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Bradycardic peri-arrest after electrical cardioversion for atrial fibrillation: a case report

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Abstract

Electrical cardioversion can precipitate prolonged bradycardia and cardiac arrest when the sinoatrial node is dysfunctional, and the escape rhythms from the atrioventricular node and ventricles fail to take over. We report the case of an elderly male who rapidly progressed into bradycardic peri-arrest after electrical cardioversion for persistent atrial fibrillation. Despite the immediate initiation of intravenous atropine and transcutaneous pacing, 2 minutes of cardiopulmonary resuscitation with further boluses of atropine and adrenaline were required before the return of spontaneous circulation. The patient was transferred to the intensive care unit, and a permanent pacemaker was implanted before discharge. Advanced age, long duration of atrial fibrillation, previous aortic valve replacement, use of anti-arrhythmic drugs, and intravenous propofol bolus were all contributing factors to this event. We summarise the relevant clinical features, risk factors, and management considerations, in the hope of promoting awareness of this rare complication of a common procedure.

Keywords: atrial fibrillation, cardiac arrest, cardiac pacing, electrical cardioversion

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Introduction

Electrical cardioversion is a relatively safe and efficient procedure that has been used to terminate atrial fibrillation (AF) since the 1960s.[1] However, bradyarrhythmia is a known complication of electrical cardioversion for AF.[2] A retrospective multicentre study conducted by Gronberg *et al.* investigating 7,660 cases of electrical cardioversion for AF showed that bradyarrhythmia occurred in 0.9% of cases, with 0.2% being bradycardia alone and the rest being asystole followed by a period of bradycardia.[2] Among the cases that resulted in asystole, most of them were of brief duration and recovered spontaneously, though 7 (0.09%) required resuscitation and 2 (0.02%) required external pacing. Another study by Shin *et al.* evaluated a total of 1,100 cases of electrical cardioversion and showed a bradyarrhythmia rate of 0.7%, where 0.3% received permanent pacemaker implantation.[3] Despite the rareness of this complication, it has the potential to cause serious harm to the patient, which marks the need for educational effort regarding its clinical features and prompt management.

Case presentation

An 83-year-old man underwent his first elective electrical cardioversion after failed pharmacological treatment for persistent rate-controlled AF, which lasted for at least 12 months and was symptomatic with shortness of breath. Past medical history included gastro-oesophageal reflux disease, hypothyroidism, aortic valve replacement, and ischaemic heart disease requiring angioplasty and coronary artery bypass grafts. His regular medications were amiodarone, bisoprolol, clopidogrel, apixaban, furosemide, ezetimibe, rosuvastatin, rabeprazole, thyroxine, and paroxetine. There was no history of presyncope or syncope suggesting bradyarrhythmia. Recent echocardiography reported normal left ventricular size and function, moderate mitral regurgitation, biatrial enlargement, and normal right ventricular size with mildly reduced systolic function. The bioprosthetic aortic valve replacement was well seated with normal haemodynamics. There was no previous anaesthetic complication. Besides the narrow-complex AF shown on electrocardiogram (ECG), the patient's physical examination and investigations were otherwise unremarkable. He consented to electrical cardioversion under monitored sedation.

In the catheterisation laboratory, the patient's monitoring included ECG, non-invasive blood pressure, pulse oximetry, and capnography through the side port of a Hudson oxygen mask (Salter Labs, MI, USA). His observations were within normal limits, and AF with a rate of 72 beats per minute (bpm) was confirmed on

cardiac monitoring. Sedation was induced with 100 mg of intravenous propofol, and the patient's blood pressure, heart rate, and capnography were maintained within normal limits after loss of consciousness.

After synchronisation, a direct current shock of 200 J was discharged through pads placed on the anterior chest wall and upper back. The slow AF rapidly progressed into a ventricular escape rhythm at a rate of 25 bpm without any signs of atrioventricular (AV) node escape rhythm. The cardiologist was able to initiate transcutaneous pacing through the cardioversion pads with minimal delay, while intravenous atropine was administered simultaneously by the anaesthetist. The demand pacing heart rate was set to 50 bpm with a maximum output of 140 mA. ECG connections and pacing pads were confirmed to be in the correct position with good skin contact. The femoral arterial pulse was palpated with a gloved hand to check for mechanical capture of external pacing.

Despite the presence of spontaneous breathing confirmed with capnography, no palpable femoral pulse or brachial blood pressure could be obtained after administration of 1.2 mg intravenous atropine. The patient was therefore considered to be in a state of bradycardic peri-arrest, and cardiopulmonary resuscitation (CPR) was initiated along with an intravenous bolus of 500 mcg of adrenaline. A laryngeal mask airway (i-gel, Intersurgical® Berkshire, UK) was inserted with minimal interruption. Return of spontaneous circulation, as evidenced by a strong central pulse, was achieved at 2 minutes after starting CPR. The first systolic blood pressure recorded after the return of spontaneous circulation was 220 mmHg with a heart rate of 120 bpm still in AF. An urgent focused echocardiogram was performed that showed an ejection fraction of approximately 50%. He was transferred to the intensive care unit after regaining consciousness.

During his intensive care stay, he developed sinus bradycardia at a rate of 40 bpm with intermittent junctional bradycardia that required isoprenaline and noradrenaline infusion to maintain his blood pressure. A permanent pacemaker was implanted 2 days later for persistent bradyarrhythmia.

Discussion

This unexpected case of bradycardic peri-arrest highlights the risk where profound bradycardia with compromised cardiac output can result from a combination of delayed sinoatrial (SA) node recovery and failure of AV node junctional rhythm.

The bradycardic peri-arrest was not anticipated in this patient, who was at narrow complex AF with a rate of 72 bpm and no history suggestive of unstable ischaemia or bradycardia. The low-to-medium dosage of amiodarone 200 mg daily and bisoprolol 2.5 mg daily in this patient with normal renal and hepatic functions was unlikely to be a direct causative factor in this event. In hindsight, the long duration of AF, advanced age, previous aortic valve replacement, use of anti-arrhythmic drugs, especially amiodarone, and the bolus dose of propofol may have all cumulatively contributed to this event.[4]

A list of causes for dysfunctional SA node, AV node, and ventricular escape are shown in Table 1. The duration of AF is one of the major predictive factors for arrhythmic complications after electrical cardioversion, which should be taken into consideration in this case given the patient's long history of AF.[5] It is also important to note that patients with underlying degenerative cardiac conduction disease such as AF with controlled ventricular response, in the absence of rate controlling agent on board, can develop significant bradycardia or sustained ventricular arrhythmias upon cardioversion.[6] Age-related degeneration and fibrosis are among the most common causative factors, while cardiac ischaemia and various cardiac procedures can also pose risks to the intrinsic pacemakers depending on the area involved.[7] A study involving 20,725 patients with AF undergoing their first electrical cardioversion found that the rate of developing bradyarrhythmia within 30 days ranges from 0.5% to 5.1% as the patient's age increases from 40 to 90 years.[8]

This case also points out the possibility that transcutaneous pacing can be inefficacious despite measures to reduce impedance, in which scenario anaesthetists should be vigilant and prepared to initiate resuscitation. Figure 1 demonstrates our modified approach to resuscitation for bradycardic peri-arrest, incorporating recommendations from guidelines and senior clinicians. It is not intended to be a substitute of current guidelines, but a summary of experience gained from reflecting on the case described in this report. It suggests that electrical treatments can be initiated simultaneously with pharmacological treatments in the setting of bradycardic peri-arrest. Failed mechanical capture, which resembles pulseless electrical activity, may occur in myocardial stunning or metabolic derangements that raise the pacing threshold.[9] Failed electrical capture may occur when there is suboptimal electrode placement or poor skin contact due to sweat or hair.[9] Early recognition of the high-risk patients and allocation to appropriate care settings, such as the catheterisation laboratory, is required to facilitate temporary or permanent transvenous pacing that may be initiated under expert guidance.

Table 1. Risk factors for intrinsic cardiac pacemaker dysfunction[4-8]

	Sinoatrial node	**Nodal escape (atrioventricular node)**	**Infra-Hisian escape (including Purkinje and ventricular escape)**
Depolarisation rate (bpm)	60–100 Narrow complex	40–60 Narrow complex	20–40 Broad complex
Common risk factors for dysfunction	• Long-standing conduction disorders • Advanced age/idiopathic fibrosis • Medication toxicities: beta-blocker, calcium-channel blocker, digoxin, amiodarone, local anaesthetic systemic toxicity • Metabolic derangements: hyperkalaemia, hypermagnesaemia, thyroid dysfunction, hypocalcaemia, hypoxia, hypothermia • Infiltrative disorders: amyloidosis, sarcoidosis, haemochromatosis • Infectious diseases: infective endocarditis, Lyme disease, Chagas disease • Inflammatory and autoimmune conditions: rheumatic heart disease, scleroderma, systemic lupus erythematosus		
Specific risk factors for dysfunction	• Excessive vagal tone • Spinal cord injury • Carotid sinus syncope • Sodium channel mutations • Ischaemia involving proximal right coronary artery	• Excessive vagal tone • Spinal cord injury • Carotid sinus syncope • Sodium channel mutations • Tricuspid valve replacement • Transcatheter aortic valve implantation • Aortic valve replacement or repair • Hypertrophic obstructive cardiomyopathy • Inferior wall ischaemia • Ticagrelor-induced bradycardia • Pre-existing left bundle branch block	• Myocarditis • Congenital heart disease • Septal ischaemia

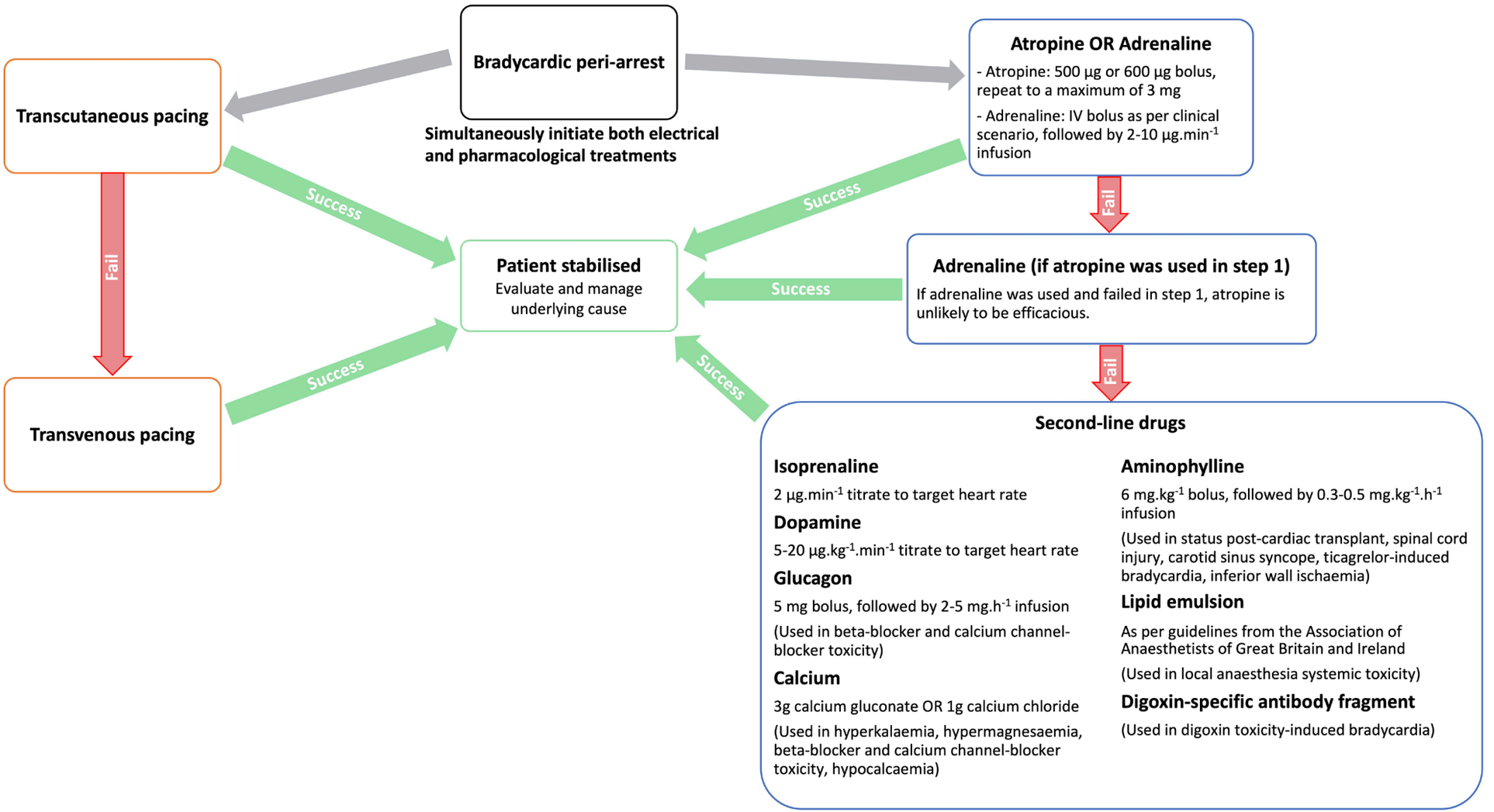

Fig. 1. Modified resuscitation for bradycardic peri-arrest.[4,10]

Atropine and adrenaline are the mainstay pharmacological treatments for bradycardic peri-arrest, as they can achieve rapid reversal of the pathophysiology. Atropine is the preferred first-line drug in many guidelines, it is readily available and can quickly counteract nodal dysfunction due to excessive vagal tone but may be ineffective in other situations, such as Mobitz type II second-degree heart block and third-degree heart block with a low Purkinje or ventricular escape rhythm.[4] Atropine may also be inefficacious in patients with cardiac transplants, as the denervated heart will not respond to vagal blockade, and probably will not work if adrenaline has already failed as the first-line drug. The role of intravenous adrenaline bolus in this case is probably more significant as it stimulates the entire myocardium despite the cause of the bradyarrhythmia.[4] Under circumstances where atropine is potentially inefficacious, such as the aforementioned higher degree heart block with ventricular escape rhythm, adrenaline may be used as an alternative first-line agent during the early course of CPR without contradicting the current guidelines.[10]

Second-line medications may be appropriate in some clinical scenarios, such as glucagon in beta-blocker or calcium channel-blocker toxicity and lipid emulsion in local anaesthetic systemic toxicity.[10] Bradyarrhythmia induced by electrolyte disturbances may be treated with calcium chloride or calcium gluconate. Aminophylline should be considered in bradyarrhythmia complicated by spinal cord injury, inferior wall myocardial infarction, and status post-cardiac transplant.[10] Other second-line options, including isoprenaline and dopamine, may be used at the clinician's discretion. Moreover, the sinus and junctional bradycardia reported during intensive care stay could be further complicated by Torsades de Pointes as a result of QT interval prolongation. The use of isoprenaline infusion and subsequent pacemaker implantation have effectively reduced the risk of this potential complication.

Conclusion

This report highlights the risk factors contributing to the development of bradycardic peri-arrest after electrical cardioversion for AF and discusses potential treatment options. Simultaneous initiation of electrical and pharmacological interventions is encouraged. Failure to capture can occur during pacing, in which scenario anaesthetists should be prepared to initiate resuscitation. Atropine and adrenaline are generally used first line, though the choice of drug may vary depending on the underlying cause.

Declarations

Informed consent for publication
Informed, sufficient, and express consent from the patient for the use of their clinical data, images, and/or other medical information consigned in the article was obtained.

Competing interests
None to declare.

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