Pulmonary function and respiratory muscle strength in chronic heart failure: comparison between ischaemic and idiopathic dilated cardiomyopathy

M Daganou, I Dimopoulou, P A Alivizatos, G E Tzelepis

Abstract
Objective—To compare pulmonary function and respiratory muscle strength in patients with ischaemic and idiopathic dilated cardiomyopathy, well matched for indices of heart failure.

Methods—The study involved 30 patients with ischaemic cardiomyopathy and 30 with idiopathic dilated cardiomyopathy. The groups were well matched for age, weight, and clinical severity of cardiac dysfunction as assessed by ejection fraction and the New York Heart Association functional class. There were more smokers in the ischaemic group (p < 0.05), but indices of pulmonary function were comparable.

Results—Mean (SD) maximum static inspiratory pressure was lower in dilated cardiomyopathy than in ischaemic cardiomyopathy (73 (20) vs 84 (22) cm H2O, p < 0.05), as was the maximum static expiratory pressure (90 (20) vs 104 (21) cm H2O, p < 0.05).

Conclusions—For a given degree of cardiac dysfunction, the respiratory muscles are weaker in patients with idiopathic dilated cardiomyopathy than in those with ischaemic cardiomyopathy.

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Keywords: respiratory muscles; congestive heart failure; pulmonary function; cardiomyopathy

Respiratory abnormalities, such as restrictive and obstructive defects, diffusion impairment, and respiratory muscle weakness, are common in patients with chronic congestive heart failure. Of these, respiratory muscle weakness has received particular attention as studies have linked it to exercise intolerance and dyspnoea in these patients.

The underlying cause of respiratory muscle weakness is uncertain, but one potential contributing factor might be reduced muscle perfusion related to low cardiac output. Furthermore, respiratory muscle weakness has been described in patients with both ischaemic cardiomyopathy and idiopathic dilated cardiomyopathy, but it is unknown whether the respiratory muscles are equally affected in these diseases. Indirect evidence in favour of differences in respiratory muscle strength between the two types of cardiomyopathy is raised by various studies, suggesting a greater skeletal muscle involvement in idiopathic dilated cardiomyopathy than in ischaemic cardiomyopathy.

We compared pulmonary function and respiratory muscle strength in two groups of patients with ischaemic cardiomyopathy and idiopathic dilated cardiomyopathy, well matched for indices of heart failure. To the extent that dilated cardiomyopathy is associated with greater muscle impairment, we anticipated greater respiratory muscle weakness in this group than in the patients with ischaemic cardiomyopathy.

Methods
We studied 60 ambulatory patients with chronic heart failure secondary to ischaemic cardiomyopathy or idiopathic dilated cardiomyopathy. All were referred to our institution for possible cardiac transplantation or assessment of heart failure. The diagnosis of ischaemic cardiomyopathy was confirmed angiographically in all patients. Four of the patients had undergone coronary artery bypass graft surgery in the past. All patients were in a stable haemodynamic condition, receiving conventional treatment, and had been free of acute exacerbations for a period of at least three months before evaluation. The study was approved by the institutional review board.

Pulmonary function testing included spirometry, lung volume, and determination of single breath diffusion capacity for carbon monoxide (DLCO). All measurements were performed with a Jaeger system (Masterlab, Jaeger, Wuerzberg, Germany). Spirometric indices were calculated from the best of three satisfactory efforts, defined as the effort associated with the highest sum of forced vital capacity (FVC) and forced expiratory volume in one second (FEV1). Static lung volumes were determined by the helium dilution method and DLCO values were adjusted for anaemia. All values are mean (SD) unless otherwise specified.

NYHA, New York Heart Association functional class.
Respiratory muscle strength was assessed by measuring maximum static inspiratory pressure (PImax) and expiratory pressures (PEmax) at functional residual capacity (FRC) and total lung capacity (TLC), respectively. The pressures were measured using a calibrated manometer connected through a cylinder to a mouthpiece; a small hole in the cylinder prevented the use of buccal muscles. Mouth pressures were measured by a technician unaware of the purpose of the study. Measurements were made in the seated position and all patients were verbally encouraged to achieve maximum strength and sustain it for about two seconds. Several minutes of rest were allowed between efforts. Repeated efforts were made until the two highest values agreed within 10%. The highest value achieved and maintained for between 5 and 10 seconds. Several minutes of rest were allowed before each measurement. The mechanisms underlying this weakness remain obscure. The reported alterations in muscle fibre type, size, and enzyme content do not appear to be related to deconditioning, malnutrition, or electrolyte abnormalities.

Respiratory muscle strength measurements using the same method were also made in a group of normal volunteers (control group, n = 16), matched for age and sex.

<table>
<thead>
<tr>
<th>Pulmonary function data</th>
<th>ICM (n = 30)</th>
<th>IDCM (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (litres)</td>
<td>3.9 (0.9)</td>
<td>3.8 (0.8)</td>
</tr>
<tr>
<td>% predicted</td>
<td>93 (16)</td>
<td>93 (18)</td>
</tr>
<tr>
<td>FEV1 (litres)</td>
<td>2.7 (0.7)</td>
<td>2.7 (0.7)</td>
</tr>
<tr>
<td>% predicted</td>
<td>88 (19)</td>
<td>88 (20)</td>
</tr>
<tr>
<td>FEV1/FVC (%)</td>
<td>70 (6)</td>
<td>71 (9)</td>
</tr>
<tr>
<td>TLC (litres)</td>
<td>5.3 (1.1)</td>
<td>5.1 (1.2)</td>
</tr>
<tr>
<td>% predicted</td>
<td>84 (13)</td>
<td>83 (15)</td>
</tr>
<tr>
<td>VA (litres)</td>
<td>5.3 (1.4)</td>
<td>5.7 (1.8)</td>
</tr>
<tr>
<td>DLCO (% predicted)</td>
<td>84 (22)</td>
<td>90 (19)</td>
</tr>
</tbody>
</table>

Values are mean (SD); p not significant for all values.}

Differences among categories of heart failure, idiopathic dilated cardiomyopathy being associated with lower respiratory muscle strength (by about 15%).

Discussion

Respiratory muscle weakness is often found in ambulatory patients with stable congestive heart failure and has been implicated in causing dyspnoea and exercise limitation in these patients. The mechanisms underlying this weakness remain obscure. The reported alterations in muscle fibre type, size, and enzyme content do not appear to be related to deconditioning, malnutrition, or electrolyte abnormalities. Because respiratory muscle weakness correlated with severity of heart failure in some studies, it has been proposed that reduced muscle perfusion may play a key role in the pathogenesis of a myopathy affecting all skeletal muscles. Indeed, a suboptimal increase in blood flow has been documented in exercising peripheral muscles, which may account for a reduction in slow oxidative (type Ia) fibres and a switch to less efficient anaerobic metabolism. However, current evidence suggests that respiratory muscle weakness may not simply be part of a generalised skeletal muscle weakness. Recent studies showed that respiratory muscle strength may not correlate with limb muscle strength and that reductions in PImax and PEmax are proportionally greater than reductions in skeletal muscle strength. These findings therefore raised the question as to whether the respiratory muscles may be more vulnerable than limb muscles to the detrimental effects of congestive heart failure.

Our study confirms previous studies reporting respiratory muscle weakness in congestive heart failure and further extends them by showing quantitative differences among categories of heart failure, idiopathic dilated cardiomyopathy being associated with lower respiratory muscle strength (by about 15%).
In idiopathic dilated cardiomyopathy was provided by two studies, in which respiratory muscle strength was assessed in a small group of patients with ischaemic cardiomyopathy and idiopathic dilated cardiomyopathy. Average values for respiratory pressures calculated from their data are shown in table 4.

Our study may have implications for training the respiratory muscles in patients with congestive heart failure. In a recent study, selective respiratory muscle training improved maximum exercise capacity and decreased dyspnoea during daily activities in patients with heart failure. Our findings of differences in respiratory muscle strength between subcategories of patients with congestive heart failure raise the question of whether the two groups respond differently to respiratory muscle training.

In conclusion, our data suggest that there are quantitative differences in respiratory muscle strength between subgroups of patients with congestive heart failure and comparable cardiac dysfunction, and that respiratory muscle weakness is greater in idiopathic dilated cardiomyopathy than in ischaemic cardiomyopathy. Further studies are required to elucidate the cause of this difference as well as to investigate any clinical implications.