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**CASE REPORT**
*Arrhythmias/electrophysiology*

# Case report: Managing profound circulatory collapse post-atrial fibrillation ablation: a methodical approach

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**Background** Circulatory collapse during/post-pulmonary vein (PV) isolation by cryo-balloon ablation is a Cardiology emergency that has multiple potential causes and requires a methodical investigative approach. Some of the complications that can arise include cardiac tamponade, bleeding/vascular injury, anaphylaxis, Addisonian crisis, acute pulmonary embolism, acute PV stenosis, oesophageal injury, and vagal reaction.

**Case summary** Here, we present a case of a 76-year-old lady who developed profound circulatory collapse during an elective pulmonary vein isolation by cryo-balloon ablation for symptomatic paroxysmal atrial fibrillation (AF). Cardiac tamponade, bleeding/vascular injury, and other less common causes were excluded. She only responded transiently to fluid resuscitation and developed intermittent bradyarrhythmias and hypotension which responded to isoprenaline. She was discharged home at Day 3 post-AF ablation after remaining well and continued to do so at follow-up.

**Discussion** Circulatory collapse during/post-PV cryo-balloon ablation is a Cardiology emergency that has multiple potential causes. The ganglionate plexi form part of the cardiac intrinsic autonomic nervous system (ANS) and are located close to the left atrial–PV junctions. The presence of vagal response has been observed to be a marker of ANS modulation although its significance on the long-term outcome post-ablation has yet to be elucidated. The true cause of our patient's profound circulatory collapse is uncertain but a vital learning point in this case is the systematic exclusion of common and potentially life-threatening complications following AF ablation. A persistent vagal reaction secondary to PV cryo-balloon ablation can usually be managed with supportive medical therapy as demonstrated in our case.

**Keywords** Vagal reaction • Atrial fibrillation • Pulmonary vein isolation • Cryoballoon ablation

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## Learning points

- Profound circulatory collapse during/post-pulmonary vein (PV) cryo-balloon ablation is a Cardiology emergency and requires a methodical investigative approach in order to make a prompt diagnosis and initiation of treatment.
- Some of the complications that can arise include cardiac tamponade, bleeding/vascular injury, anaphylaxis, Addisonian crisis, acute pulmonary embolism, acute PV stenosis, oesophageal injury, sinus node artery injury, and vagal reaction.
- Persistent vagal reaction secondary to PV cryo-balloon ablation has been described due to the close proximity of the ganglionic plexi (part of the cardiac intrinsic autonomic nervous system) to the left atrial–PV junctions.
- The vagal reaction is usually short-lasting and self-limiting and even persistent hypotension and bradyarrhythmias tend to resolve with supportive medical therapy.

# Introduction

Circulatory collapse during or post-pulmonary vein (PV) isolation by cryo-balloon ablation for atrial fibrillation (AF) is a Cardiology emergency that has multiple potential causes and requires a methodical investigative approach.[1] Some of the complications that can arise include cardiac tamponade, bleeding/vascular injury, anaphylaxis, Addisonian crisis, acute pulmonary embolism, acute PV stenosis, oesophageal injury, sinus node artery injury, and vagal reaction. Here, we present a case of a 76-year-old lady who developed profound circulatory collapse during an elective pulmonary vein isolation (PVI) by cryoballoon ablation for symptomatic paroxysmal AF.

# Timeline

| Day | Events |
|---|---|
| 0 | • Cardiology Day Unit: A 76-year-old lady attended her elective pulmonary vein (PV) isolation by cryo-balloon ablation for symptomatic paroxysmal atrial fibrillation. The patient experienced transient drops in blood pressure (80/50 mmHg with no change in sinus rate) during each PV freeze, which responded well to intravenous (IV) fluids. The patient developed further recurrent profound hypotension associated with reduced consciousness and metabolic acidosis (pH 7.2). Intravenous adrenaline (100 mcg) was administered to maintain a systolic blood pressure >100 mmHg. She received 6 L of IV fluids (including 1 unit of packed red cells), protamine 50 mg IV, and 2500 units of prothrombin complex concentrate (Octaplex) to treat a presumed acute bleeding event. Cardiac tamponade was ruled out. |
| 0 | • Coronary Care Unit (different hospital site): She was urgently transferred for further investigations and treatment which ruled out bleeding/vascular injury, acute pulmonary embolism, acute PV stenosis, oesophageal injury, and Addisonian crisis. She developed sinus bradycardia with a junctional escape rhythm at a rate of 30 b.p.m. with intermittent pauses. She responded to atropine and isoprenaline infusion. |
| 1 | • Stabilized and no further bradyarrhythmia. Isoprenaline infusion stopped. |
| 3 | • In sinus rhythm. Discharged. |
| 30 | • Well and remained in sinus rhythm on clinic follow-up. |

# Case presentation

A 76-year-old lady with a 7-year history of symptomatic paroxysmal AF underwent a percutaneous AF ablation (PVI). Her background includes hypertension and chronic obstructive pulmonary disease. The procedure was performed using the 28 mm second-generation cryo-balloon (Medtronic Arctic Front, USA) under local anaesthesia and conscious sedation. Right femoral venous access was

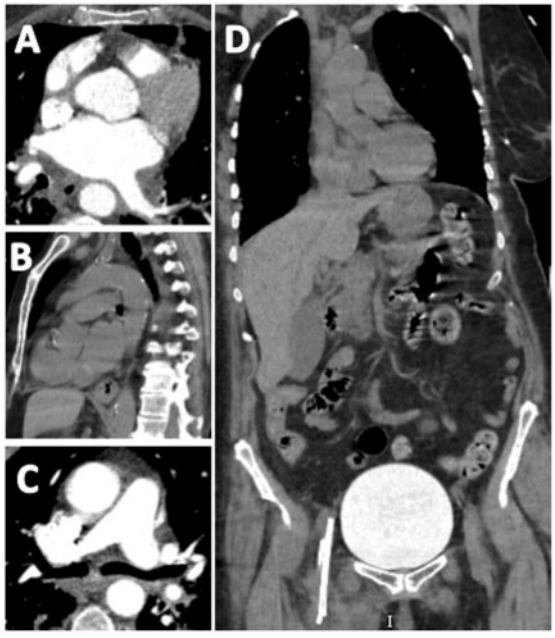


**Figure 1** Computed tomography chest, abdomen, and pelvis with contrast. (*A*) Cross-section at the level of the left atrium demonstrating patency in the left lower pulmonary vein (blue arrow) and right upper pulmonary vein (red arrow) as well the absence of an atrial-pericardial fistula. (*B*) Sagittal view of the heart showing no obvious oesophageal injury (red arrow pointing to the oesophageous) and the absence of pericardial effusion (blue arrow pointing to the pericardium). (*C*) Cross-section at the level of the main pulmonary arteries demonstrating the absence of pulmonary embolism (blue arrow pointing to the left pulmonary artery and red arrow pointing to the right pulmonary artery). (*D*) Coronal view demonstrating no obvious source bleeding. Note the femoral sheaths (blue arrow) in the right femoral vein that was kept as a form of central venous access while the patient was stabilized.

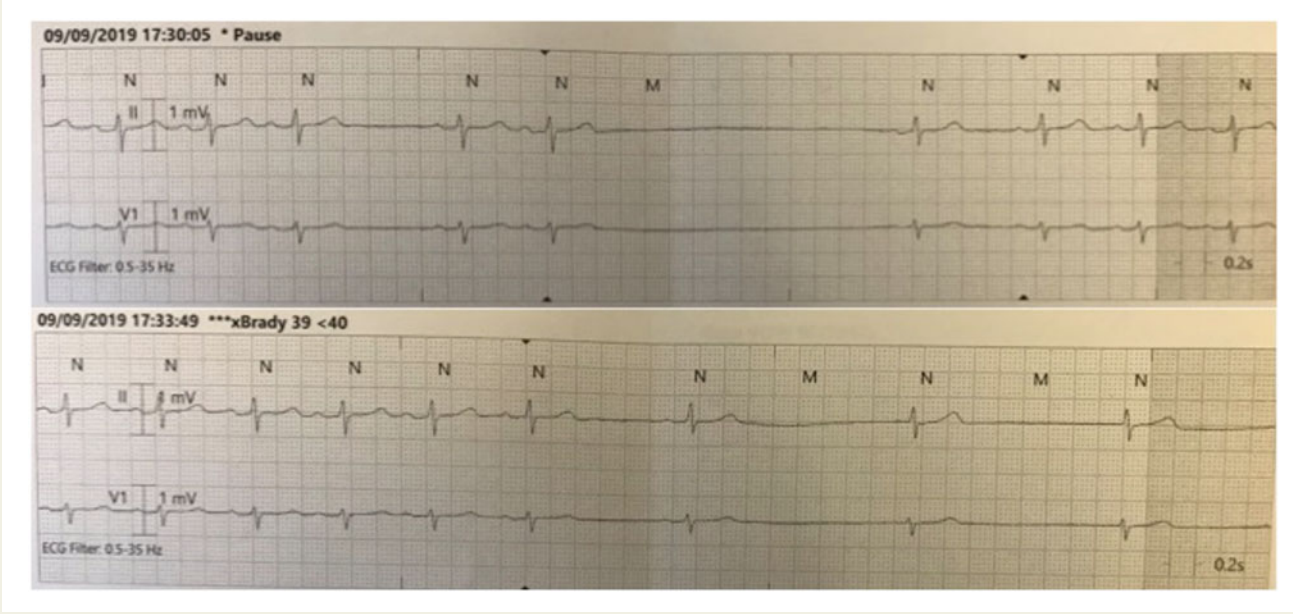


**Figure 2** Electrocardiogram—sinus bradycardia with junctional escape rhythm. Telemetry of the patient in Coronary Care Unit (CCU) demonstrated episodes of bradyarrhythmia in the form of sinus bradycardia with pause and junctional escape rhythm.

uncomplicated and obtained under ultrasound guidance. A FlexCath sheath (Medtronic, USA) and HeartSpan® transseptal needle (Merit, USA) was used for a single uncomplicated transseptal puncture (under the guidance of fluoroscopy, pressure monitoring, contrast flow into the left atrium, and aspiration of arterial blood). Pulmonary venography revealed standard left and right PV anatomy and cryoablation was performed to each PV sequentially with PVI confirmed using the Achieve™ mapping catheter (Medtronic, USA). The temperature and freeze time were as follows: left upper PV (-43°C for 180 s), left lower PV (-49°C for 180 s), right lower PV (-48°C for 180 s), and right upper PV (-47°C for 180 s). Atropine 600 mcg IV was administered prior to commencing freezing as per our routine clinical practice to reduce the incidence of peri-procedural vagal events. The patient experienced transient drops in blood pressure (80/50 mmHg with no change in sinus rate) during each PV freeze, which responded well to intravenous (IV) fluids; she went into sustained AF during the right lower PV freeze (heart rate remained stable at 80 - 100 b.p.m. in sustained AF). The electrical isolation of all four PVs was confirmed with the Achieve™ mapping catheter at the end.

Intra- and post-procedure transthoracic echocardiography did not demonstrate any pericardial effusion; phrenic nerve stimulation (using diaphragm compound motor action potential and manual palpitation) was performed during isolation of the right-sided PVs and was fine throughout. Immediately post-ablation (after all catheters were withdrawn from the left atrium) the patient developed profound recurrent hypotension (systolic blood pressure 50 mmHg) which responded transiently to IV fluids. Her physical examination was unremarkable and her femoral access site appeared fine with no clinical evidence of haematoma. The patient then developed further recurrent profound hypotension responding initially to IV fluids but was associated with the reduced conscious level and metabolic acidosis (pH 7.2). The patient was given IV adrenaline (100 mcg) to maintain a systolic blood pressure >100 mmHg. She received 6 L IV fluids (including 1 unit of packed red cells), protamine 50 mg IV, and 2500 units of prothrombin complex concentrate (Octaplex) to treat a presumed acute bleeding event.

She was urgently transferred to our coronary care unit (at a different hospital site) and spontaneously reverted to sinus rhythm. An emergency computed tomography (CT) scan of the chest, abdomen, and pelvis with contrast (*Figure 1*) was performed which excluded any bleeding source, oesophageal injury, pulmonary embolism, and acute PV occlusion. An acute Addisonian crisis was also excluded.

She developed a further hypotensive episode (systolic blood pressure 50 mmHg) which responded once again to IV fluids. Her electrocardiogram at that time revealed sinus bradycardia with junctional escape rhythm at a rate of 30 b.p.m. an intermittent pauses (*Figure 2*). She was given an IV bolus of Atropine 600 mcg and was commenced onto an isoprenaline infusion. She stabilized following this and the isoprenaline infusion was stopped the following day. She was discharged home at Day 3 post-AF ablation after remaining well. She was followed up in the clinic 1 month later and continued to remain well and in sinus rhythm.

## Discussion

Hypotension following an AF ablation can be a sign of a potentially serious complication and applying a systematic investigative approach can usually rule out the majority of causes.[1] The most common life-threatening complication associated with AF ablation is cardiac tamponade.[2] Our patient underwent repeated transthoracic echocardiography during and after the procedure which revealed good biventricular systolic function with no significant valvular abnormality and no pericardial effusion.

Vascular complications leading to hypotension are another common potential problem arising from AF ablation and include groin

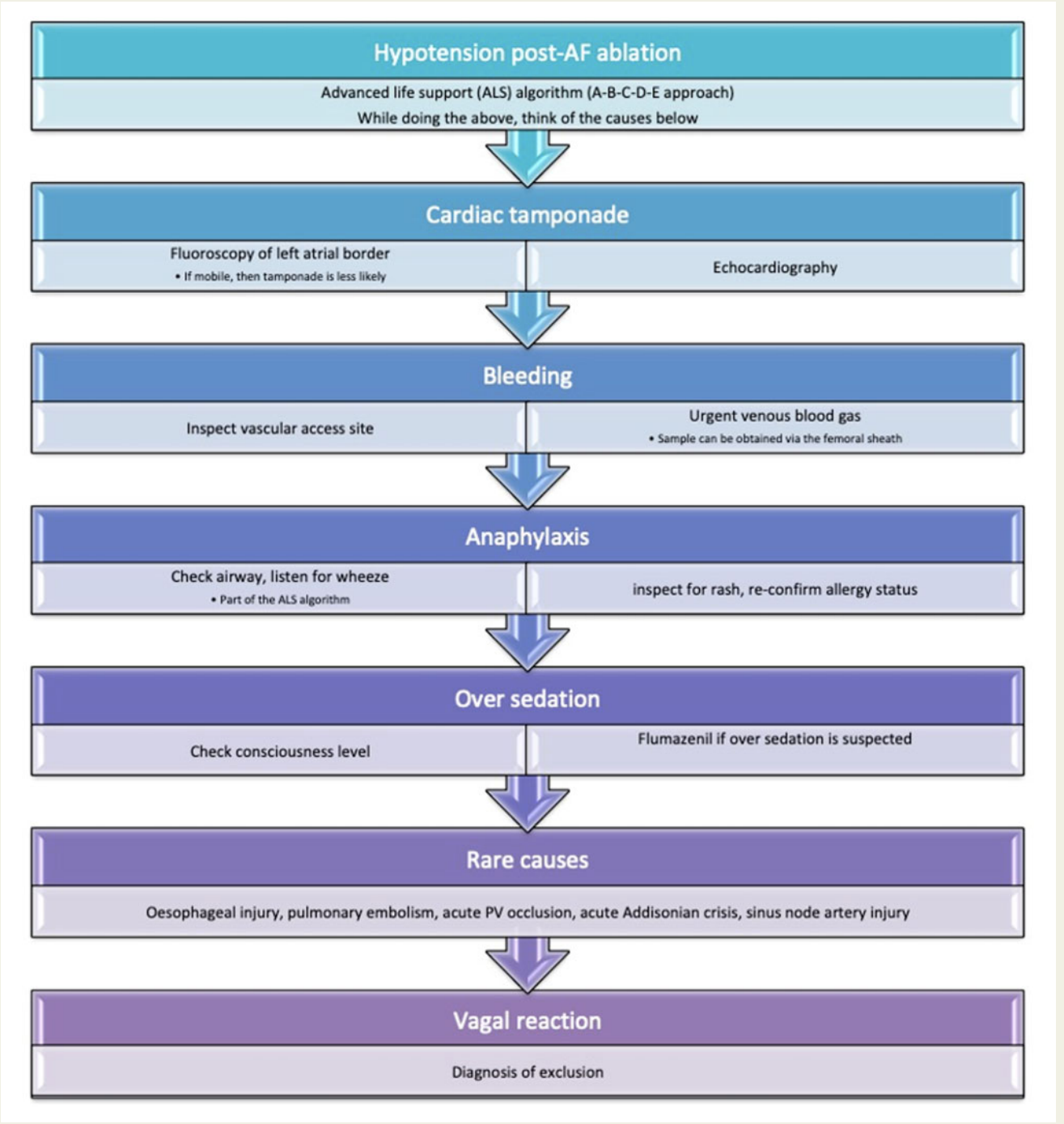


**Figure 3** Step-wise approach for the diagnostic evaluation of a patient with hypotension post-atrial fibrillation ablation.

haematoma, pseudoaneurysm, arterio-venous fistula, and retroperitoneal bleeding. Another important cause to exclude is an acute large pulmonary embolism but this would cause right ventricular dilatation (not seen in our case). Furthermore, our patient had been on uninterrupted oral anticoagulation and received IV heparin cover during the procedure. Rare causes of hypotension that are vital to consider include atrio-oesophageal or atrial-pericardial fistula,[3] or acute PV occlusion.[4] To exclude the aforementioned potential complications, an urgent CT scan of the chest, abdomen, and pelvis with contrast (*Figure 1*) was performed which excluded any bleeding source, oesophageal injury, pulmonary embolism, and acute PV occlusion. An acute Addisonian crisis was also excluded (normal serum cortisol, sodium, and potassium levels). Another rare complication is an injury to the sinus node artery but this is unlikely due to the spontaneous recovery of the patient. A suggested step-wise approach for the diagnostic evaluation of a patient with hypotension post-AF ablation is shown in *Figure 3*.

As our patient's investigations have excluded the majority of potentially life-threatening complications, one possible explanation behind her profound circulatory collapse was a persistent vagal reaction secondary to PV cryo-ablation independent of a drop in heart rate.

Potentially significant anatomical structures close to the left atrium-PV junctions are the ganglionated plexi (GPs) which form an intricate part of the cardiac intrinsic autonomic nervous system (ANS) and act as a neural interconnection system between the cardiac extrinsic and intrinsic ANS.[5] The left atrial GPs are located in the epicardial fat pads at four preferential locations close to the left atrium-PV junctions. Interestingly, GP ablation has been described as an adjunctive strategy to achieve a lower AF recurrence in patients undergoing radiofrequency-based PVI for AF where the presence of vagal reactions are thought to reflect intrinsic cardiac ANS modulation.[6] The prevalence of vagal reactions in cryo-ablation PVI using the second-generation cryo-balloon has been reported to be as high as 40% and some consider the presence of vagal reactions to be a marker of cardiac ANS modification.[7] However, robust studies on the mechanism and significance of vagal reactions on the long-term outcome post-PVI ablation are lacking and remain to be elucidated. Nevertheless, vagal reactions post-PVI ablation have been found to be usually transient and self-limiting but if treatment is required, then temporary measures such as atropine, isoprenaline, or temporary cardiac pacing can be employed.[8]

## Conclusion

One potential cause of our patient's profound circulatory collapse was a persistent vagal reaction secondary to PV cryo-ablation which was managed with supportive medical therapy after methodically excluding other potentially life-threatening complications.

## Lead author biography

Prof. Osman is Consultant Cardiologist/Electrophysiologist at University Hospital Coventry and Warwickshire NHS Trust. He set up a fully comprehensive Arrhythmia Service including all ablations and complex device implants/extractions in 2008. He was appointed Honorary Professor of Cardiology with Warwick Medical School in 2018 and has supervised several research PhDs. His main areas of interest are AF, ablation, CRT for heart failure, and sudden cardiac death risk prediction.

## Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidelines.

**Conflict of interest**: none declared.

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