Metabolic Aspects and Mechanisms

9.1 Pro-Arrhythmogenic Role of Sympathetic Activation and Baroreflex Dysfunction in Human Obesity

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Introduction. Human obesity, particularly when complicated by obstructive sleep apnoea is characterized by an increased risk of ventricular arrhythmias. Although this has been ascribed to neuroadrenergic mechanisms, no direct evidence is available on this issue in humans.

Methods. We studied 51 obese normotensive subjects (age: 49.7 ± 1.2 years, mean $\pm$ SEM), of which 28 without and 23 with monofocal premature ventricular beats (PVCs) in Lown class less than II. A polysomnographic evaluation allowed the obese subjects without PVCs to be further classified as without (n=12) and with (n=16) sleep apnoea. A similar procedure was done in obese subjects with PVCs (obese with PVC without sleep apnoea: n=10 and obese with PVC and sleep apnoea: n=13). In each patient we measured, along with anthropometric and echocardiographic variables, beat-to-beat mean blood pressure (BP, Finapres), heart rate (HR, EKG) and muscle sympathetic nerve activity (MSNA, microneurography) at rest and during baroreflex stimulation and deactivation (vasoactive technique).

Results. Obese subjects without and with PVCs were age-matched and characterized by BP and echocardiographic values, which were in the normal range and superimposable in the 2 groups. Anthropometric values were similarly altered in the 2 groups. When compared to obese without PVCs, obese with PVCs showed a slight increase in HR but a significant elevation in MSNA (45.8 ± 1.9 vs. 68.2 ± 1.5 bs/100hb, $p < 0.001$). This increase resulted further enhanced in the presence of sleep apnoea syndrome. This was accompanied by a significant HR and MSNA baroreflex impairment which was evident in obese with PVCs (-24.5 ± 3.1 and $-9.4 \pm 2.9\%$) and further improved in obese with PVCs and sleep apnoea (-35.1 ± 3.8 and $-28.7 \pm 3.1\%$).

Conclusions. Thus, in human obesity neurogenic and reflex mechanisms may participate to developing ventricular arrhythmias. This potential involvement, however, appears to be more pronounced when obesity is associated with sleep apnoea syndrome, indicating that in obesity not only baroreflex but also chemoreflex dysfunction may represent an important factor in the genesis of cardiac rhythm disturbances.