Case Report

# Epinephrine-induced electrical storm after aortic surgery


**ABSTRACT**

Electrical storm (ES) is a potentially lethal syndrome defined as three or more sustained episodes of ventricular tachycardia or ventricular fibrillation within 24 h. There are multiple inciting factors for ES, one of which involves excess catecholamine (endogenous and exogenous) effects. Exogenous catecholamines used for hemodynamic support can paradoxically engender or exacerbate an underling arrhythmia leading to ES. We report on an 63-year-old man who presented for repair of an ascending aortic dissection. After cardiopulmonary bypass separation assisted with high-dose epinephrine, ES developed requiring over 40 defibrillatory shocks. The epinephrine infusion was held and within 5 min, the ES self-terminated. ES in the context of cardiovascular surgery with the use of epinephrine for hemodynamic support has not be previously reported. Clinicians need to be cognizant of the seemingly paradoxical effect of epinephrine to induce ES. Initial ES treatment involves acute stabilization (treating or removing exacerbating factors (i.e., excess catecholamines)).



**Key words:** Electric storm; ventricular fibrillation; ventricular tachycardia


## Case Presentation

A 63-year-old man with no prior cardiac history presented for ascending aortic aneurysm repair. After uneventful anesthesia induction and initiation, cardiopulmonary bypass (CPB) was instituted, the aorta was cross-clamped, cardioplegia was administered, and the ascending aorta was incised. Aortotomy revealed a chronic circumferential ascending aortic dissection sparing the aortic valve and root. A 32-mm valve-sparing aortic graft was implanted. Following cross-clamp removal, uncontrolled hemorrhage from both graft ends occurred necessitating CPB reinstitution with the plan to perform a Bentall procedure. After cooling to 18° C, deep hypothermic arrest was initiated, and the failed graft was explanted. A 27-mm composite graft with a mechanical valve was implanted. The left coronary button was reanastomosed to the graft, the right coronary artery that constituted the posterior descending artery was identified, and a vein graft was used for its reanastomosis.

After proximal and distal anastomoses completion, rewarming, and cross-clamp removal, the patient's cardiac rhythm was ventricular fibrillation (VF) and a 20-J internal shock was administered. The patient had an accelerated junctional rhythm with a rate of 80–90 beats/min. All electrolytes were normalized and the following vasoactive infusions were initiated: Epinephrine 0.2–0.3 μg/kg/min, milrinone 0.75 μg/kg/min, vasopressin 0.04 units/min, and norepinephrine 0.2–0.3 μg/kg/min. CPB cannulas were removed and protamine was administered. Within 10 min of CPB separation, VF reoccurred requiring defibrillation. In total,


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over 40 episodes of VF occurred necessitating defibrillation. The cardiac rhythm was junctional in the interval between VF episodes. Two 150-mg boluses of amiodarone were administered and urgent electrophysiologist consultation suggested epinephrine infusion cessation. Within 5 min from ceasing all epinephrine administration, the electrical storm (ES) had terminated no further and the patient remained in an accelerated junctional rhythm [Figure 1] for the duration of the perioperative interval.

Postoperatively, the patient developed multisystem organ dysfunction necessitating renal-replacement therapy as well as implantation of a right ventricular assist device for right ventricular dysfunction. However, the patient had no further tachyarrhythmia episodes.

## Discussion

Electrical storm (ES) is defined as either three or more episodes of sustained ventricular tachycardia (VT) or VF within 24 h or two discreet VT/VF events at least 5 min apart but within a single hour or multiple discreet ventricular tachyarrhythmia events in <5 min.[1,2] ES is the most lethal of all cardiac arrhythmias; hence, early recognition, etiology determination, and prompt treatment are imperative.[3]

Multiple endogenous and exogenous factors may precipitate ES. Pharmaceutical agents, especially certain antiarrhythmics (i.e., Vaughan–Williams Class I and III agents) and agents associated with QT interval prolongation, are known triggers.[1] Postinfarction myocardial scar can predispose the conduction system to tachyarrhythmias and patients with implantable cardioverter-defibrillators (ICDs; signifying advanced cardiac disease) are at high risk with an ES incidence approaching 20%.[4] During cardiac surgery, ischemia, premature ventricular contractions (PVCs), low perfusions states, and electrolyte disturbances are all associated with endogenous catecholamine surges, which may be arrhythmogenic.[4] Beneficial alpha- and beta-agonism of exogenous catecholamines may not outweigh their negative arrhythmogenic effects if a patient is stable or between arrhythmia events in the setting of ES.

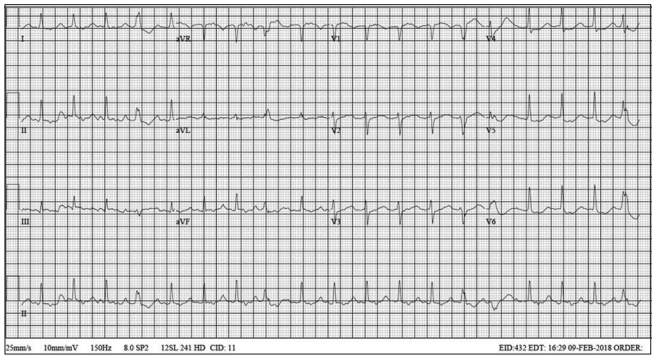


**Figure 1: EKG demonstrating the patient's accelerated junction rhythm upon cessation of epinephrine and a period of 40 episodes of ventricular fibrillation**

Catecholamines significantly alter the electrophysiological homeostasis of the cardiac conduction system and during a surge or high-dose infusion, latent genetic conditions may be unmasked.[5] Exogenous epinephrine may cause impaired repolarization, prolong QT intervals, and alter intracellular calcium and potassium concentrations, which if occurring during the T-wave, may precipitate a malignant ventricular arrhythmia.[6] ES generation is likely an aggregate of exogenous catecholamine administration, hypothermia, endogenous catecholamine surges (i.e., stress or ischemia), and latent genetic abnormalities.

With regard to hypothermia, Baderstscher *et al*. reported on a 31-year-old male who suffered cardiac arrest and was placed on a hypothermic protocol for neuroprotection.[6] With the onset of hypothermia, PVC generation followed by 10 episodes of VF requiring defibrillation occurred. The authors report that ES was incited after initiation of hypothermia and ceased once cooling was stopped.[6] While not fully understood at the cellular level, underlying "J wave syndromes" may also be a reason for VF with hypothermia.[7] Yamaki *et al*. present a patient with J waves who entered VF at low room temperature. J-point elevation was induced or exacerbated by hypothermia resulting in VF in those with early depolarization syndromes.[7]

Rapid treatment of ES is imperative with the time between ES recognition and treatment linearly related to increasing mortality.[8] Nademanee *et al*. reported on 49 ES cases divided into two treatment groups: Anti-catecholamine treatment once stabilized (sympathetic blockade) versus standard guideline-driven approach and found the former demonstrating significantly improved survival.[9] Failure to recognize ES, or confusing and treating a non-VT/VF with calcium channel blockade, may result in further arrhythmia degeneration.[4] Appropriate pharmacological treatment options are important to not only stop ES but also prevent recurrence. Beta-blockade may be useful post-ES; propranolol has been reported to reduce arrhythmia events by up to 50% via elevating the arrhythmia threshold and counteracting excess beta-agonism.[10] Other pharmacological treatment options include amiodarone, beta-blockers other than propranolol, lidocaine, magnesium, and propofol.[1,4]

Amiodarone, a class-III antiarrhythmic, partly acts to curtail norepinephrine release, block fast sodium channels, and

block L-type calcium channels.[11] With prolonged infusions, amiodarone actions on potassium channels has been reported to increase the refractory period and lengthen action potentials.[12] Amiodarone use may be the preferred treatment for refractory rhythm derangements of VF/VT in the acute setting; however, despite its proposed benefits, it can be associated with arrhythmia induction (i.e., Torsade de Pointes).[12] Makimoto *et al*.[12] described a 48-year-old woman with a dilated cardiomyopathy administered amiodarone for recurrent VT. The morphology of the inciting PVC had changed, and her VT was prolonged. After withdrawal of amiodarone and the addition of nifekalant, the VT resolved.

Our case of ES due to epinephrine administration after aortic surgery is the first to be reported. Clinicians should be aware of epinephrine's ability to incite or aggravate a malignant arrhythmia syndrome such as ES. The initial management of ES, aside from appropriate ACLS measures, should include the following: Amiodarone, beta-blockade, electrolyte normalization, catecholamine avoidance and potentially suppression (i.e., stellate ganglion blockade), followed by long-term therapy, such as ICD placement, catheter ablation, or cardiac assist devices as a bridge to transplant if no other options exist.[11]

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### Conflicts of interest
There are no conflicts of interest.

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