Hyperleptinaemia does not correlate with plasma catecholamine levels in chronic heart failure

We read with interest the recent paper by Leyva et al. which reported on chronic heart failure as a state of hyperleptinaemia, with leptin levels elevated by 81.3% compared to hyperleptinaemia, with leptin levels corrected for body mass index (5.31 ng·ml⁻¹ (kg·m⁻²)) vs 2.19 ng·ml⁻¹ (1.95 to 3.05); P<0.005) in the chronic heart failure group, whereas epinephrine was not affected. Apart from insulin, which is shown to relate to leptin levels, as in the study by Leyva et al., catecholamines are now well known to exert regulatory influences on leptin production. Consistent with these reports, correlation analysis revealed a significant negative association between leptin and plasma epinephrine (r = −0.76, P<0.05) in our healthy control subjects. However, no such relation was detectable in the heart failure patient group, thus suggesting that this regulatory influence is abolished in chronic heart failure. Similarly, we found a lack of association between leptin and catecholamines in pheochromocytoma patients with highly increased plasma noradrenaline levels but normal epinephrine levels.

These findings indicate that leptin balance is affected in chronic heart failure and several metabolic and endocrine disorders. Further research into the precise mechanisms may be crucial to the understanding of the pathogenesis, regulation, and the consequences of these hormonal derangements, and hence the contribution of leptin to the cluster of metabolic disturbances associated with chronic heart failure.

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References


Neurobiology of respiratory-pattern training in congestive heart failure

Bernardi[1] finds that respiratory-pattern training in chronic heart failure results, at least, in better coordination of the respiratory muscles and of the diaphragm, and a greater facility to slow down the breathing rate, regardless of whether or not it permanently modifies the spontaneous breathing habit. This new approach is supported by profound effects on angina by consciously focusing attention on breathing and the intervening pauses, adaptation to stress manifested by slower, deeper breathing (contributing to a 6·5-fold reduction in mortality), and the role of a slower rate of living which contributes to a fivefold increase in lifespan[2]. It is also supported by the following: the rate of speech hesitation pauses of 1 s or more, 4·79 ± 2·48 per min, 1·50 ± 0·33 s (Mean ± SD) correlates with the state of the circulation (angina/hypertension) and a sixfold incidence of clinical coronary artery disease in two groups of normal coronary-prone men followed prospectively for 10 years, P<0·05; the reduction of blood pressure is associated with longer, less recurrent pauses of about 2 s[3] which correlates with the feeling of rhythmicity; the microvascular response to the onset of neural activity is consistently delayed by several seconds; and short-term laboratory experience demonstrates that adult female speech production is sufficient to influence infant’s speech production occurring in the silent intervals between the adult’s vocalizations (average, 3·37 s). Even brief (1–5 s) spontaneous pauses in ongoing patterned behaviors are accompanied by an immediate reduction in serotonin neuronal activity to, or below, baseline levels, coordinating autonomic, motor, and sensory functions, and modulating dopamine which optimizes response organization and working memory, and regulates the microvasculature[4] implicated in slow coronary flow as a cause for anginal pain. These findings suggest that respiratory-pattern training[1] may promote a primary rhythm in the central nervous system that entrains heart rate, blood pressure, and respiratory rate, maintains cognitive unity through a callosal window between prefrontal cortices[5], and orchestrates bimanual coordination through a callosally interconnected and distributed network of frontal and parietal cortical areas of which the supplementary motor area is a part[6]. Therefore, simple training to improve breathing in chronic heart failure[1] may enhance physical and psychological well-being, whether or not it increases survival[7].

Neurobiological features of this training in chronic heart failure, a multisystem syndrome[1], are demonstrated by the association of brain stem cardiovascular control, cardiovascular reactivity in challenging tasks, cortical silent periods, mood, subclinical impairment of lung airways, and vasospasm with dopaminergic activity promoted by plasmogen and endothelin which is activated by mental stress[2,3,8,9]. These findings prompt studying long-term efficacy.