Exercise Interventions in Fibromyalgia: Clinical Applications from the Evidence

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KEYWORDS

• Fibromyalgia • Exercise interventions • Exercise prescription
• Eccentric contraction • Self-efficacy

Most clinicians are aware of the benefits of exercise for patients who have fibromyalgia (FM). Many express frustration, however, with poor patient compliance with exercise recommendations. Viewed from a broader context, regular exercise eludes at least 70% of Americans.[1] It is thus not surprising that patients with the pain, fatigue, and disrupted sleep of FM would face additional challenges in adopting and maintaining an exercise program. In fact, 83% of patients who have FM do not engage in aerobic exercise, and most of those tested have below-average fitness levels. In physical self-report or functional testing, the average 40-year-old patient who has FM was found to be as physically unfit as an 80-year-old person who does not have FM.[2,3]

This article summarizes physiologic obstacles to exercise and reviews exercise interventions in FM. In addition, the authors describe the top 10 principles for successfully prescribing exercise in the comprehensive treatment of FM and provide a practical exercise resource table to share with patients.

POTENTIAL "PHYSIOLOGIC" OBSTACLES TO ADEQUATE EXERCISE IN PATIENTS WHO HAVE FIBROMYALGIA

The common complaint of many patients who have FM is that they hurt and feel more fatigued after exercise. There are a few observations worthy of note that may be

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relevant to postexertional pain and fatigue in patients who have FM. Kosek's group[4,5] has reported that patients who have FM have reduced muscle blood flow to the infraspinatus muscle during dynamic and static exercise and that such exercise is more painful than in healthy controls. Reduced muscle blood flow in FM was originally reported in 1982 by Klemp and colleagues[6] and then by Bennett[7] and McIver and colleagues.[8] It has been hypothesized that exertional pain in FM may partially be a result of muscle ischemia. Exercise is generally considered to evoke an inhibitory effect on pain by the production of endorphins[9,10] and activation of the descending inhibitory pathways. The hypothalamic pituitary adrenergic response to exercise is blunted in patients who have FM,[11] and a concomitant reduction in the release of endorphins would be expected;[12] however, this has not so far been tested in patients who have FM. Furthermore, patients who have FM have been reported to have reduced μ-opioid receptor binding in several brain regions involved in pain modulation.[13] These findings indicate that the μ-opioid receptors are nearly saturated in patients who have FM, making them less responsive to the secretion of endogenous opioids. The descending bulbospinal pathways are critical in reducing the perception of ongoing painful stimuli.[14] Staud and colleagues[15] have reported that sustained muscular contraction induced widespread inhibition of pain perception in healthy individuals but not in patients who had FM; on the contrary, sustained muscle contraction induced an increase in the hyperalgesia of patients who had FM. The authors hypothesize that the inability of patients who have FM to develop postexercise hypoalgesia may be the result of an impaired response of the inhibitory bulbospinal pathways.

The stress response system is abnormal in FM, with perturbations in the autonomic nervous system that may be manifested as postural orthostatic tachycardia syndrome, neurally mediated hypotension, and fatigue.[11,16,17] These autonomic perturbations can contribute to poor exercise tolerance in patients who have FM. The endocrine arm of the stress system has also been reported to be dysfunctional, with cortisol and growth hormone (GH) imbalances.[18,19] Jones and colleagues[20] have reported a defective GH response to exercise in more than 90% of patients who have FM. GH is important in muscle growth and repair, and it is hypothesized that defective GH production impairs the resolution of exercise-induced muscle microtrauma.[21] Finally, levels of exercise that are excessive for a given individual result in the production of proinflammatory cytokines,[22] which results in feeling increased fatigue as a consequence of the cytokine-associated "sickness syndrome".[23] Whether these problems are a result of being deconditioned, and hence improve with gradual escalation of exercise intensity, or whether they represent a "physiologic" block resulting from having FM is not fully understood at this time. It is probable that both notions have some degree of truth and underline the necessity for a strictly graduated approach to exercise therapy in patients who have FM.

REVIEW OF FIBROMYALGIA EXERCISE INTERVENTIONS: TWO DECADES OF PROGRESS

The first consideration of exercise as a possible therapeutic intervention in FM arose from a sleep laboratory in the 1970s. Researchers repeatedly startled healthy college students awake during deep sleep. This manipulation caused muscle pain and tender points in most subjects, although, surprisingly, sparing those who were elite runners.[24] Since that time, 70 exercise interventions in FM have been published. From 1988 through 2008, a total of 4385 subjects completed those studies. Of those studies, 56 were randomized controlled trials. Modalities of exercises studied included aerobics (land and water), strength, flexibility, and various combinations of these. More recently, "movement therapies," such as Qi Gong, T'ai Chi, and yoga, have been