

Different patterns of gas exchange response to exercise in asbestosis and idiopathic pulmonary fibrosis

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ABSTRACT: To analyse the pattern of pulmonary gas exchange during maximal exercise (E_{max}) in asbestosis, we compared nine subjects with this disease (1 female/8 male), aged 54 ± 11 yrs (mean \pm SD), to nine patients (1 female/8 male) with idiopathic pulmonary fibrosis (IPF) of a similar age, height, weight and smoking history, both at rest and during E_{max} . No differences were observed in dynamic and static lung volumes between the groups. However, patients with IPF had a lower DLCOsb and KCO ($p < 0.005$ and 0.05 , respectively). At rest, both groups showed mild arterial hypoxaemia (76 ± 11 , asbestosis, vs 77 ± 11 mmHg, IPF), widened AaPO₂ (32 ± 14 vs 31 ± 13 mmHg) and slight increases in V_D/V_T (47 ± 12 vs $46 \pm 11\%$), respectively. During E_{max} , PaO₂ fell to 51 ± 7 mmHg in patients with IPF whereas those with asbestosis had PaO₂ of 73 ± 21 mmHg ($p < 0.05$). Conversely, those with asbestosis were able to reduce V_D/V_T (from 47 ± 12 to $39 \pm 10\%$, $p = 0.01$) as opposed to those with IPF (from 46 ± 11 to $47 \pm 13\%$). Furthermore, DLCOsb and AaPO₂ during E_{max} were highly correlated only in IPF ($r = -0.84$, $p < 0.01$). Despite the finding that both diseases represent a diffuse pulmonary fibrosis with a similar degree of resting ventilatory impairment, the pattern of gas exchange during exercise is different in each. These differences may be related to the underlying morphology of each process, which probably includes more airway disease and less pulmonary vascular involvement and/or a different degree of interstitial fibrotic change in asbestosis.

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Respiratory functional abnormalities in idiopathic pulmonary fibrosis (IPF) classically include a restrictive ventilatory impairment, a reduction in single-breath CO diffusing capacity (DLCOsb), a rightward and downward shift of the pressure-volume curve and a mild to moderate resting arterial hypoxaemia which deteriorates during exercise [1]. At rest, asbestosis generally displays a similar functional picture [2-5]. It has therefore been inferred that patients with asbestosis also present impaired gas exchange during exercise [2, 4, 5].

However, previous studies have mostly concerned asbestos-exposed workers (without evidence of asbestosis) [6-11] and have used non-invasive techniques to evaluate gas exchange [12], or have focused on other aspects of exercise testing [10, 13, 14]. Thus, a comparative analysis of gas exchange during exercise between patients with asbestosis and IPF, matched for anthropometric data, smoking habits and the degree of resting ventilatory impairment is lacking. Asbestosis also involves several pathological and functional peculiarities which differ in part from those observed in IPF. The early peribronchiolar fibrosis seen in asbestosis [4, 8] supports the development of

airway disease [15], and airflow obstruction may be observed in this condition [16-18].

Given that the arterial oxygen tension (PaO₂) response to exercise is less predictable in the face of chronic airways obstruction [19-21], we wondered whether gas exchange during exercise in asbestosis might differ from that in IPF [1]. The present study was undertaken to specifically address this question. A better understanding of the gas exchange response to exercise in asbestosis is potentially useful since the legal, social and physiological issues concerning occupational lung diseases are rapidly becoming complex [22].

Methods

Population studied

We analysed the results of nine consecutive patients with asbestosis (one female/eight males) who were matched with nine non-consecutive subjects with IPF (one female/eight males). A case-by-case matching was not possible because of the relative rarity of both diseases. However, it was deliberately intended that

Table 1. - Anthropometric, clinical, radiographic, bronchoalveolar lavage (BAL) and pulmonary function test data (mean±SD) for patients with asbestosis and idiopathic pulmonary fibrosis

	Asbestosis		Idiopathic pulmonary fibrosis		p<
Age yrs	54±11		52±11		-
Height cm	165± 9		164± 6		-
Weight kg	75± 9		74±11		-
Tobacco pack-yr	30±20		26±23		-
Course § months	57±45		9±14		0.05
BAL					
% PMN	9±2		8±3		-
% Lymph	7±4		7±5		-
FVC l	2.6±1.1	(60±20%)	2.6±0.3	(63±11%)	-
FEV ₁ l	1.8±0.8	(56±23%)	2.1±0.3	(66±11%)	-
FEV ₁ /FVC %	71±11		80±6		-
V ₅₀ l·s ⁻¹	2.4±1.6	(60±40%)	4.2±2.0	(108±51%)	-
V ₇₅ l·s ⁻¹	0.5±0.4	(52±45%)	0.9±0.4	(87±45%)	-
TLC l	4.7±1.7	(68±20%)	4.6±1.1	(72±16%)	-
RV/TLC %	42±6		39±8		-
sGaw cmH ₂ O ⁻¹ ·s ⁻¹	0.18±0.10		0.24±0.18		-
DLCosb ml·min ⁻¹ ·mmHg ⁻¹	18.8±2.6	(70±11%)	12.5±3.9	(45±14%)	0.005
KCO min ⁻¹ ·mmHg ⁻¹	5.2±1.9	(97±32%)	3.8±1.5	(69±23%)	0.05

§ Course: period of time elapsed from the first objective evidence of disease to testing; %PMN and %Lymph: percentage of neutrophils and lymphocytes, respectively, in BAL fluid (normal values: %PMN: ≤3%; %Lymph: ≤12%) [21]. Values between brackets correspond to percentage of predicted [27, 28]. DLCosb: Single breath diffusing capacity for CO.

both groups were matched as closely as possible for sex, age, height, weight and smoking habits (table 1). The whole study was performed as part of the full diagnostic assessment and outpatient care protocol for interstitial lung disease in our institution. Therefore, all the patients were informed of the characteristics and nature of the lung function tests and gave oral consent. All of them were in a stable clinical condition.

Asbestosis

This group included nine subjects exposed to asbestos for an average of 11 ± 8 yrs (range, 3–25 yrs), whilst 20 ± 10 yrs (range, 7–33 yrs) had elapsed since the first known exposure; six of them were fibro-cement asbestos workers (1, 3, 6–9), two were textile asbestos workers (4 and 5) and the remaining subject (2) had been working as a loader in a harbour. Chrysotile and crocidolite fibres were the main fibres used in the fibro-cement factory, whereas chrysotile and amosite fibres were used in the textile factory. The diagnosis of asbestosis was made according to the following criteria [23]: a) a history of asbestos exposure; b) radiographic evidence of bilateral diffuse irregular interstitial opacities (coded 1/1 or higher, ILO/UC International Classification 1980) [24]; and c) absence of cardiopulmonary disease resulting in these abnormalities. Supportive, but not compulsory criteria were the presence of end-inspiratory ('late') crackles, as well as forced vital capacity (FVC) and/or

DLCosb below 80% of predicted values [23]. Bronchoalveolar lavage (BAL) was carried out in six patients, four of whom (1, 3, 5 and 6) have been reported elsewhere [23]. BAL showed the presence of asbestos bodies in all the patients tested. An open lung biopsy in patient 3 gave evidence of a diffused interstitial fibrosis with asbestos bodies [25]. None of the patients was receiving systemic steroids and bronchodilators, if any, were withdrawn 12 h before the study.

Idiopathic lung fibrosis

The diagnosis of 'lone' IPF was established, in part, according to the criteria proposed by FULMER *et al.* [26]: a) presence of progressive exertional breathlessness, without history of hypersensitivity lung disease, chronic pulmonary infection, left ventricular failure and/or exposure to any agent known to cause pulmonary fibrosis; b) existence of a diffuse interstitial pattern on chest X-ray film (coded 1/1 or higher, ILO/UC International Classification 1980), with no left ventricular enlargement; and c) presence of reduced lung volumes and/or single-breath CO diffusing capacity (DLCosb). BAL was carried out in all patients other than 14. Transbronchial biopsy showed morphologic changes suggestive of IPF and ruled out any granulomatous process in three patients (11, 12 and 17). In patient 16 an open lung biopsy revealed diffuse interstitial fibrosis without associated granulomata or vasculitis. Patients 10, 14 and 18 had a familial form of IPF. At the time of the study, three

patients (13, 15 and 16) were on regular steroid therapy (15–20 mg per day prednisolone).

General assessment

All patients answered a medical questionnaire about respiratory symptoms and were submitted to a detailed physical examination. In order to evaluate the course of the disease, we elected to pinpoint its onset from the first objective evidence, according to CARRINGTON *et al.* [27]; namely, the first abnormal chest roentgenogram, the first documented physical abnormality and/or the first measured pulmonary physiologic deficit. The latter was significantly longer in patients with asbestosis (table 1), suggesting therefore a more benign clinical course. It is of note, however, that this evaluation probably underestimates the actual length of the evolution of the disease [27]. All subjects had routine posteroanterior and lateral chest X-ray films and standard ECG records. Chest X-ray films were evaluated according to the ILO/UC 1980 Classification for Pneumoconioses [24] by three independent readers without knowledge of the clinical and functional findings. The final assessment of each roentgenogram represented the median interpretation of these three observers. Pulmonary function tests included forced vital capacity (FVC), forced expiratory volume in one second (FEV₁) and static lung volumes (Hewlett-Packard 47804A, Pulmonary System Desk, Palo Alto, CA), plethysmographic functional residual capacity and airway resistance (Body test, E. Jaeger, Würzburg, W. Germany) and single-breath carbon monoxide diffusing capacity (DLCOsb) (Respirometer Model A, PK Morgan Ltd, Chatham, Kent, UK). All reference values for forced spirometry, static lung volumes and DLCOsb correspond to those of our own laboratory [28, 29].

Exercise study

All the patients performed an incremental exercise test on a cycle ergometer whilst lying semi-recumbent (E. Jaeger, Würzburg, W. Germany). Exercise began at a power output of 0 Watts and increased by 20 Watts each minute [30]. Patients were encouraged to continue for as long as possible, both cardiac rhythm (Hewlett-Packard 7830A, Palo Alto, CA) and ear oximetry (BIOX Oximeter II A, Ohmeda, Boulder, CO) being continuously monitored. A low resistance, low dead-space (21 ml), non-rebreathing valve (E. Jaeger, Würzburg, W. Germany) was used, and minute-by-minute values for ventilation (\dot{V}_E) and mixed expired oxygen (FE_{O₂}) and carbon dioxide (FE_{CO₂}) fractions were obtained. From the latter, oxygen uptake (\dot{V}_{O_2}) and carbon dioxide output (\dot{V}_{CO_2}) were calculated (μ -Dataspir, E. Jaeger, Würzburg, W. Germany). Reference values for the incremental exercise test were those of JONES *et al.* [31]. Variations of less than ± 5 beats in heart rate and of less than $\pm 0.1\%$ in FE_{CO₂} and FE_{O₂} were

taken as indicative of an adequate steady state [30].

After ensuring local collateral circulation, arterial blood samples were anaerobically drawn from an indwelling radial artery catheter (Seldicath 1.3 mm, Plastimed), both at rest and during maximal exercise (Emax), and were assayed for PaO₂, PaCO₂ and pH (IL 1302 pH Blood Gas Analyser, Milano, Italy) within 10 min. The alveolar-arterial oxygen pressure difference (AaPO₂) was calculated using the standard alveolar air equation [30]: Alveolar PO₂ = PiO₂ - PaCO₂ · (FiO₂ + (1 - FiO₂/RQ)), where PiO₂ is the inspired PO₂, FiO₂ the fraction of inspired oxygen (room air), and RQ the measured respiratory exchange ratio ($\dot{V}_{CO_2}/\dot{V}_{O_2}$). The dead space/tidal volume ratio (V_D/V_T) was derived by means of the Bohr equation [30]: V_D/V_T = (PaCO₂ - PE_{CO₂})/PaCO₂, where PE_{CO₂} is mixed expired CO₂ pressure.

Statistical analysis

Data are expressed as mean \pm SD. Non-parametric tests were used to analyse differences between groups (Mann-Whitney test) or those intra-group changes induced by exercise (Wilcoxon signed rank test). Linear regression was used when appropriate. Probability values lower than 0.05 were considered significant in all cases.

Results

General findings

Because of the matching process, sex (one female/eight males), age, height, weight and smoking habits were very similar in both groups (table 1). The clinical course was significantly longer in asbestosis (57 \pm 45 vs 9 \pm 14 months, $p < 0.05$), probably suggesting a more rapid and aggressive progression in IPF. Parenchymal radiographic involvement was more prominent in patients with IPF. All but one of them had scores equal to or greater than 2/2, whilst four patients with asbestosis showed scores lower than 2/2 [24]. However, the four asbestosis patients showed mild pleural thickening, which was bilateral in two subjects and unilateral in the two others. BAL fluid included a comparable proportion of neutrophils in both groups (table 1), suggesting a similar degree of mild to moderate alveolitis [1]. The observed values agree with previous reports in each group [23, 32, 33]. The mean percentage of lymphocytes in BAL fluid was within normal limits [1].

Routine lung function data

Five patients with asbestosis had a restrictive ventilatory pattern (two mild, one moderate, two severe), two showed an obstructive ventilatory impairment (one mild, one severe), and the two others a mixed ventilatory defect (one mild, one severe). In contrast, six of the patients with IPF disclosed a moderate and one a mild restrictive pattern. Of the remaining two subjects, one showed a normal

ventilatory capacity and the other a mild mixed defect. As a group, no significant differences were observed in dynamic and static lung volumes, either expressed as absolute or as percentage of predicted values (table 1). However, although differences just failed to reach statistical significance, the asbestosis group tended to show more airflow limitation than the IPF group (table 1). As for DLCOsb, one subject with asbestosis showed normal values, five had a mild defect and two had moderate reductions. Most of them normalized their values when the alveolar volume was taken into account (KCO). In contrast, six patients with IPF showed a severely impaired DLCOsb, whilst only two had a moderate reduction and one a mild defect. DLCOsb did not improve when it was divided by the alveolar volume (KCO) in IPF. As a consequence, patients with IPF presented significantly lower DLCOsb and KCO values than those with asbestosis (table 1).

Exercise test

Results at rest. Heart rate, breathing frequency, minute ventilation (\dot{V}_E), oxygen uptake (\dot{V}_{O_2}) and carbon dioxide production (\dot{V}_{CO_2}) were similar in both groups (table 2). Although some patients had normal resting arterial blood gas values, most of them showed mild to moderate hypoxaemia (below 80 mmHg), widened AaPO₂ (above 20 mmHg) and slight increases in V_D/V_T (above 35%).

Results at symptom-limited exercise. Exercise was stopped whenever the patient complained of dyspnoea, weakness, leg pain and/or showed marked (lower than 80%) ear oximetry desaturation. As expected, heart rate, breathing frequency, \dot{V}_E , \dot{V}_{O_2} and \dot{V}_{CO_2} increased in each group with respect to resting conditions ($p < 0.005$), although both respira-

tory rate and \dot{V}_E during exercise were greater in IPF ($p < 0.025$ and < 0.05 , respectively). It is of interest that \dot{V}_{O_2} during E_{max} was similar in both groups, expressed either as absolute values or percentage of predicted (table 2). In other words, each group performed the same amount of exercise (representing almost 60% of their maximal predicted [31]). However, PaO₂ fell dramatically (from 77 ± 11 to 51 ± 7 mmHg, $p < 0.001$) and AaPO₂ rose (from 31 ± 13 to 60 ± 11 mmHg, $p < 0.001$) during exercise in the IPF group but remained essentially unchanged in the asbestosis group (from 76 ± 11 to 73 ± 21 mmHg and from 32 ± 14 to 35 ± 23 mmHg, respectively). Analysis of individual data in patients with asbestosis showed that AaPO₂ decreased during exercise in two, remained unchanged in four and deteriorated in three. In contrast, exercise induced a deleterious effect in all patients with IPF, in keeping with former studies [1, 14, 26, 34]. On the other hand, subjects with asbestosis were able to lower V_D/V_T with exercise ($p = 0.01$) unlike those with IPF (table 2), a finding also previously reported [1, 14, 26, 34]. Moreover, DLCOsb expressed as percentage of predicted was inversely correlated with AaPO₂ during E_{max} only in IPF ($r = -0.84$, $p < 0.01$) (fig. 1).

Discussion

Our study highlights the fact that the pattern of gas exchange during exercise is different in asbestosis and idiopathic pulmonary fibrosis (IPF), even though both groups were matched for age, sex, height, weight, smoking habits and the severity of resting ventilatory impairment. Patients with IPF showed a dramatic fall in PaO₂ during exercise, whilst those with asbestosis did not. In addition, V_D/V_T fell with exercise only in patients with asbestosis, who also presented a lower increase in minute ventilation

Table 2. - Mean (\pm SD) metabolic, circulatory and gas exchange data for patients with asbestosis and idiopathic pulmonary fibrosis (IPF), at rest and during exercise.

	Rest			Exercise		
	Asbestosis	IPF	p<	Asbestosis	IPF	p<
Heart rate bpm	78 \pm 14	83 \pm 17	-	121 \pm 14 (74 \pm 6%)	123 \pm 7 (75 \pm 11%)	-
Resp. rate bpm	22 \pm 6	21 \pm 6	-	34 \pm 11	46 \pm 11	0.025
\dot{V}_E l·min	11 \pm 2	13 \pm 3	-	37 \pm 8 (68 \pm 34%)	49 \pm 14 (67 \pm 17%)	0.05
\dot{V}_{O_2} ml·min ⁻¹	373 \pm 48	302 \pm 59	-	1074 \pm 373 (56 \pm 20%)	1089 \pm 266 (59 \pm 14%)	-
\dot{V}_{CO_2} ml·min ⁻¹	240 \pm 40	250 \pm 51	-	986 \pm 348	1054 \pm 277	-
arterial pH	7.42 \pm 0.02	7.42 \pm 0.03	-	7.39 \pm 0.04	7.37 \pm 0.06	-
PaO ₂ mmHg	76 \pm 11	77 \pm 11	-	73 \pm 21	51 \pm 7	0.05
PaO ₂ mmHg	37 \pm 3	35 \pm 4	-	38 \pm 3	37 \pm 5	-
AaPO ₂ mmHg	32 \pm 14	31 \pm 13	-	35 \pm 23	60 \pm 11	0.05
V _D /V _T %	47 \pm 12	46 \pm 11	-	39 \pm 10	47 \pm 13	-

P values denote the significance of differences between patients with asbestosis and IPF in each condition (rest/exercise). The significance of changes induced by exercise within each group is not shown. Values between brackets correspond to percentage of maximum predicted during exercise [20].

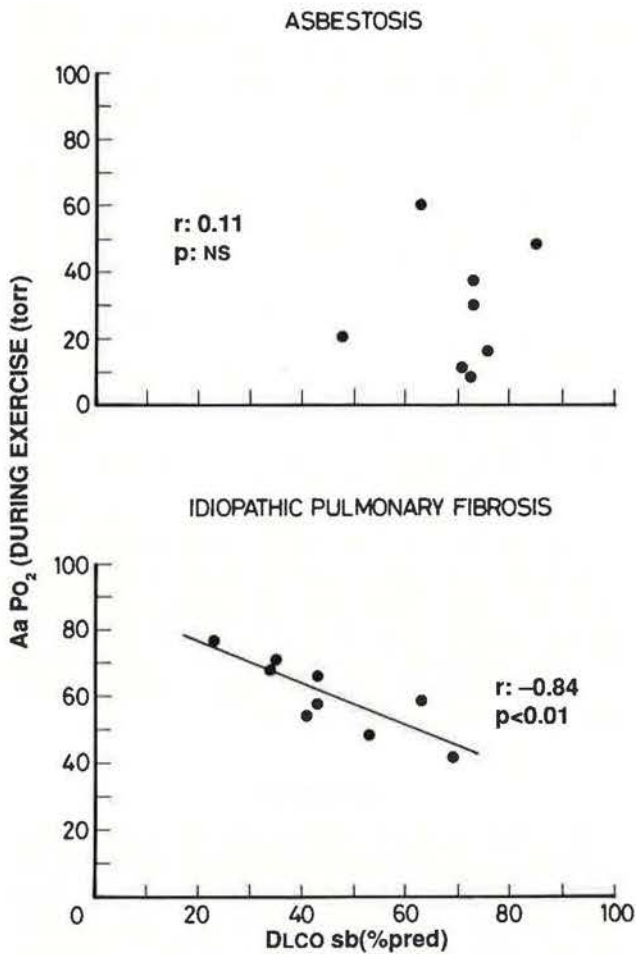


Fig. 1. Relationship between DLCOsb (expressed as percentage predicted) and AaPO₂ (actual values) during maximal exercise in asbestosis (top) and IPF (bottom). In the former individuals, DLCOsb was measured in all but one ($n = 8$).

(table 2). These observations are at variance with the generalized assumption [2, 4, 5] that the response to exercise in asbestosis is similar to that observed in IPF [1]. In 1984 RISK *et al.* [14] included eighteen individuals with asbestosis among a series of 168 patients with different interstitial lung disease, trying to ascertain which resting functional variable might predict gas exchange impairment during exercise. They did not attach specific significance to the variations of AaPO₂ during exercise in patients with asbestosis. However, their plot of AaPO₂ at rest and during exercise also suggests the absence of a consistent deterioration of gas exchange during exercise in asbestosis. To our knowledge, however, our study is the first in which such clear-cut differences between asbestosis and IPF are shown.

Our data also suggest that the different structural derangement observed in asbestosis and IPF may play a key role in their different gas exchange response to exercise. Although we do not have adequate pathologic specimens to substantiate this hypothesis, several morphological and functional findings may support it.

Firstly, it has been postulated that oxygen transfer during Emax in interstitial lung disease is maximally affected by fibrosis and honeycombing and less so by alveolitis [14]. Moreover, AaPO₂ during exercise seems to reflect quite well the pathologist's overall estimate of functional impairment [27]. In keeping with this hypothesis, our patients with IPF showed more abnormal AaPO₂ values during exercise than those with asbestosis, who probably have less architectural derangement (as graded by the ILO/UC X-ray Classification) but the same degree of alveolitis (same percentage of neutrophils in BAL fluid). Therefore, it might well be that the observed differences are somehow related to a less severe interstitial fibrotic stage in asbestosis, an argument which is also in keeping with a more rapid and aggressive progression of the fibrotic process in IPF patients, as shown by their shorter duration of disease (table 1).

Secondly, in contrast to IPF, patients with asbestosis showed the expected [35] decrease in V_D/V_T during exercise. This paradoxical behaviour of V_D/V_T in IPF has previously been related to the pulmonary vascular abnormalities usually seen in this disease [1, 34, 36]. The latter changes might in turn result in hypoxaemia because of alveolar end-capillary oxygen diffusion limitation through its well-known effect upon both mixed venous oxygen tension (P \bar{v} O₂) and the capillary transit time of the red blood cell, especially during exercise [36–38]. Unfortunately, cardiac output was not measured, so that we do not know its role in relation to P \bar{v} O₂ changes, but it is of note that patients with IPF showed a lower DLCOsb, which may well also reflect a lower surface area of pulmonary capillaries.

Thirdly, it has been shown that exposure to asbestos may cause airways disease [15] giving rise to the presence of airflow obstruction [16–18]. In fact, our patients with asbestosis showed a less uniform restrictive ventilatory impairment than those with IPF. Consequently, it may be possible that most of the asbestos patients had ventilation-perfusion (V_A/Q) mismatch at rest, which improved during exercise, as happens in some patients with chronic obstructive pulmonary disease (COPD) [19–21]. This would be consistent with previous exchange respiratory ratio (RQ) measurements obtained in patients with asbestosis [39]. Moreover, it has been shown that IPF frequently shows minimal V_A/Q inequality, both at rest and during exercise [36–38]. Thus, it seems that only those subjects with IPF who incur more resting V_A/Q inequality, sometimes because of coexisting COPD, might exceptionally show an increase in PaO₂ during exercise [27].

Finally, it is of note that DLCOsb was lower in patients with IPF and that it was inversely correlated with AaPO₂ during exercise only in this disorder (fig. 1). Conceivably, this correlation was not evident in asbestosis because factors other than resistance to gas transport across the alveolar-capillary bed, including V_A/Q inequality, also modulate DLCOsb [26]. The

possibility that it might also have emerged in patients with asbestosis, if more subjects with lower DLCOsb had been included, cannot be ruled out. However, an analysis of our individual data shows that only two patients with asbestosis had a DLCOsb lower than 65% of predicted (63 and 48%, respectively) and that P_{aO_2} fell markedly during exercise in the former (from 70 to 49 mmHg) whereas it remained unchanged in the latter (84 to 84 mmHg). In addition, despite the fact that mean DLCOsb was different in each group (table 1), mean $AaPO_2$ was quite close at rest but clearly differed during exercise (table 2). These findings suggest that the mechanisms underlying pulmonary gas exchange in these two disorders may not necessarily be coincidental and that a marked reduction in resting DLCOsb does not need to be a functional marker of arterial desaturation during exercise, at least in asbestosis.

To summarize, the present investigation shows that, in contrast to IPF, pulmonary gas exchange in asbestosis is not systematically impaired during exercise. Conceivably, this may be related to the presence of more airways disease and less pulmonary vascular involvement and/or a lower degree of interstitial fibrotic changes in asbestosis, which in turn may result in a different pattern of \dot{V}_A/\dot{Q} mismatching.

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RÉSUMÉ: Pour l'étude des échanges gazeux pulmonaires au cours de l'effort maximal (Emax) dans l'asbestose, nous avons

comparé 9 sujets atteints de cette affection (1 femme et 8 hommes) âgés de 54 ± 11 ans ($x \pm SD$) à 9 patients (1 femme et 8 hommes) atteints de fibrose pulmonaire idiopathique, d'un âge, d'une taille, d'un poids, et d'une anamnèse de tabagisme similaires, à la fois au repos et au cours de l'effort maximal. L'on n'a observé aucune différence dans les volumes dynamiques et statiques entre les deux groupes. Toutefois, les patients atteints de fibrose pulmonaire idiopathique avaient une DLCOsb et une KCO plus basses ($p < 0.005$ et 0.05 , respectivement). Au repos, les deux groupes ont une hypoxémie artérielle légère (asbestose: 76 ± 11 , vs fibrose pulmonaire 77 ± 11 , mmHg), une augmentation de la ΔaPO_2 (32 ± 14 vs 31 ± 13 mmHg) et de légères augmentations du rapport V_D/V_T (47 ± 12 vs $46 \pm 11\%$). Pendant l'effort maximal, la PaO_2 tombe à 51 ± 7 mmHg chez les patients atteints de fibrose idiopathique, alors que ceux atteints d'asbestose ont une PaO_2 de 73 ± 21 mmHg ($p < 0.05$). D'autre part, les sujets atteints d'asbestose peuvent réduire leur rapport V_D/V_T (de 47 ± 12 à $39 \pm 10\%$, $p = 0.01$), alors que ceux atteints de fibrose idiopathique ne le font pas (46 ± 11 vs $47 \pm 13\%$). D'autre part, la DLCOsb et la ΔaPO_2 au cours de l'effort maximal ne sont en excellente corrélation que dans la fibrose pulmonaire idiopathique ($r = -0.84$, $p < 0.01$). Quoique les deux affections représentent des fibroses pulmonaires diffuses entraînant un degré similaire d'insuffisance ventilatoire au repos, les modifications des échanges gazeux au cours de l'effort y sont différentes. Cette différence pourrait être en relation avec la morphologie sous-jacente à chacun des deux processus, qui entraîne probablement plus de maladies bronchiques et moins d'atteints vasculaires pulmonaires, ainsi que/ou des degrés différents de modifications interstitielles fibreuses dans l'asbestose.