Clin Rheumatol (2000) 19:253–257

Clinical
Rheumatology

Original Article

Patients with Fibromyalgia Benefit from Aerobic Endurance Exercise

L. Meiworm[1], E. Jakob[2], U. A. Walker[1], H. H. Peter[1] and J. Keul[2]

[1]Division of Rheumatology and Clinical Immunology and [2]Division of Rehabilitative and Preventive Sports Medicine, Department of Medicine, University Hospitals, Freiburg, Germany

Abstract: Fibromyalgia (FM) is a disorder characterised by diffuse widespread musculoskeletal aching and stiffness and multiple tender points [1]. Its pathophysiology is poorly understood. The influence of aerobic endurance exercise on pain in patients with FM was investigated. Twenty-seven patients (25 female, 2 male) participated in a controlled clinical study and performed 12 weeks of jogging, walking, cycling or swimming following a given schedule. Twelve sedentary FM patients (11 female, 1 male) served as controls. Before and after training both the study and the control groups were evaluated spiroergometrically. Tender point pain was quantified by dolorimetry. The painful body surface was estimated by a pain body diagram, and its intensity by a visual analogue scale and a ranking scale. Patients trained for an average of 25 min two to three times a week, with an average intensity of 50% of maximal oxygen uptake (VO_{2max}). Unlike the control group, the training group exhibited a decrease in heart rate and VO_2 and an increase in respiratory quotient during submaximal workload. Maximal performance capacity and VO_{2max} remained unchanged, whereas the wattpulse (watt/heart rate) improved at maximal workload. Pain parameters remained unchanged in the control group, but in the training group the mean number of positive tender points (15.4/12.7), the mean pain threshold of the gluteal tender point (2.89 kp/3.50 kp) and the painful body surface (18%/15% body surface) decreased significantly. Subjective general pain condition deteriorated in two patients but improved in 17. Our results suggest a positive effect of aerobic endurance exercise on fitness and well-being in patients with FM.

Keywords: Exercise; Fibromyalgia; Metabolism

Introduction

Fibromyalgia (FM) is characterised by chronic musculoskeletal pain and is clinically defined by the ACR criteria established in 1990 [1]. Different central and peripheral, but mainly muscular, abnormalities have been suggested to contribute to the aetiopathogenesis of FM. Light and electron microscopic investigations of FM muscle biopsies revealed non-specific type II fibre atrophy, a moth-eaten appearance, rubber bands and ragged-red fibres [2–9]. Several findings link FM pain to local muscle ischaemia and tender points. Biochemical investigations show that low levels of high-energy phosphates [10,11], low concentration of cellular oxidative enzymes [12] and a diminished local muscular glucose metabolism [13] are associated with FM. Tender points (TP) in FM have been shown to be hypoxic [14] and muscular blood flow was found to be low [15]. The theory of muscular ischaemia is, however, questioned by findings of elevated tissue oxygen pressure in paraspinal muscles of FM patients [16]. Furthermore, some investigators have observed no abnormalities in muscular oxygen metabolism [17] but have found impaired muscle relaxation [15]. One common characteristic of FM patients is low physical fitness owing to poor aerobic performance capacity [18–22]. Furthermore, muscular deconditioning and microtrauma have been suggested as

Correspondence and offprint requests to: Professor Dr H. H. Peter, Abteilung Rheumatologie und Klinische Immunologie, Medizinische Universitätsklinik der Albert-Lundwigs-Universität, Hugstetter Straße 55, 79106 Freiburg, Germany. Tel: +49–761–270–3448; Fax: +49–761–3446.

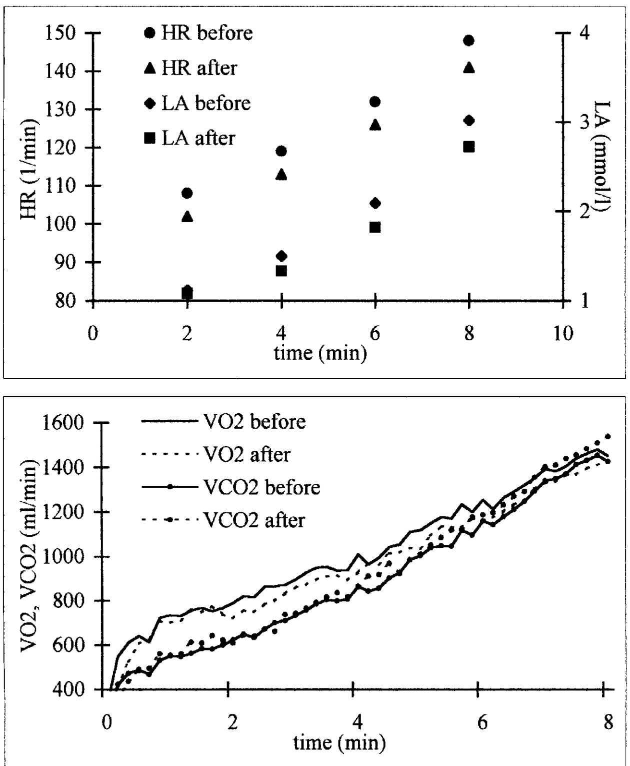
a direct cause of FM [23], which led us to investigate whether FM might be improved by aerobic endurance exercise [24,25].

Materials and Methods

Over a 1-year period 76 patients were diagnosed with FM in our rheumatology clinics. Of these, 33 (29 female, 4 male) chose not to participate in the study. Eventually 31 participants (29 female, 2 male) were subjected to a 3-month aerobic endurance exercise; 27 (25 female, 2 male) completed the study. Twelve patients (11 female, 1 male) maintained their usual lifestyle and served as sedentary controls (Table 1). This group showed a motivation level similar to the members of the training group, as they all started training according to the same schedule after participating in the study.

Table 1. Anthropometric data (mean ± SD)

	Non-participants n = 33	Participants n = 27	Controls n = 12
Age (yrs)	47 (±10)	45 (±10)	49 (±7)
Height (cm)	167.4 (±8.5)	166.3 (±7.4)	166.9 (±5.6)
Weight (kg)	69.5 (±10.5)	64.5 (±8.9)	74.3 (±8.2)



HR = heart rate; LA = lactic acid concentration; VO2 = oxygen uptake, VCO2 = carbondioxide emission

Fig. 1. Spiroergometry, training group, within 1 week before and after training.

Within 1 week before and after the training period the diagnosis of FM was established according to the ACR criteria [1]. We also quantified the pain threshold of all 18 TP with a Fischer dolorimeter [26]. Pain threshold values, including negative TP, were rated in kp in order to calculate averages. All participants marked their pain-affected areas using a body diagram, which permitted registration of pain spreading as a percentage of body surface [27]. The participants rated their subjective pain level on a visual analogue scale (VAS) [28]. They also recorded their subjective general pain condition on a ranking scale (± 3 = much better/worse, ± 2 = better/worse, ± 1 = some better/worse, 0 = unchanged).

Within 1 week before and after training all participants completed a spiroergometric evaluation on an electromagnetic braking revolution-independent bicycle ergometer (Excalibur, Lode). Workload started at 25 W and was increased in 25 W intervals every 2 minutes until volitional exhaustion. Over the course of the test ventilation and breath gases were analysed with a spirometric device (Oxycon Sigma, Mijnhardt).

Lactic acid concentration (LA) was enzymatically analysed from a 50 μl blood sample taken from a hyperaemic earlobe at the end of each workload interval. Heart rate (HR) was recorded by ECG. The data on each workload level up to 100 W (Fig. 1) and at maximum performance were analysed.

Training

The training programme was designed to provide patients with a simple schedule for self-managed domestic training. Training disciplines are walking, jogging, cycling and swimming. The members of the training group were advised to choose their own training schedule within the limits of the given protocol (Table 2). During the first half of the 12-week training period and aerobic intensity was increased every 2 weeks

Table 2. Training programme

Level	Week	Walking (min)	Jogging (min)	Cycling (min)	Swimming distance (m)
1	1 + 2	2 × 15	2 × 15	2 × 30	2 × 50
	3 + 4	3 × 15	3 × 15	3 × 30	2 × 50
	5 + 6	3 × 20	3 × 15	3 × 30	2 × 75
	7–12	3 × 30 or more	3 × 20 or more	3 × 40 or more	2 × 100 or more
2	1 + 2	3 × 20	2 × 20	2 × 30	3 × 50
	3 + 4	3 × 25	3 × 20	3 × 30	3 × 75
	5 + 6	4 × 30	3 × 20	3 × 40	3 × 100
	7–12	4 × 40 or more	3 × 30 or more	3 × 50 or more	3 × 125 or more
3	1 + 2	4 × 20	4 × 15	3 × 30	3 × 100
	3 + 4	4 × 30	4 × 20	4 × 40	3 × 150
	5 + 6	4 × 40	4 × 30	4 × 50	3 × 200
	7–12	4 × 50 or more	4 × 30 or more	4 × 60 or more	3 × 250 or more

through the use of longer or more frequent training sessions. The second half of the study maintained a constant schedule.

Patients were advised to maintain regular training patterns and times without intermittent resting. According to their individual fitness (history of sport, ergometric performance) all patients were allowed to enter the study at an exercise level they felt able to accomplish. In order to estimate the real training workload, patients recorded each training session and their HR at the end of training. The patients in the control group kept to their regular lifestyle.

Statistics

Dolorimetric and spirogerometric pre- and post-test data were analysed using the Wilcoxon test. Anthropometric parameters were tested with the Whitney test. The rank scale was computed using Wilcoxon's single sample test. All calculations were done with the statistical program SPSS for Windows (SPSS Inc., 1993–97).

Results

The anthropometric data (Table 1) between the patient groups showed no significant differences.

Training

Three patients did not fulfil the required training, executing either too-short training sessions, or sessions with inadequate intensity (HR <100/min), and were excluded from the analysis, as was a fourth patient who suffered from an upper respiratory tract infection at the time of the second spiroergometry. The remaining 27 individuals exercised at an average of two to three training units per week, with a duration of 20–30 minutes. Their mean training HR was 115/min. At the spiroergometric testing this corresponded to an average of 50% maximal oxygen uptake (VO_{2max}) and an average of 1.4 mmol/l LA. Therefore training intensity has to be rated moderate [29,30]. Among the different training disciplines 42% of training time was spent cycling, 39% jogging and walking and 19% swimming.

Table 3. Spiroergometry at volitional exhaustion (mean ± SD)

	Patients		p
	Before training	After training	
Performance (W)	122.92 (±31.25)	128.30 (±36.88)	n.s.
HR (1/min)	161.85 (±20.61)	159.19 (±19.05)	n.s.
WP (Watt/HR)	0.76 (±0.17)	0.81 (±0.17)	<0.01
LA (mmol/l)	5.69 (±2.23)	5.76 (±2.13)	n.s.
VO_2 (ml/min*kg)	29.43 (±7.40)	28.24 (±7.64)	n.s.
	Controls		p
	Examination 1	Examination 2	
Performance (W)	112.3 (±12.3)	114.7 (±9.2)	n.s.
HR (1/min)	154.43 (±10.77)	152.43 (±11.15)	n.s.
WP (Watt/HR)	0.73 (±0.19)	0.75 (±0.19)	n.s.
LA (mmol/l)	5.29 (±1.86)	5.65 (±1.19)	n.s.
VO_2 (ml/min*kg)	24.59 (±6.74)	25.30 (±4.93)	n.s.

Hr, heart rate; LA, lactic acid concentration; VO_2, oxygen uptake; WP, Wattpulse. Measurements were done within 1 week before and after training.

Training did not result in improved maximal ergometric performance (Watt max), but we found improved values for the Wattpulse (Watt/heart rate) at maximal workload ($p<0.01$) (Table 3). After training HR at rest remained almost unchanged (81.17 bpm/80.48 bpm). The mean LA at equal submaximal workload levels only showed a trend to lower levels, but the mean HR decreased significantly by 6 bpm ($p<0.05$) (Fig. 1). Mean VO_2 was significantly ($p<0.05$) lower after training (48 ml/min), whereas VCO_2 remained unchanged (Fig. 1), resulting in a significant ($p<0.05$) rise (0.07) of the mean respiratory quotient ($RQ = VCO_2/VO_2$). Within the sedentary control group there was no statistical change in any parameter during the course of the study.

Pain

Four patients in the training group did not meet the ACR criteria after completion of the full training programme, but two of them demonstrated a distinct subjective improvement in their general pain condition. Training significantly ($p<0.01$) decreased the mean number of positive TP, but in five patients TP number increased

Table 4. Dolorimetry: pain threshold (mean ± SD)

	Patients			Controls		
	Before training	After training	p	Examination 1	Examination 2	p
TP (kp)	2.45 (±0.77)	2.63 (±1.14)	n.s.	2.34 (±0.76)	2.30 (±0.78)	n.s.
Number of TP	15.41 (±2.27)	12.74 (±4.77)	0.01	15.66 (±2.39)	15.33 (±2.74)	n.s.
VAS	5.38 (±2.0)	5.13 (±2.53)	n.s.	5.73 (±1.97)	5.03 (±1.77)	n.s.
Body surf (%)	18.69 (±9.83)	14.97 (±9.1)	0.05	25.63 (±12.20)	19.20 (±10.81)	n.s.

TP, tender point; VAS, visual analogue scale; body surf (%), percentage of pain-affected body surface. Measurements were done within 1 week before and after training.

Table 5. Pain threshold values (average of left and right body side, mean ± SD) and number of positive thresholds (<4 kp) of individual bilateral TP sites in 27 patients of the training group

	TP – thresh.	TP – thresh.			Pos. TP	Pos. TP	
	Before training	After training	Diff.	p	Before	After	*p*
Occiput, bilat.	2.6 (±1.20)	2.6 (±1.26)	0	n.s.	52	48	n.s.
Low cervical, bilat.	2.02 (±0.7)	1.98 (±0.92)	−0.04	n.s.	54	54	n.s.
Trapezius, bilat.	2.13 (±0.71)	2.18 (±1.03)	0.05	n.s.	54	50	n.s.
Supraspinatus, bilat.	2.5 (±1.02)	2.63 (±1.41)	0.13	n.s.	53	46	n.s.
Second rib, bilat.	1.96 (±1.09)	2.28 (±1.24)	0.32	n.s.	53	48	n.s.
Lateral epicondyle, bilat	2.71 (±1.45)	3.02 (±1.58)	0.31	n.s.	45	42	n.s.
Gluteal, bilat.	2.89 (±1.4)	3.5 (±1.82)	0.61	<0.01	48	42	n.s.
Greater trochanter, bilat.	2.79 (±1.07)	3.18 (±1.56)	0.39	n.s.	46	43	n.s.
Knee, bilat.	2.44 (±9.3)	2.32 (±0.99)	−0.12	n.s.	52	47	n.s.

Measurements were done within 1 week before and after training.

(Table 4). The mean pain threshold of the 18 TP per person did not change significantly after training (Table 4). The VAS score also remained unchanged. Interestingly, we found a significantly decreased pain threshold for the gluteal TP after training (p<0.01), whereas other eight sites showed no significant change (Table 5). The painful body area also decreased significantly after training (p<0.05).

Seventeen individuals in the training group showed a substantial (p<0.05) subjective pain improvement on the ranking scale (2 × +3, 7 × +2, 10 × +1); eight in the group remained unchanged, whereas one felt −1 and one −2. One patient in the control group felt +2, three +1, one −1 and seven remained unchanged.

Discussion

Our aim was to test whether pain in FM patients could be improved by aerobic endurance exercise. By using a guided but self-managed training programme we aimed to motivate the patients [31], providing an easy schedule and circumstances under which they felt comfortable. The training instructions aimed at achieving a moderate training intensity in consideration of their individual performance capacities [30].

The training schedule was successful as it improved the aerobic fitness of the patients. It lowered the mean HR and VO_2 at submaximal workload levels as a result of adaptation, and improved performance economy. It also improved the Wattpulse (Watt/HR) after training at maximal workload. Exercise had no serious side-effects and resulted in a positive influence on patient wellness, TP count and painful body area.

Several hypothesis may explain our observations. Patients apparently perceive that pain is caused by strain [23], which prompts them to avoid any exercise causing more pain. In contrast, we found that more than half the patients were not only highly motivated to participate in a guided exercise programme, but also were able to complete a training schedule of moderate intensity. Our controlled exercise programme might therefore break through a vicious cycle of pain, perpetuating a lack of physical activity, related depression and more pain.

Different studies show that FM pain is influenced by sleep [32–34], therefore better sleep, improved by regular exercise [35], might contribute to pain reduction.

A better local muscular blood flow [29] and less susceptibility to muscular microtrauma as a result of regular training [36], might also improve symptomatic pain. FM patients show an impaired local glucose metabolism [13]. In healthy subjects insulin secretion, glucose uptake and metabolism are increased by moderate exercise [37]. The training of our patients was characterised by relatively short sessions of 20–30 minutes' duration, leading to predominant carbohydrate metabolism during exercise. Therefore, training increased RQ measured during the spiroergometry, indicating improved glucose utilisation. This adaptation possibly improves impaired muscle relaxation by increasing cellular ATP levels.

Disciplines that predominantly train the lower extremities (cycling, walking, jogging) where chosen by 81% of patients. Accordingly, we found a decreased pain threshold only for the gluteal TP. This may also support the hypothesis of a training-dependent metabolic effect on pain in FM patients. FM patients benefit from regular aerobic endurance exercise with moderate intensity, as it has no apparent negative effects, improves physical fitness and pain symptoms, and maybe also selfconfidence. Further investigations will aim at optimising the training programme and its intensity.

References

1. Wolfe F, Smythy HA, Yunus MB, et al. The American College of Rheumatology 1990: criteria for the classification of fibromyalgia. Arthritis Rheum 1990;33:160–72.
2. Bartels EM, Danneskiold-Samsoe B. Histological abnormalities in muscle from patients with certain types of fibrositis. Lancet 1986;1:755–7.
3. Bengtsson A, Henriksson KG, Larsson J. Muscle biopsy findings in primary fibromyalgia and other forms of nonarticular rheumatism. Scand J Rheumatol 1986;15:1–6.
4. Drewes AM, Andreasen A, Schröder HD, Hogsaa B, Jennum P.

Pathology of skeletal muscle in fibromyalgia: a histo-immunochemical and ultrastructural study. Br J Rheumatol 1993;23:479–83.
5. Jacobsen S, Bartrels EM, Danneskiold-Samsoe B. Single cell morphology of muscle in patients with chronic muscle pain. Scand J Rheumatol 1991;20:336–43.
6. Norregaard J, Bulow PM, Mehlsen J, Danneskiold-Samsoe B. Biochemical changes in relation to a maximal exercise test in patients with fibromyalgia. Clin Physiol 1994;14:159–67.
7. Pongratz D, Fischer P, Späth M, Müller-Höcker J. Gibt es ein myopathologisches Substrat der primären generalisierten Fibromyalgie? Zeitschr Rheumatol 1994;53:20.
8. Späth M, Obermaier-Kusser B, Fischer P, Pongratz D. Are point mutations or deletions of mitochondrial DNA of any significance for fibromyalgia? J Muskuloskeletal Pain 1995;1:116–17.
9. Yunus MB, Kalyan-Raman UP, Kalyan-Raman K, Masi AT. Pathological changes in muscle in primary fibromyalgia syndrome. Am J Med 1986;81:38–42.
10. Bengtsson A, Henriksson KG, Larsson J. Reduced high-energy phosphate levels in the painful muscles of patients with primary fibromyalgia. Arthritis Rheum 1986;29:817–21.
11. Eisinger J, Plantamura A, Ayavou T. Glycolysis abnormalities in fibromyalgia. J Am Coll Nutr 1994;13:144–8.
12. Lindh MH, Johansson G, Hedberg M, Henning GB, Grimby G. Muscle fiber characteristics, capillaries and enzymes in patients with fibromyalgia and controls. Scand J Rheumatol 1995;24:34–7.
13. Frey LD, Locher JTh, Hrycaj P, et al. Bestimmung der regionalen Glucose-Metabolisierungsrate der Lumbalmuskulatur bei Patienten mit generalisierter Tendomyopathie (GTM) mittels dynamischer 18F-FDG PET. Zeitschr Rheumatol 1992;51:238–42.
14. Lund N, Bengtsson A, Thorborg P. Muscle tissue oxygen pressure in primary fibromyalgia. Scand J Rheumatol 1986;15:165–73.
15. Elert JE, Rantapaa-Dahlqvist SB, Henriksson-Larsen K, Larentzon R, Gerdle BU. Muscle performance, electromyography and fibre type composition in fibromyalgia and work-related myalgia. Scand J Rheumatol 1992;21:28–34.
16. Brückle W, Suckfull M, Fleckenstein W, Weiss C, Müller W. Gewebe-pO2-Messung in der verspannten Rückenmuskulatur (m. erector spinae). Zeitschr Rheumatol 1990;49:208–16.
17. King S, Fisher BD, Burnham R, Maclean I, Bell G, Warren S. Cytokine and tissue oxygenation levels in myofascial pain and fibromyalgia syndromes in females as compared to controls. J Muskuloskeletal Pain 1995;1:36–7.
18. Bennett RM, Clark SR, Goldberg L, et al. Aerobic fitness in patients with fibrositis. A controlled study of respiratory gas exchange and 133xenon clearance from exercising muscle. Arthritis Rheum 1989;32:454–60.
19. Jacobsen S, Jensen KE, Thomsen C, Danneskiold-Samsoe B, Henriksen O. 31P magnetic resonance spectroscopy of skeletal muscle in patients with fibromyalgia. J Rheumatol 1992;19:1600–3.
20. Mannerkorpi K, Burckhardt CS, Bjelle A. Physical performance characteristics of women with fibromyalgia. Arthritis Care Res 1994;3:123–9.
21. Mengshoel AM, Forre O, Komnaes HB. Muscle strength and aerobic capacity in primary fibromyalgia. Clin Exp Rheumatol 1990;8:475–9.
22. Sietsma KE, Cooper DM, Caro XJ, Liebling MR, Louie JS. Oxygen uptake during exercise in patients with primary fibromyalgia syndrome. J Rheumatol 1993;20:860–5.
23. Bennett RM, Jacobsen S. Muscle function and origin of pain in fibromyalgia. Baillière's Clin Rheumatol 1994;8:721–46.
24. McCain GA, Bell DA, Mai FM, Halliday PD. A controlled study of the effects of a supervised cardiovascular fitness training program on the manifestation of primary fibromyalgia. Arthritis Rheum 1988;31:1135–41.
25. Granges G, Littelfohn GO. A comparative study of clinical signs in fibromyalgia/fibrositis syndrome, healthy and exercising subjects. J Rheumatol 1993;20:344–51.
26. Fischer A. Pressure threshold meter, its use for quantification of tender spots. Arch Phys Med Rehabil 1986;67:836–8.
27. Heberer G, Köhle W, Tscherne H (Hg.) Verbrennungen. In: Chirurgie und angrenzende Gebiete. Stuttgart: Thieme, 1989;118–19.
28. Revill SI, Robinson JO, Rosen M, Hogg MIJ. The reliability of a linear analogue scale for evaluating pain. Anaesthesia 1976;31:1191–8.
29. Stegemann J. Leistungsphysiologie. Stuttgart: Thieme, 1991; Ch 6.
30. Fletcher GF, Balady G, Froelicher VF, Hartely LH, Haskell WL, Pollock ML. Exercise standards. Circulation 1995;91:580–615.
31. Burckhardt CS, Bjelle A. Education programmes for fibromyalgia patients: description and evaluation. Baillière's Clin Rheumatol 1994;8:935–55.
32. Branco J, Atalaia A, Paiva T. Sleep cycles and alpha-delta sleep in fibromyalgia syndrome. J Rheumatol 1994;21:1113–7.
33. Moldofsky H, Scarisbrick P. Induction of neurasthenic musculoskeletal pain syndrome by selective sleep deprivation. Psychosom Med 1976;38:35–44.
34. Moldofsky H, Scarisbrick P, England R, Smythe H. Musculoskeletal symptoms and non-REM sleep disturbance in patients with ''fibrositis syndrome'' and healthy subjects. Psychosom Med 1975;37:341–51.
35. Shapiro CM, Bortz R, Mitchell D, Bartel P, Jooste P. Slow-wave sleep: a recovery period after exercise. Science 1981;214:1253–4.
36. Jakob E, Berg A, Keul J. Regenerative Möglichkeit spezieller Organsysteme – Muskulatur. Dtsch Zeitschr Sportmed 1993; 44:116–18.
37. Oshida Y, Yamynouchi K, Haymamizu S, Sato Y. Long term mild jogging increases insulin action despite no influence on body mass index or VO2 max. Am J Phys 1989:2206–10.

Received for publication 24 February 1999
Accepted in revised form 23 September 1999