Sino-aortic denervation enhanced the pressor response elicited by the chemical stimulation of caudal ventrolateral medulla in chloralose anesthetized Wistar-Kyoto rats but not in spontaneously hypertensive rats

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The neurons of rostral ventrolateral medulla (RVLM) have been considered as the tonic vasomotor center, since tonic activities were required for maintaining a resting blood pressure. The caudal ventrolateral medulla (CVLM) neurons inhibit the activities of the RVLM neurons bilaterally, thus the stimulation of the CVLM neurons cause a depressor response. Some studies had suggested that a diminished inhibition, compared to Wistar-Kyoto rat (WKY), from the CVLM to RVLM neurons was responsible to the hypertension in spontaneously hypertensive rat (SHR). In this study, we estimated the effect of sino-aortic denervation (SAD) on the CVLM depressor response in WKY and SHR.

Ten WKY and 10 SHR were used, and 5 rats of each strain were prepared for SAD, respectively. Under anesthesia (α -chloralose) and artificial ventilation, left femoral artery was cannulated for measuring an arterial pressure (AP), then mean arterial pressure (MAP) and heart rate (HR) were calculated electrically from the AP. Phenylephrine ($20 \mu\text{g/Kg}$) was injected intravenously for estimating an effects of SAD on baroreceptor reflex in each intact and SAD groups. The CVLM neurons were stimulated chemically with L-glutamate ($0.25\text{--}8.0 \text{ nmol}/50 \text{ nl}$), delivered from a calibrated glass micropipette holding with a stereotaxic micromanipulator. All injection sites were verified histologically.

The SAD suppressed a bradycardic response during the pressor response induced by phenylephrine injection ($-3.56 \pm 2.72 \text{ b/mmHg}$ in intact groups v.s. $-0.51 \pm 0.26 \text{ b/mmHg}$ in SAD groups, $p < 0.05$). The SAD increased a resting MAP in WKY ($88.79 \pm 6.36 \text{ mmHg}$ in intact group < $127.07 \pm 11.46 \text{ mmHg}$ in SAD group, $p < 0.01$), but did not in SHR ($145.71 \pm 6.36 \text{ mmHg}$ in intact group v.s. $143.67 \pm 6.29 \text{ mmHg}$ in SAD group). Resting MAP in both SHR groups were significantly higher than the SAD-WKY group ($p < 0.01$). With the chemical stimulation of CVLM, MAP and HR were decreased in all groups. Two-way ANOVA appeared that the depressor responses were significant in all groups by a dose-factor ($p < 0.01$), but post-stimulation levels of MAP were different between some groups by a group-factor ($p < 0.01$). Post-stimulation levels of MAP in WKY-intact group was lowest, and those in WKY-SAD group was intermediate, and those in SHR-intact and SHR-SAD groups were similar and highest.

In summary, SAD elevated the resting MAP and enhanced the CVLM depressor response in WKY, but not in SHR. These data suggest that the diminished inhibition from CVLM to RVLM neurons in SHR was resultant from an alternation of baroreceptor input to the CVLM.