Annals of the Rheumatic Diseases 1989; **48:** 632–635

Pulmonary function and maximal transrespiratory pressures in ankylosing spondylitis

D VANDERSCHUEREN,[1] M DECRAMER,[2] P VAN DEN DAELE,[1] AND J DEQUEKER[1]

From the [1]Division of Rheumatology and the [2]Respiratory Division, Department of Internal Medicine, K U Leuven, Belgium

SUMMARY Clinical measurements and pulmonary functions, including maximal transrespiratory pressures, were studied in 30 patients (age 43 (SD 10) years) with ankylosing spondylitis. Vital capacity (VC) was slightly reduced to 79 (16)% and forced expiratory volume in one second (FEV_1) was similarly reduced to 82 (20)% such that the average FEV_1/VC ratio was 77·8 (6·65). Total lung capacity was slightly reduced to 85 (13)%. Transfer factor of the lung for carbon monoxide (TLCO) averaged 88 (17)% and TLCO per unit lung volume was 114 (26)%. Reductions in lung volumes correlated well with clinical measurements. Both maximal expiratory pressures (PE_{max}) and inspiratory pressures (PI_{max}) were clearly reduced to 56 (17)% and 76 (28)% respectively. This suggests that spirometrically determined volumes were better preserved than respiratory muscle strength in ankylosing spondylitis. It is speculated that the reduction in respiratory muscle strength may be due to intercostal muscle atrophy.

Ankylosing spondylitis is a systemic rheumatic disease. Involvement of the lungs with a reduction in lung volumes is a well known manifestation of this disease, presumably resulting from a reduction in thoracic mobility.[1] No data are available, however, on respiratory muscle strength in patients with ankylosing spondylitis.

This study was designed to evaluate respiratory muscle function and strength by measurement of the maximal transrespiratory pressures as well as lung volumes in ankylosing spondylitis.

Patients and methods

We examined 30 patients meeting the criteria of definite ankylosing spondylitis according to the American Rheumatism Association (New York). Table 1 shows their clinical features. The following clinical measurements were obtained by one observer: expansion in centimetres at the level of axilla, nipple, diaphragm, and umbilicus; Schöber index; and fingertop to floor distance (Table 2).

All patients underwent routine spirometry and determination of residual volume using a helium dilution technique. Transfer factor of the lung for carbon monoxide was measured with a single breath test and related to the normal values of Billiet *et al.*[2] Maximal airway pressures were measured as described previously.[3] Briefly, we used a mercury manometer connected to a stiff rubber mouth piece (external diameter 3 cm) without leaks. Pressures were sustained for two seconds. Subjects wore a noseclip while the measurements were made.

Maximal expiratory pressures (PE_{max}) were measured at total lung capacity (TLC) and functional residual capacity (FRC), inspiratory pressure (PI_{max}) at residual volume (RV) and FRC. In addition, for inspiratory pressures the subject was instructed to make an inspiratory effort with the

Table 1 *Clinical data of the patients**

	Mean (SD)
Age (years)	43 (9·8)
Weight (kg)	74·6 (14·3)
Height (cm)	1·69 (7·3)
Age at onset of symptoms (years)	28·2 (9)
Age at onset of treatment (years)	34·3 (9·8)
Difference between age at onset and age at start of treatment (years)	6 (6·6)

*The patients comprised 25 men, four women; M:F=6·25:1.

Accepted for publication 16 November 1988.
Correspondence to Dr D Vanderschueren, Division of Rheumatology, Department of Internal Medicine, Catholic University of Leuven, UZ Pellenberg, B-3041 Pellenberg, Belgium.

Table 2 *Expansions, fingertop to floor distance, and Schöber index*

	Mean (SD) (cm)	*Minimum mean value (cm)*	*Maximum mean value (cm)*
Expansion at the level of axilla	3·24 (2·10)	−0·2	7
Expansion at the level of nipple	3·75 (2·40)	−1·5	9
Expansion at the level of diaphragm	3·79 (2·08)	0	7·5
Expansion at the level of umbilicus	4·12 (2·2)	1·5	9
Fingertop to floor distance	19·50 (14·4)	0	50·5
Schöber index	3·44 (1·66)	0	6·5

thorax and not to use the cheeks, thus avoiding glottis closure and pressure generation by the cheeks alone. Maximal pressures were expressed as a percentage of the predicted values determined by a similar technique as described previously.[4]

Results

SPIROMETRY

A slight reduction in spirometrically determined lung volumes was present. Vital capacity (VC): 79 (16)% (3·87 (1·08 litres), total lung capacity (TLC): 85 (13)% (5·62) (1·15) litres, and forced expiratory volume in one second (FEV_1): 82 (20)% (3·04 (0·98) litres) were similarly reduced. Residual volume (RV) (97·7 (27·4)% of predicted or 1·52 (0·46) litres) was normal and as a consequence the RV/TLC ratio (0·28) was slightly increased. The transfer factor of the lung for carbon monoxide (TLco) (88 (17)% of predicted) slightly decreased but the TLco per unit lung volume slightly increased (114 (26)%). Table 3 summarises these results.

Table 3 *Pulmonary function results indicated in litres and as a percentage of the predicted values*

	Mean (SD)	*Mean (SD) (%)*
VC (litres)	3·87 (1·08)	79·4 (16·0)
FEV_1 (litres)	3·04 (0·98)	82·2 (20·0)
FEV_1/VC	0·78 (0·07)	
RV (litres)	1·52 (0·46)	97·7 (27·4)
TLC (litres)	5·62 (1·15)	85·0 (12·7)
RV/TLC	0·28 (0·08)	
TLco (ml/mm Hg.min)	28·54 (6·68)	87·8 (17)
TLco per unit lung volume (ml/mm Hg.min)	6·97 (1·18)	114 (25·6)

VC=vital capacity; FEV_1=forced expiratory volume in one second; RV=residual volume; TLC=total lung capacity; TLco=transfer factor of the lung for carbon monoxide.

RELATION BETWEEN CLINICAL MEASUREMENTS AND SPIROMETRY

Clinical measurements (Table 2) correlated with pulmonary function tests (Table 3). The best correlations were observed between vital capacity and chest expansion ($p<0·001$), followed by the correlation between vital capacity and Schöber index ($p<0·01$). A good inverse relation between vital capacity and fingertip to floor distance was present ($p<0·01$). We noticed, however, that patients with a markedly reduced thorax expansion (14 mm or less) still had a vital capacity that was 62% of predicted values. Table 4 summarises these correlations. Table 5 summarises the relation between thorax expansion and lung volumes.

MAXIMAL TRANSRESPIRATORY PRESSURES

Maximal transrespiratory pressures were significantly

Table 4 *Summary of regression coefficients*

	VC	*FEV_1*	*TLC*	*RV*	*TLco/LV*	*Fingertip to floor distance*	*Schöber index*
Expansion at the level of axilla	0·62	0·60	0·65	0·22	−0·38	−0·35	0·60
Expansion at the level of nipple	0·65	0·64	0·66	0·17	−0·47	−0·34	0·56
Expansion at the level of diaphragm	0·53	0·53	0·61	0·32	−0·42	−0·13	0·50
Expansion at the level of umbilicus	−0·38	−0·33	−0·33	0·02	0·31	0·42	−0·23
Fingertop to floor distance	−0·43	−0·435	−0·33	0·08	0·20	—	−0·42
Schöber index	0·61	0·58	0·44	−0·21	−0·37	−0·42	—

VC=vital capacity; FEV_1=forced expiratory volume in one second; RV=residual volume; TLC=total lung capacity; TLco=transfer factor of the lung for carbon monoxide; LV=lung volume.

Table 5 *Mean (SD) pulmonary function values expressed as percentage of predicted value for different excursions at the nipple level. Values are mean (SD)*

Thorax expansion (mm)	*VC**	*FEV_1*	*TLC*	*T_{LCO}/LV*
Correlation	0·65	0·64	0·66	−0·47
<14	62·33 (8·62)	62 (8·8)	74 (9·17)	137 (23·26)
14–62	77·45 (15·53)	79·65 (19·76)	80·65 (11·38)	115·2 (26·89)
>62	94·67 (5·16)	100·83 (7·52)	99·83 (6·18)	99·67 (11·78)

*For abbreviations see Table 4.

Table 6 *Maximal transrespiratory pressures. Values are mean (SD)*

	Mean (SD) (cmH_2O)
PE at TLC	110·5 (36·8) (or 56 (17)% of predicted values)
PE at FRC	92·0 (30·8)
PI at FRC	−75·1 (29·4)
PI at RV	−88·6 (32·5) (or 76 (28)% of predicted values)

PE=expiratory pressure; TLC=total lung capacity; FRC=functional residual capacity; PI=inspiratory pressure; RV=residual volume.

reduced: mean (SD) PE at TLC averaged 110·5 (36·8) cm H_2O and PE at FRC averaged 92·0 (30·8) cm H_2O; PI at FRC averaged −75·1 (29·4) cm H_2O and PI at RV −88·6 (32·5) cm H_2O. If these results are related to normal values according to sex and age this means that PI_{max} (PI at RV) was reduced to 76 (28)% and PE_{max} (PE at TLC) was reduced to 56 (17)%. Twenty seven of 30 patients had a PE_{max} value of two standard deviations or more below the mean; 16 of 30 patients had a PI_{max} value of two standard deviations or more below the mean according to the normal values of Rochester and Arora.[4] Table 6 summarises these results.

Discussion

In agreement with previous studies,[5–10] the present study shows that patients with ankylosing spondylitis have minimal restrictive pulmonary defects, the most pronounced abnormality being a reduction in vital capacity. Moreover, there was no evidence of air flow obstruction. In previous studies no important changes in pulmonary compliance could be shown.[5 7] Furthermore, we found good correlations between pulmonary function tests and clinical measurements, especially the correlation between vital capacity and thorax expansion together with the correlation between vital capacity and Schöber index. These correlations are similar to those found in other studies.[6 7 9]

These correlations between vital capacity and thorax expansion of Schöber index may be affected by variability in spinal mobility and chest expansion as these vary with age and sex, and, moreover, may be influenced by artefacts related to measurement techniques.[11 12] These artefacts were minimised by measuring chest wall expansion while the patient stood with hands on head, and arms flexed in a frontal plane.

Unexpectedly, however, patients with ankylosing spondylitis with a markedly reduced thorax expansion (less than 14 mm) still had an average vital capacity of 62% of the predicted value. This could either mean that the diaphragm contributes the greatest part to vital capacity or that it partly compensates for the loss of thoracic expansion. The latter phenomenon is certainly present during tidal breathing as it is a common clinical observation that patients with ankylosing spondylitis have enhanced inspiratory abdominal outward motion. Previous studies have suggested that the diaphragmatic contribution to inhaled volume increases as a direct consequence of ankylosis of the thoracic cage.[5 6 8 9 13–15] The compensatory effort of the diaphragm is a probable explanation of the fact that patients with ankylosing spondylitis have only mild respiratory discomfort or none at all. We have occasionally noticed that patients with ankylosing spondylitis with unilateral diaphragmatic paralysis have pronounced dyspnoea together with a marked reduction of the vital capacity.

It is known that other deformities, such as kyphoscoliosis, are usually accompanied by more severe abnormalities of lung function.[4] This is probably partly due to the presence of thoracic deformity and may be partly due to other factors, such as fixation changes of the thorax in ankylosing spondylitis at a lung volume higher than the relaxation volume of the thorax.[6 8] Necropsy studies of patients with ankylosing spondylitis would be most interesting to determine whether or not these patients show hypertrophy of the diaphragm.

Thus the observed reduction of vital capacity is probably the result of two opposing influences: the

rigidity and reduced expansion of the thoracic cage and the compensatory effort of the diaphragm. The reduction of vital capacity appears to be only slightly affected or not influenced at all by the treatment or duration of the disease.[6 9]

Finally, this study shows that patients with ankylosing spondylitis have clearly reduced maximal transrespiratory pressures,[3 4] indicating decreased respiratory muscle strength. If indeed the diaphragmatic strength were to be unchanged or even increased, as follows from the reasoning developed above, the decreased respiratory muscle strength would be due to reduced strength or atrophy of intercostal or accessory muscles, or both. Although the present data do not provide direct evidence of intercostal muscle atrophy and do not allow us to conclude why such atrophy may be present, it is tempting to speculate that immobilisation of these muscles due to thoracic rigidity and decreased inspiratory intercostal and accessory activation leading to disuse may be an important factor contributing to it.

References

1 Rosenow E, Strimlan C V, Muhm J R, Ferguson R H. Pleuropulmonary manifestations of ankylosing spondylitis. *Mayo Clin Proc* 1977; **52:** 641–9.

2 Billiet L, Barsien W, Naedts J. Effet de taille, du sex et de l'âge sur la capacité de diffusion pulmonaire de l'adulte normal. *Physiologie Paris* 1963; **55:** 194–200.

3 Decramer M, Demedts M, Rochette F, Billiet L. Maximal transrespiratory pressures in obstructive lung disease. *Bull Eur Physiopathol Respir* 1980; **16:** 479–80.

4 Rochester D F, Arora N S. Respiratory muscle failure. *Med Clin North Am* 1983; **67:** 573–99.

5 Stewart R M, Ridyard J B, Pearson J D. Regional lung function in ankylosing spondylitis. *Thorax* 1976; **31:** 433–6.

6 Josenhans W T, Wang C S, Josenhans G, *et al.* Diaphragmatic contributions to ventilation in patients with ankylosing spondylitis. *Respiration* 1971; **28:** 331–46.

7 Feltelius N, Hedenström H, Hillerdal G, Hällgren R. Pulmonary involvement in ankylosing spondylitis. *Ann Rheum Dis* 1986; **45:** 736–40.

8 Gacad G, Hamosh P. The lung in ankylosing spondylitis. *Am Rev Respir Dis* 1973; **107:** 286–9.

9 Franssen M J A M, Van Herwaarden C L A, van de Putte L B A, *et al.*, Lung function in patients with ankylosing spondylitis. A study of the influence of disease activity and treatment with non steroidal anti-inflammatory drugs. Disease activity and response to therapy in ankylosing spondylitis. Nijmegen: Stichting studentenpers, 1985. (Thesis.)

10 Hart F D, Emerson P A, Gregg I. Thorax in ankylosing spondylitis. *Ann Rheum Dis* 1963; **22:** 11–18.

11 Moll J M H, Wright V. Normal range of spinal mobility. *Ann Rheum Dis* 1971; **30:** 381–6.

12 Moll J M H, Wright V. An objective clinical study of chest expansion. *Ann Rheum Dis* 1972; **31:** 1–8.

13 Grimby G, Fighl Meyer A R, Blomstrand A. Portioning of the contributions of ribcage and abdomen to ventilation in ankylosing spondylitis. *Thorax* 1974; **29:** 179–84.

14 Davis D. Ankylosing spondylitis and lung fibrosis. *Q J Med* 1972; **41:** 407.

15 Zorab PA. The lungs in AS. *Q J Med* 1962; **31:** 272–3.