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Autonomic nervous system profile in fibromyalgia patients and its modulation by exercise: a mini review.

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Abstract

This review imparts an impressionistic tone to our current understanding of autonomic nervous system abnormalities in fibromyalgia. In the wake of symptoms present in patients with fibromyalgia (FM), autonomic dysfunction seems plausible in fibromyalgia. A popular notion is that of a relentless sympathetic hyperactivity and hyporeactivity based on heart rate variability (HRV) analyses and responses to various physiological stimuli. However, some exactly opposite findings suggesting normal/hypersympathetic reactivity in patients with fibromyalgia do exist. This heterogeneous picture along with multiple comorbidities accounts for the quantitative and qualitative differences in the degree of dysautonomia present in patients with FM. We contend that HRV changes in fibromyalgia may not actually represent increased cardiac sympathetic tone. Normal muscle sympathetic nerve activity (MSNA) and normal autonomic reactivity tests in patients with fibromyalgia suggest defective vascular end organ in fibromyalgia. Previously, we proposed a model linking deconditioning with physical inactivity resulting from widespread pain in patients with fibromyalgia. Deconditioning also modulates the autonomic nervous system (high sympathetic tone and a low parasympathetic tone). A high peripheral sympathetic tone causes regional ischaemia, which in turn results in widespread pain. Thus, vascular dysregulation and hypoperfusion in patients with FM give rise to ischaemic pain leading to physical inactivity. Microvascular abnormalities are also found in patients with FM. Therapeutic interventions (e.g. exercise) that result in vasodilatation and favourable autonomic alterations have proven to be effective. In this review, we focus on the vascular end organ in patients with fibromyalgia in particular and its modulation by exercise in general.


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