Contribution of peripheral and central chemoreceptors to sympatho-excitation in heart failure

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Chronic heart failure (CHF) is a major public health problem. Tonic hyper-activation of sympathetic neural outflow is commonly observed in patients with CHF. Importantly, sympatho-excitation in CHF exacerbates its progression and is strongly related to poor prognosis and high mortality risk. Increases in both peripheral and central chemoreflex drive are considered markers of the severity of CHF. The principal peripheral chemoreceptors are the carotid bodies (CBs) and alteration in their function has been described in CHF. Mainly, during CHF the CB chemosensitivity is enhanced leading to increases in ventilation and sympathetic outflow. In addition to peripheral control of breathing, central chemoreceptors (CCs) are considered a dominant mechanism in ventilatory regulation. Potentiation of the ventilatory and sympathetic drive in response to CC activation has been shown in patients with CHF as well as in animal models. Therefore, improving understanding of the contribution of the peripheral and central chemoreflexes to augmented sympathetic discharge in CHF could help in developing new therapeutic approaches intended to attenuate the progression of CHF. Accordingly, the main focus of this review is to discuss recent evidence that peripheral and central chemoreflex function are altered in CHF and that they contribute to autonomic imbalance and progression of CHF. (Figure presented.).