. When in doubt, a magnet can be applied to slow the ventricular rate and unmask the atrial rhythm. Of course, also the programmer with the marker channel can be used. Magnet application can easily help to distinguish PMT (stops immediately) from SVT (SVT continues with asynchronous dual chamber pacing).

Upper rate behaviour

In pacemaker tracking modes such as DDD, an upper tracking rate has to be programmed, meaning the pacemaker will only track the atrial rate by ventricular pacing up to this programmed rate. Above the upper tracking rate, a pseudo-Wenkebach phenomenon will occur (figure 8A) as a consequence of specific pacemaker refractory periods (i.e. the post ventricular atrial refractory period). The AV interval prolongs until one atrial beat is not followed by a ventricular pacing activity. When the heart rate further increases, a 2:1 block can occur, leading to a sudden drop in the ventricular rate (figure 8B). Depending on the pacemaker settings, this 2:1 block can also occur without a preceding pseudo-Wenkebach block. These phenomena can be seen in patients with shock that are dependent on

ventricular pacing and have sinus tachycardia or another form of SVT. The best solution to the problem is treating the cause of the tachycardia. Sometimes reprogramming of the device is necessary.

Sensor induced tachycardia

Patients that have a rate-response feature programmed 'ON', can have increased heart rates on the ICU. Especially patients with minute ventilation sensors can experience increased heart rates at rest if they have a high breathing rate. In addition vibrations from ICU devices that are transmitted to the patient might induce tachycardia if an accelerometer is used. These problems can easily be resolved by switching off rate response during ICU admission.

Automatic threshold and sensitivity testing

In most CIEDs an automatic threshold test and automatic sensitivity test can be programmed. These features intermittently change pacing behaviour and should not be confused with pacemaker malfunctioning. Specifically repeated events with similar time intervals (e.g. every 21 hours) should raise suspicion of such feature. This can easily be checked in the CIED settings.

Ventricular suppression algorithms

As right ventricular pacing is associated with adverse outcomes [13,14], manufacturers have various programmable algorithms that decrease right ventricular pacing in patients with sufficient intrinsic AV conduction who do not have a CRT device and are not pacing dependent. In general, these algorithms intermittently pause or delay ventricular pacing to allow for intrinsic conduction to occur. These algorithms might thus temporarily induce long AV intervals or even a missed ventricular beat.

Rate hysteresis

To minimize unnecessary pacing at night or when the patient is at rest, pacemakers often allow to program a rate hysteresis below the lower rate. This means that the heart rate can drop below the lower rate up to the hysteresis rate before pacing starts at the lower rate. For example, in a patient

with lower rate 60 bpm and rate hysteresis at 50 bpm, the pacemaker will only start to pace at 60 bpm when the rate drops below 50 bpm.

SPECIFIC CONDITIONS IN THE ICU

Shock

Some CIED patients will have chronotropic incompetence. As a consequence, they cannot augment their heart rate during shock states. Because cardiac output is mostly increased by heart rate rather than stroke volume, the chronotropic incompetence might hamper adequate cardiac response to shock. This could be overcome by increasing the pacemaker lower rate, while monitoring the hemodynamic effects. Importantly, if AV conduction is preserved, it might be warranted to program a higher pacing rate with long AV delays, thereby avoiding right ventricular pacing which will induce ventricular dyssynchrony and worsen haemodynamics, especially in patients with reduced left ventricular ejection fraction [15]. Therefore the decision to increase the pacemaker lower rate should be tailored to each patient specifically.

Mechanical circulatory support

Contemporary pacemakers and ICDs can be safely used with all mechanical support devices and oversensing due to electromagnetic interference is very rare [16]. However, ICD therapy is often programmed 'off' in patients with left ventricular assist devices as these devices provides full hemodynamic support.

Electrical cardioversion

For external electrical cardioversion in patients with CIEDs, an anteroposterior position of the patches or paddles is preferred to minimize current shunting to the leads. With current biphasic shocks and use of bipolar leads, electrical cardioversion can be performed safely [17,18]. The shock can also be delivered by the ICD which minimizes the risk of ICD damage during the external shock.

Cardiopulmonary resuscitation

Cardiopulmonary resuscitation (CPR) can be safely performed in all patients with a CIED. In ICD patients, internal shocks can occur but pose no risk for the resuscitator besides some uncomfortable tingling sensations [19]. These can be avoided by wearing gloves and avoiding contact with the patient's bed.

Insertion of central venous lines

To prevent damage to the leads or the device and to reduce infection risk, central venous lines are best inserted on the opposite site of the CIED. Inserting a pulmonary artery catheter should be done carefully not to dislocate the leads and preferably under fluoroscopic guidance.

Surgery

During surgery, the use of electrocautery equipment can induce electromagnetic oversensing leading to pacing inhibition and/or inappropriate ICD shocks. Therefore, it is mandatory that the CIED patient is seen by a cardiac rhythm specialist at forehand to reprogram the pacemaker to an asynchronous pacing mode if the patient is pacemaker dependent and to switch ICD therapy off prior to surgery, with close perioperative cardiac monitoring [20].

Magnetic resonance imaging

Most new cardiac CIEDs are designed to be magnetic resonance imaging (MRI) conditional, meaning an MRI can be performed under predefined conditions. Of note, sometimes the pulse generator can be MRI conditional, but the leads might be older and MRI non-conditional. In this case, the system is considered as MRI non-conditional. Currently, the MRI conditional CIEDs are only approved for MRI's with a strength up to 1.5 and some up to 3 Tesla. Before MRI, ICD therapy should be switched off and the pacing mode needs to be reprogrammed to an asynchronous mode (pacing dependent patients) or turned off [21]. Most CIEDs now also have a programmable 'MRI mode'. Importantly, during MRI, monitoring is indicated and the CIED should be interrogated afterwards to rule out any device or lead failure. Recently, MRI has also been found to be feasible and safe in non-conditional CIEDs with the

use of a prespecified safety protocol [22]. Patients with abandoned leads, fractured leads or epicardial leads should not undergo an MRI [21]. In case of a recent implantation (< 6 weeks) benefits should be carefully weighed against potential risks.

End of life care

During end of life care, pacemakers and CRT's don't need to be reprogrammed or turned off as they prevent disabling symptomatic bradycardia and don't prevent or delay natural death [23] . In contrast, ICD therapy should be switched off to avoid unnecessary shocks and discomfort.

CONCLUSION

This review discussed an approach to common CIED problems in the ICU. The key messages are summarized in the central figure.

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CONFLICT OF INTEREST:

The authors declare that there are no conflicts of interest.

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TABLES

Table 1 Basic concepts of device therapy

Device	Common class I guideline indications	Goal	Normal behaviour	Magnet behaviour*
Pacemaker	Symptomatic sinus node disease 2nd degree AV block, Möbitz type II 3rd degree AV block Atrial fibrillation with slow AV conduction	Prevent bradycardia	Pacing if HR is below programmed lower rate Ventricular tracking of atrial rate in DDD mode	Asynchronous pacing
ICD	Symptomatic HF with LVEF ≤ 35% Unprovoked VA with haemodynamic compromise	Prevent SCD	ATP or shock if ventricular arrhythmia is detected Pacing if HR is below programmed lower rate Ventricular tracking of atrial rate in DDD mode	Switch off tachy-arrhythmia detection
CRT	LVEF ≤ 35% and LBBB > 130 ms LVEF < 40% with pacemaker indication and expected high ventricular pacing rate	Resynchronize LV Prevent bradycardia Prevent pacing cardiomyopathy	Continuous biventricular pacing Pacing if HR below programmed lower rate Ventricular tracking of atrial rate in DDD mode	CRT-P: Asynchronous pacing CRT-D: Switch off tachy-arrhythmia detection

References [24–27]. AV: atrioventricular; ATP: anti-tachy pacing; CRT: cardiac resynchronisation therapy; HF: heart failure; HR: heart rate; ICD: implantable cardioverter defibrillator ; LVEF: left ventricular ejection fraction; SCD: sudden cardiac death; VA: ventricular arrhythmia.

* In both Boston Scientific and Abbott devices, magnet response can be programmed 'OFF'

Table 2: Effect of common drugs on pacing and defibrillation thresholds

	Pacing threshold	Defibrillation threshold
Anti-arrhythmics		
1. Class Ia		
- Disopyramide	=/↑	=
- Procainamide	=/↑	=
- Quinidine	=/↑	↑
2. Class Ib		
- Lidocaïne	=	↑
- Mexiletine	=/↑	↑
3. Class Ic		
- Flecaïnide	↑	↑
- Propafenone	↑	=
4. Class II		
- Propanolol	=	↑
- β1-selective BB	=	=
5. Class III		
- Sotalol	=	↓
- Amiodarone		
○ Oral	=	↑
○ Intravenous	=	=
- Dronedarone	=	=
6. Class IV		
- Verapamil	=	↑
- Diltiazem	=	↑
Vasopressors and inotropes		
Isoproterenol	↓	↓
Dobutamine	↓	↓
Noradrenaline	↓	↓
Adrenaline	↓	=/↑*
Milrinone	ND	ND
Levosimendan	ND	↑
Others		
Digoxin	=	=
Atropine	↓	↑

* Adrenaline can decrease defibrillation threshold when administered during cardiac arrest by ventricular fibrillation. References: [28–30]. BB: beta blocker; ND: no data.

Table 3: Causes of undersensing

Lead dislodgment
Lead disfunction: insulation defect or fracture
Metabolic: hyperkalaemia, acidosis
Class Ic anti-arrhythmic drugs
Myocardial infarction
Change of intrinsic signal: bundle branch block, VT, VF (ventricular) and AF (atrial)
Pulse generator failure

Table 4: Causes of unsuccessful shock

Increased defibrillation threshold: - Drugs - Metabolic: hyperkalaemia, hypocalcaemia, hypomagnesemia - Myocardial ischaemia/hypoxia - Pneumothorax - Pleural effusion **Device failure:** - Pulse generator failure - Shock lead failure: dislodgement, insulation defect, lead fracture, loose setscrew

FIGURES

Central figure: Overview of cardiac device troubleshooting on the ICU

BiV pacing: biventricular pacing; CO: cardiac output; CRT: cardiac resynchronisation therapy; CRT-P: CRT pacemaker; CRT-D: CRT defibrillator; ECG: electrocardiogram; ICD: implantable cardioverter defibrillator; ICU: intensive care unit; SVT: supraventricular tachycardia; VT: ventricular tachycardia

Figure 1: Common pacemaker malfunctions

Panel A: Loss of capture in a patient with permanent atrial fibrillation and VVI pacemaker with lower rate set at 70 bpm. None of pacemaker spikes followed by ventricular activation and a slow ventricular escape rhythm. Note the sensing is preserved as the first ventricular complex resets the pacing interval of the subsequent pace.

Panel B: No right ventricular output in a patient with a pacemaker in DDD mode and sinus tachycardia that suddenly develops complete AV block.

Panel C: Undersensing in VVI pacemaker mode with lower rate 60 bpm. The pacemaker is pacing at a constant rate of 60 bpm in a patient with sinus rhythm at 80 bpm. Intrinsic ventricular activation does not alter the pacing cycles, indicating absence of sensing. Capture is preserved as every pacing spike is followed by ventricular activation, except for those in a refractory period during the ST-segment (arrowheads). A fusion beat can also be noted (asterix).

Figure 2: Approach to pacemaker loss of capture

* Normal impedance depends on lead type and manufacturer. In general: High impedance is > 2000 Ohm or > 300 Ohm increase. Low impedance is < 200 Ohm or > 300 Ohm decrease**.**

Figure 3: Approach to no pacemaker output

*Asynchronous pacing modes should be programmed above the patient's own heart rate. #Normal impedance depends on lead type and manufacturer. In general: High impedance is > 2000 Ohm or > 300 Ohm increase. Low impedance is < 200 Ohm or > 300 Ohm decrease**.**

Figure 4: Electrocardiographic features of right ventricular apical pacing and biventricular pacing

Right apical pacing typically has a left superior axis with a negative complex in the inferior leads and a positive complex in lead I, aVL and aVR. In the precordial leads V1-4 is negative, while V5-6 can be both negative or positive. In contrast, biventricular pacing has a right superior axis with a negative complex in I and a positive complex in aVR. Lead V1 has a positive complex.

Figure 5: Approach to ICD therapy

* Prior episodes of potential subclinical ventricular arrhythmias should also be assessed for appropriate detection and therapy. EGM: electrogram; EM: electromagnetic; ICD: implantable cardioverter defibrillator.

Figure 6: T-wave oversensing

Supraventricular tachycardia with 1:1 conduction and oversensing of T-waves leading to detection of ventricular fibrillation (FD) at a rate twice the supraventricular rate and charging of the defibrillator. On the bottom an inappropriate shock (CD) of 36.4J is delivered. Ab: atrial sense in blanking period; Ar: atrial sense in refractory period; CE: Charge ended; Fs: ventricular sense above fast ventricular tachycardia rate; Vs: ventricular sense.

Figure 7: Lead noise oversensing

Oversensing of noise (asterix) in between ventricular signals (arrowheads) caused by lead dysfunction, leading to false detection of ventricular fibrillation and charging of the defibrillator.

Figure 8: Pacemaker pseudo malfunctions

Panel A: Sinus tachycardia above the upper tracking rate of 130 bpm leading to a peudo-Wenkebach 4:3 block. Note there are atrial fusion beats, indicated by an atrial pace (Ap). As: atrial sense; Ar: atrial sense in refractory period.

Panel B: Sinus tachycardia in a pacemaker dependent patient with every other atrial beat falling in a refractory period (Ar), leading to a 2:1 block.

Panel C: The ECG on top shows the imitation of a pacemaker mediated tachycardia (PMT) by a premature ventricular beat (PVC) (arrow), followed by a sudden ventricular paced tachycardia at the upper tracking rate (130 bpm). On the bottom, the EGM tracing shows 2 consecutive PVC's (arrowhead and arrow), followed by a retrogradely conducted atrial activation (asterix), initiating a PMT. Eventually the PMT is recognized by a device algorithm that pauses ventricular pacing, restoring normal rhythm. As: Atrial sense; BP: biventricular pace.

Panels A and B were reproduced from Medtronic Academy with permission.