Review Article

Induced Therapeutic Hyperkalemia as a Method for Treatment of Refractory Arrhythmias after Aortic Declamping in Cardiac Surgical Procedures: A Narrative Review

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Abstract

Cardiac surgical procedures are among the most complex operations with a detailed list of perioperative care. Weaning from CPB is associated with a number of disturbances in cardiac rhythm. One of the most challenging cardiac rhythm disturbances is the ventricular arrhythmia (tachycardia/fibrillation). Cardioversion and antiarrhythmic agents may be ineffective after aortic declamping; systemic hyperkalemia may be a therapeutic option. Here, we review the available studies regarding this issue and the possible underlying mechanisms of this therapeutic approach.

Keywords: Electrophysiological Refractory Period; Arrhythmia; Hyperkalemia

Please cite this article as: Dabbagh A, Foroughi M, Imani A. Induced Therapeutic Hyperkalemia as a Method for Treatment of Refractory Arrhythmias after Aortic Declamping in Cardiac Surgical Procedures: A Narrative Review. J Cell Mol Anesth. 2019;4(3):96-9.

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Introduction

Cardiac surgical procedures are among the most complex procedures with a detailed list of perioperative care. One of the main procedures during cardiac surgery is the cardiopulmonary bypass (CPB); mandating a series of sophisticated tasks to be done in an appropriate order. However, CPB has its own disadvantages and events; weaning from CPB is among them and highly depends on the adequacy of different parameters including cardiac contractility, temperature, cardiac rhythm, and arterial blood gas. The most frequent disturbances in cardiac rhythm may be attributed to electrolyte imbalance, mechanical injury to conductive tissues, defect in myocardial preservation, ischemic reperfusion injury, and other cellular mechanisms. As teamwork, the surgeon and cardiac anesthesiologist should evaluate these differential diagnoses and rule out the possible etiologies one by one, as the treatment of each cause may be completely different from others. One of the most challenging rhythm disturbances is the ventricular arrhythmia (tachycardia/ fibrillation) in the ischemic-reperfusion setting. The commonly used approaches include cardioversion and a number of anti-arrhythmic agents; electrical cardioversion may lead to minor myocardial injury or dysfunction (1, 2); which is a challenge in daily practice in patients with underlying cardiac disease (3-5). What should be done if there is no response to these classic interventions while the patient is on CPB and usually, the adequate perfusion provided by the CPB machine, relieves our concerns about potential organ hypoperfusion; including the CNS?

Here, we reviewed the studies considering therapeutic hyperkalemia as the method for treatment of refractory arrhythmias (VT storm) occurring after aortic de-clamping until weaning from CPB.

Discussion

Successful treatment of ventricular fibrillation through direct use of potassium (K) into the heart chamber has been reported as early as 1961; it was an anecdotal case report during inguinal hernia repair in a 3 years old Babyboy (6). In 1979, Lazar, et al. performed an animal study on 20 samples and concluded that using a small dose of continuous blood cardioplegia leading to re-arrest of the heart leads to better outcomes in the reversal of VT storm, needing less consumption of energy for ventricular defibrillation (7).

In another experimental study, Marill et al studied the role of chemical defibrillation as an alternative approach for the treatment of intra-arrest VF waveform. They concluded that potassium supplemented by calcium (administered in their study as KCl+CaCl2) leads to results comparable with standard therapy regarding the return of spontaneous circulation (ROSC) and "post-ROSC hemodynamics" (8).

Yang et al, demonstrated an experimental model of resuscitation after cardiac arrest in rats, in which hyperkalemia could exert the following 4 results (9):

- promoted enlargement of the VF amplitude
- expedited spontaneous conversion
- raised the rate of successful defibrillation
- decreased the needed energy for VF defibrillation

In 1984, Robicsek reported a technique of re-clamping and infusing 37°C cardioplegia until reaching standstill; then pacing and returning to a normal rhythm in refractory ventricular tachycardia (10). In 1995, Ovrum, et al. reported a case series of 200 cardiac surgical patients in two groups (100 in each); one group has received 20 mEq of KCl into the aortic root and the other did not receive it. The result was the significant decreased need to use electrical energy for the treatment of post-declamping ventricular arrhythmias (11). Watanabe and colleagues injected a high concentration of 20 mEq KCl into the root of the aorta to treat refractory VF; they did this technique in 3 valve surgery patients after declamping of the aorta and needed no defibrillation in order to resolve sinus rhythm (12). Turkoz, et al. reported secondary cross-clamping added with an infusion of an extra dose of cardioplegia to treat VT storm (13, 14). Gadhinglajkar, et al. reported similar results regarding the use of warm cardioplegia into the root of the aorta in 3 adult patients undergoing aortic valve replacement surgery (15). In a clinical study on 8465 adult patients undergoing cardiac surgery patients with CPB, all the study patients having VF after declamping of aorta received potassium infusion (20 mmol plus an extra 10 mmol if needed). The patients in this study had a low rate of direct countershock and compared with other patient groups, had a lower rate of demand for pacing during weaning from bypass; the results suggested potassium infusion as an "effective and convenient first-hand measure" in treatment of post declamping VF in cardiac surgery patients undergoing CPB (16).

Underlying Mechanism

Refractory arrhythmia after CPB is a main clinical problem, which could be considered as a sign of an underlying imbalance in myocardial action potentials. In fact, during cardiac surgery with CPB, the infusion of cardioplegia to the root of the aorta is a common and long-lived practice, leading to Diastolic arrest, protect the myocardium, prepare motionless and bloodless field to facilitates meticulous operation (17); however, this approach leads to an abnormal imbalance in normal resting and action potential of myocardial cells. After the reinstitution of normal rhythm due to the vanished effects of cardioplegia, the ischemia-reperfusion effects are added to the cardioplegia-induced electrolyte imbalance (18-20). Nevertheless, how could we explain the antiarrhythmic effects of extra KCl doses?

One may consider the effects of adding extra KCl simply as an asystole-inducing agent; leading to asystole and after the normal action potential is reinstituted, the normal sinus rhythm initiates spontaneously (21). Though this seems a plausible explanation, there might be other underlying mechanisms. One main mechanism is the effects of potassium inward rectifying channels (KIR) on cardiac arrhythmias (22, 23). KIR channels constitute a simple but diverse ion channel superfamily of 7 subtypes (24). The first KIR has been cloned in 1993 (25); but research in this field is still awaiting novel and surprising results, especially in the field of cardiac excitability (26). Though they are found in a number of

organ tissues, KIR2, KIR3, and KIR6 are the cardiac-specific subtypes (23, 27, 28). Ventricular KIR channels have an important role in cardioprotection during ischemic preconditioning and in shortening of action potential during ischemia-reperfusion periods (29, 30). There are new perspectives for potential drug effects on myocardial KIR channels (31), which could be one of the main possible explanations for the antiarrhythmic effects of hyperkalemia inducing asystole after declamping of patients and treatment of refractory ventricular tachycardia.

In summary, the main electrophysiological effects of systemic hyperkalemia could be categorized under these three items (32):

- depolarization of resting membrane potential (Em) via less negative values of the Nernst equilibrium potential for K+ (EK)
- shortening of action potential duration
- changes in conduction velocity

Through increasing K+ channel conductance and inducing post-repolarization refractoriness, hyperkalemia can prolong the effective refractory period. Since activation and inactivation states of the Na+ fast channels depend on [K+]o magnitude, hyperkalemia induces biphasic effects on conduction velocity. In other words, it is increased at lower [K+]o (up to 8 mmol/L) and reduced at higher [K+]o levels (32).

Conclusion

Our proposed strategy was to create systemic hyperkalemia with or without inducing hypothermia. Infusion of high dose KCL (20 mEq) induces cardiac diastolic arrest and relief of ventricular arrhythmia. Once the cardiac arrest is well established, conventional ultrafiltration should be restored in such a way that hyperkalemia would be corrected gradually. The sinus normal rhythm appears in most of the patients; though other supportive considerations should be available like any other routine patient; including drips of antiarrhythmic agents; cardioverter defibrillator, and even, heart mechanical support such as intra-aortic balloon pump.

The systemic hyperkalemia is a known and relatively routine strategy for myocardial protection in redo surgeries with patent LIMA (left internal mammary artery) anastomosis in order to avoid manipulation around the LIMA area, as its damage would be associated with significant mortality. The disadvantage of this method is prolonging CPB time to remove the excess concentration of K from serum. Nevertheless, when there is no response to conventional treatment modalities, it could be the unique magic option.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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