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Lung mechanics and their relationship to lung volumes in pulmonary sarcoidosis

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ABSTRACT: Pulmonary sarcoidosis was studied with respect to lung mechanical properties and to the influence of these on lung volumes. Sixty six patients, with histological support for the diagnosis of sarcoidosis, and radiological signs of pulmonary involvement, *i.e.* stage II or III, were studied. The static pressure/volume (P/V) curves showed that the static elastic recoil pressure ($P_{el,L}$) tended to be increased at a given percentage of predicted total lung capacity (TLC). Reduction of static lung compliance (Cst_L) was a typical finding. At maximal inspiration $P_{el,L}$ was abnormally low in 20 subjects, including in the main those with recent onset of the disease and older patients. The possibility of a greater inflammatory activity at the site of mechanical receptors in the lungs and airways of these patients is proposed. Pulmonary resistance, measured at a given $P_{el,L}$, was usually increased signifying bronchial involvement. TLC, residual volume (RV) and functional residual capacity (FRC) were lower in current smokers and ex-smokers than in lifelong nonsmokers. This may be due to synergistic effects of the inflammatory processes caused by smoking and sarcoidosis. A reduced vital capacity (VC) mainly reflected a low Cst_L but also obstruction with increased RV. Forced expiratory volume in one second (FEV_1) reflected lung stiffness and obstruction equally. Lung mechanics revealed functional abnormalities which were not obvious from the standard tests, particularly in patients with respiratory symptoms.

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Reduced vital capacity and total lung capacity in pulmonary sarcoidosis were reported in the earliest physiological studies in 1940 [1]. Since that time numerous investigators have described various other abnormalities, the major ones being decreased lung compliance, reduced diffusing capacity, abnormal gas exchange and airway obstruction [2, 3]. Some abnormalities are easily measured, whilst the detection of others requires methods not often used clinically. This may partly explain the lack of reports on lung mechanics including analysis of the pressure/volume curve in a large group of subjects with pulmonary sarcoidosis.

The routine procedure for functional evaluation of interstitial lung disease at our hospital is study of the pulmonary static pressure/volume curve and the relationship between lung resistance and static recoil pressure ($P_{el,L}$). These curves reflect elastic properties of the parenchyma, and bronchial involvement. Lung mechanics are investigated in all patients referred to the hospital with radiological signs of pulmonary sarcoidosis. The degree of functional impairment, together with other parameters reflecting disease activity, is used as a guideline for the institution of therapy. We therefore have experience of measurements of lung mechanics in pulmonary sarcoidosis from a large group of subjects. Patients with only lymph node enlargement without parenchymal infiltrations are not usually receiving treatment. Their disease is prone to spontaneous subsidence [4], and lung mechanics are not usually studied in these patients.

The aim of the present study is to elucidate the elastic and resistive properties of the lung and to show how these are reflected in lung volumes in pulmonary sarcoidosis. The subjects in the study comprise a wide spectrum of patients, which is necessary to establish these relationships. Another objective was to evaluate the usefulness of a complex mechanical analysis in clinical practice and in the judgement of development of pulmonary fibrosis.

Material and methods

The criteria for inclusion in the study were radiological signs of pulmonary sarcoidosis, *i.e.* stage II or stage III chest radiographs, and histological support for the

Table 1. – Clinical data in the 66 patients divided according to age, smoking habits, symptoms, radiographic stage, duration of disease and treatment

	Age, yrs		Smoking habits		Symptoms		Radiographic stage	
	≤40	>40	non smokers	current, ex-smokers	no	yes	Stage II	Stage III
	n=35	n=31	n=41	n=25	n=23	n=43	n=27	n=38
Age, yrs								
mean	32	55	45	39	38	45	41	43
±SD	6	8	13	14	13	14	16	12
≤40 yrs			20	15	15	20	14	21
>40 yrs			21	10	8	23	13	17
Smoking habits								
nonsmokers					15	26		
current, ex-smokers					8	17		
Radiographic stage								
Stage II			14	13	11	16		
Stage III			26	12	12	26		
Disease duration								
<24 months	18	14	20	12	12	20	18	14
≥24 months	17	17	21	13	11	23	9	24
Steroid therapy								
prior	7	5	7	5	4	8	4	8
current	2		1	1	1	1	1	1

Three patients who at the time of functional evaluation had normalized their radiographs from stage II to stage 0 are included in the Stage II group. One subject's radiograph was lost.

diagnosis of sarcoidosis, mainly by positive Kveim test and/or lymph node biopsy. Sixty six of the patients referred to us between September 1974 and December 1981 met these criteria and were thus studied. Their clinical data are shown in table 1. The mean age of the patients was 43 yrs (range 20–71 yrs). There were 36 men and 30 women, all white. Forty one subjects were nonsmokers, 14 ex-smokers and 11 current smokers. In the data analysis, ex-smokers are treated as current smokers. Twenty three patients had no symptoms of cough, or dyspnoea, whilst the other 43 had minor to moderate respiratory symptoms. We estimated the duration of disease from the clinical picture and chest radiographs to be less than 2 yrs in 32 of the subjects. In fifteen patients the duration was five yrs or more, the longest duration being 18 yrs. Twelve patients had previously been prescribed oral steroids at other hospitals. Their maintenance dose had been low, 5–10 mg prednisolone per day. Two other patients were on treatment at the time of investigation with 5 and 10 mg prednisolone. In one subject the information about the treatment was lost. The main reason for treatment had been hypercalcaemia in two subjects, ulcerative colitis in one and pulmonary involvement in the others. The duration of treatment had varied from a few months in half of the patients to more than 4 yrs in two.

Chest radiography

Chest radiographs obtained when the patients were admitted to the Department of Lung Medicine were used for inclusion in this study. A second radiograph was obtained at the time of physiological investigation. All were standard chest radiographs in anteroposterior and lateral projections. A standard lung stand with a film-focus distance of 340 cm and air-gap technique was used and the films were exposed at 135 kV. The second radiograph was used for the study and simultaneously reviewed by two co-operating radiologists unaware of the results of the lung function tests. The following classification was used: stage 0=normal radiograph; stage I=lymph node enlargement without parenchymal abnormalities; stage II=interstitial parenchymal changes with or without enlarged lymph nodes and without evidence of fibrosis; stage III=indications of fibrosis apart from other parenchymal changes [5].

At the time of the functional evaluation 24 patients showed stage II findings, 13 with minor infiltrations, 10 with moderate and one with extensive parenchymal changes. Thirty eight had stage III appearance and of these 13 had minor, 20 moderate and 4 extensive infiltrations. Twenty two of the patients with stage III

findings had minor radiographic signs consistent with pulmonary shrinkage, such as hilar and/or diaphragmatic elevation. Fifteen subjects had moderate, and one had severe, signs of shrinkage. One patient showed no other abnormality than hilar and diaphragmatic elevation. Twelve patients with moderate to extensive infiltrations showed moderate shrinkage. Three patients had cleared their radiographs from stage II to stage 0 after entering the study. These patients are included below in the stage II group. In one case the radiograph was lost.

Lung function tests

A complete investigation of pulmonary mechanics was made in all patients, this being the routine procedure.

Static lung volumes, *i.e.* total lung capacity (TLC), functional residual capacity (FRC), residual volume (RV), and vital capacity (VC) were measured with a body plethysmograph [6]. Dynamic lung volumes, *i.e.* forced expiratory volume in one second (FEV_1), and FEV_1%VC were measured with a Bernstein spirometer.

Pulmonary mechanics were studied with the flow regulator method described by JONSON [6]. The pulmonary static pressure volume (P/V) curve was determined in the body plethysmograph. The flow was limited to 1 $l \cdot s^{-1}$ during a long expiration from maximal inspiration and the airway was closed at intervals of 0.3 s. An oesophageal balloon was used for measurement of the static elastic recoil pressure ($P_{el}L$) during the periods of zero flow. Without leaving the mouthpiece the patient then returned to the normal breathing level and the airway was shut by the flow regulator. The patient was asked to make a slow panting and the thoracic gas volume was determined. The P/V curve was then automatically constructed from the spirogram, $P_{el}L$ and the thoracic gas volume. The volume axis was expressed as percentage of predicted TLC [7, 8]. Static lung compliance (Cst_L) was calculated from the interval 0.5–1.5 kPa (5–15 cmH_2O) of the P/V curve. For determination of pulmonary resistance (R_L) the oesophageal balloon was used. R_L was measured through expirations covering most of the vital capacity at a standardized flow rate of 1 $l \cdot s^{-1}$. R_L was related to the simultaneously measured $P_{el}L$, again obtained during flow interruptions. The resulting curve, R_L *versus* $P_{el}L$, was constructed and R_L at $P_{el}L$ of 0.74 kPa (7.5 cmH_2O) was determined.

Table 2. – Pulmonary function data in sarcoidosis are presented as mean±SD for all patients together and for different groups of the patients according to age, smoking habits and symptoms

		All patients	Age, yrs		Smoking habits		Symptoms	
			≤40	>40	non-smokers	current, exsmokers	no	yes
		n=66	n=35	n=31	n=41	n=25	n=23	n=43
Cst_L %pred	mean	65***	64***	65***	66***	61***	75*** [***]	59***
	±SD	20	17	22	23	12	17	19
$P_{el}TLC$ %pred	mean	97	110 [***]	81**	94	101	105	92
	±SD	35	35	30	35	36	32	37
R_L %pred	mean	212***	167** [**]	265***	224***	193**	153* [**]	245***
	±SD	149	117	166	160	130	92	165
TLC %pred	mean	85***	84***	86***	88*** [*]	81**	86***	86***
	±SD	14	14	15	17	8	15	14
VC %pred	mean	85***	87***	82***	85***	84***	90*** [*]	82***
	±SD	17	17	17	20	11	10	20
RV %pred	mean	96	88** [*]	105	105 [***]	81***	89*	99
	±SD	33	27	37	34	23	27	35
FRC %pred	mean	92***	88**	98	101 [***]	78***	88**	95*
	±SD	27	26	29	29	17	25	29
FEV_1 %pred	mean	80***	83***	77***	79***	82***	87*** [*]	76***
	±SD	21	21	21	23	16	11	24
FEV_1%VC %pred	mean	93***	93**	93**	91***	97	97	91***
	±SD	14	13	15	14	14	10	15

Significant deviations of the mean values from those predicted are indicated and also significant differences within subgroups. Significant correlations are indicated by *: $p<0.05$; **: $p<0.01$; ***: $p<0.001$. Cst_L: static lung compliance; $P_{el}TLC$: static recoil pressure at total lung capacity; R_L: pulmonary resistance; TLC: total lung capacity; VC: vital capacity; RV: residual volume; FRC; functional residual capacity; FEV_1: forced expiratory volume in one second.

All subjects had given informed consent to the measurements.

Statistical methods

For statistical analysis Student's t-test for paired and unpaired comparisons and single and double correlation tests were used. Differences with a probability level of $p<0.05$ are marked *, $p<0.01$ **; and $p<0.001$ ***. Since a large number of significance analyses have been performed p values >0.01 should be regarded with particular caution.

The range of a parameter was calculated as the interval between the 5th and the 95th percentiles of observed values.

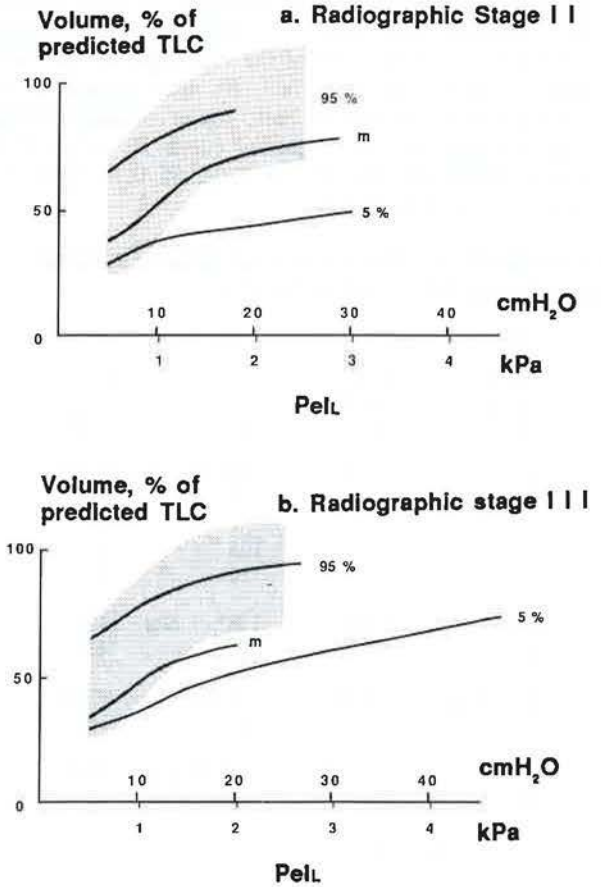

Fig 1. – For each of the radiographic groups the patient with the median position of the curve in the volume dimension, m, illustrates typical P/V curves. The range is illustrated by curves from two other patients representing the 5th and the 95th percentiles. The shaded area indicates the 95% confidence interval for healthy subjects. a. In stage II more than 50% of the curves are within the normal range. b. In stage III more than 50% of the curves are below the normal range at higher values of PeiL. In both groups the slope of the P/V curves is low, *i.e.* compliance is reduced.

Predicted normal values

The results are reported as percentage of predicted values (%pred). Lung volumes were predicted according to BERGLUND *et al.* [9] and GRIMBY and SÖDERHOLM [10]. The normal P/V curves represent data from KNUDSON *et al.* [11] pooled with data from our department [7]. Reference values of PeiL at a maximal inspiration (PeiTLC) were calculated according to DE TROYER and YERNAULT [12]. Data on RL refer to material on healthy subjects, in part reported previously [7]. Values outside the 95% confidence interval for healthy subjects were regarded as abnormal.

Results

Lung function findings in all patients

Pulmonary function data for all patients together, and for subgroups of the patients arranged according to age, smoking habits and symptoms, are presented in table 2.

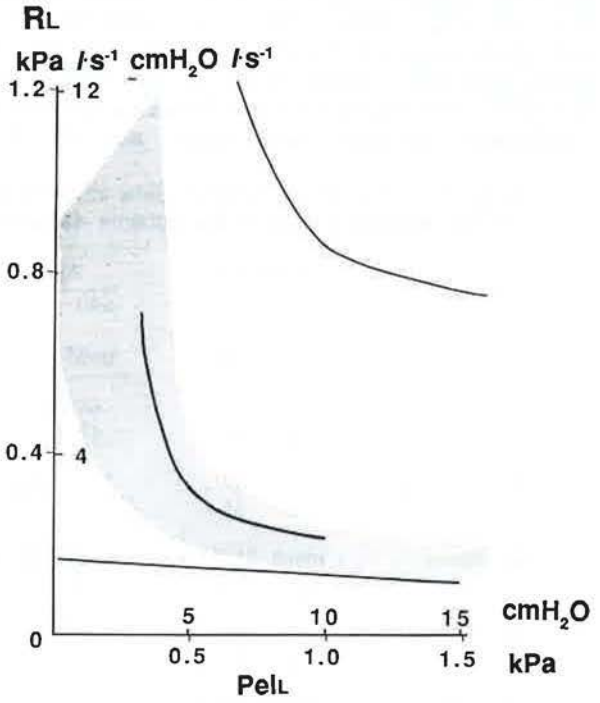

Fig 2. – Lung resistance is measured through an expiration starting at a high PeiL. Three curves from men are shown together with the normal range for men (stippled area indicating the 95% confidence interval for healthy subjects). One selected curve shows normal resistance at any given PeiL. Another curve shows a high resistance as in obstructive bronchial disease. The curve which does not show the normal increase in resistance at low values of PeiL may indicate stiff airways [38].

In the entire group of patients the most striking finding was reduction of compliance, which is further illustrated by figure 1. PeiL at maximal inspiration (PeiTLC) was on average 97% of that expected but the range was wide, 47–172 %pred. RL was on average 212% of predicted (range 59–550 %pred). Twenty four, *i.e.* 37%, of the patients had abnormally high values of RL, and 15 of these had never smoked. Most patients, both obstructive and non-obstructive, showed the classical feature of rapidly increasing RL at falling values

of $P_{el,L}$, (fig. 2). In three patients R_L did not increase in the normal way when $P_{el,L}$ approached zero. All lung volumes except RV were reduced. The greatest reduction was seen in FEV_1 (80 %pred) followed by VC and TLC (both 85 %pred).

Lung function findings in clinical subgroups

The main findings in the subgroups are as follows:
1. Patients over the age of 40 yrs had lower $P_{el,TLC}$ than younger ones. Older patients also had higher R_L and higher RV than the younger ones.
2. Current smokers and ex-smokers showed lower TLC, RV and FRC compared to lifelong nonsmokers.
3. Symptomatic subjects had both lower compliance and more severe obstructive changes than those who were asymptomatic.
4. Patients with known duration of disease less than 2 yrs had lower $P_{el,TLC}$ than subjects with longer duration (87 %pred and 106 %pred, respectively, $p<0.05$) (this is not shown in table 2). All but three of the 20 subjects with abnormally low values of $P_{el,TLC}$ had a short history of disease and/or were older than 40 yrs.

No significant differences were seen between treated and untreated patients.

Lung function data in radiographic subgroups

$P_{el,TLC}$ was significantly lower in stage II than in stage III (table 3). Other parameters showed no significant differences between the two stages. A separate analysis of the patients with stage III disease showed that those with minor radiographic signs of shrinkage had higher Cst_L, TLC, VC, FEV_1, and FEV_1%VC than those with more pronounced shrinkage. $P_{el,TLC}$ was not seen to vary with the degree of shrinkage.

Typical features of the P/V diagram in stage II and stage III patients are shown in figure 1a and 1b. For each group three curves from individual patients are displayed. One of the curves represents the median position and the other curves the 90% confidence interval of the observations in the patients volume-wise. In both radiographic groups the P/V curves fall within the normal volume range at low values of $P_{el,L}$. At higher values of $P_{el,L}$ the volumes tend to be reduced, particularly in patients with stage III disease. Thus, the

Table 3. – Pulmonary function data presented as mean±SD for the patients according to their radiographic appearance

		Stage II		Stage III		
			all	minor shrinkage		moderate-severe shrinkage
		n=27	n=37–38	n=22		n=15–16
Cst_L %pred	mean	65***	65***	71	[*]	55
	±SD	17	21	21		18
$P_{el,TLC}$ %pred	mean	83*	[**] 108	105		112
	±SD	32	34	33		36
R_L %pred	mean	184**	227***	200		267
	±SD	137	155	111		201
TLC %pred	mean	86***	84***	88	[*]	79
	±SD	16	14	15		11
VC %pred	mean	88**	83***	89	[**]	74
	±SD	18	16	10		19
RV %pred	mean	95	95	95		96
	±SD	24	37	34		42
FRC %pred	mean	91*	92**	93		89
	±SD	26	26	27		23
FEV_1 %pred	mean	85***	77***	86	[**]	66
	±SD	20	21	15		23
FEV_1%VC %pred	mean	94	92***	96	[*]	87
	±SD	16	13	10		14

The three patients who had cleared their radiographs from stage II to stage 0 are included in the stage II group. One subject's radiograph was lost. Significant deviations of the mean values from normal in stage II and stage III are given, as are significant differences between the two stages and between the two subgroups of stage III. Significant correlations are indicated by *: $p<0.05$; **: $p<0.01$; ***: $p<0.001$. For abbreviations see table 2.

dominant abnormality of the P/V curve is the slope: Cst_L is 65% of the predicted value (range 31–93 %pred and 32–106 %pred, respectively) in both stage II and stage III.

Relationship between lung volumes and lung mechanics

The reductions in TLC, VC and FEV_1 are statistically related to the decrease in Cst_L, although the coefficient of determination, (r^2), indicates that only 27% of the variation in TLC can be attributed to variations in Cst_L, (table 4). The variation in VC is more closely related to compliance (r^2=0.45). TLC is also correlated to volumes read from the P/V curve at specific values of Pel_L. The volume at a Pel_L of *e.g.* 0.5 kPa (5 cmH_2O) correlates to TLC (r^2=0.45, p<0.001). The r^2 values indicate that TLC reflects the position of the curve volume-wise to a larger extent than it reflects compliance. Pel_{TLC} does not correlate to Cst_L, but there is a weak negative correlation to R_L, (table 4).

Table 4. – Relations between different pulmonary function data in sarcoidosis and Cst_L and R_L

	Relationship to Cst_L %pred n=65–66 r^2	Relationship to R_L %pred n=65–66 r^2
Cst_L % pred	-	-
Pel_{TLC} %pred	0.01	0.06*
R_L %pred	0.09*	-
TLC %pred	0.27***	0.02
VC %pred	0.45***	0.29***
RV %pred	0.00	0.11**
FRC %pred	0.04	0.01
FEV_1 %pred	0.37***	0.44***
FEV_1%VC %pred	0.03	0.34***

Correlations to Cst_L and R_L are indicated by r^2=coefficient of determination, showing to what extent the variation in a parameter can be explained by Cst_L %pred or R_L %pred. Significant correlations are indicated by *: p<0.05; **: p<0.01; ***: p<0.001.

As expected FEV_1 and FEV_1%VC correlate strongly to R_L. High resistance is also associated with a low VC. A weak correlation is found between RV and R_L. When an orthogonal double regression analysis is performed between RV (dependent variable), R_L and volume at Pel_L=0.5 kPa (5 cmH_2O) (independent variables) one finds that the latter factor is also reflected in RV (p<0.01, r^2=0.22). The slight reduction in FRC can not be statistically associated with Cst_L but rather with the position of the P/V curve in the volume range. Thus FRC correlates with volume at a Pel_L=0.5 kPa (5 cmH_2O) (r^2=0.46).

The number of patients with abnormal values of VC, TLC and Cst_L is shown in figure 3. All subjects with low VC had either abnormal TLC or abnormal Cst_L. To the number of patients with abnormal VC, determination of TLC added 12 abnormal observations, and determination of Cst_L contributed yet another six. Nine patients with abnormal TLC had normal Cst_L values. The reduced values of TLC in these cases reflect the fact that the P/V curves fall below the normal range. The six patients with low Cst_L but normal TLC and VC were all symptomatic.

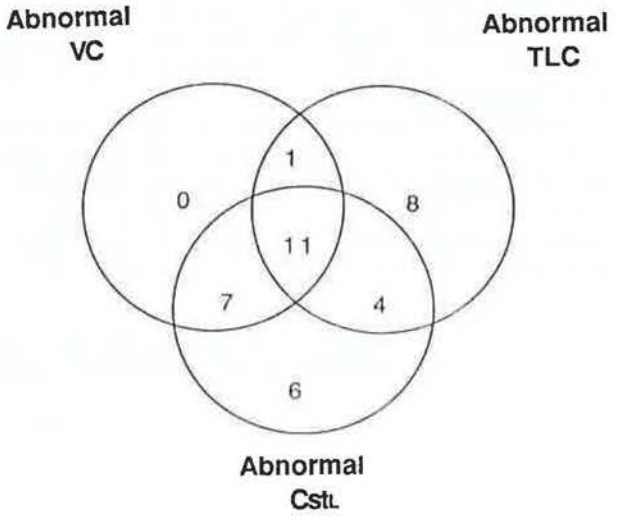

Fig 3. – Number of patients with abnormal values of VC, TLC and Cst_L. Total number=65.

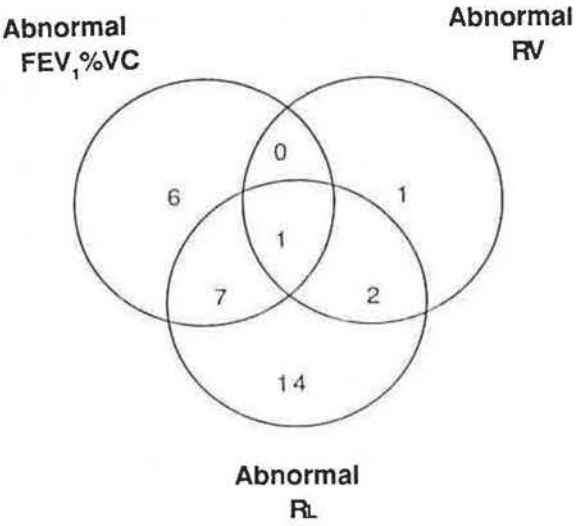

Fig 4. – Number of patients with low values of FEV_1%VC, high values of RV and R_L. Total number=65.

In 14 patients R_L alone revealed bronchial obstruction (fig. 4). The six subjects with low FEV_1%VC in spite of normal RV and R_L values showed a significantly lower Pel_{TLC} than the other 60 subjects, 72

%pred compared to 99 %pred ($p<0.01$). The P/V curves of these patients do not show loss of recoil. Emphysema cannot, therefore, explain the findings. Of the 14 patients who showed an increase in R_L alone (fig. 4), only three were asymptomatic, two of whom had a minor increase in R_L.

Discussion

Reduced compliance is a well recognized abnormality in sarcoidosis [13–16]. Compliance is thought to reflect the elasticity of lung tissue and the number of functioning alveoli. A correlation between compliance and lung volumes has been reported [14, 16]. However, some authors who plot the volume in percentage of measured TLC against Pel_L, claim that the P/V curve is fairly normal [17–19]. The patient's measured TLC is affected by elastic changes within the lungs and also by the pressure developed by the respiratory muscles, reflected by Pel_{TLC}. The P/V curve displayed with volume in percentage of measured TLC is, by construction, normal at TLC and, therefore, offers a limited image of the overall abnormalities in the lung parenchyma [7, 8, 20].

Our finding of a close relationship between VC and Cst_L is in agreement with other observations [13–16] and implies that a low VC is in most instances a sign of stiff lungs. However, in our study Cst_L is a more sensitive indicator of stiff lungs. If the position of the complete P/V curve is also taken into account the sensitivity of mechanical studies increases further. In accordance with the results of others [15, 16], we found that patients with a low compliance are usually symptomatic. In our study, TLC, RV and FRC were found to reflect the position of the P/V curve along the volume axis, more so than its slope (compliance). This seems logical as each of the three volumes reflects an absolute volume at different recoil pressures, namely Pel_{TLC}, Pel_L at RV (which is about zero) and Pel_L at muscle relaxation, respectively. On the other hand, VC reflects a volume change relative to the pressure change created by the respiratory muscle, and is more closely correlated to compliance as this also represents volume change relative to pressure change.

Patients with lung restriction may compensate by means of an increase of the muscle force at full inflation [12, 15], although considerable variability has been noted [21]. Pel_{TLC}, reflecting this force, was on average normal in our patients. A reduced muscle force or reflex inhibition of inspiration may explain why, in the present study, Pel_{TLC} was lower in older subjects and in the subgroup of patients with a short duration of disease. The latter group of patients may, at least, have a greater inflammatory activity in their lungs and airways at the site of mechanical receptors. Indeed, after laryngectomy TLC is reduced during a period when patients have airway inflammation but unchanged P/V curves [22].

Three factors influencing VC have been recognized, a low Cst_L being the most important. Bronchial involvement has an additional influence *via* its effect on RV, as has the expanding pressure, *i.e.* Pel_{TLC}. No statistical relationship could be shown between a low VC or TLC on the one hand and Pel_{TLC} on the other. As a low VC may not only be caused by a low Pel_{TLC} but may also lead to a high Pel_{TLC} [12, 15], this is not surprising.

Some patients with a low Pel_{TLC} have reduced FEV_1%VC but normal R_L. A low ability to inflate the lungs may lead to reduced maximal expiratory flow rates and cause a low FEV_1%VC in the absence of significant airway disease.

In our study, TLC, RV and FRC are lower in smokers than in nonsmokers. Similar findings have been described previously [23], and also increased volumes [24]. The latter study suggests a synergism between sarcoidosis and smoking, leading to increased occurrence of airway disease and to hyperinflation of the lung. Others noted no significant differences in volumes between patients with diverging smoking histories [25]. By use of computed tomography [26] pulmonary density has been shown to be higher in healthy smokers than in nonsmokers. Histological examination of lungs from young smokers has shown pathological changes not solely confined to the airways, but also peribronchiolar inflammation and fibrosis [27]. Various alterations in number and function of lung immune and inflammatory cells of smokers have been reported [27, 28]. Some smoking patients with sarcoidosis have very high values of serum angiotensin converting enzyme and pulmonary gallium-67 uptake [25]. This indicates an intense disease activity and, again, an additional or even synergistic effect of smoking and sarcoidosis. Further studies are needed to confirm our finding of reduced volumes in smokers, and to explain the mechanism behind it.

Our study shows a high prevalence of airway obstruction in patients with sarcoidosis. It may reflect involvement of both small and large airways. Endobronchial sarcoidosis, extrinsic compression of the airways, fibrotic scarring, extension of a peribronchial granulomatous process and nonspecific hyperresponsiveness may all contribute [29]. In early physiological studies, little emphasis was placed on the airways although, in one early investigation, it was suggested that the increase of RV could be due to obstructive lesions [30]. Later airway obstruction has been documented by different methods [18, 23, 24, 29, 31–36]. The incidence is apparently variable, and not all studies of airway mechanics have shown evidence of obstruction [17]. In the present study symptomatic patients had higher R_L values than the rest. Surprisingly few studies of the airways in patients with sarcoidosis give information about the patient's symptoms. Abnormal airway function was demonstrated by at least one test in all subjects in a study comprising only symptomatic patients [35]. In another large study, dyspnoea was frequently associated with expiratory slowing due to intrinsic bronchial obstruction in addition to pulmonary restriction [36]. Our result is also in keeping with the finding of an inverse correlation between exercise capacity and variables reflecting bronchial obstruction [37].

The lack of increase in R_L at low values of Pel_L

found in three patients in this study of sarcoidosis is more common in sceleroderma [38]. The pattern is thought to represent increased bronchial rigidity. The increased maximal expiratory flow in relation to PeIL found in some patients with sarcoidosis in another study [17] could be explained by the same mechanism.

In pulmonary sarcoidosis a comprehensive investigation of lung mechanics displaying the complete P/V and RL/PeIL curves can reveal physiological abnormalities not shown by a less extensive examination. In clinical routine, however, investigation of lung mechanics in asymptomatic subjects is unlikely to add important information to that obtained by measurements of lung volumes. In symptomatic patients, on the other hand, lung mechanics can reveal elastic and resistive abnormalities which would not be anticipated from measurements of lung volumes alone.

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Mécanique pulmonaire et sa relation au volume pulmonaire dans la sarcoïdose pulmonaire. L. Bradvik, P. Wollmer, B. Simonsson, U. Albrechtsson, K. Lyttkens, B. Jonson.

RÉSUMÉ: Les propriétés mécaniques du poumon dans la sarcoïdose et leurs influences sur les volumes pulmonaires ont fait l'objet de cette étude. 66 patients où le diagnostic de sarcoïdose était confirmé par un examen histologique et présentant des signes radiologiques d'atteinte pulmonaire (stade II ou III) ont été étudiés. Les courbes de pression-volume statique ont montré que la pression de recul élastique statique (Pe1L) tendait à augmenter à un pourcentage donné de la capacité pulmonaire totale prédite (TLC). La réduction de la compliance pulmonaire statique (CstL) est une observation typique. A l'inspiration maximale, Pe1L est anormalement basse chez 20 sujets parmi lesquels principalement des patients dont la maladie a commencé récemment et des patients plus âgés. La possibilité d'une augmentation de l'activité inflammatoire au siège des récepteurs mécaniques des poumons et des voies aériennes est proposée chez ces patients. La résistance pulmonaire, mesurée à une Pe1L donnée s'avère habituellement augmentée, ce qui signe une atteinte bronchique. TLC, volume résiduel et capacité résiduelle fonctionnelle sont plus bas chez les fumeurs et les ex-fumeurs que chez les non-fumeurs: ceci pourrait être dû aux effets synergiques des processus inflammatoires attribuables également à la fumée et à la sarcoïdose. Une réduction de la capacité vitale traduit principalement un abaissement de CstL mais aussi une obstruction avec augmentation du volume résiduel. Le VEMS traduit à la fois la rigidité pulmonaire et l'obstruction. L'étude de la mécanique pulmonaire a révélé des anomalies fonctionnelles qui n'étaient pas évidentes lors des tests standard, principalement chez les patients atteints de symptômes respiratoires.

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