Role of delayed afterdepolarization-induced triggered activity in the development of cardiac arrhythmias.

Scheen Timothy*

Department of Medical Sciences, University of Siena, Siena, Italy

Introduction

Cardiac arrhythmias are a group of disorders characterized by an abnormal rhythm of the heart, which can lead to serious complications such as heart failure, stroke, and sudden cardiac death. One of the mechanisms that can contribute to the development of cardiac arrhythmias is delayed afterdepolarization-induced triggered activity. Delayed After Depolarization (DAD) refers to a small depolarization that occurs in the cardiac myocyte during the diastolic phase of the cardiac cycle, after repolarization has been completed. DADs are caused by the release of calcium from the sarcoplasmic reticulum, which can activate the sodium-calcium exchanger and lead to a transient inward current that depolarizes the cell. Normally, DADs are suppressed by the action of the sodium-potassium pump, which removes the excess intracellular calcium and restores the resting membrane potential [1].

However, under certain conditions, such as increased intracellular calcium levels, sympathetic stimulation, or drugs that affect calcium handling, the amplitude and frequency of DADs can increase, leading to the generation of triggered activity. Triggered activity refers to a series of depolarizations that are initiated by a single event, such as a DAD, and can propagate through the myocardium, causing an arrhythmia. The role of DAD-induced triggered activity in the development of cardiac arrhythmias has been extensively studied in various animal models and in human cardiac tissue. Several mechanisms have been proposed to explain how DADs can trigger an arrhythmia, including re-entry, automaticity, and focal activity [2].

Re-entry is a mechanism that involves the formation of a circular path of conduction in the myocardium, which can sustain a propagating wave of depolarization. When a DAD occurs during the refractory period of the cardiac cycle, it can create a new wave front of depolarization that can propagate along a different path than the normal conduction system. If this path intersects with the original wave front, a reentrant circuit can be formed, which can lead to sustained arrhythmias such as ventricular tachycardia or fibrillation. Automaticity is a mechanism that refers to the ability of cardiac cells to generate spontaneous depolarizations in the absence of an external stimulus. When DADs become large enough to reach the threshold for action potential generation, they can trigger a series of depolarizations that can initiate an arrhythmia. This mechanism is particularly relevant in tissues that have a high level of sympathetic stimulation, such as the sinoatrial node or the Purkinje fibers [3].

Focal activity is a mechanism that involves the generation of a localized source of depolarization in the myocardium. When a DAD occurs in a single cell or a small group of cells, it can create a focal point of depolarization that can propagate to neighboring cells and initiate an arrhythmia. This mechanism is often observed in tissues that have a high level of heterogeneity in ion channel expression or calcium handling, such as the border zone between infarcted and healthy myocardium. The contribution of DAD-induced triggered activity to the development of cardiac arrhythmias can depend on the underlying disease or condition. For example, in heart failure, there is a dysregulation of calcium handling that can lead to an increase in DAD frequency and amplitude, as well as a decrease in the expression of the sodium-potassium pump. This can create a substrate for the formation of reentrant circuits or focal sources of activity, which can lead to ventricular arrhythmias [4].

In long QT syndrome, a genetic disorder that affects the repolarization of the cardiac action potential, DAD-induced triggered activity can play a role in the initiation of torsades de pointes, a type of ventricular tachycardia that can degenerate into ventricular fibrillation. In this condition, the prolonged repolarization can create a window of vulnerability during which DADs can occur and initiate an arrhythmia. The role of DAD-induced triggered activity in the development of cardiac arrhythmias has important clinical implications for the diagnosis and treatment of arrhythmias. Electrophysiological studies, which involve the insertion of catheters into the heart to record electrical signals, can be used to identify the presence and mechanism of DAD-induced arrhythmias. For example, the induction of ventricular fibrillation during an electrophysiological study can indicate the presence of a reentrant circuit or a focal source of activity [5].

Conclusion

Delayed afterdepolarization-induced triggered activity can play an important role in the development of cardiac arrhythmias. The mechanisms by which DADs can initiate an arrhythmia include re-entry, automaticity, and focal activity, and the contribution of DAD-induced arrhythmias can depend on the underlying disease or condition. Diagnosis

***Correspondence to:** Scheen Timothy. Department of Medical Sciences, University of Siena, Siena, Italy, E-mail: timothy.scheen@unisi.it

***Received:** 29-Mar-2023, Manuscript No. AAINIC-23-97689; **Editor assigned:** 31-Mar-2023, Pre QC No. AAINIC-23-97689(PQ); **Reviewed:** 14-Apr-2023, QC No. AAINIC-23-97689; **Revised:** 19-Apr-2023, Manuscript No. AAINIC-23-97689(R); **Published:** 26-Apr-2023, DOI:10.35841/aainic-6.2.143*

***Citation:** Timothy S. Role of delayed afterdepolarization-induced triggered activity in the development of cardiac arrhythmias. J Invasive Noninvasive Cardiol. 2023;6(2):143*

and treatment of DAD-induced arrhythmias can involve a combination of pharmacological and non-pharmacological interventions, and the choice of treatment can depend on the underlying mechanism and the risk of sudden cardiac death. Further research is needed to improve our understanding of the role of DADs in the pathophysiology of cardiac arrhythmias and to develop more effective and personalized treatments for patients with these conditions.

References

1. Tsuboi M, Antzelevitch C. Cellular basis for electrocardiographic and arrhythmic manifestations of Andersen-Tawil syndrome (LQT7). Heart Rhythm. 2006;3(3):328-35.
2. Gadsby DC, Cranefield PF. Electrogenic sodium extrusion in cardiac Purkinje fibers. J Gen Physioly. 1979;73(6):819-37.
3. Burashnikov A, Antzelevitch C. Late-phase 3 EAD. A unique mechanism contributing to initiation of atrial fibrillation. Pacing Clin Electrophysiol. 2006;29(3):290-5.
4. Garfinkel A, Kim YH, Voroshilovsky O, et al. Preventing ventricular fibrillation by flattening cardiac restitution. Proc Natl Acad Sci. 2000;97(11):6061-6.
5. Ogawa M, Morita N, Tang L, et al. Mechanisms of recurrent ventricular fibrillation in a rabbit model of pacing-induced heart failure. Heart rhythm. 2009;6(6):784-92.

Citation: *Timothy S. Role of delayed afterdepolarization-induced triggered activity in the development of cardiac arrhythmias. J Invasive Noninvasive Cardiol. 2023;6(2):143*