Role of sarcoplasmic reticulum in small muscular artery in young spontaneously hypertensive rats. Yasuaki Toyoda, Hiroki Shima, Hisashi Sasajima, Akira Baba, Takuzo Hano, Yuui Ueno, and Ichiro Nishio. Division of Cardiology, Department of Medicine, Wakayama Medical College, Wakayama City, 640.

Sarcoplasmic reticulum (SR) in small muscular artery plays an important role in regulating cytosolic Ca\(^{2+}\) through its Ca\(^{2+}\) release and sequestration. Especially, we suppose that SR has Ca\(^{2+}\) buffering effect by sequestration of cytosolic Ca\(^{2+}\) in time of elevated cytosolic Ca\(^{2+}\) concentration.

To examine the contribution of SR Ca\(^{2+}\) sequestration to evoked contraction in a small muscular artery, we measured the evoked tension before and after depleting SR Ca\(^{2+}\) stores by thapsigargin (Tg), SR Ca\(^{2+}\)-ATPase inhibitor. Isometric tension was measured in the rings of the second branch of superior mesenteric artery in Sprague-Dawley (SD) rats. Evoked tensions induced by 0.2\(\mu\)M serotonin and 10mM caffeine (Caf) were measured before and after 1\(\mu\)M thapsigargin (Tg). In SD rats, Caf response was diminished by Tg. After depleting SR Ca\(^{2+}\) stores, serotonin response was augmented in the presence of Tg. However, the augmentation of serotonin response was completely abolished by extracellular Ca\(^{2+}\) extrusion. Therefore, we suggest that, in SD rats, SR regulates evoked contraction mainly by sequestration of elevated cytosolic Ca\(^{2+}\) in a small muscular artery.

We studied the SR role of Ca\(^{2+}\) sequestration in regulation of cytosolic Ca\(^{2+}\) in young spontaneously hypertensive rats (SHR) and Wistar Kyoto rats (WKY). In the rings of the first branch of superior mesenteric artery in 5-week-old SHR and age-matched WKY, we measured evoked tensions by high potassium (20mM, 40mM, 70mM, 100mM; for 5 min.) and 10mM Caf (for 1 min.) before and after 1\(\mu\)M Tg. Systolic blood pressure was almost the same level in SHR (124±3mmHg) and WKY (118±3mmHg). Caf-induced contraction was significantly lower in SHR (77±9mg) than WKY (141±27mg), indicating that caffeine sensitive SR Ca\(^{2+}\) store is lower in SHR than in WKY. The magnitudes of high potassium-induced contraction in each concentration were not different in both rats (SHR: 20mM 2±2mg, 40mM 189±35mg, 70mM 454±23mg, 100mM 470±34mg, WKY: 20mM 3±2mg, 40mM 240±32mg, 70mM 451±32mg, 100mM 472±42mg). However, the rate of 40mM potassium-induced contraction was significantly slower in SHR than WKY. In both rats, by Tg, the resting tensions were almost unaffected and Caf-induced contractions were almost diminished. After treatment with Tg, the magnitudes of high potassium-induced contraction in each concentration were augmented in SHR (20mM 28±17mg, 40mM 332±35mg, 70mM 511±29mg, 100mM 512±24mg), but not in WKY (20mM 0±0mg, 40mM 233±18mg, 70mM 411±40mg, 100mM 421±47mg). Moreover, the rate of 40mM potassium-induced contraction was augmented in SHR, but not in WKY.

We suggest that SR Ca\(^{2+}\) sequestration is higher in SHR than in WKY. We postulated that the SR prevents the development of hypertension in SHR via a mechanism that SR strongly buffers the evoked rise in cytosolic Ca\(^{2+}\) in SHR.