

Effects of diffuse pleural thickening on respiratory mechanics

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ABSTRACT: Five patients with radiographic and computed tomography (CT) evidence of extensive pleural thickening were studied. All had a restrictive ventilatory defect. The single breath carbon monoxide transfer factor (TLCO) was reduced in each case (mean 76% predicted) but in four patients the transfer coefficient (KCO) was increased (mean 116% predicted), which is consistent with lung 'en cuirasse'. Static pressure-volume curves of the lungs showed that the maximum transpulmonary pressure was greater than normal and pulmonary compliance was reduced; the curves were therefore indistinguishable from those obtained in patients with pulmonary fibrosis. Transdiaphragmatic pressures during maximal inspiratory efforts were moderately impaired and the proportions of each tidal breath contributed by anteroposterior (AP) motion of rib cage and abdomen were similar to normal in both erect and supine postures, suggesting that diaphragmatic mobility was well preserved.

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Diffuse bilateral pleural thickening can result from asbestos exposure, when it may be seen as a discrete entity without conventional radiographic evidence of underlying pulmonary interstitial fibrosis [1]. Pleural thickening has also been suggested as a cause of the 'shrinking lung' syndrome seen in some patients with systemic lupus erythematosus [2], although radiographically visible pleural abnormalities are slight or absent in such patients [3].

There is conflict in the literature as to whether maximum static transpulmonary pressure (PL_{max}) is increased [4] or decreased [5] in subjects with pleural disease without diffuse interstitial pulmonary fibrosis, and hence whether measurement of PL_{max} is a useful clinical test for distinguishing 'pleural restriction' from intrapulmonary restriction. We have studied a group of patients with extensive pleural disease to help resolve this controversy and to further assess the functional consequences of bilateral pleural thickening.

Patients and methods

We investigated chest wall motion, mechanical function of the lungs and transdiaphragmatic pressures during static maximum efforts in five patients with extensive bilateral thickening, in whom this appeared to be the main cause of lung volume restriction. Four of the five patients had a history of exposure to respirable asbestos. Each patient underwent computed tomography (CT) scanning of the thorax to assess the pleural thickening and to identify any abnormality of the underlying lungs. No patient had clinical or conventional radiographic

evidence of interstitial pulmonary fibrosis but minor peripheral scarring was shown by CT scanning in the lung fields of two patients (Nos 1 and 3). All subjects had extensive bilateral pleural thickening visible on the chest radiograph and confirmed by CT scan (figs 1 and 2). Measurements from the pleural abnormality (PA) radiographs showed pleural thickening extending over an average of 71 (range 36-100)% of the vertical dimension of the rib cage laterally, with an average maximum thickness of 10.3 (range 5-20) mm. It was not possible to visualize the diaphragmatic surface directly but 'blunting' of the costophrenic angles was visible bilaterally in four patients and unilaterally in the fifth.

Forced expiratory volume in one second (FEV₁) and vital capacity (VC) were measured using a bellows spirometer (Vitalograph). Total lung capacity (TLC) and residual volume (RV) were determined by whole body plethysmography [6]. The transfer factor for carbon monoxide (TLCO) was measured by the single breath technique [7], using the alveolar volume (VA) measured by helium dilution during the single breath [8]; the transfer coefficient (KCO) was calculated as TLCO/VA. Reference values for lung volumes were taken from COTES [9] and for TLCO and KCO from BRADLEY *et al.* [10].

Pleural pressure (Ppl) was estimated by measurement of oesophageal pressure using a balloon containing 0.3-0.5 ml air, positioned in the lower oesophagus at a distance from the nares determined by the formula (height-5) cm [11]. Static pressure-volume (PV) curves of the lungs were constructed from measurements during stepwise expiration. The values obtained were compared with PV curves



Fig. 1. Posteroanterior chest radiograph of patient No. 1 showing extensive pleural thickening with no evidence of intrapulmonary shadowing.



Fig. 2. CT cut of mid thorax in patient No. 1 showing concentric pleural thickening.

constructed from the reference values for lung recoil pressures at various lung volumes obtained by de TROYER and YERNAULT [12]. Static compliance (CL) was measured as the slope of the static expiratory PV curve over the volume range functional residual volume (FRC) to (FRC+0.5) l.

A second balloon in the stomach was used to record gastric pressure; subdiaphragmatic abdominal pressure (Pab) was obtained from gastric pressure by subtracting the pressure difference (usually 10–15 cmH₂O) between the balloons recorded during relaxation at FRC [13]. Studies were performed with the subject seated in a variable volume plethysmograph;

each subject made several maximal inspiratory efforts at various lung volumes against an occluded mouthpiece. The subjects were requested to inspire as forcefully as possible and no specific instructions were given about technique. Corresponding values of Ppl and Pab were then plotted against absolute thoracic gas volume (expressed as a percentage of predicted TLC) with lines of best fit drawn by eye and the results compared with the ranges of values found in eleven normal subjects [14].

Finally, the proportions of each tidal breath contributed by the rib cage and abdomen were estimated by two pairs of linearised magnetometers measuring anteroposterior (AP) motion at the levels of the nipples and 2 cm above the umbilicus in both upright sitting and supine positions. The magnetometer signals were calibrated by isovolume manoeuvres in each posture. Measurements were made with the subject resting quietly with no mouthpiece or noseclip and averages of ten representative breaths in each posture were calculated. The results were compared with the normal data for twenty elderly men reported by SHARP *et al.* [15].

Results

The results of spirometry, lung volumes, CO transfer, static compliance and maximum lung recoil pressure are shown in table 1. There were significant reductions in FEV₁, VC (mean 64% predicted) and TLC (mean 75% predicted). Two patients had evidence of mild airflow obstruction with FEV₁/VC between 65 and 70%. The TLCO was reduced (mean 76% predicted) but the KCO was on average increased (mean 116% predicted).

The static PV curves of the lungs (with volume plotted as a percentage of predicted TLC) lay outside the normal range, with, in each case, a reduction in static compliance and an increased maximum transpulmonary pressure (fig. 3). Values of Ppl and Pab during maximum static inspiratory efforts at various lung volumes are shown for each individual in figure 4. Values for maximum transdiaphragmatic pressure (Pdi) recorded averaged 84 (range 65–106) cmH₂O, slightly lower than the normal values of Pdi max during spontaneous maximum inspiratory efforts of 83–201 cmH₂O reported by GIBSON *et al.* [14].

The proportions of the tidal volume contributed by anteroposterior movement of rib cage and abdomen in each patient are shown in table 2. Despite extensive pleural thickening, change in rib cage volume was the main contributor to the tidal volume in the upright position and the results were broadly similar to normal.

Discussion

In all five patients reported here extensive bilateral pleural thickening appeared to be the main cause of the reduction in lung volumes. Although there was no clinical or radiographic evidence of diffuse interstitial

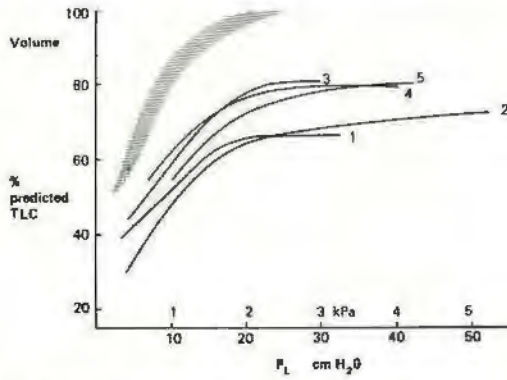


Fig. 3. Static expiratory pressure-volume curves of five patients compared with normal range for the appropriate age group [12]. Volume is plotted as % predicted TLC to minimize variation due to body size.

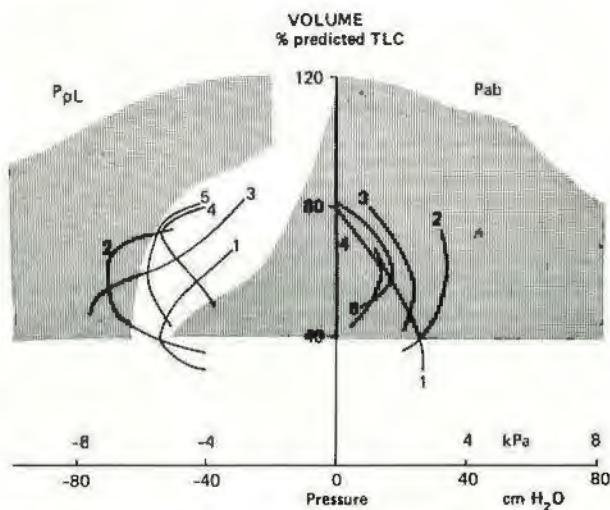


Fig. 4. Values of Ppl (left) and Pab (right) during static maximum inspiratory efforts at various lung volumes with lines of best fit drawn by eye, compared to normal ranges [14]. Horizontal distances between Ppl and Pab represent transdiaphragmatic pressures.

lung disease, minor pulmonary scarring was detected by CT scanning in two of the patients. Pathological studies of diffuse pleural fibrosis due to asbestos [16] suggest that some degree of interstitial fibrosis in the immediately subpleural alveoli is the rule. The extent to which this localized alveolar fibrosis contributes to abnormal lung function is speculative. The abnormalities described in this and other reports with pleural fibrosis should therefore be interpreted in a pragmatic fashion, as those expected in patients where diffuse pleural thickening is the dominant feature. Routine lung function tests in our patients showed abnormalities similar to those previously described with a restrictive ventilatory defect and some reduction of CO transfer factor but the Kco was greater than predicted in four of the five subjects and was normal in the fifth individual. Elevation of Kco is seen in normal subjects, if the measurement is made at volumes less than TLC [17] and in disease it is compatible with extrapulmonary restriction, which results in incomplete alveolar expansion at TLC.

Table 1. - Lung volumes, CO transfer factor, static compliance and maximum recoil pressure

Patient	age	VC	% pred	FEV ₁ % pred	FEV ₁ /VC	TLC % pred	RV % pred	FRC % pred	Tlco % pred	Kco % pred	CL	PLmax	%pred
n	yr	l			%						cmH ₂ O ⁻¹	cmH ₂ O	
1	45	2.65	60	52	68	67	85	79	69	128	0.122	32	111
2	48	2.85	71	75	82	73	77	81	79	125	0.139	52	186
3	68	2.15	65	61	65	81	110	81	82	127	0.131	29	128
4	44	3.0	67	59	70	80	115	108	65	94	0.132	40	137
5	50	2.15	54	62	88	81	139	120	55	108	0.096	42	153

% pred: percentage of predicted value; VC: vital capacity; FEV₁: forced expiratory volume in one second; TLC: total lung capacity; RV: residual volume; FRC: functional residual capacity; Tlco: transfer factor; Kco: specific transfer factor; CL: compliance; PL: elastic recoil pressure

Table 2. - Contributions of rib cage (% RC) and abdomen (% AB) to tidal breathing

Patient	Upright		Supine	
	%RC	%AB	%RC	%AB
1	67	33	15	85
2	73	27	26	74
3	67	33	25	75
4	69	31	42	58
5	70	30	28	72

For 20 normal elderly men [14] values of %RC upright are 74 ± 3 and for %RC supine, 21 ± 3 (mean \pm SD)

The validity of oesophageal pressure as a guide to pleural pressure is questionable in the presence of pleural disease but pleural thickening would seem to attenuate rather than to exaggerate pressure changes, *i.e.* to lead to underestimation rather than overestimation of lung recoil pressures. Comparison of the differences between oesophageal and mouth pressures during maximum static efforts with the differences during relaxation in one subject showed agreement, further supporting the validity of oesophageal pressure measurements. In practice, the curves obtained were indistinguishable from those found in patients with diffuse interstitial pulmonary fibrosis [18], with reduced static compliance and increased lung recoil pressures (fig. 3). A secondary reduction in lung compliance is well recognized with other extrapulmonary causes of restriction such as respiratory muscle weakness [19] and therefore does not necessarily imply alveolar fibrosis. The estimated maximum transpulmonary pressure was greater than normal, a finding in keeping with the results of WRIGHT *et al.* [4], but the overall shape of the PV curves (fig. 3) was different and we did not find the sigmoid curves reported in three of their four subjects. The increased PLmax in our patients contrasts with the results of COLP *et al.* [5] in three patients with apparent pleural restriction. However, the patients presented in detail in their paper had more complex problems than simple pleural thickening and these may have influenced their results.

The hallmark of extrapulmonary restriction due to other mechanisms is a reduction in PLmax: this is seen in patients with respiratory muscle weakness or with scoliosis, as well as in normal subjects with the chest wall strapped. The raised PLmax in the patients described here might therefore be a consequence of thickening of the visceral pleura. The reductions in Pdi during maximum static inspiratory efforts suggest some impairment of inspiratory muscle function and similar findings have also been reported in patients with pulmonary fibrosis [20]. Nevertheless, it appears that in both situations the inspiratory muscles retain

sufficient power to generate supranormal values of PLmax at the reduced total lung capacity. The normal relative contributions of rib cage and abdomen to tidal breathing in the present series of patients suggest the retention of diaphragmatic mobility.

Pleural thickening has been postulated as a cause of the 'shrinking lung' syndrome found in some patients with systemic lupus erythematosus (SLE); investigation of lung mechanics in such patients [3], however, shows reduction in PLmax and more marked impairment of maximum inspiratory pressures than was seen in the patients with extensive pleural thickening described here. It is, therefore, unlikely that the abnormalities found in the 'shrinking lung' syndrome are directly attributable to pleural thickening.

The ability to recognize pleural restriction without diffuse pulmonary fibrosis may have important clinical implications since surgical removal of the thickened pleura may be undertaken in order to alleviate symptoms. It appears that the PV curve of the lungs alone is unlikely to be helpful in making this distinction. An increased PLmax, together with a raised KCO should, give useful functional evidence to support the CT scan appearances in recognizing pleural restriction as the dominant mechanism of lung volume reduction in such patients.

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RÉSUMÉ: Cinq patients présentant des signes évidents radiographiques et CT d'épaississement pleural ont été examinés. Tous souffraient d'une déficience restrictive ventilatoire. Le facteur de transfert en monoxyde de carbone pour une seule inspiration (TLCO) était réduit dans chaque cas (moyenne prédite 76%) mais chez quatre patients, ce coefficient de transfert (KCO) était augmenté (moyenne prédite 116%), caractérisant le poumon 'en cuirasse'. Les courbes du volume de pression statique des poumons ont montré que la pression maximale transpulmonaire était plus élevée que la normale et que la compliance pulmonaire était réduite; il s'ensuit que ces courbes ne différaient en aucune façon de celles obtenues chez des patients atteints de fibrose pulmonaire. Les pressions transdiaphragmatiques pendant les efforts respiratoires maxima étaient modérément gênées et les proportions de chaque respiration contribuéées par les mouvements AP du gril costal et de l'abdomen s'approchaient de la normale chez les sujets en position debout ou couchée, suggérant ainsi que la mobilité du diaphragme était bien conservée.