Relation between chest expansion, pulmonary function, and exercise tolerance in patients with ankylosing spondylitis

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Abstract

Thirty three patients with definite ankylosing spondylitis (AS) were examined to establish the relation between restriction of chest expansion, limitation of lung function, and working capacity or exercise tolerance. As in previous studies there was a significant association between chest expansion and lung vital capacity. There was also a significant association between vital capacity and exercise tolerance as measured by a subject's maximum oxygen capacity (Vo_2max). Both vital capacity and Vo_2max were expressed as a percentage of predicted normal values using patients' height before disease. In this study chest expansion did not have a significant effect on exercise tolerance. The results suggested that patients who took a modest amount of exercise regularly could maintain a satisfactory work capacity despite very restricted spinal and chest wall mobility.

It is recommended that greater emphasis should be given to encouraging patients with AS to maintain cardiorespiratory fitness as well as spinal mobility.

Ankylosing spondylitis (AS) is a condition characterised by inflammation of ligamentous insertions or entheses and joints, especially of the axial skeleton. Bony ankylosis may occur in the numerous joints around the thorax, resulting in limited movement of the chest wall. A chest expansion of less than 2·5 cm is one of the clinical factors in the New York criteria for AS,[1] though it has been suggested that such a rigid borderline between normal and abnormal is not supported by clinical studies.[2 3] The typical respiratory function abnormalities in AS are those of a restrictive defect. Studies by Feltelius have shown an association between the limitation of chest expansion and restriction of vital capacity.[4] Reports have been made of abnormalities in skeletal muscle and cardiac function in patients with AS (P A Revell *et al*, British Society for Rheumatology meeting, 1987). Apical pulmonary fibrosis is also associated with AS, though the prevalence is low.[6 7]

Patients with AS rarely complain of respiratory symptoms or functional impairment unless there is coexistent cardiovascular or respiratory disease. For most patients the pain, stiffness, and fatigue associated with the disease is the most limiting factor.

This study undertook to evaluate whether restriction of chest expansion affected vital capacity and exercise tolerance of patients with AS.

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Patients and methods

Patients with definite AS according to New York criteria were identified and invited to participate in the study. Exclusion criteria included coexistent cardiac or respiratory disease, treatment with β blocking agents, or the presence of such severe arthritis in the legs as to make it impossible to exercise on a bicycle. Those patients fulfilling the criteria and giving informed consent were entered into the study. Patients were all examined between 9 and 11 am.

CLINICAL ASSESSMENT

A full history was taken and a full examination performed. Particular note was taken of the patients' smoking history, occupation, and sporting activities. Previous treatment with radiotherapy or exposure to occupational lung disease was also noted.

SPINAL MOBILITY

A warm up period was not used before the examination. Chest expansion was measured with a tape-measure placed circumferentially around the chest wall at the fourth intercostal space.[3] Flexion and extension of the lumbar spine was measured with a tape-measure by a modified Shober method.[8] A spondylometer was used to assess lumbar-thoracic flexion and extension.[9 10] No attempts were made to measure lateral flexion of the spine.

PULMONARY FUNCTION TESTS

The following pulmonary function tests were performed: peak expiratory flow rate; dynamic spirometry (forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), FEV_1/FVC (%)); transfer factor of the lung for carbon monoxide; static spirometry (vital capacity, residual volume, total lung capacity, and functional residual capacity) performed using the helium rebreathing method.

Predicted normal values were obtained for all the above tests using the patient's height before disease where this was reliably known. Arm span measurement was not found to be a useful predictor of height before disease.[11]

EXERCISE TESTS

The measurement of a subject's maximum oxygen consumption during a minute of work or exercise sustained at the maximum tolerated level is recognised as the value of the maximum

exercise tolerance (Vo_2max). In this study exercise testing was performed on an electronically braked bicycle ergometer.[12] The bicycle saddle was adjusted for the patient's height. A nose clip was applied and the patient allowed to breath through a mouth piece. The mouth piece has a valve system to allow inspired air to enter and expired air to be expelled through separate channels. The expired air passes to a mixing chamber where a small pump extracts gas, dries it, and feeds it to oxygen and carbon dioxide analysers. Mean concentrations of oxygen and carbon dioxide at minute intervals are available for inspired and expired air, thus providing a value for oxygen consumption at each minute of exercise. The system also allows the measurement of ventilatory rate and tidal volume.

The advantages of the exercise bicycle are firstly, that work rate remains constant over a wide range of pedalling frequencies from 45 to 75 rpm. Secondly, the supported torso reduces the differences that weight may introduce and enables measurement of blood pressure, electrocardiogram (ECG), and oxygen tension to be performed more easily. An incremental exercise test was used.[13] The programme consisted of two minutes at rest to establish steady state values for oxygen consumption. This was followed by one minute pedalling with no resistance and then a progressive increase in work load by 15 watt increments every minute. The test was stopped when the patient could no longer continue exercising. The maximum working capacity was the highest work load sustained for one minute and was expressed as the maximum oxygen consumption (Vo_2max).

A 12 lead ECG was performed at rest and an ECG was displayed throughout the exercise programme. The exercise was stopped if the patient developed chest pain or any cardiac symptoms, or in the event of ECG changes, such as runs of three or more successive ventricular extrasystoles or more than six ventricular extrasystoles in one minute. Capillary oxygen tension was measured from an oxygen photometer attached to the ear lobe. At the end of the exercise patients completed a visual analogue scale representing the degree of breathlessness (perceived exertional rating) and were asked for their reason for stopping exercise.

INVESTIGATIONS

Radiographs of chest and lateral thoracic spine were obtained. Thoracic kyphosis was measured as the angle subtended by lines drawn from the anterior bodies of the fourth and 12th thoracic vertebrae. All patients entering the study had had recent radiographs of the sacroiliac joints, lumbar and thoracic spine, and these were not therefore repeated.

A full blood count and erythrocyte sedimentation rate (ESR) was obtained.

FORMULAE AND STATISTICAL METHODS

Maximum predicted heart rate (HRmax) was calculated using the formula[14]:

$$HRmax = 202 - (age \times 0{\cdot}72) \text{ beats/minute}$$

Maximum predicted exercise tolerance was calculated from the formula described by Jones *et al*,[14] taking into account the patient's age, sex, and height:

$$Vo_2max = height \times 0{\cdot}046 - age \times 0{\cdot}021 - 0(M)/0{\cdot}62(F) - 4{\cdot}314$$

where height is in cm and age in years.

Correlation coefficients for parametric data were obtained in comparisons of the results from clinical, lung function, and exercise tests.

Student's t test was used to determine the significance level between the group with AS and normal reference values for pulmonary function tests. This test was also used to compare the mean values obtained for chest expansion, vital capacity, and exercise tolerance in smokers and non-smokers and in patients classed as sedentary or exercisers.

Results

Forty one patients were seen and examined. Eight had to be excluded from the study: three did not fulfil New York criteria for AS, two had severe coexistent respiratory disease, one was receiving β blockers for hypertension, and two patients were unable to exercise owing to severe hip and knee disease.

Thirty three patients (28 men, five women) entered the study. Ages ranged from 26 to 74 years (mean (SD) 46·8 (10·9)). Disease duration ranged from 8 to 42 years (mean (SD) 20·1 (8·6)). Two patients had juvenile onset of the disease and seven had received radiotherapy treatment. All patients were HLA-B27 positive, 12 (36%) had had iritis, and 20 (61%) had some disease of peripheral joints.

Most patients 22 (67%) smoked, four (12%) were ex-smokers, and seven (21%) were non-smokers. Sixteen patients were judged to take a modest amount of exercise—for example, walking or cycling over three miles each day or playing golf, squash, tennis, or swimming three or more times each week. Seventeen patients were assessed to lead sedentary occupations and to take no regular exercise. None of the group complained of any respiratory features during exercise.

Table 1 shows the intraobserver error for clinical measurements. It is of interest to note that in this study intraobserver error was similar for measurements of spinal flexion whether a tape-measure or spondylometer was used. Previous reports have suggested that error is less with the spondylometer[8] than with the tape-measure.

CLINICAL AND LABORATORY MEASUREMENTS

Table 2 shows the ranges and means for all

Table 1 Intraobserver error for clinical measurements

		Coefficient of variation (%)
Chest expansion	Tape-measure	14·0
Lumbar flexion	Tape-measure	6·0
Lumbar extension	Tape-measure	7·3
Lumbar-thoracic flexion	Spondylometer	6·0
Lumbar-thoracic extension	Spondylometer	12·5

Table 2 Results of clinical and laboratory measurements

	Mean	SD	Range	Number of patients
Chest expansion (cm)	3·05	(1·63)	0·83–6·5	32†
Chest expansion of 2·5 cm or less (cm)	1·53	(0·47)	0·83–2·5	13
Lumbar flexion (cm)	4·03	(2·15)	0·5–8·0	33
Lumbar extension (cm)	0·91	(0·64)	0·0–2·3	33
L–T* flexion (deg)	22·71	(14·1)	3·0–47	33
L–T extension (deg)	6·48	(5·0)	0·0–24	33
Thoracic kyphosis (deg)	41·70	(12·4)	20·0–65	24
ESR* (mm/h)	24·20	(15·6)	3·0–62	22

*L–T=lumbar thoracic; ESR=erythrocyte sedimentation rate.
†One patient unable to take a deep breath despite normal chest movements.

Table 3 Results of pulmonary function tests

Spirometry	Mean	SD	Percentage of normal	Significance (p value)
Peak flow (l/min)	495	80	98	NS
FEV_1* (litres)	2·79	0·65	80·2	<0·001
FVC* (litres)	3·7	0·8	88·6	<0·001
FEV_1/FVC (%)	74·6	8·9	90·7	<0·001
Vital capacity (litres)	3·64	0·79	82·3	<0·001
Residual volume (litres)	1·86	0·46	94	<0·05
Total lung capacity (litres)	5·48	1·0	84·4	<0·001
Diffusion capacity (TLCO)* (ml/mmHg.min)	8·25	1·5	82·7	<0·001

*FEV_1=forced expiratory volume in one second; FEV=forced vital capacity; TLCO=transfer factor of the lung for carbon monoxide.

Table 4 Correlation between chest expansion and spinal mobility

	Number of patients	Correlation coefficient	Significance (p value)
Chest expansion *v* lumbar flexion	32	0·6	<0·001
Lumbar flexion TM* *v* SPOND*	33	0·95	<0·001
Lumbar extension TM *v* SPOND	33	0·87	<0·001
Lumbar flexion *v* extension	33	0·78	<0·001
Chest expansion *v* thoracic kyphosis	24	0·16	NS
Chest expansion *v* disease duration	31	0·14	NS
Chest expansion *v* ESR*	22	0·13	NS

*TM=tape-measure; SPOND=spondylometer; ESR=erythrocyte sedimentation rate.

Table 5 Correlation between chest expansion, vital cap .y, and exercise tolerance

	Number of patients	Correlation coefficient	Significance (p value)
VC*† *v* Vo_2max*†	33	0·53	<0·01
VC (%) *v* Vo_2max (%)	33	0·68	<0·001
Chest expansion *v* VC*	32	0·71	<0·001
Chest expansion *v* VC (%)	33	0·79	<0·001
Chest expansion *v* Vo_2max*	32	0·29	NS
Chest expansion *v* Vo_2max (%)	32	0·38	NS

*Expressed as absolute values
†VC=vital capacity; Vo_2max=maximum oxygen capacity.

Table 6 Comparison of mean values for chest expansion, VC (%), and Vo_2max* (%) between sedentary and active patients with ankylosing spondylitis*

	Exercisers (n=16) Mean (Range)	Non-exercisers (n=17) Mean (Range)	Significance (p value)
Age (years)	46·9 (29–70)	47·8 (26–74)	NS
Chest expansion (cm)	3·4 (0·83–6·5)	2·83 (0·87–6·3)	<0·5
VC (%)	89·2 (52–113)	77 (56–114)	<0·02
Vo_2max (%)	83·3 (54–115)	58·6 (30–109)	<0·001

*VC=vital capacity; Vo_2max=maximum oxygen capacity.

clinical and laboratory measurements. Only 13 of 32 patients had a chest expansion of 2·5 cm or less. One patient had paradoxical chest movements despite no severe restriction of chest or spinal mobility. Radiographs of the chest and resting electrocardiograms were normal in all patients.

RESULTS OF PULMONARY FUNCTION TESTS

Table 3 shows the ranges and means of pulmonary function tests. Eleven patients (10 men, one woman) had a vital capacity more than two standard deviations outside the normal range.[13]

RESULTS OF EXERCISE TESTING

Twenty of the 32 (63%) patients who completed a visual analogue scale scored more than 75% and reported that they had stopped exercise owing to breathlessness. The remaining 12 patients stopped exercise for other reasons, such as painful muscles or joints. Only 24 of 31 (77%) patients reached 75% or more of their predicted maximum heart rate. Maximum heart rate was not calculated in two patients owing to technical problems with the monitor. None of the group developed any clinical cardiac problems or ECG abnormalities during exercise testing. Similarly, oxygen tension as measured by the photometer remained normal throughout the test.

The maximum work load achieved ranged from 60 to 225 watts with a mean value of 128 watts. The maximum oxygen capacity (Vo_2max) ranged from 10·3 to 37·6 with a mean (SD) of 23·4 (6·8) ml/min/kg.

CORRELATION COEFFICIENTS BETWEEN MEASURED VARIABLES

As expected there was a close correlation between physical variables such as chest expansion, lumbar flexion and extension. There was no significant correlation between chest expansion and any of the following variables: disease duration, thoracic kyphosis, or ESR (table 4).

This study confirmed previous reports of a significant association between chest expansion and vital capacity. This result was not altered whether the vital capacity was expressed in absolute values or as a percentage of predicted normal values. Vital capacity showed a significant association with exercise tolerance, whether expressed as an absolute value or as a percentage of predicted normal values. This result was not significantly altered when the following subgroups were assessed separately: smokers and non-smokers, exercisers and sedentary patients, and patients who achieved more or less than 75% of their predicted maximal heart rate on exercise.

Chest expansion showed no significant association with exercise tolerance either when measured in absolute values or as a percentage of predicted normal values (table 5). This result did not alter on separate evaluation of the various subgroups.

Neither vital capacity nor exercise tolerance showed any correlation with disease duration, thoracic kyphosis, or ESR.

Patients who took regular exercise were compared with the group who were judged to take minimal exercise. Table 6 shows the results from each group. No significant difference was found between the two groups for age or chest expansion ($0·1<p<0·5$). There were equal numbers of smokers in the two subgroups. A significant difference between the groups was

Table 7 Comparison of mean values for chest expansion, VC (%), and Vo_2max* (%) between smokers and non-smokers with ankylosing spondylitis*

	Smokers (n=22) Mean (Range)	*Non-smokers (n=11) Mean (Range)*	*Significance (p value)*
Age (years)	45·6 (29–70)	49 (26–74)	NS
Chest expansion (cm)	3·0 (0·83–6·5)	3·23 (1·3–5·75)	NS
VC (%)	80·9 (52–113)	85·5 (58–114)	<0·5
Vo_2max (%)	68·3 (40·3–115)	68·4 (30·3–109)	NS

*VC=vital capacity; Vo_2max=maximum oxygen capacity.

found for both vital capacity ($p<0·02$) and exercise tolerance ($p<0·001$). Both measurements are expressed as a percentage of predicted normal values. Active patients had a higher mean Vo_2max (83·3%) than the sedentary group (58·6%). In comparison, there was no significant difference in chest expansion or exercise tolerance between the groups of smokers and non-smokers. Ex-smokers were evaluated with the non-smokers, but this did not alter the results. Rather surprisingly, pulmonary function tests such as vital capacity (%), FEV_1 (%), and peak flow rates showed only weakly significant differences between smokers and non-smokers ($0·1<p<0·5$) (table 7). Pulmonary function tests are represented by vital capacity only.

Discussion

Rigidity of the thorax occurs in ankylosing spondylitis with bony ankylosis of the thoracic vertebrae, costovertebral, costotransverse, sternoclavicular, and sternomanubrial joints. Progressive kyphosis adds deformity to the rigidity of the thorax. As the disease progresses movements of the thoracic cage diminish until, in severe cases, respiration becomes entirely diaphragmatic. Previous studies on large numbers (>2000) of patients with AS showed no predisposition to pulmonary disease apart from apical fibrosis, which occurs rarely with a prevalence of about 1%.[6 7]

Regional lung ventilation in patients with AS is normal unless there is pre-existing apical fibrosis.[15] Respiratory function in AS shows typical restrictive changes, but pulmonary compliance, diffusion capacity, and arterial blood gases are normal.[16] Recent work has suggested mild disease of the lung parenchyma and small airways in AS.[4]

Patients with AS rarely develop respiratory failure or complain of dyspnoea, but the effect of the restrictive lung disease has received little attention. Hart *et al* measured pulmonary function before and after exercise and reported improvements in many of their patients.[17 18] Jossenhans studied 222 patients with AS before and after physiotherapy and found that spirometry remained unchanged despite improvements in chest wall and spinal mobility.[19]

Electromyography recordings from the diaphragmatic and intercostal muscles of patients with AS and chronic obstructive airways disease are reported to show inspiratory muscle fatigue during exercise.[20] Grassino *et al* proposed that fatigue of inspiratory muscles limited exercise tolerance.

Studies on patients with rheumatoid arthritis and osteoarthritis found a reduced exercise tolerance, especially in the group with rheumatoid arthritis compared with controls.[21] Exercise tests have also been performed in patients with diffuse interstitial lung disease,[22] but we have been unable to trace previous studies on exercise testing in patients with AS.

The study reported here on a group of patients with AS confirms previous reports of an association between the restriction of chest expansion and vital capacity. There was also a significant correlation between vital capacity and exercise tolerance (Vo_2max), but not between chest expansion and exercise tolerance. These results did not change when various subgroups were assessed separately—for example, smokers and non-smokers, those with chest expansion greater or less than 2·5 cm, active and sedentary patients, or patients who achieved more or less than 75% of their predicted maximum heart rate during exercise. When the patients with AS were subdivided into active and sedentary groups, however, there were significant differences in the group means for exercise tolerance, with the active group achieving a higher Vo_2max than the sedentary group ($p<0·001$). There was a less marked but significant difference in mean values for vital capacity ($p<0·02$), but chest expansion was similar in the two groups ($p<0·5$).

A comparison of smokers and non-smokers (including ex-smokers) showed no significant difference between them for mean values of chest expansion, vital capacity, or exercise tolerance. Similarly, other pulmonary function tests, including peak flow rates, FEV_1/FVC ratio, and transfer factor, showed no significant difference between smokers and non-smokers.

There are many problems in trying to assess a person's exercise tolerance. Firstly, there is the unfamiliarity with the form of exercise using a bicycle ergometer. It may be expected that people cycling regularly would perform better. In this study only three of the 33 patients cycled regularly.

Secondly, there is the problem of patient 'motivation' as some patients will push themselves closer to their maximum working capacity than others. More practice on the ergometer and testing on more than one occasion might have helped some patients to improve their recorded Vo_2max but would have introduced a training effect. Some of these problems might have been avoided with the use of controls but it would have been essential to control the sex, height, weight, smoking history, and amount of sport or exercise taken.

In view of these difficulties we felt justified in comparing a patient's results for pulmonary function tests and exercise tolerance with predicted normal values.

The third major problem was the high proportion (67%) of cigarette smokers in the group. A survey of 3333 people aged 16 to 65, randomly selected from the Southampton district, found that 28% of the group smoked (J Howell, unpublished data). The proportion of

men in the survey population was 52·4% and in the group of smokers 54·5%. An earlier survey (1984) found that 38% of 2000 adults in the same district smoked.[23] In a large study of all patients with definite AS (n=150) in the Southampton district 52% are smokers (B L Kidd, unpublished data). It is interesting to note the higher number of smokers (67%) among our group of patients with AS. Why such a disproportionately large number of smokers entered this study and whether this seriously affected the results is not known. These results suggest that although restriction of chest expansion may result in reduction of vital capacity, it is not a major factor determining exercise tolerance. In this study there were examples of patients taking a moderate amount of daily exercise who were able to achieve a Vo_2max close to their predicted normal despite having very restricted chest expansion.

Conclusion

This study showed that in a group of patients with AS there is an association between chest expansion and vital capacity, and between vital capacity and exercise tolerance. It failed to show an association between chest expansion and exercise tolerance. Many patients with very restricted chest wall movements were able to obtain satisfactory work capacities.

The results suggest that efforts should be directed not only towards improving spinal mobility but also towards increasing cardiorespiratory fitness in patients with AS. Counselling and encouragement for patients with AS to take more regular exercise and to participate in sporting activities should be given greater emphasis.

Although this study failed to show any significant difference in pulmonary function tests between smokers and non-smokers, it would seem prudent to encourage patients with AS to stop smoking in view of the theoretical risk of compounding a restrictive with an obstructive pulmonary defect, as well as the other known health hazards of cigarette smoking.

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