Marked Impairment in Baroreflex Control of Heart Rate in Spontaneously Hypertensive Rats with Chronic Myocardial Infarction.

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Myocardial infarction (MI) is one of the important complications for determining the long-term prognosis of hypertensive patients. In addition to impaired baroreflex observed in hypertension, recent studies have shown that chronic MI also blunts the baroreflex and is responsible for fatal arrhythmia and sudden death, which are important factors for the long-term prognosis of the patients with chronic MI.

However, few studies have reported the baroreflex function in hypertensive animals with chronic MI. In the present study, we examined the combined effect of hypertension and MI on the baroreflex function.

Methods: The left coronary artery was ligated in 15-week-old male spontaneously hypertensive rats (SHR) and in age matched male normotensive Wistar-Kyoto (WKY) rats.

Four weeks after, catheters were placed in the femoral artery and vein for the measurement of mean arterial pressure (MAP) and infusion of drugs.

More than twenty four hours after the operation, we examined the baroreflex control of heart rate (HR) in the conscious state. HR was recorded when mean arterial pressure (MAP) was decreased and increased with nitroprusside and phenylephrine. These data were analyzed using four-parameter logistic function curves. The largest slope of the curve was defined as the maximal gain of the baroreflex.

Then, another catheter was placed in the left ventricle via the right carotid artery to measure left ventricular end diastolic pressure (LVEDP).

After the experiments, the heart was taken out, and infarct size and heart weight were measured. Infarct size was determined by Triphenyltetrazolium chloride staining.

Results: MAP of SHR with chronic MI was smaller than that of SHR with sham operation (148 ± 9 vs. 107 ± 5 mmHg, $p < 0.01$) and shifted MAP-HR relation curve (midrange, 147 ± 3 vs. 124 ± 2 mmHg, $p < 0.01$) to a lower pressure level in SHR, which was not seen in WKY rats (MAP; 109 ± 6 vs. 108 ± 5 mmHg, NS, MAP-HR relation curve; midrange, 110 ± 4 vs. 110 ± 3 mmHg, NS). The maximal gain of the baroreflex control of HR was smaller in SHR with sham operation than that in WKY rats, and the more remarkable attenuation was recognized in SHR with chronic MI (SHR; 2.3 ± 0.1 vs. 0.3 ± 0.1 bpm/mmHg, $p < 0.01$, WKY; 3.5 ± 0.2 vs. 1.8 ± 0.1 bpm/mmHg, $p < 0.01$).

The infarct size was smaller in SHR than that in WKY rats (29 ± 2 vs. 36 ± 2 %, $p < 0.05$), and LVEDP was higher in SHR with chronic MI than that in WKY rats with chronic MI (22 ± 2 vs. 16 ± 2 mmHg, $p < 0.05$).

Heart-to-body weight ratio was increased concerning both left and right ventricles in the rats with chronic MI (SHR; 2.36 ± 0.07 vs. 2.66 ± 0.07 mg/g, $p < 0.05$; 0.62 ± 0.03 vs. 0.74 ± 0.02 mg/g, $p < 0.01$, WKY; 1.91 ± 0.07 vs. 2.20 ± 0.07 mg/g, $p < 0.05$; 0.58 ± 0.02 vs. 0.66 ± 0.01 mg/g, $p < 0.01$, respectively).

Conclusions: As compared with the case of WKY rats, MAP was decreased in SHR with chronic MI. This is probably due to decreased cardiac output, and we presume that SHR with chronic MI suffered from a more serious impairment in cardiac function, although the infarct size in SHR was smaller than that in WKY rats.

Chronic MI more greatly impairs the baroreflex control of HR in SHR than in WKY rats. It is suggested that the marked impairment in baroreflex function is one of the important factors for the poor prognosis of hypertensive patients with chronic MI.

The increase in heart weight caused by the remodeling after MI was seen both in left and in right ventricles.