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Streptomycin inhibits electrophysiological changes induced by stretching of chronically infarcted rat hearts

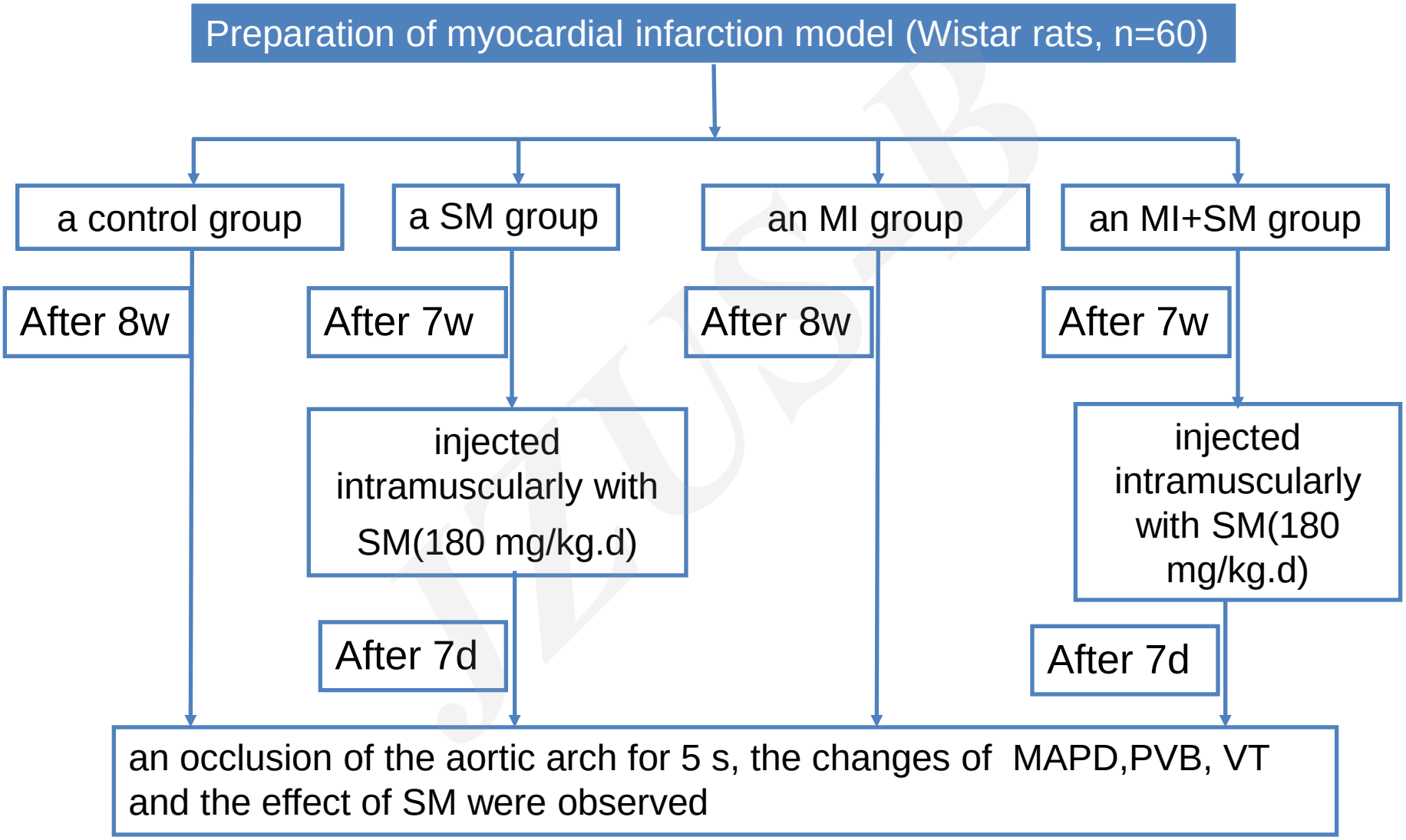
Key words: Arrhythmia, Mechanoelectric feedback, Monophasic action potential, Myocardial infarction, Streptomycin

- Myocardial stretch can initiate the changes of myocardial electrophysiological properties, which is called mechanoelectric feedback (MEF). MEF is enhanced in chronic MI which contributes to the appearance of arrhythmias.

- Streptomycin (SM) has been known to inhibit stretch-induced electrophysiological changes as a blocker of SACs. However, this effect of blocking of SACs with SM has seldom been observed in vivo and results are often contradictory.

- The aim of this study was to observe the effect of stretching on the monophasic action potential (MAP) and the effect of the application of SM in vivo on electrophysiological changes in chronically infarcted rat hearts.

METHOD


Preparation of myocardial infarction model (Wistar rats, n=60)
a control group
a SM group
an MI group
an MI+SM group
After 8w
After 7w
After 8w
After 7w
injected intramuscularly with SM(180 mg/kg.d)
injected intramuscularly with SM(180 mg/kg.d)
After 7d
After 7d
an occlusion of the aortic arch for 5 s, the changes of MAPD,PVB, VT and the effect of SM were observed

RESULTS AND CONLUSIONS

- The MAPD90 decreased during stretching in both the control (from 50.27±5.61 ms to 46.27±4.51 ms, $P < 0.05$) and MI groups (from 65.5±6.38 ms to 57.5±5.76 ms, $P < 0.01$). SM inhibited the decrease in MAPD90 during inflation (46.27±4.51 ms vs. 49.53±3.52 ms, $P < 0.05$ in normal and 57.5±5.76 ms vs. 61.9±5.33 ms, $P < 0.05$ in MI hearts).

- The occurrence of PVBs and VT in the MI group increased compared with that in the control group (PVB: 7.93±1.66 vs. 1.8±0.86, $P < 0.01$ and VT: 7 vs. 1, $P < 0.05$). SM decreased the occurrence of PVBs in both normal and MI hearts (0.93±0.59 vs. 1.8±0.86 in normal hearts, $P < 0.05$ and 5.4±1.18 vs. 7.93±1.66 in MI hearts, $P < 0.01$).

- Stretch-induced MAPD changes and arrhythmias were observed in chronically infarcted myocardium. The use of SM in vivo decreased the incidence of PVBs but not of VT. This suggests that SACs may be involved in mechanoelectric feedback (MEF) but that there might be other mechanisms involved in causing VT in chronic myocardial infarction.