with a spinal reflex bladder, mean values from a series of consecutive 10–50 ml/min fill cystometries seem to be useful. This method may be used in studies of the causal relation between a high detrusor pressure and renal complications and also for evaluation of any treatment which is assumed to change the detrusor pressure.

REFERENCES


Adress for offprints:
Mikael Thyberg
Department of Rehabilitation Medicine
University Hospital
S-58185 Linköping
Sweden

RESPIRATORY FUNCTION IN CHRONIC PRIMARY FIBROSYNALGIA

Miroslaw Lurie,1 Kenneth Caidahl,2 Göran Johansson3 and Björn Bake1

From the Department of Rehabilitation Medicine and Clinical Physiology, Sahlgrens’ University Hospital, Gothenburg, Sweden

ABSTRACT. Since patients with severe chronic primary fibrosynalgia (CPF) report effort dyspnoea, respiratory function was studied in 87 consecutive women with CPF according to Yannas’ criteria. Bernstein spirometry, maximum inspiratory (MIP) and expiratory (MEP) pressures were obtained in patients, and in a reference group of 61 healthy women. MIP was considerably lower in patients than in controls (3.6 ± 2.0 kPa, p < 0.0001), as was MEP (3.1 ± 2.1 vs. 8.3 ± 2.2 kPa, p < 0.0001). Patients who had previously reported dyspnoea at a bicycle exercise test showed significantly lower values of respiratory pressures than patients without dyspnoea. Respiratory pressures were reproducibly low in CPF patients. Spirometric values were normal among patients and controls. We conclude that maximum expiratory and inspiratory pressures are low in CPF, a finding which may indicate respiratory muscle dysfunction in this syndrome.

Key words: chronic primary fibrosynalgia (CPF), dyspnoea, respiration, respiratory pressures, spirometry.

Patients, mainly females, with chronic muscle pain of unknown etiology form an increasing problem to the medical health care system. The symptoms are often referred to as the Primary Fibrosynalgia (CPF) syndrome (19). The pain in this syndrome is often localized to the paravertebral muscles of the cervical and lumbar spine and the extremities but often also to the intercostal and abdominal muscles and the costovertbral junctions. Besides the pain, complaints of muscular fatigue and weakness are common, and morphologic changes verify muscular disease (10). Many patients regard their overall physical performance as reduced when compared to before onset of the symptoms. We have observed that dyspnoea is a prevalent symptom among patients with CPF, and that this symptom is associated with low physical performance on bicycle ergometer test (8). These findings raised the hypothesis that patients with CPF have impaired ventilatory function or respiratory muscle dysfunction. We have therefore made a controlled study of pulmonary function and maximum respiratory pressures in patients with CPF.

MATERIAL AND METHODS

We studied 87 consecutive female patients, who fulfilled the criteria of CPF (8, 19), and were admitted to our rehabilitation clinic for vocational guidance and pain treatment. The patients were admitted mainly from orthopedic surgeons but also from rheumatologists and general practitioners. Secondary fibrosynalgia, as well as other musculoskeletal, neuro-muscular and neurological disease were excluded by routine clinical examination and medical history. Inflammatory rheumatic disease was excluded by laboratory tests. Pulmonary disease was excluded by medical history and physical examination.

The patients, 42 immigrants and 45 Swedes, were all blue-collar workers. They were on sick leave or on early retirement since at least 12 months. The pain symptoms of CPF were of long duration (mean 3.5 years, range 2–11 years).

As a control group we selected 61 healthy Swedish women, mainly hospital staff, without symptoms from muscles, joints or respiratory tract. Despite an effort to recruit age matched controls they were somewhat younger than patients (p < 0.001), Table 1. Patients were significantly shorter (p < 0.001) and heavier (p < 0.001) than controls, while smoking habits did not differ significantly.

All subjects passed pulmonary function tests including measurement of vital capacity (VC), forced expiratory volume during one second (FEV1) and maximum inspiratory and expiratory pressures (MIP, MEP). VC and FEV1 were recorded by means of a water-sealed Bernstein spirometer. Three acceptable recordings were obtained from each male and the highest values were used for further analysis. The values were expressed as percentage of the predicted normal values according to Bergland et al. (1). The maximum respiratory pressures were obtained at ordinary end-expira- tion (functional residual capacity (FRC) level) with the sub- jects seated breathing in a mouth-piece connected to a three-way valve. A pressure-transducer connected to a pressure transducer, in turn connected to an ink-jet recorder (Stentschmidt). The experienced techni-
Table I. Physical characteristics and smoking habits. Values are mean (range)

<table>
<thead>
<tr>
<th>Physical characteristics</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Smoking habits (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=487)</td>
<td>(n=46-65)</td>
<td>(n=46-176)</td>
<td></td>
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<tr>
<td></td>
<td>45.9</td>
<td>163.5</td>
<td>68.2</td>
<td>Non-smokers 46.9</td>
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<tr>
<td></td>
<td></td>
<td>(26-65)</td>
<td>(46-176)</td>
<td>Ex-smokers 12.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(46-65)</td>
<td>(46-115)</td>
<td>Active smokers 40.7</td>
</tr>
<tr>
<td>Controls (n=61)</td>
<td>36.8</td>
<td>167.0</td>
<td>61.8</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>(19-62)</td>
<td>(55-181)</td>
<td></td>
</tr>
</tbody>
</table>

RESULTS

Maximum inspiratory and expiratory pressures were low and significantly reduced compared to the controls (3.6±2.0 vs. 8.0±2.2 kPa, p<0.0001; and 3.1±2.2 vs. 4.3±2.2 kPa, p<0.0001). There was no correlation between respiratory pressures and age, height or weight in the control group. Mean respiratory pressures of the control group were therefore used as predicted normal values without adjustments. Maximum respiratory pressures as well as VC and FEV1, are shown in relation to predicted normal values in Fig. 2. VC and FEV1 of both groups were normal according to the reference values which we use for clinical investigations (5). VC and FEV1 among controls were slightly higher than among the patients.

Almost two thirds (61%) of the patients perceived dyspnea during a bicycle exercise test. They were compared to non-dyspneic patients (39%) as regards spirometric data and maximum respiratory pressures. Maximum respiratory pressures were lower in the dyspneic group (p<0.05), while VC and FEV1 did not differ, Fig. 3.

Within the patient group, MIP (p<0.002) and MEP (p<0.0001) were lower among the immigrants. VC and FEV1 did not differ between Swedes and immigrants. Dyspnea as a reason for interruption of the bicycle exercise test was equally distributed among Swedes and immigrants.

Reproducibility results are given in Table II. There was no significant difference between the first and second measurement in any group. In 45/48 patients myopathy of the trapezius muscle was present as judged by EMG. No correlation was observed between the presence of myopathy and VC, FEV1, respiratory pressures or dyspnea.

Fig. 1. Maximum inspiratory pressure (MIP) was measured as the lowest pressure which was held during 1 s.

Fig. 2. Vital capacity (VC), and forced expiratory volume during 1 s (FEV1) were slightly higher in the control group both p<0.05 despite normal findings in patients according to our reference values (5). Maximum inspiratory (MIP) and expiratory (MEP) pressures in the 87 women with chronic primary fibromyalgia (CPF) were, however, markedly lower than in the control group of 61 healthy women (both p<0.001).

Fig. 3. Those of the CPF patients who reported dyspnea during an exercise stress test (n=46) were compared to those who did not (n=31). All values are expressed as % of predicted normal. VC and FEV1 did not differ between the groups, while MIP and MEP were slightly lower (both p<0.03) in the dyspneic group. Abbreviations as in Fig. 2.
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<table>
<thead>
<tr>
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<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Smoking habits (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Non-smokers</td>
</tr>
<tr>
<td>Patients (n=87)</td>
<td>45.9</td>
<td>163.5</td>
<td>68.2</td>
</tr>
<tr>
<td>(26-65)</td>
<td>(46-170)</td>
<td>(46-115)</td>
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<tr>
<td>Controls (n=63)</td>
<td>36.8</td>
<td>167.0</td>
<td>61.8</td>
</tr>
<tr>
<td>(19-62)</td>
<td>(53-181)</td>
<td>(49-79)</td>
<td></td>
</tr>
</tbody>
</table>

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Maximum inspiratory and expiratory pressures were low and significantly reduced compared to the controls (3.6±2.0 vs. 8.0±2.2 kPa, p<0.0001; and 3.1±2.1 vs. 4.8±2.3 kPa, p<0.0001). There was no correlation between respiratory pressures and age, height or weight in the control group. Mean respiratory pressures of the control group were used as predicted normal values without adjustments. Maximum respiratory pressures as well as VC and FEV₁ are shown in relation to predicted normal values in Fig. 2. VC and FEV₁ of both groups were normal according to the reference values we use for clinical investigations (5). VC and FEV₁ among controls were slightly higher than among the patients.

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Table II. Repeated measurement (I and II) of maximal respiratory pressures in a subsample of 32 healthy women and 10 women with CFP

<table>
<thead>
<tr>
<th>Measurement (mean ± SD)</th>
<th>Correlation r</th>
<th>Intrapatient variance (kPa)</th>
<th>Variation coefficient (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (kPa)</td>
<td>II (kPa)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>Controls</td>
<td>All</td>
<td>Patients</td>
</tr>
<tr>
<td>2.2±1.0</td>
<td>2.3±1.8</td>
<td>0.32</td>
<td>1.16</td>
</tr>
<tr>
<td>7.2±2.3</td>
<td>5.9±2.3</td>
<td>0.62</td>
<td>1.33</td>
</tr>
<tr>
<td>All</td>
<td>6.1±3.0</td>
<td>6.7±3.3</td>
<td>0.80</td>
</tr>
<tr>
<td>Maximum inspiratory</td>
<td>Patients</td>
<td>Controls</td>
<td>All</td>
</tr>
<tr>
<td>pressure</td>
<td>2.9±1.6</td>
<td>3.0±1.2</td>
<td>0.72</td>
</tr>
<tr>
<td>Controls</td>
<td>7.8±2.2</td>
<td>8.0±1.9</td>
<td>0.69</td>
</tr>
<tr>
<td>All</td>
<td>6.7±2.1</td>
<td>6.9±2.7</td>
<td>0.83</td>
</tr>
</tbody>
</table>

All patients fulfilled the criteria of CFP as suggested by Yamas (19). Other neurological, orthopedic or rheumatological diseases were excluded as far as possible by routine clinical procedures. None of the patients had respiratory disease. As a study group, therefore, they seemed homogeneous.

Patients with CFP often have several symptoms in common with neurocirculatory asthenia (16), such as chest pain, vegetative symptoms, and dyspnea. Since some symptoms of CFP, such as hallucinations and severe neuroasthenia may indicate a psychiatric disorder, such an etiology of the CFP syndrome has been suggested (11), but seems less likely (1). Psychologic factors are necessarily involved in a multisymptomatic disease centered around aches and pain, but also...
possible influence on the investigation from such factors could hardly explain the rather homogeneous pattern of low respiratory pressures in the presence of normal VC and FEV. The similar reproducibility of the measurements between patients and controls makes it also unlikely that mental factors were of major importance.

The intravariability variances for MEP and MIP were similar in patients and controls, while the variation coefficient was high in patients due to low mean values in this group. This means that the method will not separate between patients but between patients and controls. We found normal values of VC and FEV, among the CFP women, indicating normal dynamic (i.e. shortening of muscle fibres) capacity of their respiratory muscles. On the other hand their maximum respiratory pressures were low as compared to those found in healthy women, a finding indicating low isometric (i.e. no appreciable shortening of muscle fibres) capacity of their respiratory muscles. This is in accordance with the observation by Jacobsen & Danneskiold-Samsøe (12) that particular isometric muscular capacity is lowered in CFP patients. Furthermore, it is in line with observations from work-situations where the patient often finds static loads to increase the pain, exhaustion and subjective swelling of their muscles. Presence of muscular involvement in the CFP syndrome seems fairly convincing from EMG findings of myopathy in the present study, and microscopic (13) as well as biochemical (2, 3, 14) evidence of muscular abnormalities. It was recently shown that handgrip strength is reduced in CFP patients, as is muscular relaxation rate (7).

It has been observed that patients with CFP show signs compatible with dysfunction at the brain stem level (4, 15, 18). The dyspnoe observed among patients with CFP could therefore be the result of a disturbed function of the respiratory centre in the brain stem. We evaluated this possibility by CO2 stimulation of the central drive on the respiratory muscles. However, the CO2 response-curve was normal in 12 randomly chosen patients. Thus, a dysfunction of the chemical drive at the brain stem level cannot explain the inefficiency of the respiratory muscles. Despite this, central nervous processing may, together with respiratory muscle dysfunction, explain the dyspnoea noted.

It is concluded that CFP patients seem to have an isometric type of dysfunction of their respiratory muscles, since respiratory pressures were reproduci-

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Address for offprints: Keesth Cusalak, M.D.
Department of Clinical Physiology
Sahlgrens' University Hospital
S-41345 Gothenburg, Sweden

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