

Regional lung function in ankylosing spondylitis

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Stewart, R. M., Ridyard, J. B., and Pearson, J. D. (1976). *Thorax*, 31, 433–437. Regional lung function in ankylosing spondylitis. Xenon-133 was used to study regional pulmonary function in nine patients with chest cage rigidity due to ankylosing spondylitis. In comparison with normal subjects, the patients showed an overall diminution in lung volume and the proportion of inhaled xenon reaching the lung apices was reduced but the distribution of injected xenon was normal. The possible relationship between these findings and apical lung disease in ankylosing spondylitis is mentioned.

In the present paper and the one which follows (Ridyard and Stewart, 1976), an attempt has been made to determine the relative effects on regional lung function of diaphragmatic and thoracic breathing in naturally occurring clinical situations. The first paper describes patients with a fixed thoracic cage due to ankylosing spondylitis, and therefore predominantly diaphragmatic breathing, and the second paper describes patients with diaphragmatic paralysis and a normal thoracic cage.

Ankylosing spondylitis is a chronic arthritis which may affect the costovertebral joints and cause fixation of the thoracic cage. Fibrosis and cavitation of the lung apices can also complicate the disease (Campbell and MacDonald, 1965), and it has been suggested (Zorab, 1962) that this could be due to disturbed ventilation secondary to the costovertebral ankylosis. In the only previous report of regional lung function in ankylosing spondylitis (Lauritzen *et al.*, 1968), the patients also had apical lung fibrosis.

The present paper describes a study of regional ventilation and perfusion, using xenon-133, in a group of patients with ankylosing spondylitis but apparently healthy lungs.

SUBJECTS

Physicians in the Liverpool area were asked to refer any patient who fulfilled both the Rome and New York criteria for ankylosing spondylitis (Moll

and Wright, 1973) associated with limited chest expansion. Subsequently, 12 such patients agreed to participate. One patient could not be studied because of extreme kyphosis. Two further patients were excluded from analysis because of concomitant disease: one had mitral stenosis and the other severe chronic airways obstruction. The remaining nine patients were all males with a mean age of 44.4 years (range 34–52 years). Five were current cigarette smokers, one was a previous smoker, and three lifetime non-smokers. Five had had radiotherapy to the spine (Table I).

Fourteen normal volunteers with a mean age of 35.7 years (range 23–60 years) were studied as controls. All were lifetime non-smokers with no cardiorespiratory symptoms, normal chest radiographs, and no abnormality of lung function.

TABLE I

Subject	Age (yr)	Maximum Height (inches)	Smoking History (cigarettes per day)	Radiotherapy
DE	45	—	15–20	Nil
RF	48	70½	Nil	Nil
RM	41	68½	Nil for 6 yr	1957, 1962, 1971 to lumbosacral spine
AM	44	66½	25	1971 lumbosacral and thoracic spine
DDE	36	69	10	1965 entire spine
PP	34	66½	Nil	1972 entire spine
JB	47	68½	Nil	1967 entire spine
WM	54	71½	10 (2 yr only)	Nil
DK	54	70	10	Nil

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METHODS

Lung volumes were obtained by standard techniques of spirometry and helium dilution (Bates and Christie, 1950) and carbon monoxide transfer factor by the single-breath method of Ogilvie *et al.* (1957). Normal values were predicted from nomograms relating lung function to height and age for adult males of Western European descent (Cotes, 1975). Because ankylosing spondylitis may cause loss of height, previous maximum height was used.

Diaphragmatic excursion was evaluated by standard postero-anterior inspiratory and expiratory chest radiographs. Apical lesions were excluded by apical lordotic views. Chest expansion was measured in the fourth intercostal space as described by Moll and Wright (1972).

An electrocardiogram, latex agglutination test, erythrocyte sedimentation rate, haemoglobin, white blood cell count, and differential were obtained in most patients.

Regional ventilation and perfusion studies were carried out in the supine posture, scanning 28 cm of chest from base to apex by the method of Dollery and Gillam (1963). Scans were first obtained following a single breath inhalation of xenon-133 at a concentration of 0.5 to 1.0 millicurie per litre. Regional alveolar volume was then determined by scanning after rebreathing this same concentration of xenon-133 for at least seven minutes. Perfusion scans were obtained immediately after the injection of 2 mCi of xenon-133 in solution in 1 ml of normal saline.

The patients were instructed to breathe naturally after the injection scan. After 30 seconds of relaxed breathing a final 'washout' scan was recorded to provide a 'dynamic' index of regional ventilation (Evans, 1973); this was expressed as the percentage of injected xenon-133 retained after 30 seconds. In other words, high scores reflect less efficient ventilation in contrast to the

inhalation and perfusion scans where high scores indicate relatively better function. Detailed accounts of the technique are available in previous publications from this laboratory (Brown, Kirk, and Seaton, 1969; Gaziano, Seaton, and Ogilvie, 1970; Hall, 1974).

For regional analysis the lung was divided into three zones of equal length—base, midzone, and apex. The measurements were established from the equilibration scan. Regional ventilation or perfusion indices were expressed per unit of regional alveolar volume as described by West (1966). The distribution indices for ventilation and perfusion were averaged for each of the three zones and expressed as a percentage of the mean distribution index for the whole lung.

RESULTS

Blood counts and electrocardiograms were normal and the latex agglutination tests negative. Erythrocyte sedimentation rates ranged from 1–30 mm per hour with a mean of 12.9 mm (Westergren).

Diaphragmatic excursion, as determined by inspiratory and expiratory radiographs, ranged from 6.2 to 1.2 cm (mean 3.85 cm). Mean chest expansion was 1.64 cm with a range of 0.9 to 3.5 cm.

Lung volumes and transfer factors are shown in Table II. In every case vital capacity was decreased (mean 65.6% predicted). Similar results were found for both total lung capacity (mean 70% predicted) and functional residual capacity (mean 69.4% predicted). Mean residual volume was diminished to 77.4% predicted but, in two patients, residual volume was increased to 114 and 149% predicted. The forced expired volume in one second (FEV₁) as percentage of vital capacity was normal or high in all but one patient. Transfer factor (carbon monoxide diffusing capacity) expressed as percent predicted ranged from 63% to 92% (mean 80%).

TABLE II

Subject	VC		FEV ₁		RV		FRC		TLC		TF % Pred.
	Litres	% Pred.	Litres	VC	Litres	% Pred.	Litres	% Pred.	Litres	% Pred.	
DE	2.70	64	2.45	91	0.97	33	1.87	54	4.27	70	69
RF	3.15	67	2.75	87	0.90	39	2.40	60	3.95	56	66
RM	1.70	36	1.70	100	3.00	149	3.50	94	4.65	70	89
AM	3.75	86	2.25	62	2.12	114	3.22	93	5.62	90	89
DDE	2.60	54	2.35	90	1.25	64	2.25	60	3.95	59	81
PP	3.60	80	2.50	69	0.75	42	1.40	40	3.95	63	82
JB	4.15	95	3.40	82	1.93	89	2.93	78	6.13	94	92
WM	2.70	58	1.85	69	2.14	92	2.99	75	4.84	69	90
DK	2.45	52	1.70	70	1.73	75	3.08	77	4.13	59	63
Mean	2.98	65.6	2.33	80.0	1.64	77.4	2.63	69.4	4.61	70.0	80.0

When the means of the ventilation indices for each of the three zones are compared to those of the normal subjects (Fig. 1 and Table III) there is no significant difference at the base. However, the mid zone and upper zone values differ significantly (P less than 0.001), the former being greater and the latter less than normal. There is no significant difference in perfusion in any zone when the mean values are compared to those of the normal subjects (Fig. 2 and Table III).

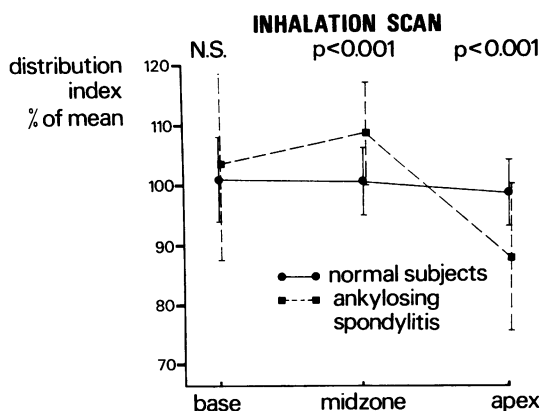


FIG. 1. Mean values for inhalation scan indices. The normal subjects are shown with a continuous line and those with ankylosing spondylitis with a broken line. The vertical lines show the standard deviations.

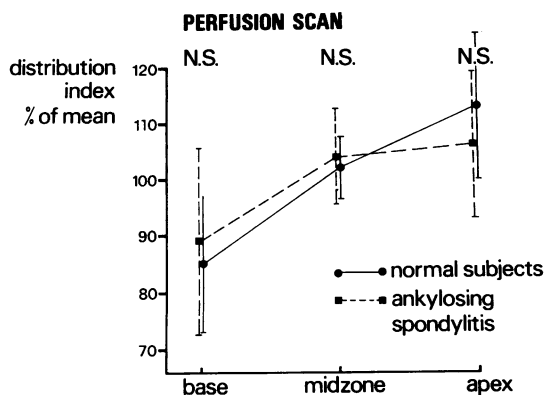


FIG. 2. Mean values for the perfusion scan indices. The normal subjects are shown with a continuous line and those with ankylosing spondylitis with a broken line. The vertical lines show the standard deviations.

TABLE III

	Base	Middle	Apex
Inhalation indices			
Normal subjects	101.1 ± 7.3	100.5 ± 5.4	98.4 ± 5.5
Ankylosing spondylitis	103.5 ± 16.1	108.5 ± 8.6	87.8 ± 12.3
Perfusion indices			
Normal subjects	84.9 ± 12.0	102.0 ± 5.5	113.1 ± 13.1
Ankylosing spondylitis	89.2 ± 16.7	104.2 ± 8.6	106.7 ± 13.4

Tabulated data for regional distribution indices in the individual patients can be obtained on application to Dr. J. B. Ridyard.

There was abnormal retention of xenon at 30 seconds ($>30\%$) at six apical zones, four mid zones, and four bases. The mean values were 28% for the apices, 24% for the mid zones, and 22% for the bases. These differences, although in keeping with the results of the inhalation scan, were not significant.

DISCUSSION

Previous measurements of lung volume in ankylosing spondylitis have given varying results. One difficulty is that some observers determine the predicted volumes on the present height, not taking into account the loss of height due to spinal deformity. Our results, based on previous maximum height, showed a diminution in all lung volumes. A decrease in total lung capacity and vital capacity agrees with most studies (Rogan, Needham, and McDonald, 1955; Renzetti *et al.*, 1960; Hart, Emerson, and Gregg, 1963; Miller and Sproule, 1964; Sharp *et al.*, 1964; Gacad and Hamosh, 1973). Travis *et al.* (1960) and Zorab (1962) found vital capacity decreased although total lung capacity was usually normal.

Our finding of a reduced functional residual capacity and residual volume is unusual as others have found them normal or even increased. However, Sharp *et al.* (1964) found that residual volume and functional residual capacity were only increased when the total lung capacity was not diminished. Among our subjects no direct relationship could be found between residual volume or functional residual capacity, and the FEV₁, chest expansion, diaphragmatic excursion, or smoking history; however, the only subject with a significantly increased residual volume was the heaviest smoker in the group.

Grimby, Fugl-Meyer, and Blomstrand (1974) have shown that the rib cage, which normally contributes 75% of resting tidal volume, contributed only 56% in subjects with ankylosing spondylitis. Sackner *et al.* (1974) found that voluntary dia-

phragmatic breathing in normal subjects shifted the distribution of inhaled xenon-133 from the apex towards the base. We have made a similar observation in the present study of patients with ankylosing spondylitis. Presumably, this is due directly to bony ankylosis of the thoracic cage with a subsequent greater contribution of diaphragmatic motion to inhalation. An attempt was made to show that decreasing chest expansion correlated with decreasing apical ventilation: however, due to the minimal interpatient variation in chest expansion (range less than 2.5 cm) no such correlation was possible.

An alternative explanation for this abnormality is airways obstruction predominantly affecting the apices, as may occur in emphysema (Gaziano *et al.*, 1970). In this case the 'washout' scan, which is a more sensitive indicator of airways obstruction than the inhalation scan (Evans, 1973), should reflect this abnormality. However, there was no significant difference in xenon washout between apex and base in the patients with ankylosing spondylitis. Smoking can also be eliminated as the cause of the redistribution of inhaled xenon; the three life-time non-smokers showed an apical ventilation defect similar to that of the smokers, and a χ^2 test revealed no significant difference between these two groups. Likewise the four patients who had received radiotherapy to the thoracic spine showed no significant difference from the five not given this treatment.

The distribution of injected xenon, unlike the distribution of inhaled gas, conformed to the normal pattern for the supine posture (increasing perfusion from base to apex) with no significant defect of apical perfusion (Fig. 2 and Table III).

It is known that patients with ankylosing spondylitis may develop bilateral apical pulmonary fibrosis with cavitation. Several possible causes for this have been suggested. As noted earlier, Zorab (1962) considered that these changes were either a primary manifestation of the disease or a result of an alteration in pulmonary mechanics. Scobie (1971) showed that a defect in oesophageal peristalsis is common in ankylosing spondylitis and suggested that recurrent aspiration could result in pulmonary fibrosis. More recently, Davies (1972) concluded that the pulmonary lesions were an extra-articular manifestation of the disease process.

In conclusion, it is suggested that impaired thoracic cage excursion due to ankylosing spondylitis results in a relative impairment of apical ventilation and that this may be one factor in the pathogenesis of apical fibrosis in this disease.

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