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ELECTROCONDUCTIBILITY OF POLY N-ISOPROPYLACRYLAMIDE THERMORESPONSIVE HYDROGEL INHIBITS THE OCCURRENCE OF MALIGNANT ARRHYTHMIAS POST MYOCARDIAL INFARCTION

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Objectives Recent research proved the protective effects of synthesized hydrogels (Gels) formyocardial infarction (MI) animals due to the inhibition of ventricular remodeling. However, cardiac malignant arrhythmia is one of the most serious complications after MI, but the exact mechanism underlying the increased vulnerability of arrhythmias is not clear. Synthesised hydrogel, as a kind of non-bioactive material, whether hydrogel intra-myocardium injection could result in electrical reentry between the non-bioactive material and the infarcted myocardium or not, it

is still under controversy. The present study was performed to investigate the alteration of electrophysiological characteristics in infarcted myocardium after Gel injection at the acute phase of MI.

Methods New Zealand White Rabbits (2.5 ± 0.5 Kg) were used and divided into sham operation (SO) group accepted Gel or phosphate-buffer saline (PBS) and MI group accepted Gel or PBS. After left anterior descending coronary artery (LAD) was ligated, 200 μ l 3% (w/v) Gel or PBS solution was injected around the infarcted myocardium by intra-myocardium injection. Rabbits in SO groups were subjected to the same procedure except that the silk suture around LAD was loose. Effective refractory period (ERP), monophasic action potential duration at 90% repolarisation (MAPD₉₀) and transmural dispersion of repolarisation (TDR) were measured in three layers myocardium respectively by programmed electrical stimulation at 30 min, 3 h, and 6 h after injection. Arrhythmias were recorded by surface electrocardiogram during the surgery.

Results Data manifested that ERP of left ventricle was significantly shortened post-MI, but the alteration can be reversed after Gel injection. MAPD₉₀ in infarcted myocardium was significantly shortened, especially in mid-myocardium. Gel can homogeneously prolong MAPD₉₀ in three layers of myocardium and consequently, Gel inhibited repolarisation heterogeneity post-MI. In addition, Gel blunted the increasing of TDR post-MI and the effects were continuously enhanced as time goes on. Besides that, arrhythmias score indicated the use of Gel obviously reduced the occurrence of ventricular malignant post-MI.

Conclusions As a non-bioactive biomaterial, poly N-isopropylacrylamide thermoresponsive hydrogel injected into normal or damaged myocardium is safe. Additionally, intra-myocardium injection of the thermoresponsive hydrogel could promote electrophysiological repair of infarcted myocardium due to the amelioration of electrical heterogeneity among the three layers of myocardium. We deem that the thermoresponsive hydrogel potentially inhibits malignant arrhythmias by reducing reentry; furthermore, it is a suitable consideration for MI therapy strategy.