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Nervous System

10.1 Sympathetic Responses to Ventricular Extrasystolic Beats in Obese Subjects without and with Sleep Apnoea

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Introduction. Ventricular extrasystolic beats (PVCs) trigger, along with an early and a late blood pressure (BP) fall and overshoot, an early sympatho-excitation and a late period of sympatho-inhibition. The present study was designed to quantify the sympathetic response to PVCs in obesity, uncomplicated or complicated by sleep apnoea, i.e. conditions in which the adrenergic overdrive may be responsible for the increased of arrhythmogenic threshold.

Methods. In 22 obese normotensive subjects (age 50.1±2.1 years, mean±SEM), 14 of which with and 8 without sleep apnoea, and in 14 age-matched healthy controls (C), both in Lown class less than II, we evaluated BP (Finapres), heart rate (HR, EKG) and muscle sympathetic nerve traffic (MSNA, microneurography) responses to isolated monofocal PVCs.

Results. MSNA, quantified as bursts incidence corrected for HR, was significantly increased in obese without and with sleep apnoea (61.7± 2.2 and 74.8±2.5 bs/100hb respectively, $p<0.01$ for both) as compared to C (42.0± 3.1 bs/100hb). In C, the PVC-induced BP fall and overshoot was accompanied by a sympatho-excitation (total MSNA activity: +132.2± 18%) followed by a period of sympatho-inhibition (average duration: 11834±1036 ms). The responses were markedly impaired in obese subjects, in which the magnitude of the sympatho-excitation and particularly the duration of the subsequent sympatho-inhibition were strikingly reduced (average reduction obese with sleep apnoea: -42.4% and -68.8%; obese with out sleep apnoea: -34.7% and -54.8%, respectively, $p<0.001$). In obese subjects without sleep apnoea and, to a lesser extent, in obese with sleep apnoea the reduction in MSNA responses to PVCs was related to impaired baroreflex sensitivity.

Conclusions. Thus MSNA responses to PVCs are impaired in human obesity particularly when complicated by obstructive sleep apnoea syndrome. They also suggest that baroreflex and chemoreflex mechanisms are involved in these neuroadrenergic alterations, which may be responsible for the increased arrhythmogenic profile of human obesity.