Ablation of brainstem C1 neurons improves cardiac function in volume overload heart failure

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Activation of the sympathetic nervous system is a hallmark of heart failure (HF) and is positively correlated with disease progression. Catecholaminergic (C1) neurons located in the rostral ventrolateral medulla (RVLM) are known to modulate sympathetic outflow and are hyperactivated in volume overload HF. However, there is no conclusive evidence showing a contribution of RVLM-C1 neurons to the development of cardiac dysfunction in the setting of HF. Therefore, the aim of this study was to determine the role of RVLM-C1 neurons in cardiac autonomic control and deterioration of cardiac function in HF rats. A surgical arteriovenous shunt was created in adult male Sprague-Dawley rats to induce HF. RVLM-C1 neurons were selectively ablated using cell-specific immunotoxin (dopamine-? hydroxylase saporin [D?H-SAP]) and measures of cardiac autonomic tone, function, and arrhythmia incidence were evaluated. Cardiac autonomic imbalance, arrhythmogenesis and cardiac dysfunction