Atrial pacing to suppress ventricular arrhythmias in the critically ill patients: a case report

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Background	Atrial and ventricular arrhythmias are common in the critically ill due to a variety of factors including sepsis, myocardial ischaemia, renal dysfunction, and electrolyte disturbances. Anti-arrhythmic medications can be useful to control arrhythmias but can result in bradycardia and haemodynamic compromise. A paced atrial rhythm alongside normal atrioventricular conduction can be helpful to treat bradycardia, prevent arrhythmias, and support cardiac output.
Case summary	A 55-year-old gentleman with pseudomonas pneumonia, respiratory failure necessitating mechanical haemodynamic support, and subsequent coronary ischaemia presented to the intensive care unit. Paroxysms of atrial fibrillation and ventricular arrhythmias caused haemodynamic embarrassment and presented an ongoing clinical challenge as anti-arrhythmic medications resulted in bradycardia and Torsade de Pointes. Atrial pacing mediated intrinsic conduction via the His-Purkinje system inhibited ventricular ectopy and further arrhythmia breaking the tachycardia—bradycardia cycle; this stabilized the patient, facilitated ongoing intensive therapy unit care and promoted recovery.
Conclusion	Atrial pacing mediated intrinsic conduction via the His-Purkinje system is an effective approach to suppress ventricular ectopy and sustained arrhythmias whilst protecting the patient from haemodynamically compromising bradycardia.
Keywords	Atrial pacing • Ventricular arrhythmias • Atrial fibrillation • Bradycardia • Pneumonia • Case report
ESC Curriculum	3.2 Acute coronary syndrome • 5.3 Atrial fibrillation • 5.6 Ventricular arrhythmia • 5.9 Pacemakers

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Learning points

- Amiodarone is often employed to suppress atrial and ventricular arrhythmias; however, it is a class III anti-arrhythmic agent (with additional class II action) which can cause bradycardia, hypotension, and the prolongation of QT interval, leading to Torsade de pointes.
- Atrial pacing is a helpful approach to protect against medication-induced bradycardia in patients with normal atrioventricular conduction.
- Atrial pacing mediating intrinsic conduction (and activation of the ventricles via the His-Purkinje system) can suppress ventricular ectopy and Torsade de pointes in order to support cardiac output and ongoing intensive therapy unit care.

Introduction

Cardiac arrhythmias are common in critically ill patients especially if superimposed ischaemic events occur. The milieu of sepsis, vasopressors, renal hypoperfusion, electrolyte disturbances, and side effects of medications, triggers and maintains atrial and ventricular arrhythmias. Amiodarone is an effective medication to control arrhythmias but can induce bradycardia and hypotension.[1] Atrial pacing is useful to treat bradycardia and to control both atrial and ventricular arrhythmias.[2] We describe a case report depicting the importance of this therapeutic intervention.

Timeline

Time	Events
Local intensive care unit (ICU) Days 0–5	Admission to ICU with right-sided community acquired necrotizing pneumonia and multiple pulmonary emboli, following a viral prodrome.
Cath lab Day 4	Inferior ST elevation myocardial infarction, drug eluting stent (DES) to left circumflex coronary artery (LCx).
Day 5	Respiratory deterioration requiring intubation and invasive ventilation.
Tertiary centre referral ICU Days 6–75	Veno-venous extracorporeal membrane oxygenation (VV-ECMO).
Day 11	Atrial fibrillation (AF) with ventricular rate 140–150 bpm, ventricular ectopy (VE), and a run of ventricular tachycardia. Increasing noradrenaline requirement (mean arterial pressure 59 mmHg). Amiodarone loading given.
Cath lab. Day 12	Repeated angiogram—patent LCx stent and left anterior descending disease treated with 2 DES.
Day 13	AF with fast ventricular rate terminated with esmolol and resultant bradycardia.
Day 16	VF arrest following episode of Torsade de Pointes (corrected QT interval 550 ms). One cycle of CPR and direct current cardioversion followed by return of spontaneous circulation. Post arrest ECG—AF with high VE burden.
Day 23	Persistent VE, AF, and bradycardic episodes with increase in noradrenaline requirement.
Day 24	**Insertion of atrial pacemaker, AAI 90 bpm**
Day 26–30	Cessation of VE, AF and reduction in noradrenaline requirement.
Day 47	Decannulated from ECMO.
Day 56	Insertion of secondary-prevention implantable cardioverter defibrillator
Local ICU Day 75	Repatriated to local ICU

Case presentation

A 55-year-old Caucasian gentleman presented to his local emergency department with a 1-week history of common cold, with symptoms of myalgia, fatigue, headaches, pleuritic chest pain, and breathlessness on exertion. He had no relevant past medical history apart from mild asthma, controlled on Beclomethasone 100 µg, 2 puffs bd, and salbutamol 100 µg, 2 puffs prn, and he was an ex-smoker, of 25-pack years. On examination, his body mass index was 24.6 kg/m^2, blood pressure, 130/80 mmHg, pulse, 110 bpm, regular, and temperature of 38.2°C. Chest auscultation revealed decreased air entry on the right lung with coarse crackles. Cardiovascular examination showed normal heart sounds and no murmurs. Admission 12-lead electrocardiogram (ECG) showed sinus tachycardia at 113 bpm, PR interval of 124 milliseconds (ms), QRS duration 99 ms, corrected QT interval (QTc) of 382 ms, and no ST-T wave abnormalities. Initial blood tests showed serum creatinine of 85 umol/L, alanine aminotransferase, 216 IU/L, potassium level, 5.4 mmoL/mL, magnesium, 1.2, corrected calcium, 2.26, sodium, 138, C-reactive protein, 184 mg/L, haemoglobin, 86 g/L, (normocytic, normochromic), platelets, 670 × 10^9/L, WBC, 18 × 10^9/L, (neutrophilia), international normalised ratio 1.4, and activated partial thromboplastin time 27.2 s. Initial chest radiograph showed right sided consolidation, and both sputum and blood cultures revealed growth of *Pseudomonas aeruginosa*. He was diagnosed with a community acquired *P. pneumonia*, treated with oxygen, intravenous fluids, and antibiotics, meropenem and clarithromycin.

Four days later, he developed central chest pain and was diagnosed with an inferior ST elevation myocardial infarction. Coronary angiogram indicated an occlusion to the mid segment of left circumflex artery (LCx) requiring placement of a drug-eluting stent, as well as bystander left anterior descending (LAD) disease, not necessitating intervention. He suffered from atrial fibrillation (AF) and ventricular bigeminy post-procedure, both were paroxysmal and self-terminating. Dual antiplatelet therapy, aspirin and ticagrelor, was commenced.

Over the following 24 h, the patient suffered rapid respiratory deterioration requiring intubation and ventilation. Despite maximum oxygenation, bronchoscopy, diuresis, and antibiotic escalation, he remained difficult to oxygenate with worsening type 1 respiratory failure even with proning attempts and was unresponsive to inhaled nitric oxide. COVID-19 tests were negative. Murray[3] score, or lung injury score, based on a set of parameters including extent of alveolar consolidation, hypoxaemia, positive end-expiratory pressure, and respiratory compliance score denoted severe lung injury with a score of 2.5. RESP score, which predicts survival of patients receiving extracorporeal membrane oxygenation (ECMO) for respiratory failure was 5, indicated 76% survival after ECMO.[4] A decision was made to establish veno-venous ECMO therapy and retrieve to a severe acute respiratory failure tertiary centre.

Chest computed tomography showed multiple segmental and sub-segmental pulmonary emboli affecting the left lower lung lobe and right lower lobe anterior sub-segmental embolus, but with no evidence of right heart strain. The right lung exhibited features of necrotizing pneumonia, pleural effusion, and the left showed minimal basal consolidation and peripheral ground glass opacification (*Figure 1*).

Transthoracic echocardiogram showed severely impaired left ventricular systolic function and mild dilatation of the right ventricle with
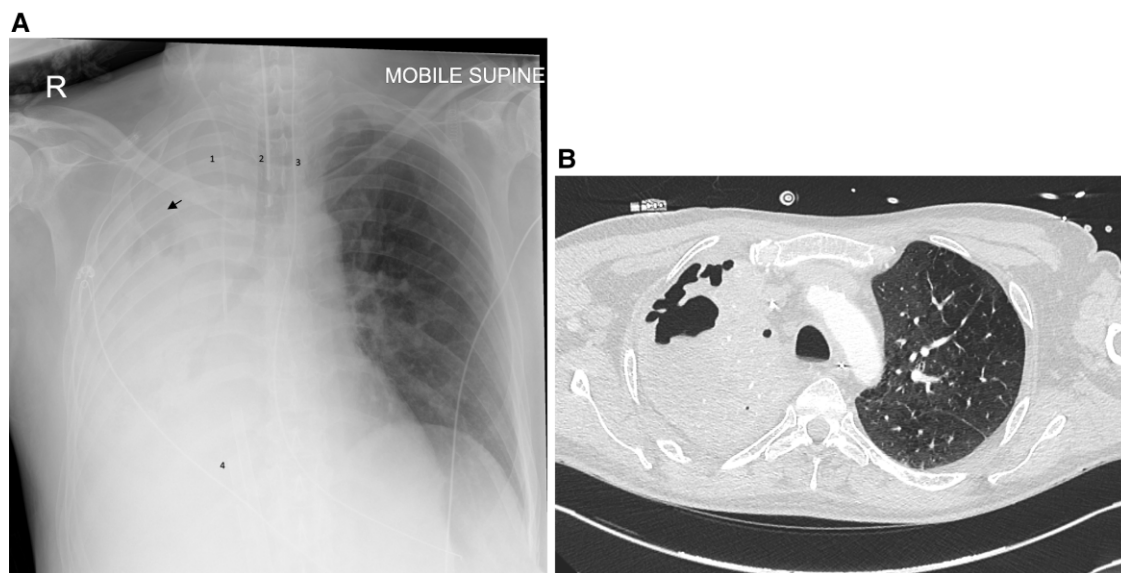
Figure 1 Necrotizing right-sided pneumonia. (*A*) Chest radiograph after transfer to tertiary centre, Day 6. Note the opacification of the entire right lung field denoting consolidation, with cavitation seen in the right upper lobe (arrow). Also note the endotracheal tube, denoted by 1; nasogastric tube, 2; right internal-jugular central line, 3; and two extracorporeal membrane oxygenation cannulae, 4 in the inferior vena cava-right atrium junction. (*B*) Chest computed tomography, Day 6. Note the consolidation with multiple gas locules and hypoattenuating foci scattered throughout the right lung section.

moderate to severe impairment of systolic function. Cardiac output was 3.5 L/min, and pulmonary pressures were not significantly raised. Twelve-lead ECG showed sinus rhythm with T-wave inversion in the inferior leads and QTc of 515 ms. Amiodarone loading dose, 300 IV followed by 900 mg IV, was used to control AF and ventricular ectopy (VE), and it was the likely cause of QT prolongation, in addition to recent ischaemia. Antibiotic regimen was amikacin, ceftazidime, and linezolid, and these were less likely to cause QT prolongation. No vasopressors other than noradrenaline were used. Serum electrolytes at this time showed potassium level of 5.3 mmoL/mL, magnesium, 1.6, corrected calcium, 2.49, and temperature was 36.1°C.

Over the next week, he suffered from further episodes of AF with rapid ventricular rates, 140–150 bpm, increased burden of VE, with persistent periods of ventricular bigeminy, and an episode of self-terminating ventricular tachycardia (VT) with a drop of mean arterial pressure below 59 mmHg and increased noradrenaline requirements (0.06–0.18 µg/kg/min). Electrolyte levels and renal function were continuously optimized by electrolyte replenishment and initiating renal replacement therapy. Amiodarone reloading, in addition to metoprolol 25 mg Ter Die Sumendus/three times a day, alternating with esmolol 50 mcg/kg/min, was administered to suppress arrythmias; however, this resulted in bradycardia, hypotension, first degree atrioventricular (AV) block, and further prolongation of the QTc to 550 ms. Lidocaine infusion was ineffective at reducing VE burden.

His worsening cardiogenic shock was likely the result of episodes of AF, runs of VT, and frequent VE on a background of severely impaired left ventricular systolic function. In an effort to exclude ischaemia as a possible cause, a repeat coronary angiography showed

patent LCx stent, and two drug-eluting stents were placed in the LAD to optimize perfusion (*Figure 2*); however, arrhythmia burden did not improve. Short-acting beta-blockers were restarted, with the addition of amiodarone when QTc is below 480 ms. As a consequence of amiodarone infusion, prolongation of the QTc to 550 ms induced an episode of Torsade de pointes deteriorating to ventricular fibrillation, responsive to electrical cardioversion (*Figure 3*), after 1 min of cardiopulmonary resuscitation. Spontaneous circulation was restored, and post-arrest ECG showed AF with high VE burden.

He was challenging to manage, with beta-blocker and amiodarone treatment resulting in sinus pauses and bradycardia compromising the cardiac output needed during his critical illness and possibly increasing susceptibility to AF, VE, and QT prolongation. A multidisciplinary team decision was made to place an atrial pacemaker, with atrial pacing rate set at 90 bpm to facilitate intrinsic conduction, support cardiac output, and suppress AF and VE. A 52 cm-lead was placed through the left internal Jugular vein to the right atrial appendage, with good measurements (Impedance, 560 ohms, p wave amplitude, 4.5 mV, and pacing threshold, 0.5 V at 0.4 ms) and connected to an externalized pacemaker with pacemaker code for atrial pacing (AAI) pacing mode. Other alternative vascular access sites could be left or right subclavian or axillary veins. The right internal Jugular vein was reserved for the central venous line.

The patient's condition improved over the following weeks, with a reduction in vasopressor support, decreased arrythmia burden, and normalization of QTc interval (<460 ms) (*Figure 4*). The subsequent stay in the critical care unit was uneventful. The patient was decannulated from ECMO following a 41-day run. Given his recurrent haemodynamically significant ventricular arrhythmias and severely impaired left ventricular function, decision was made to implant a
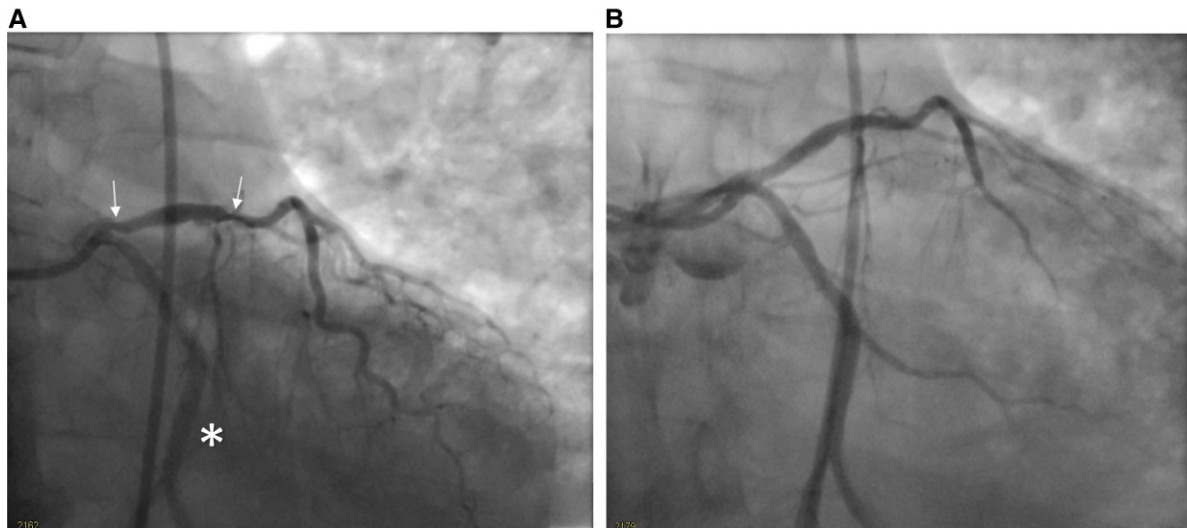
Figure 2 Left coronary angiogram (*A*) before and (*B*) after percutaneous coronary intervention to left anterior descending artery. Note the two tight lesions in osteal and mid left anterior descending artery (arrows). Left circumflex artery stent shown in mid segment (asterisk).
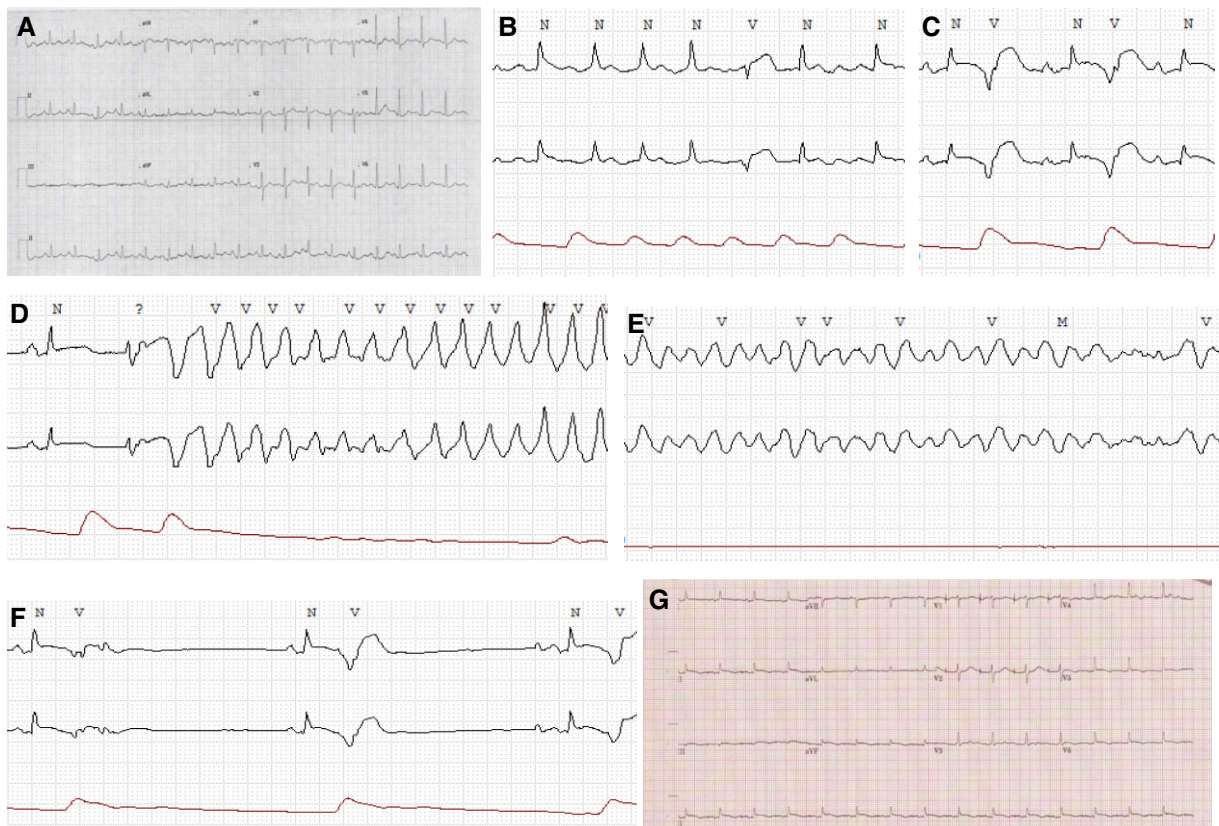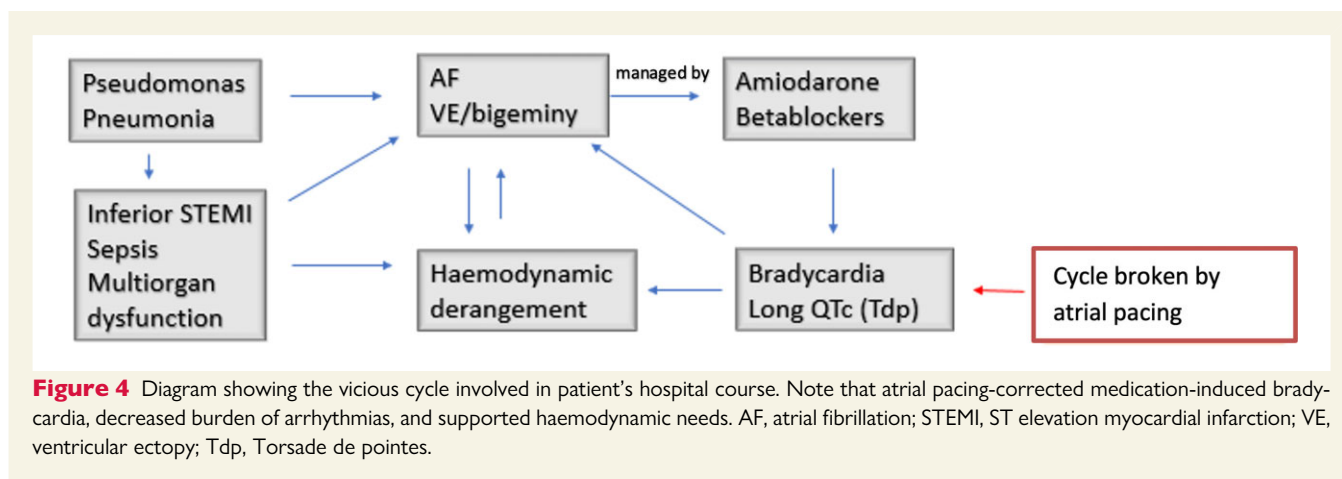

Figure 3 Rhythm monitoring at different time points, including ECG and telemetry strips with invasive blood pressure tracing. (*A*) Twelve-lead ECG on admission showing sinus tachycardia and normal corrected QT interval (382 ms). (*B*) Episode of paroxysmal atrial fibrillation with ventricular ectopic beat. (*C*) Sinus rhythm with first degree AV block and ventricular bigeminy. (*D*) Sinus rhythm with initiation of Torsade de pointes due to an early-coupled ventricular premature beat, degenerating into (*E*) ventricular fibrillation. (*F*) Sinus bradycardia with ventricular ectopy falling in the vulnerable period of repolarization on the preceding T wave. (*G*) Twelve-lead ECG showing atrial pacing followed by intrinsic conduction and a physiological, normal morphology and duration of QRS without ventricular ectopy.

Figure 4 Diagram showing the vicious cycle involved in patient's hospital course. Note that atrial pacing-corrected medication-induced bradycardia, decreased burden of arrhythmias, and supported haemodynamic needs. AF, atrial fibrillation; STEMI, ST elevation myocardial infarction; VE, ventricular ectopy; Tdp, Torsade de pointes.

secondary prevention implantable cardioverter-defibrillator. He was then discharged to his local hospital for continued rehabilitation. Unfortunately, the patient died after he was repatriated to his local hospital. He died of sepsis.

Discussion

Pseudomonas aeruginosa pneumonia has been associated with thromboembolic events and acute ST elevation myocardial infarction.[5] Critically ill patients often have frequent atrial and ventricular arrhythmias due to increased triggered activity, caused by multiple factors including sepsis, ischaemia, metabolic derangement, autonomic dysfunction, and effect of medications.[6] The development of bradycardia was due to combined effect of both betablockers and amiodarone in trial to control arrhythmias. AF, with fast ventricular rates, and persistent ventricular bigeminy compromised the hemodynamic state. Intravenous amiodarone helps to suppress both atrial and ventricular arrhythmias but also causes bradycardia, hypotension, QT prolongation, and negative inotropic effects.[1,7] Other antiarrhythmic medications as short-acting betablockers helped to reduce arrhythmias, but they caused further bradycardia and hypotension. Class Ic drugs like lidocaine was tried but was not helpful in this case, and other alternative would have been mexiletine to supress VE. Atrial pacing is a well-known therapeutic option to suppress atrial and ventricular arrhythmias through inhibition of ectopic beats which can act as precursors for more sustained arrhythmia.[2,8] The use of atrial pacing in our patient was crucial to break the vicious cycle of arrhythmias and medication-induced bradycardia permitting safer use of antiarrhythmic medications and suppressing triggered activity causing atrial and ventricular arrhythmias.[9] Atrial pacing was equally important to help mediate an increase in cardiac output in the face of multi-organ failure and sepsis.

Patients require 1:1 AV node conduction in order for an atrial paced rhythm to be consistently conducted through the His-Purkinje network to the ventricular myocardium. In this capacity, atrial pacing can indeed be very helpful to suppress VE and sequential Torsade de Pointes, that can ensue in these situations. If there is evidence of high-degree AV block, a backup (right) ventricular pacing lead is often required. In patients with a compromised cardiac output, a temporary dual chamber system may offer benefit due to preserved AV synchrony. However, minimizing ventricular pacing is important in patients with impaired heart function as it is well-known that RV pacing is deleterious in these patient groups[10] and can indeed be pro-arrhythmogenic.

Acquired QT interval prolongation in this case can be due to amiodarone, heart failure, and ischaemia,[11,12] and these could have triggered the episode of Torsade de pointes.[13] Other important causes include hypokalaemia and hypomagnesemia, antibiotics as macrolides and fluoroquinolones, antifungals, antidepressants, neuroleptics and prokinetic agents, and none of these were used in our case.[12] Cardiac pacing has also demonstrated use in supressing ventricular arrhythmias in congenital long QT syndrome.[14] An implantable cardioverter defibrillator is recommended to prevent sudden death in patients who experience haemodynamically unstable ventricular arrhythmias, in the absence of a reversible cause, especially for those with LV function less than 35%.[15]

Conclusion

Forced atrial pacing mediated intrinsic conduction via the His-Purkinje system can mediate an effective treatment to suppress dangerous ventricular arrhythmias, prevent bradyarrhythmias, and support cardiac output in critically ill patients.

Lead author biography

Omar Riad is a clinical fellow in interventional cardiac electrophysiology at Royal Brompton Hospital, Guy's and St Thomas' NHS foundation trust, London, UK, and a clinical lecturer of cardiology at Ain Shams University Hospitals, Cairo, Egypt. He is interested in the care of patients with rhythm abnormalities including catheter ablation, pacing, and device therapy for heart failure. He had his MD degree at Ain Shams University with joint supervision from Imperial College London, about radiofrequency and cryoballoon ablation of atrial fibrillation.

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