Original Article

Relationship between Chest Expansion and Respiratory Muscle Strength in Patients with Primary Fibromyalgia

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Abstract: In this paper the assessment of the relationship between chest expansion with maximal inspiratory (MIP) and expiratory pressures (MEP) in primary fibromyalgia (FM) syndrome is discussed. Chest expansion (CE) measurements, spirometric values, and MIP and MEP values in 30 female patients with primary FM are compared with 29 healthy age-matched female controls. Patients with FM had lower CE, MEP and MIP values than controls. CE correlated significantly with MIP and MEP values. There was no significant difference between groups in spirometric values. Our results indicate that patients with FM have impaired respiratory muscle strength, and measurement of CE may be a useful clinical parameter. Despite its limitations CE may reflect respiratory muscle strength. It is worth following up these data in a wider and controlled series, with ancillary tests in addition to the MIP and MEP.

Keywords: Chest expansion; Fibromyalgia; Spirometry

Introduction

Fibromyalgia (FM) is characterised by widespread musculoskeletal pain, aching, and stiffness in the neck, mid and lower back, chest, arms and legs [1,2]. In recent studies FM has been reported as a common cause of chest wall pain and even dyspnoea [3–7]. Despite normal

spirometric values, patients with FM had lower values of maximum inspiratory (MIP) and expiratory pressures (MEP) than matched healthy controls. Values of MIP and MEP reflect respiratory muscle strength, and therefore low values have been explained by respiratory muscle weakness or dysfunction [5,6]. Recently we reported that patients with FM have reduced chest expansion with respect to healthy age- and sex-matched controls [8]. To our knowledge there is no prior study assessing the relationship of CE with MIP and MEP in FM. In this study we assessed the relationship between chest expansion and MIP, MEP values. In addition, pulmonary function tests, MIP, MEP and chest expansion of FM patients were compared with age- and sex-matched healthy controls.

Patients and Methods

Thirty female patients, aged 22–52 years, who met the 1990 ACR [1] criteria for the classification of FM, were examined and compared with 29 healthy, age- and BMI-matched female subjects (ages 24–50 years). All of these newly referred patients and controls were non-smoking premenopausal women, had no history or evidence of respiratory or cardiac illness, and no current therapy (pharmacological or physical training).

All the subjects were interviewed and examined for spinal deformities and other diseases that might account for the pain. All were naive to the measurement of maximal respiratory pressures. Blood was sampled and tested for erythrocyte sedimentation rate, blood count, thyroid hormone levels, rheumatoid factor, antinuclear

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antibody, creatine kinase, calcium and hepatic enzymes. ECG and chest films of all subjects were provided.

Tender points (TP) were examined using the protocol described by Wolfe et al. [1]. A score for number of TP was obtained and could range from 11 to 18. A myalgic score, which was a measure of pain in each tender point using a three-point scale of pain severity, and could range from 11 to 54, was also obtained.

Pulmonary function tests were assessed by a computerised spirometer (Sensor-Medics V_{max} 29, Yorba Linda, CA). The values were expressed as a percentage of the predicted normal values according to Kory/Polgar. The acceptable recordings were obtained from each manoeuvre and the highest values were used for further analysis [9,10].

The chest expansion was measured with the subjects standing with the hands on the hips. The tape measure was placed at the inframammary line in young females and at the fourth intercostal space in women with pendulous breasts. Chest expansion was taken as the difference to the nearest 0.1 cm between full expiration and inspiration. The score for three tries was recorded and the best of these was taken as the index of chest expansion [11].

The maximum respiratory pressures were carried out by using a digital mouth-pressure meter (Sensor-Medics MPM, Yorba Linda, CA). The maximum inspiratory pressure was measured with the subjects sitting and breathing through a mouthpiece connected to the digital MPM, just after clamping the nose. The maximum expiratory pressure was measured following end-inspiration by the same procedure. Both of the measurements were carried out three times and mean values were calculated as MIP and MEP in cmH₂O [12].

The data were analysed on a personal computer using SPSS software. An independent sample *t*-test was used for intergroup comparisons. Values were correlated using partial correlation coefficients to adjust for age and body mass index (BMI). A two-tailed P < 0.05 was considered statistically significant.

Results

Baseline data of the subjects are shown in Table 1. Number of tender points was 13.3 ± 1.9 (range 11-17) and total myalgic score was 25.5 ± 5.3 (range 17-32). Blood test results, ECG and chest films were normal in all subjects. Patients with FM had lower chest expansion, MEP and MIP values than the healthy controls, and these were statistically significant. There was no significant difference between groups in spirometric values, age or BMI, as shown in Table 1. There was a significant correlation between chest expansion and values of MIP (r: 0.49, P < 0.01) and MEP (r: 0.57, P < 0.001) (values are the partial correlation coefficients, adjusted for age and BMI) (Fig. 1). There was no significant correlation between total myalgic score and MIP, MEP or chest expansion.

In the control group there was no significant correlation between MIP, MEP or CE (r:0.23 and r:0.29, P > 0.05, respectively).

Ten patients (33%) complained of chest wall pain during inspiration and expiration. There was no significant difference in MIP or MEP values between these 10 patients (54.1 ± 17.2 and 70.0 ± 17.1, respectively) and the rest of the patients (n = 20) (54.1 ± 16.1 and 69.1 ± 14.5, respectively), P > 0.05. There was also no significant difference in CE between these patient subgroups (3.1 ± 0.9, n = 10 and 2.9 ± 0.8, n =20, P > 0.05).

Discussion

Although palpation of the chest wall and related structures is mentioned as a routine clinical examination procedure in FM, which is a common cause of chest wall pain, measurement of chest expansion has not been considered as a routine application. Patients with FM have no objective signs of inflammation and have unrestricted movement of joints, but alterations in

Table 1. Baseline data (means and standard deviations) on subjects

FM patients $(n = 30)$ Mean (SD) (range) Controls $(n = 29)$ Mean	(CD) ()
	(SD) (range)
Age 36.4 (9.5) (22–52) 38.4 (10.0) (24–50) DMU 26.6 (4.7) (10.4 24.2) 28.2 (5.1) (21.6 25.6)	
BMI $20.6 (4.7) (19.4-34.2)$ $28.3 (5.1) (21.6-35.6)$ Chest expansion (cm) [‡] $2.9 (0.8) (2-5)$ $4.3 (0.8) (3-5.5)$	
MIP $(cmH_2O)^{\dagger}$ 54.1 (16.2) (37–93.3) 69.1 (19.2) (36.2–88.6)	
$\begin{array}{ccc} \text{MEP} \ (\text{cmH}_2\text{O})^* & 69.4 \ (15.1) \ (44-91.2) & 94.4 \ (22.6) \ (71-136) \\ \text{VC} \ * & 107.3 \ (11.5) \ (95.128) & 114.1 \ (11.9) \ (95.133) \end{array}$	
FVC* 112.7 (13.8) (95–132) 113.5 (11.7) (95–133)	
$FEV_1^* 103.1 (7.3) (88-110) 104.7 (9.8) (89-121)$	
FEV_1/FVC 87.7 (8.7) (73–94)82.3 (3.3) (77–89)MEE*08.5 (21.6) (70.121)101.6 (14.4) (01.130)	
MET 25%-75% $26.3 (21.0) (70-121)$ $101.0 (14.4) (91-150)$ PEF* $87.8 (17.5) (72-119)$ $98.1 (13.0) (78-117)$	

^{* %} predicted

BMI:body mass index, MIP:maximal inspiratory pressure, MEP:maximal expiratory pressure, VC:vital capacity, FVC:forced vital capacity, FEV1:forced expiratory volume in one second, MEF 25%–75%:maximal expiratory flow at 25%–75% of the vital capacity, PEF:peak expiratory flow rate.

 $^{^{\}dagger} P < 0.05$

 $^{^{\}ddagger} P < 0.01$



Fig. 1. Relationship of chest expansion with MIP (0.49, p < 0.01) and MEP values (0.57, P < 0.001).

muscle strength and maximal voluntary muscle contraction have been widely studied and have demonstrated that these parameters are lower than expected [13–15]. Respiratory muscle strength has been evaluated by means of maximal expiratory and inspiratory pressures in patients with FM [5,6]. These authors concluded that patients with FM have lower MIP and MEP values, which may indicate respiratory muscle dysfunction and diaphragmatic muscular insufficiency, especially in patients suffering from dyspnoea. Even though our patients have not reported dyspnoea, they had lower MIP and MEP values with respect to the healthy matched controls. Reduced chest expansion and significant correlation with MIP and MEP values indicate that low CE measurement may be related to the reduced respiratory muscle strength in patients with FM. MIP and MEP reflect different muscle groups and may have different clinical values regarding the type of pulmonary

(i.e. obstructive or restrictive) or diaphragmatic involvement. Physiologically, the respiratory muscle pump may be considered to have an inspiratory and an expiratory component, and the expiratory muscles are critical for an effective cough, but their failure in isolation is not normally considered a cause of ventilator dependence [16]. Regarding inspiratory muscles, the diaphragm is the most important muscle in healthy humans, and during quiet respiration accounts for 60%-70% of lung volume change, which has a vital importance in respiration. The extradiaphragmatic inspiratory muscles are the scalenes and parasternal intercostals, which are invariably active even during quiet breathing in healthy subjects, and the sternomastoids, which are recruited in response to increased load [17]. Many pulmonary rehabilitation programmes include the assessment of MIP as an outcome measure for ventilatory muscle training [18]. Accordingly, correlation between CE and MIP may be considered to have a more significant value with respect to MEP.

Maximal respiratory pressures (MIP and MEP) are non-invasive methods both to assess the strength of inspiratory and expiratory muscles and to document the effects of pulmonary diseases, neuromuscular disorders and ageing on respiratory muscle strength [19]. Several factors, such as differences in technique, motivation and cooperation of subjects, number of times the subject attempts the manoeuvre, learning effects and the degree of fatigue may affect maximal respiratory pressure measurements, so that the normal range for these manoeuvres is wide and varies between laboratories [20–22]. Besides these technical and motivational factors there are also several additional factors that influence the measurements, such as diaphragmatic configuration, air trapping, and severe chronic obstructive or restrictive pulmonary diseases [22]. However, it is important to be cautious in interpreting low values of maximal respiratory pressure as an indication of respiratory muscle weakness, especially in uncontrolled or special patient groups with respiratory and neuromuscular disorders. Our study was a controlled one and the pulmonary status of the subjects was cleared with spirometric measurements. But it is worth following up these data with ancillary tests to separate some individual factors (i.e. electrical stimulation of the respiratory muscles during manoeuvres in order to be sure that the results are exactly maximal values).

Patients with FM have markedly decreased voluntary muscle strength [13–15], a possible explanation for which might be the motivational factors, reflex inhibition due to pain or fear of pain, and an impaired central drive for action [9,10]. Studies using electrical muscle stimulation indicate that the low voluntary muscle strength is due to submaximal contraction, most probably to supraspinal factors [13,14,23,24]. Reflex inhibition because of pain may be supported with an observation that chest expansion in FM patients was unconsciously self-limited in an attempt to prevent the chest wall pain [7,8]. However, we found no significant difference in maximal respiratory pressure in patients

who had chest pain during manoeuvres and those who did not. Also, there was no correlation between maximal respiratory pressure and total myalgic score. Therefore, reflex inhibition caused by the fear of pain may be properly considered, rather than pain itself. We have also demonstrated an improvement in chest expansion measurements consistent with the improvement in pain, depression and fatigue scales of the patients treated with omega-3 fatty acids [25]. This result may support the notion that CE measurement may be a useful clinical parameter.

In conclusion, we found reduced chest expansion, MIP and MEP in patients with FM, indicating that such patients do have impaired respiratory muscle strength. Measurement of CE may be a useful clinical parameter, as despite its limitations CE may reflect respiratory muscle strength. It is worth following up these data in a wider controlled series with ancillary tests in addition to the MIP and MEP.

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