a day, also for 4 weeks. EPCs in circulation were quantified by fluorescence-activated cell sorter (FACS) analysis for rabbits at the end of the 4-week-experiments. Capillary densities in ischemic heart region were also counted for all groups.

Results The numbers of CD34/FLK-1 cell in peripheral blood of trained rabbits in groups PIT, MI and PIT-MI were 3.8 ± 0.6 fold increase, 6.1 ± 2.8 fold increase and 6.9 ± 1.7 fold increase, respectively with respect to group SO. Neo-angiogenesis as assessed by capillary density in ischemic myocardium which elevated accompanied with EPCs were found to be 326 ± 76 in group SO, 327 ± 59 in group PIT, 516 ± 77 in group MI and 824 ± 106 (mm^2) in group PIT-MI at the end of the experiments for all groups, respectively.

Conclusion Physiological ischemia training could evoke the endogenous EPCs significantly which homed to the injured heart fraction for protection by angiogenesis.

Rehabilitation of cardiovascular disease

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PHYSIOLOGICAL ISCHEMIA TRAINING ENHANCED CIRCULATING EPCS AND NEOVASCULATION

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Background Recently researches showed that physiological ischemic training increased VEGF and capillary density (CD) in the remote regions. Physiological ischaemic training was referred to the ischemic isometric contraction on the normal muscles, which yield some factors into circulation, reached the remote area and enhanced collateral circulation. But the cytology mechanism is not clear. Our aim is to investigate the effects of physiological ischemic training on endogenous endothelial progenitor cells (EPCs) and neovascularisation with myocardial ischemia (MI).

Methods Thirty-six adult male Zealand rabbits were used for the investigation and were randomly grouped into four groups: group SO had neither physiological ischemia training (PIT) nor myocardial ischemia (MI) and served as a control group; group PIT underwent PIT only; group MI underwent MI only; and group PIT-MI underwent both PIT and MI. Physiological ischemia training was induced isometric contraction on normal skeletal muscles by electrical stimulation (40% maximum current strength, 1 ms, 40 Hz) for 4 min twice a day, 5 days a week, for 4 weeks. Pathological MI was induced by left ventricular branch intermittent occlusion for 2 min twice