THE EFFECTS OF SILICA DUST EXPOSURE ON SMALL AIRWAYS

JOSÉ ROBERTO DE BRITO JARDIM M.D., Ph.D. • Ericson Bagatin • Luiz Eduardo Nery • Neil Ferreiro Novo • Yara Juliano

ESCOLA PAULISTA DE MEDICINA

Division of Respiratory Medicine and Division of Biostatistics. Rua Botucatu, 740—3º andar, 04023 São Paulo, SP, Brazil

INTRODUCTION

Recently Churg et al[2] have shown that dust-exposed workers may have markedly abnormal small airway pathologic findings. When this group was matched by age, sex and smoking habit to a dust-exposed worker group without pathologic abnormality they concluded that the first group had abnormalities of air flow greater than those induced by smoking alone. In fact we still do not know what role may smoking play on the pathogenesis of silica induced airway changes. Nery et al[7] have very recently shown that smoking and silica exposure may have an addictive effect on pulmonary epithelial permeability of ceramic workers.

The purpose of this investigation was to analyze the small airway function of non smoking silicotic ceramic workers with at least ten years of dust exposure.

MATERIAL AND METHODS

We studied 46 non smoking ceramic workers with the diagnosis of silicosis based on occupational history of silica dust exposure and on radiographic features. Their mean age were 48.3 ± 8.6 years (mean ± standard deviation) and the mean exposure time of the groups was 22.6 ± 5.4 (range from 10 to 40 years). Five patients had tuberculosis in the past and one was bronchitic.

Standard spirometric tests were performed using a 9.0 1 Godart spirometer and measured forced vital capacity (FVCO, forced expiratory volume in one second (FEV_1) and forced expiratory flow at 25-75% (FEF 25-75%); it was calculated the ratio FEV_1/FVC. Forced spirometric curve was repeated 10 minutes after the inhalation of a beta two adrenergic drug (fenoterol—200 mcg) and FVC, FEV_1 and FEF 25-75% were calculated by the isovolume technique.[4]

Maximal expiratory flow volume curves were obtained with a "bag in box" system.[9] The bag dislocation was sensed by a Validyne differential pressure transducer ± 2 cmH_2O and volume was integrated electronically. Curves were registered on a X-Y Tektronics Storage oscilloscope and copied on a plastic card. They were obtained breathing air and then after three inspiratory vital capacities maneuvers of a mixture of 80% of helium and 20% of oxygen (HeO_2).[5] The best fit of three curves for air and HeO_2 were drawn and they were superimposed and matched at residual volume if the vital capacities were unequal. We calculated the flow at 50% and 25% of the curve breathing air ($\dot{V}$max 50% and $\dot{V}$max 25%) and at the volume were both curves had the same flow (Viso$\dot{V}$).[7]

Chest radiograms were classified according to ILO Classification, 1980.[3]

RESULTS

As there was a large variability in the radiological appearances concerning profusion, size and shape of the regular and irregular small opacities we grouped them according to the predominant lesion. We found 25 individuals with 1/1 profusion, 13 with 2/2 profusion and 8 with 3/3 profusion; 37 of them (80%) had a predominance of the p/p small opacities.

Spirometric parameters were considered as normal if the predicted values for FVC and FEV_1 were above 80%, above 60% for FEF 25-75% and above 70% for the ratio FEV_1 /FVC. Thirty five individuals (76.1%) were classified as having normal spirometry and 11 (23.9%) as having some degree of pulmonary impairment[9] predominantly obstructive and 2 restrictive).

The opposite was observed with the analyzis of flow-volume curve and isoflow volume; considering as an abnormal result the presence of at least one altered parameter ($\dot{V}$max 50%, $\dot{V}$max 25% or Viso$\dot{V}$) we observed that the abnormal results (71.7%) predominated over the normal ones (28.3%). These percentages differed significantly from the ones obtained with spirometry ($p < 0.05$).

The analysis of $\dot{V}$max 50%, $\dot{V}$max 25% and Viso$\dot{V}$ in the 33 individuals with normal spirometry show that 24 of them had already some degree of airflow abnormality (Figure 1).

$\dot{V}$max 25% could significantly detect more abnormalities than the other 2 parameters ($p < 0.05$) (Table I).[6]

After 200 mcg of inhaled fenoterol 61.4% of the group (27 out of 44) increased their FEF 25-75% by at least 15% (46.1 ± 36.6% increase); 24 out of 27 increased by 25% or more. Twenty individuals of the responsive group had normal spirometry (74.1%) and showed an increase in FEF 25-75% of 47.2 ± 38.4%, a very similar increase presented by the 7 responsive silicotics with abnormal spirometry, 40.1 ± 30.4% (Table II).

There was no association between the small opacities pro-

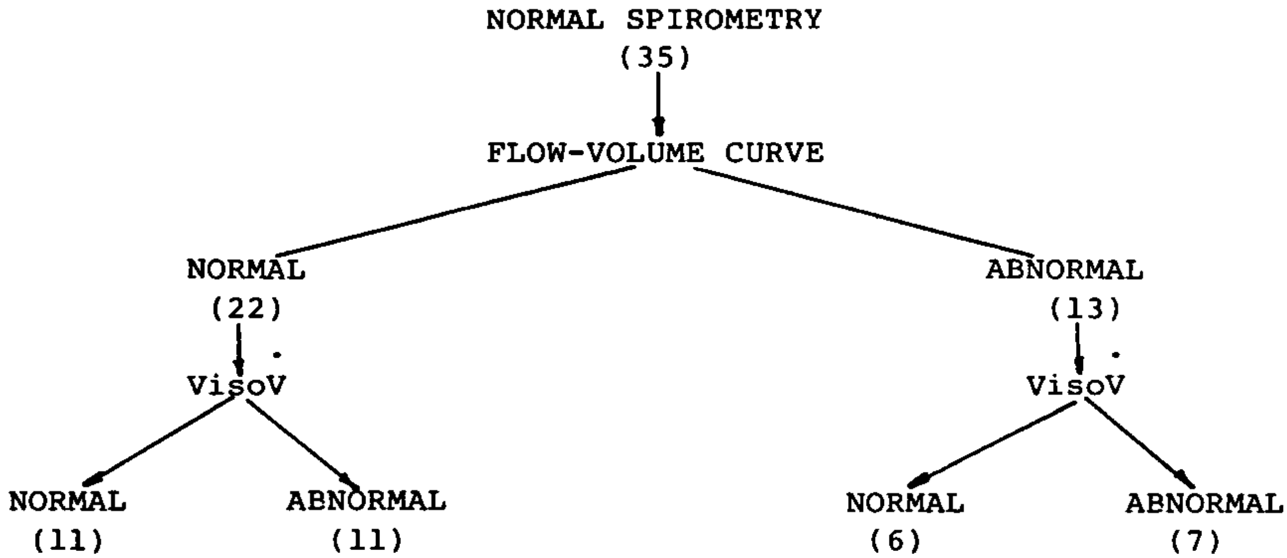

Figure 1. Diagram showing the distribution of the 35 individuals with normal spirometry after they performed a flow volume curve breathing air and helium for the analysis of $\dot{V}$max 50%, $\dot{V}$max 25% and Viso$\dot{V}$.

Table I
Distribution of Normal and Abnormal Values of $\dot{V}$max 50%, $\dot{V}$max 25% and Viso$\dot{V}$ in 46 Individuals Exposed to Dust Silica

	ABNORMAL VALUE	NORMAL VALUE	TOTAL
$\dot{V}$max 50%	12	34	46
$\dot{V}$max 25%	22	24	46
Viso$\dot{V}$	21	25	46

fusion and the response to bronchodilator (Table III) or flow volume-isoflow abnormalities (Table IV).[10]

DISCUSSION

We have shown that flow-volume curves breathing air and a mixture of HeO_2 are able to detect a large number of individuals with airflow obstruction that otherwise would be classified as normals. Isoflow volume was abnormal in 50% of the individuals with normal spirometry while $\dot{V}$max 25% alone would detect 33.3% of them. The combined use of $\dot{V}$max 50%, $\dot{V}$max 25% and Viso$\dot{V}$ were altered in 24 individuals with normal spirometry, a 66% early detection. Despite the fact that Viso$\dot{V}$ has been described for a long time as a tool for early diagnosis of small airway disfunction it has not largely been used in occupational medicine.[7,11]

There seem to be no influence of the length of exposure since the group exposed from 10 to 20 years and the one exposed over 20 years presented very similar Viso$\dot{V}$ values, 11.7% and 12.7%, with 40% of abnormal individuals in each group.

The second point we want to stress is the increased bronchial motor tonus these individuals presented; approximated 60% of the ones with normal spirometry (the same percent for the whole group) responded to inhaled fenoterol with an increase in their FEF 25-75%. There was a slight negative correlation between the initial value and the FEF 25-75%

Table II

Association of Bronchodilator Response (Concerning a 15% Increase in FEF 25–75%) to the Normality or Not of the Spirometric Values

BRONCHODILATOR RESPONSE	SPIROMETRY		
	NORMAL	ABNORMAL	TOTAL
POSITIVE	20 (60.6)	7 (63.6)	27
NEGATIVE	13 (39.4)	4 (36.4)	17
	33	11	44

Table III

Association of FEF 25–75% Increase (Positive Response) to Bronchodilating Drug and Small Opacities Profusion in the Chest X-ray of 44 Non-smoking Silicotics (Non Significant)

	X-RAY PROFUSION			
	1/1	2/2	3/3	TOTAL
POSITIVE	13	9	5	27
NEGATIVE	11	4	2	17
TOTAL	24	13	7	44

Table IV

Association of Altered $\dot{V}$max 50%, $\dot{V}$max 25% or Viso$\dot{V}$ to Small Opacities Profusion in the Chest X-ray of 46 Non-smoking Silicotics (Non Significant)

$\dot{V}$/V and Viso$\dot{V}$	X-RAY PROFUSION			
	1/1	2/2	3/3	TOTAL
NORMAL	18	7	8	33
ABNORMAL	7	6	0	13
TOTAL	25	13	8	46

response (r: −30, $p < 0.05$). As these individuals were not smokers and had no symptoms of asthma we conclude that silica dust is the causal agent of this abnormal response.

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