

Respiratory muscle strength in chronic heart failure

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Abstract

Background – Several studies have suggested that the respiratory muscles are weak in patients with heart failure, but the aetiology and clinical relevance of this weakness are unclear. In order to see if respiratory muscle weakness in this context is part of a more generalised myopathic process, respiratory and limb muscle strength were compared in patients with heart failure. The relation between respiratory muscle strength, breathlessness on exercise, and exercise capacity was also examined.

Methods – Twenty patients (three women) with New York Heart Association (NYHA) class II–IV heart failure of mean age 63 years were studied. Respiratory muscle strength was assessed using maximum inspiratory and expiratory mouth pressures (MIP and MEP) and transdiaphragmatic pressure during sniffs (sniff PDI). These parameters were compared with cardiac output (indirect Fick) and with limb muscle strength as assessed by grip strength. The patients also performed two exercise tests during which they rated their breathlessness on a Borg scale.

Results – Mean (SD) cardiac index was 2.2 (0.4) l/min/m². MIP and MEP were 66 (27) and 99 (29) cm H₂O, respectively. Sniff PDI was 103 (21) cm H₂O and was positively correlated with grip strength and cardiac output (Spearman rank correlation coefficients 0.527 and 0.451, respectively). None of the indices of respiratory muscle strength were related to exercise time or breathlessness during exercise.

Conclusions – The respiratory muscles are weak in patients with heart failure. This weakness reflects a more generalised myopathic process, possibly related to reduced cardiac output. However, respiratory muscle weakness does not appear to be an important factor in the aetiology of breathlessness on exercise.

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Breathlessness on exercise is a prominent complaint of patients with heart failure, but the aetiology of this symptom remains unclear. One possible cause which has not been fully explored is respiratory muscle dysfunction. Ventilation during exercise is higher in patients with heart failure than in normal subjects, and although only a proportion of these patients stop because of breathlessness, respiratory

muscle weakness might nevertheless be important in determining their exercise capacity.

In 1980 De Troyer *et al* inferred from oesophageal pressure measurements that patients with mitral stenosis have weak inspiratory muscles.¹ More recently, reduced maximum mouth pressures have been reported both in patients with mitral stenosis and in those with heart failure from other causes.^{2–6} It remains unclear, however, why the respiratory muscles are weak in these patients. Furthermore, little attempt has been made to link this weakness with breathlessness or exercise limitation.

We have therefore studied respiratory muscle strength in patients with heart failure and looked to see if weakness is related to poor cardiac output. We have also compared respiratory and limb muscle strength in these patients to see if respiratory muscle weakness is part of a generalised myopathic process. Finally, we have investigated whether respiratory muscle strength is related to either exercise capacity or dyspnoea on exercise.

Methods

PATIENTS

Twenty patients with chronic heart failure (three women) with a mean age of 63 years were studied. The mean (SD) height and weight were 1.69 (0.05) m and 73.6 (10.6) kg respectively. All gave informed consent to the study, which was approved by the hospital ethical committee. The aetiology of heart failure was ischaemic heart disease (n=14), valvular lesions (n=2), hypertension (n=2) or dilated cardiomyopathy (n=2). All patients had an enlarged heart on chest radiography and echocardiographic evidence of impaired left ventricular function. Their New York Heart Association (NYHA) classification of heart failure severity was II (n=8), III (n=10) or IV (n=2). All were taking diuretics, 13 angiotensin converting enzyme inhibitors and three digoxin. The serum potassium level was normal in all patients. For each patient all investigations were carried out over a maximum period of 10 days. All patients were clinically stable with no history of recent upper respiratory tract infection, and their treatment did not change during the period of the study. None suffered from pulmonary or neuromuscular disease or other systemic diseases which could have affected muscle function.

LUNG VOLUMES

Forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) were

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measured using a bellows spirometer (Vitalograph, Buckingham, UK). Functional residual capacity (FRC), residual volume (RV), and total lung capacity (TLC) were measured by helium dilution (PK Morgan, Gillingham, Kent, UK).

RESPIRATORY MUSCLE STRENGTH

Maximum inspiratory pressure (MIP) was measured at FRC using a flanged mouthpiece with a small leak connected to a pressure transducer (PK Morgan, Gillingham, Kent, UK). Maximum expiratory pressure (MEP) was measured at TLC using the same apparatus. After education in the techniques for performing each manoeuvre, several recordings were made until at least three tracings with reproducible pressure plateaus were obtained. The best values of MIP or MEP, sustained for at least one second, were compared with normal ranges obtained using the same technique.⁷

Transdiaphragmatic pressure (PDI) was recorded using catheter-mounted pressure transducers (Gaeltec, Skye, UK) during 10 maximal sniffs commenced at FRC.⁸ The values obtained (sniff PDI) were compared with those recorded in the same laboratory using the same equipment in 50 normal subjects.⁹ Within subject reproducibility for MIP, MEP, and sniff PDI in our laboratory is within 10%.

LIMB MUSCLE STRENGTH

Limb muscle strength was assessed by measuring grip strength in the dominant hand with a dynamometer (Therapeutic Equipment, Clifton, USA). Patients were seated with the elbow supported and the forearm horizontal. The best of five maximal contractions, sustained for five seconds with five seconds of rest between, was compared with predicted values taken from Mathiowetz *et al.*¹⁰

EXERCISE CAPACITY

Patients were exercised to symptom limited maximum using two different treadmill exercise tests, performed in random order on different days. The two tests comprised an incremental (modified Bruce) protocol, and a fixed protocol in which the slope and rate were set at the equivalent of stage four of the incremental test. Ventilation was calculated by integration of the signal from a pneumotachograph attached to a mouthpiece and valve. The expired gas was passed through a mixing chamber to a mass spectrometer (VG Quadrupoles, Middlewich, Cheshire, UK) sampling at one second intervals. Readings were made over the last minute of each stage of the incremental protocol and after two and five minutes of the fixed protocol. For each reading mixed expired gas concentrations, tidal volume, and respiratory rate were averaged over one minute. Borg scale ratings for breathlessness were also obtained at the end of each stage of the incremental protocol, and after two and five minutes of the fixed protocol.

CARDIAC OUTPUT

Resting cardiac output was measured by the indirect Fick principle, using carbon dioxide as the indicator. Ventilation and mixed expired carbon dioxide concentrations were measured with the subject at rest using the same equipment as in the exercise tests. Carbon dioxide production was measured over one minute, during which time arterial carbon dioxide tension was estimated from the end tidal concentration. Mixed venous carbon dioxide tension was then estimated by asking the subject to rebreathe from an anaesthetic bag, initially containing 10% carbon dioxide. This method of calculation of cardiac output has been shown to correlate well in our laboratory with the thermodilution technique.¹¹

DATA ANALYSIS

Statistical comparisons were made using the FASTAT package on a microcomputer, taking 0.05 as the level of statistical significance. Comparisons of mean values with normal range data were made using unpaired *t* tests. Spearman rank order correlation coefficients were used to investigate the relationship between indices of respiratory muscle strength and the other variables measured. Results are expressed as mean (SD).

Results

Table 1 shows the lung volumes of the patients and table 2 the indices of muscle strength. MIP, MEP, and sniff PDI were all significantly reduced from normal ($p < 0.01$). Grip strength was also reduced from normal ($p < 0.01$) in proportion to the reduction in sniff PDI (figure): (Sniff PDI (% pred) = $44.8 + 0.53 \times$ grip

Table 1 Mean (SD) lung volumes ($n = 20$)

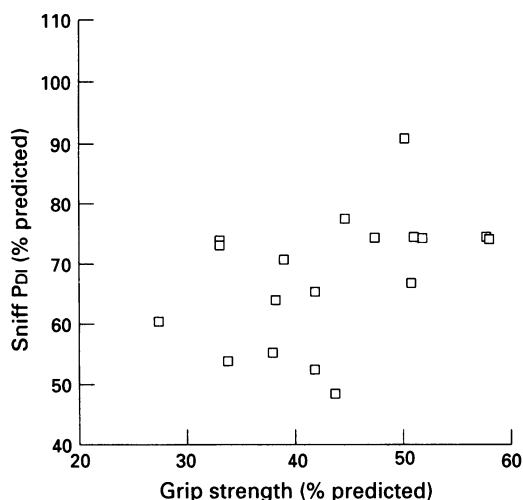
	Mean (SD)
FEV ₁ (% pred)	82.4 (20.5)
FVC (% pred)	81.9 (13.8)
FEV ₁ /FVC (%)	73.6 (10.6)
TLC (% pred)	91.0 (17.9)
FRC (% pred)	77.4 (11.7)
RV (% pred)	82.8 (10.9)
RV/TLC (%)	38.1 (5.06)

FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity; TLC = total lung capacity; FRC = functional residual capacity; RV = residual volume.

Table 2 Mean (SD) respiratory and limb muscle strength ($n = 20$)

MIP	
cm H ₂ O	66 (27)
% pred	64 (24)
MEP	
cm H ₂ O	99 (29)
% pred	70 (17)
Sniff PDI	
cm H ₂ O	103 (21)
% pred	71 (17)
Hand grip	
Newtons	37 (10)
% pred	43 (9)

MIP = mean inspiratory pressure; MEP = mean expiratory pressure; PDI = transdiaphragmatic pressure.



Relationship between grip strength and sniff PDI

Table 3 Mean (SD) exercise time, ventilation and oxygen consumption (n = 18)

	Incremental protocol	Fixed protocol
Exercise time (s)	496 (273)	387 (287)
\dot{V}_{max} (l/min)	37 (10)	40 (11)
$\dot{V}_{\text{O}_2\text{max}}$ (ml/min/kg)	13.2 (3.5)	14.8 (3.3)

strength (% pred), $r=0.527$, $p<0.05$). The mean cardiac index was 2.2 (0.4) l/min/m² which was related to sniff PDI: (Sniff PDI (% pred) = $54.7 + 7.3 \times$ cardiac index (l/min/m²), $r=0.451$, $p<0.05$). By contrast, neither MIP nor MEP were significantly related to grip strength or cardiac index. None of the indices of respiratory muscle strength correlated with lung volumes.

Two patients were unable to complete the exercise tests because of angina or claudication. Table 3 gives details of the exercise tests in the other 18 subjects. There was no significant relation between any index of respiratory muscle strength and exercise duration, maximum ventilation achieved or maximal oxygen uptake ($\dot{V}_{\text{O}_2\text{max}}$). Borg scores at each stage were not related to any index of respiratory muscle strength nor to lung volumes.

Discussion

We have used measurement of sniff PDI to confirm that, in patients with heart failure, inspiratory muscle strength is reduced. It has

been suggested that this merely reflects changes in lung volume, altering the position of the muscles on their operational length-tension curves to a suboptimal position. In our patients the differences in lung volumes from predicted values (table 1) were not great, but the reduction in TLC could explain at least some of the lowering of MEP. However, the reduction in FRC would tend to increase the MIP and sniff PDI values obtained, whereas we found that these parameters were reduced.

Respiratory muscle weakness in heart failure could also be explained by poor perfusion of these muscles. Compared with previous studies (table 4), there is no clear relation between the degree of weakness found and the severity of heart failure in the patients studied. Nevertheless, one previous study³ also found a weak correlation between MIP and cardiac index, suggesting that muscle perfusion may indeed be involved in the aetiology of respiratory muscle weakness.

Does respiratory muscle weakness merely reflect a more generalised myopathy? "Cardiac cachexia" is a well recognised clinical condition and abnormalities of peripheral muscles have been described in patients with heart failure. The only other report of paired respiratory and limb muscle strength measurements is that of Hammond *et al*² who noted mildly impaired grip strength with the respiratory muscles being more severely affected. Grip strength was slightly higher in our patients, but a similar pattern was observed when this was compared with the inspiratory muscles.

If we accept that the respiratory muscles are weak in patients with heart failure, and that the work of breathing in these patients is increased,¹² muscle fatigue can be proposed as a possible factor limiting exercise tolerance. Mancini *et al*⁴ and Davies *et al*¹³ have shown that mouth pressures decrease during exercise in patients with heart failure, but these volitional manoeuvres are highly dependent on patient effort and are very weak evidence of muscular fatigue. Davies *et al* found, using gasps through a Starling resistor, that the relaxation rate of mouth pressure slowed at peak exercise, suggesting that this reflected inspiratory muscle fatigue. Mancini *et al*, however, using more direct techniques, failed to find any reduction in the force generated by the diaphragm in response to supramaximal phrenic nerve stimulation. In our study maximum ventilation during exercise was not related to respiratory muscle strength, and no convincing evidence has yet been presented to support the suggestion that respiratory muscle fatigue limits exercise tolerance in heart failure.

McParland *et al*⁵ showed that maximum mouth pressure correlated with the baseline dyspnoea index, but this could reflect a common aetiological factor rather than a causal relationship. Mancini *et al*¹⁴ found that Borg ratings of effort during exercise were related to pre-exercise MIP and MIP, but also to FEV₁. We did not confirm this finding. Interestingly, their study showed that dyspnoea was the symptom which limited exercise in 50% of normal subjects and the same proportion of

Table 4 Published data on maximum mouth pressure in heart failure

Author	n	NYHA class (mean)	LVEF (%)	$\dot{V}_{\text{O}_2\text{max}}$ (ml/min/kg)	MIP (cm H ₂ O)	MEP (cm H ₂ O)
Present study	20	2.7	?	13.2	66	99
Hammond <i>et al</i> ²	16	?	26	?	41	59
Nishimura <i>et al</i> ³	13	3.5	?	11.9	60	84
Nishimura <i>et al</i> ³	10	2	?	18.6	92	104
Mancini <i>et al</i> ¹⁴	10	2.7	18	15.6	81	161
McParland <i>et al</i> ⁵	9	2.7	26	?	80	114
Ambrosino <i>et al</i> ⁶	25	2	23	?	88	118
Ambrosino <i>et al</i> ⁶	20	3	20	?	70	103

NYHA = New York Heart Association; LVEF = left ventricular ejection fraction; $\dot{V}_{\text{O}_2\text{max}}$ = maximum oxygen consumption; MIP = maximum inspiratory pressure; MEP = maximum expiratory pressure.

patients with mitral stenosis. This was the case in only 20% of patients with heart failure from other causes, fatigue being a more common limiting symptom. This finding suggests that perhaps patients with long standing valvular heart disease, such as those studied by De Troyer *et al.*¹ should be considered separately when evaluating respiratory muscle function in heart failure. Breathlessness may be less important in heart failure from other causes, and in any case is likely to be multifactorial in origin. Our data suggest that respiratory muscle strength is not one of these factors.

In conclusion, we confirm that the respiratory muscles are indeed weak in patients with chronic heart failure, and that this weakness is related to cardiac output. The weakness reflects a global myopathy with respiratory and limb muscle strength being reduced in proportion. Simple tests of strength suggest that respiratory muscle weakness neither limits exercise capacity nor, indeed, is involved in the aetiology of breathlessness on exercise. More work is needed on respiratory muscle endurance in heart failure, and paired measurements of strength and endurance before and after treatment should also be performed. When these data are available it should be apparent whether or not specific interventions to improve respiratory muscle function – for example, endurance training regimes – are likely to be worthwhile in alleviating symptoms in patients with heart failure. Respiratory

muscle weakness appears not to limit exercise capacity or, indeed, to be involved in the aetiology of breathlessness on exercise.

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