

## Direct acute effects of cyclosporine on recording of extracellular field potentials of atrioventricular node, isolated from rabbit, during experimental atrial fibrillation

S. Shariat Nejad<sup>1</sup>, V. Khori<sup>1</sup>, S. Shariat Nejad<sup>1</sup>, A. Alizadeh<sup>1</sup>, N. Shahini<sup>2,3,\*</sup>

<sup>1</sup>Cardiovascular Research Center, Golestan University of Medical Sciences, Gorgan, Iran <sup>2</sup>Student Research Committee, Golestan University of Medical Sciences, Gorgan, Iran Committee, Mashhad University of Medical Sciences, Mashhad, Iran

**Background and Aims:** Atrial fibrillation is one of the most common types of supraventricular tachycardia that is more prevalent in adults. The incidence of atrial fibrillation is 2%-1.5% in all of the world also it is well known as one of the most important and most common chronic cardiac arrhythmia. Since one causing of sudden cardiac death is arrhythmias, so we want to survey direct acute effects of cyclosporine on recording of extracellular field potentials of atrioventricular node ,isolated from rabbit during experimental atrial fibrillation.

**Methods:** In this experimental study , male New Zealand rabbits (1.5-2.5 kg)were randomly selected and used cumulative concentrations of cyclosporine, (0.5-10 mm) (7=n). recording of extracellular field potentials in specific stimulation protocols (recovery, hidden area, and atrial fibrillation), in both groups without drug (control)and in the presence of the drug, was performed on the electrophysiological properties of atrioventricular node. All results were calculated as mean  $\pm$  standard. Data analysis was performed with EXCEL and GRAPH PAD.

**Results:** Cyclosporine significantly increased HHmean (average distance between two consecutive records of the category) from the  $231.8 \pm 5.7$  to  $277.4 \pm 14.6$  / milliseconds.also increased duration of functional nonstimulation (AF FRP) from  $138.3 \pm 7.5$  to  $161.2 \pm 10.31$  milliseconds and duration of effective nonstimulation (AF ERP)was significantly decreased by cyclosporine.

**Conclusions:** Cyclosporine reduced ventricular rate during atrial fibrillation through increasing of hidden area and nodal nonstimulatation, so it can be considered as an antiarrhythmic drug. Possible mechanism could be through intermediate. Interstitial pore blockage of mitochondrial permeability.

**Keywords:** Atrial node-Ventricular; Intermediate pore of mitochondrial permeability; Cyclosporine; Atrial fibrillation