Award Presentations (Poster)

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Hiroshi and Aya Irisawa Memorial Award for Excellent Paper on Research in Circulation in the Journal of Physiological Sciences

SP-1

A new calpain inhibitor protects left ventricular dysfunction induced by mild ischemia-reperfusion in in situ rat hearts.

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Our previous studies showed that a new soluble calpain inhibitor, SNJ-1945 (SNJ), attenuated cardiac dysfunction after cardioplegia arrest-reperfusion by inhibiting the proteolysis of α-fodrin in in vitro study. Nevertheless, to explore realistic therapeutic approaches for clinical use, the in vivo study design is indispensable. The aim of the present in situ study was to investigate whether SNJ attenuated left ventricular (LV) dysfunction (stunning) after specially developed mild ischemic-reperfusion (mI-R) in in siu rat hearts. SNJ (60 μmol/l, 5 ml i.p.) was injected 30 min before gradual and partial coronary occlusion at proximal left anterior descending artery (mI). To confirm LV dysfunction, we examined curvilinear end-systolic pressure-volume relationship by increasing afterload 60 min after reperfusion. In the mI-R group, specific LV functional indices at midrange LV volume (mLVV), end-systolic pressure (ESPmLVV), and end-systolic pressure-volume area (PVAmLVV): a total mechanical energy per beat, linearly related to oxygen consumption) significantly decreased, but SNJ reversed these decreases to time control level. Furthermore, SNJ prevented the α-fodrin degradation and attenuated degradation of Ca2+ handling proteins (LTCC and SERCA2a) after mI-R. Our results indicate that the recovery from LV dysfunction induced by mI-R injury is associated with inhibition of the proteolysis of α-fodrin in in situ rat hearts. We conclude that SNJ could be a promising tool to protect the heart from the stunning. No COI.

Award Presentations (Poster)
Aya Irisawa Memorial Promotion Award for Excellence by Women Physiologists

SP-2

From Sodium-Calcium exchanger to Shakuyaku-Kanzo-to

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