FILE NAME: Asbestos Cement Pipe and Sheet (ACPS)

DATE: 1977 Aug

DOC#: ACPS031

DOCUMENT DESCRIPTION: Published Article - The Acute Effects of Chrysotile Asbestos Exposure on Lung Function

The Acute Effects of Chrysotile Asbestos Exposure on Lung Function[1]

KEITH W. HARLESS,[2] SUETARO WATANABE, AND ATTILIO D. RENZETTI, JR.[3]

Pulmonary Division of the University of Utah College of Medicine, Salt Lake City, Utah 84132

Received August 8, 1977

ENVIRONMENTAL RESEARCH 16, 360 – 372 (1978)

The Acute Effects of Chrysotile Asbestos Exposure on Lung Function[1]

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Lung function was determined in 23 men 1.5 months after an intense 5-month exposure to chrysotile asbestos using spirometry, plethysmography for determination of lung volumes and specific conductance, single breath nitrogen washout for determination of closing volume, and rest and postexercise arterial blood gas analysis. Although no man showed evidence of restriction, 12 of the 23 had documented airflow obstruction. Eight months following exposure, 16 of the 23 had a repeat pulmonary evaluation. This included the repetition of all previous testing and determination of the diffusion capacity for carbon monoxide and pulmonary compliance. Three men developed airflow obstruction, as determined by the closing volume, during the interval of study. Seventeen of the 23 had airflow obstruction documented on either the initial or follow-up study. Of these 17, 12 were nonsmokers, current light smokers (less than 10 pack years), or ex light smokers. It is concluded that acute intense chrysotile asbestos exposure causes airflow obstruction.

INTRODUCTION

Chronic exposure to asbestos fiber is a well-known health hazard. Such exposure can cause a diffuse interstitial fibrosis (asbestosis) of the lung, carcinoma of the lung and gastrointestinal tract, mesothelioma, plaques, and calcifications of the pleura and asbestos corns of the skin (Becklake, 1976). Although these chronic effects of asbestos are recognized, we are not aware of any reports describing the effects of an acute and limited exposure to asbestos fibers.

This report is concerned with studies of lung function of construction workers immediately following and 8 months after an intense 5-month exposure to chrysotile asbestos fibers. The results indicate that airflow obstruction may be an early manifestation of such exposure.

MATERIALS AND METHODS

Asbestos exposure. The exposure occurred during construction of the Automotive Trades Building at the Utah Technical College in Orem, Utah between December 1975 and May 1976. The building is large, and inside finishing work was being completed during these months. Concrete asbestos Flexboard manufactured by the Johns–Mansville Corp. and containing 30% chrysotile asbestos was being used to cover all inside walls of the building for fire protection. The asbestos Flexboard was cut inside the building with an unventilated portable power saw for

[1] Supported in part by the Parker B. Francis Foundation.

[2] A Parker B. Francis Foundation Fellow in Pulmonary Research. Present address: Bend Memorial Clinic, 1501 Northeast Medical Center Drive, Bend, Oregon.

[3] Author to whom requests for reprints should be sent.

0013-9351/78/0163–0360$02.00/0

installation around electrical outlets, ventilation ducts, rafters, doors, and corners. Cutting occurred throughout the 5 months on most days. The building was kept closed because of the cold winter months.

The concentration of asbestos in the air was not quantitated during the period of exposure. Workers stated it was always dusty, making it difficult at times to see across the building. Some workers reported leaving the building occasionally because of discomfort in breathing the dust. The chips and dust from the cutting were swept into piles periodically. Only one man reported occasionally wearing a protective respirator and observed no other workers doing so.

Immediately following the job closure, air sampling was conducted by the Environment Health Services Branch of the Utah State Division of Health and all workers were advised by their respective employers to be examined by a physician. The method used for the sampling was that stipulated in the Occupational Safety and Health Act (OSHA) (U.S. Occupational Health and Safety Administration, 1972) on 0.8-μm membrane filters. The results of the air sampling activities are summarized in Table 1. Additionally, grab samples of the dust on the floor of the building were analyzed and contained from 1 to 30% asbestos fiber.

The OSHA standard for airborne asbestos exposure is 5.0 fibers greater than 0.5 μm in length per cubic centimeter of air (f/cc) as an 8-hr time-weighted average and a 10 f/cc maximum for any-time exposure. On July 1, 1976, the average limit was reduced to 2.0 f/cc and the maximum exposure standard of 10 f/cc remained the same.

Since the air samples were taken after job closure during simulated conditions (but without the asbestos Flexboard being cut or men actively working), the results in Table 1 are probably underestimates of the actual fiber density encountered by the men.

TABLE 1
ASBESTOS CONCENTRATIONS IN THE AIR AT CONSTRUCTION SITE WHERE RECENT EXPOSURE OCCURRED[a]

Date	Activity during sampling	Minutes of collection	fibers/cc[b]
5-7-76	Sweeping and moving scaffolding	20	3.84
		14	6.18
	Drilling holes in the Flexboard	25	0.57
5-25-76	After all cleaning completed		
	Walking around on inspection	15	0.00
	Slapping Flexboard walls	7	0.64
	During collection of grab samples	74	0.06
	Area sample	78	0.02
6-3-76	During installation of fiberglass insulation	28	0.15

[a] Sampling completed by the Utah State Division of Health.

[b] Occupational Safety and Health Acts Standard is 2 asbestos fibers/cc of air as an 8-hr time-weighted average and 10 fibers/cc as an any-time exposure.

Historical information from employers and from each examined man was used to estimate the duration of each man's exposure to the asbestos. The number of hours worked and exposed to the asbestos per day was multiplied by the number of working days and is reported as total hours for each man. For analysis, the exposures of the men were categorized as light (less than 199 hr), moderate (200 to 499 hr), and heavy (greater than 500 hr). Those men cutting the asbestos Flexboard were considered to have heavy exposure.

Subjects. Approximately 79 men were exposed and 23 of these referred themselves for pulmonary evaluation. Their initial examination occurred an average of 45 days (±21) after their last exposure day. This evaluation included a history, physical examination, pulmonary function testing, and standard chest radiography. Seven months following the initial evaluation, 16 of the original 23 accepted a return invitation. History, physical examination, pulmonary function testing, and chest radiography were repeated with the additional measurements of carbon monoxide diffusing capacity (D_LCO), and static, quasi-static, and dynamic pulmonary compliance.

The demographic data of the 23 workers (all men) initially evaluated are summarized in Table 2. The length of the acute asbestos exposure was variable for individual men. The mean was 518 hr (±344; range, 6 to 896 hr). The median was 623 hr and the mode was 896 hr. Four men had once been exposed to asbestos but considered it to be light exposure, i.e., less than 1 week. None of these four knew the fiber type(s) or density for their prior exposure. Eight men (35%) had never smoked cigarettes. Ten (43%) were smoking at the time of the study but six of these had smoked 10 pack-years or less. Five (22%) were ex-smokers, having smoked 10 pack-years or less in the past (average = 7.4 pack-years; range, 3 to 20; average time after quitting = 8 years; range, 2 to 19 years). Nine men (39%) had no prior history of respiratory symptoms or disease. Seven (30%) had a history of seasonal rhinitis. However, only one had ever consulted a physician because of symptoms and none of the seven had had symptoms for at least 5 months prior to testing. Four (17%) had chronic bronchitis, and three (13%) had a remote history of pneumonia.

Only one man (No. 2) reported having continuous symptoms during the exposure which were related to exacerbation of his asthma. Three others (Nos. 9, 12, and 23) experienced temporary difficulty breathing during their asbestos exposure. One (No. 23) had acute pharyngitis associated with the exposure, causing him to quit work and following which he had a spontaneous recovery.

All men were asymptomatic for acute respiratory infections at the time of their evaluation and had been so for at least the previous 2 months.

Chest radiographs were interpreted by a board-certified radiologist without knowledge of the men's exposure history or lung function.

Pulmonary function studies. The following procedures were done in the seated position:

(1) Spirometric measurements were made with either a 13.5-liter Collins spirometer or a 13.5-liter waterless spirometer manufactured by Cardio–Pulmonary Instruments. The forced vital capacity (*FVC*), forced expired volume in 1 sec (FEV_1), and the flow between 25 and 75% of the *FVC* ($FEF_{25-75\%}$) were measured

TABLE 2

AGE, PHYSICAL CHARACTERISTICS, VOCATION, ASBESTOS EXPOSURE, CIGARETTE SMOKING HABITS, AND PRIOR HISTORY OF RESPIRATORY DISEASE IN 23 MEN WITH RECENT ASBESTOS EXPOSURE

TABLE 2

AGE, PHYSICAL CHARACTERISTICS, VOCATION, ASBESTOS EXPOSURE, CIGARETTE SMOKING HABITS, AND PRIOR HISTORY OF RESPIRATORY DISEASE IN 23 MEN WITH RECENT ASBESTOS EXPOSURE

Subject No.	Identification	Age (yr)	Height (cm)	Weight (kg)	Vocation	Exposure (hr)	Previous exposure Hx.	Cigarette smoking hx. (yr ppd)[a]	Previous respiratory disease or symptoms
1	MB	25	180	81.4	Carpenter	896	No	0	Seasonal rhinitis
2	WC	32	184	75.0	Electrician	896	No	0	Seasonal rhinitis, asthma
3	FC	50	173	84.0	Lather (cutting)	200	No	0	None
4	LF	47	163	69.0	Supervisor	448	No	17 (½)	None
5	CF	32	185	63.2	Electrician	896	No	0	Seasonal rhinitis
6	GG	23	180	90.0	Carpenter	448	No	6 (¾)	None
7	AG	23	183	70.0	Electrician	160	No	0	None
8	DH	39	178	86.0	Electrician	623	No	15 (1½)	Chronic bronchitis
9	JH	34	168	65.0	Electrician	896	No	0	None
10	KK	41	178	96.6	Pipefitter	896	Yes	25 (1½)	Chronic bronchitis
11	WK	39	180	88.0	Welder	712	Yes	25 (1½)	Chronic bronchitis
12	LM	45	185	112.5	Sheet metal worker	896	No	Ex-sm[b]; 19 years[c]; 10 (1)	None
13	KP	23	172	70.0	Electrician	896	No	Ex-sm; 6 years; 7 (¾)	Seasonal rhinitis
14	NS	24	177	83.0	Laborer	70	No	Ex-sm; 7 years; 1 (1)	Seasonal rhinitis
15	DS	39	175	83.2	Sheet metal worker	672	No	0	Pneumonia, 1971
16	MT	35	169	67.7	Carpenter (cutting)	896	Yes	Ex-sm; 6 years; 6 (¼)	Seasonal rhinitis
17	GA	47	180	91.6	Welder	125	No	25 (¾)	None
18	ED	23	189	77.0	Carpenter	672	Yes	10 (1)	None
19	JE	36	170	74.0	Mason	6	No	10 (1)	Chronic bronchitis
20	JM	22	185	83.0	Mason	9	No	Ex-sm, 2 years; 3 (1½)	None
21	BR	28	171	89.1	Laborer	208	No	10 (1)	Seasonal rhinitis; pneumonia, 1974
22	MR	23	183	87.3	Laborer	160	No	0	None
23	AP	26	175	73.9	Electrician	240	No	4 (1)	Seasonal rhinitis, pneumonia, 1963

[a] ppd, packs per day.
[b] Ex-sm = Ex-smoker.
[c] Number of years since stopping smoking.

manually from the spirometric record. The reported data are from the best test based upon the sum of *FVC* and FEV_1 of two or three spirometric records. Spirometric testing was completed before and after administration of 0.68 mg of aerosolized isoetharine with phenylephrine (Bronkometer). Normal values used were from the ITS Manual (Intermountain Thoracic Society, 1975).

(2) Functional residual capacity (*FRC*), airway resistance (*Raw*), and thoracic gas volume (V_{TG}) at which *Raw* was determined were measured by plethysmography according to the methods of DuBois *et al.* (1956a, b) using a constant volume plethysmograph described by Schmidt and Cohn (1961). Specific conductance (*SGaw*) was calculated from *Raw* and V_{TG}.

(3) Total lung capacity (*TLC*) was determined by adding the plethysmographically determined *FRC* to the spirometrically determined inspiratory capacity (*IC*). Normal values used for *FRC* and *TLC* were from the ITS Manual.

(4) Closing volume of the lung (*CV*) was measured by the nitrogen method. The subject exhaled at a constant flow rate, approximately 0.5 liters/sec into a Fleisch No. 3 pneumotachograph after maximal inspiration of 100% oxygen initiated at residual volume (*RV*), the signal of which was displayed on an oscilloscope facing the subject, integrated through a Hewlett–Packard VR 4000 Digital Pneumotach, and recorded on the *x* axis of a Hewlett–Packard 7045A X–Y Recorder. The percentage nitrogen (N_2) was measured at the mouth using an ionizing N_2 meter (Cardio–Pulmonary Instruments Nitrogen Analyzer 410), the output of which was displayed on the *y* axis of the X–Y Recorder. The apparatus dead space between the mouth and oxygen source was about 40 ml. Closing volume was taken as that volume at which a sudden inflection in N_2 concentration above the slope of the alveolar plateau occurred. It was expressed as *CV/VC%*. Closing capacity *(CC)* was obtained by adding residual volume *(RV)* to the *CV* and was expressed as *CC/TLC%*. *RV* was obtained by subtracting the spirometrically determined expiratory reserve volume *(ERV)* from the measured *FRC*. Closing volume tests were administered by the authors and all measurements were checked for accuracy by one of us. Normal values for the *CV/VC%* and the *CC/TLC%* were those of Buist and Ross (1973). At least two satisfactory tracings were obtained, each 5 min apart, and the mean of the CV measurements was taken as the final result.

(5) Arterial blood gases before and immediately after exercise were determined with the Radiometer BMS3 Mk2 Blood Micro System blood gas analyzer. The exercise performed was 1 min of stepping up and down a 20-cm-high stool.

(6) D_LCO was determined using the single-breath technique described by Ogilvie *et al.* (1957). The expired gas sample was collected in a 2-liter bag and analyzed in a Beckman Medical gas analyzer LB-2 for CO. Normal values used were from the ITS Manual.

(7) The pulmonary compliance was measured using an esophageal balloon (length, 10 cm; perimeter, 2.25 cm filled with 0.5 cc of air sealed over a polyethylene catheter, PE 200, i.d. = 1.40 mm) connected to a Statham PM131TC (±2.5 psid) transducer according to the method of Milic-Emili *et al.* (1964) and a Fleisch No. 3 pneumotachograph, whose signal was integrated for volume through a fast-responding integrator (Hewlett–Packard VR 4000). Pressure, volume, and flow were displayed on an oscilloscope and recorded on light-sensitive paper with

the use of elect
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Subject No.	LUNG AND ... TLC^a / TLC_{pr} (%)
Normals	100
1	***
2	*
3	111
4	156
5	**
6	98
7	105
8	139
9	107
10	88
11	123
12	122
13	81
14	107
15	120
16	106
17	132
18	98
19	96
20	107
21	94
22	109
23	116
Average	107
SD	12

[a] Abbreviation
vital capacity; *FI*
and 75% of the *I*
capacity; *CC/TL*
[b] Intermountai
[c] Watanabe *et*
[d] Normal valu
[e] Asterisks (*
[f] N indicates r
[g] Flows impro
[h] An arrow (↑
capacity (%) res

the use of electronics manufactured by Electronics for Medicine. The static expiratory compliance (*Cst*) was measured by a stepwise expiration from *TLC* by a method similar to that of Turner *et al.* (1968). The operator controlled the expiration by periodically closing the airway for 3 sec while the subject relaxed against the closed airway. Measurements were made over a range of 0.750 liter above *FRC*. A quasi-static expiratory compliance was also measured by a slow expira-

TABLE 3
LUNG VOLUMES, SIROMETRY, SPECIFIC CONDUCTANCE, CLOSING VOLUMES, AND CLOSING CAPACITIES IN 23 MEN AFTER RECENT ASBESTOS EXPOSURE

Subject No.	TLC^a / TLC_{pred} (%)	FRC / FRC_{pred} (%)	FVC / FVC_{pred} (%)	FEV_1 / FVC (%)	$FEF_{25-75\%}$ / FVC (%)	$SGaw$ (liters/sec/cm of H_2O/liter)	CV/VC (%)	CC/TLC (%)
Normals	100	100	100	>70[b]	>65[b]	>0.127[c]	*[d]	*[d]
1	**[e]	**	112	81	86	**	N[g]	N
2	**	**	93	70	44	**	N	N
3	111	100	115	83	93	0.127	N	N
4	156	249	100	67	35[f]	0.121	↑[h]	↑
5	**	**	122	83	92	**	N	N
6	98	102	98	80	76	0.225	N	N
7	105	134	98	92	120	0.177	↑	↑
8	139	188	123	70	49[f]	0.107	↑	↑
9	107	130	103	73	53	0.191	N	↑
10	88	100	111	72	52	0.113	↑	↑
11	123	171	122	62	30[f]	0.119	↑	↑
12	122	128	136	73	54	0.210	N	N
13	81	100	79	73	81	0.232	N	N
14	107	107	120	82	80	0.190	N	N
15	120	140	114	75	62	0.205	N	↑
16	106	102	121	68	106	0.239	N	N
17	132	161	107	68	43	0.134	**	**
18	98	125	120	89	110	0.208	N	N
19	96	123	98	76	59	0.190	↑	↑
20	107	137	114	75	59	0.146	N	↑
21	94	90	93	87	112	0.248	N	N
22	109	123	115	79	75	0.161	N	N
23	116	148	118	87	106	0.240	N	N
Average	107	133	111	77	73	0.180	—	—
SD	12	37	13	7	26	0.044	—	—

[a] Abbreviations used: *TLC*, total lung capacity; *FRC*, functional residual capacity; *FVC*, forced vital capacity; FEV_1, forced espired volume in 1 sec; $FEF_{25-75\%}$, average expiratory flow between 25 and 75% of the *FVC*; *SGaw*, specific conductance of the airways; *CV/VC*, closing volume per vital capacity; *CC/TLC*, closing capacity per *TLC*.

[b] Intermountain Thoracic Society, 1975.

[c] Watanabe *et al.*, 1974.

[d] Normal values dependent upon age (Buist and Ross, 1973).

[e] Asterisks (**) indicate study was not completed.

[f] N indicates normal result.

[g] Flows improved at least 25% after administration of aerosolized isoetharine.

[h] An arrow (↑) indicates elevated closing volume/vital capacity (%) or closing capacity/total lung capacity (%) result.

tion from *TLC*. The dynamic compliance (*Cdyn*) was measured by the method of Mead and Whittenberger (1953). Dynamic compliance was measured at 15 and 60 breaths/min ($Cdyn_{15}$ and $Cdyn_{60}$, respectively). The breathing frequencies were maintained voluntarily, being cued by a tape recording or by one of the authors. During the *Cdyn* maneuvers, the subject was instructed to maintain a constant tidal volume and to keep his *FRC* level constant. Static expiratory compliance, *Cst*, *Cqst*, and *Cdyn* were each determined after three slow vital capacity breaths. A mean of at least two different measurements for *Cst* and *Cqst* and eight breaths for *Cdyn* was used to express the final result reported. Static expiratory compliance, *Cqst*, and *Cdyn* were expressed in cubic centimeters per centimeter of H_2O and *Cdyn* was also expressed as a percentage of *Cst* (*Cdyn/Cst%*). Normal values for *Cst* and *Cdyn* used were from Begin *et al.* (1975).

RESULTS

The results of the initial pulmonary function studies are summarized in Table 3. No man had restrictive chest disease, as determined by *FVC*, *FRC*, or *TLC*. Twelve (50%) had airflow obstruction demonstrated. Most of these men had their airflow obstruction documented by two or more tests. All but one of these had an abnormal $FEF_{25-75\%}$. Of the twelve, three were nonsmokers and did not have a history of past respiratory symptoms or disease. Two were light smokers, and three were ex-smokers who smoked lightly in the past. Eleven (40%) did not manifest airflow obstruction, including five smokers, two ex-smokers, and four nonsmokers. There were no significant differences in the hours of asbestos exposure for those with airflow obstruction (528 ± 348 hr) and those without airflow obstruction (507 ± 331 hr). Additionally, a dose response could not be demonstrated in those with airflow obstruction. Seven had had heavy, one had moderate, and four had light asbestos exposure.

The results of the arterial blood gas analysis before and after exercise, in general, showed the group had normal oxygenation of the arterial blood before and after exercise as well as normal ventilation and pH of the arterial blood. Five, who did not have a history of pulmonary disease, had mild hypoxemia at rest. Two of these had no other documented abnormality, whereas these others had airflow obstruction. Five others had a significant decrease in their arterial oxygen tension (PaO_2) after exercise. Of these, three had airflow obstruction and two did not, although the latter two had smoked. Again, a dose–response relationship was not evident.

The results of pulmonary function measurements in the 16 subjects who returned for follow-up study were compared to the initial data given in Table 4. These follow-up studies were conducted an average of 196 days (±23) after the initial examination. The asbestos exposure for this returning group averaged higher (656 ± 290 hr) than that for the seven who did not return (203 ± 209 hr), and this difference in exposure was significant ($P < 0.005$). Although the average change in weight between the initial and follow-up observations did not change significantly, three subjects gained more than 3 kg. It may be seen that the results of measurements of the *FVC*, FEV_1, $FEF_{25-75\%}$, *Raw*, *TLC*, and PaO_2 at rest and after exercise did not change from the first to the second study for the group. Functional residual capacity decreased slightly and *SGaw* increased slightly for

TABLE 4
RESULTS OF PULMONARY FUNCTION MEASUREMENTS AT 1.5 AND 8 MONTHS FOLLOWING ASBESTOS EXPOSURE IN 16 MEN[a]

Time after exposure	Initial measurement at 1.5 months	Follow-up measurement at 8 months
Weight (kg)	80.3 ± 12.6	81.7 ± 13.9
FVC (liters)[b]	5.19 ± 0.75	5.24 ± 0.74
FVC/FVC_{pred} (%)	110 ± 15	111 ± 13
FEV_1 (liters)	3.95 ± 0.51	3.95 ± 0.68
FEV_1/FVC (%)	75 ± 7	75 ± 7
$FEF_{25-75\%}$ (LPS)	3.64 ± 1.45	3.42 ± 1.28
$FEF_{25-75\%}/FVC$ (%)	70 ± 25	66 ± 24
TLC (liters)[c]	7.16 ± 1.18	6.76 ± 1.10
TLC/TLC_{pred} (%)[c]	107 ± 19	106 ± 15
FRC (liters)[c]	3.59 ± 1.02	3.14 ± 0.74
FRC/FRC_{pred} (%)[c]	135 ± 43	114 ± 27
SGaw (liters/sec/ of H_2O/liter)[c]	0.1667 ± 0.0624	0.2005 ± 0.0575
CV/VC (%)	17.11 ± 7.26	21.89 ± 7.96[c,*]
CC/TLC (%)	38.78 ± 8.13	42.25 ± 8.93
PaO_2 at rest (mmHg)[d]	74 ± 8	75 ± 8
PaO_2 with exercise (mmHg)	74 ± 9	76 ± 9

[a] The data are expressed as the mean ± SD.
[b] The abbreviations are the same as those given in Table 3.
[c] Results in 13 men in whom measurements were made.
[d] Arterial oxygen tension.
* $P < 0.05$.

the group, but neither change was significant. The group's average closing volume did show a significant increase from 17.11% ±7.26% to 21.89% ± 7.96% ($P < 0.05$).

The closing volume data are listed in Table 5 for the 16 returning men during their first and follow-up study. Assuming that there is a 2 to 3% variation during the testing and measuring Ducic *et al.*, 1975; McCarthy *et al.*, 1975; Burki *et al.*, 1975) plus a 0.5% increase due to the older age (about 1 year) and that larger changes are significant, seven men had significant increases in their closing volume. Of these, six (86%) either were nonsmokers at the time of both studies or were light smokers. Three (Nos. 8, 12, and 14) had gained 4.0, 6.5, and 6.0 kg, respectively. The others had gained 1 kg or less. Again, a dose response to the asbestos exposure could not be demonstrated.

The results of the D_LCO and lung compliance measurements are listed in Table 6. All values for the D_LCO are within the normal range. All men had normal values for *Cst*. Nine demonstrated frequency dependence of compliance (Table 7), as judged by the results obtained from measurements in normal nonsmoking male subjects of comparable ages (Begin *et al.*, 1975). Four (44%) of these had never smoked and had no prior history of lung disease. One was a smoker but for only 4.5 pack-years, and another was an ex-smoker who had smoked 1 pack-year and had quit 7 years previously.

The radiographic examinations were abnormal in four men (Nos. 4, 11, 17 and 23). All were cigarette smokers and one (No. 23) had a history of possible pleural

TABLE 5
CLOSING VOLUME DATA AT 1.5 AND 8 MONTHS FOLLOWING ASBESTOS EXPOSURE IN 16 MEN

Subject No.	1.5 months (*CV/VC%*)	8 months (*CV/VC%*)	Change[a] (*CV/VC%*)
1	10.52	12.50	1.98
2	11.22	8.50	−2.72
3	12.35	27.80	15.45
4	26.55	27.46	0.91
5	10.86	14.00	3.14
6	4.35	31.60	27.25
7	29.79	15.17	−14.62
8	24.00	25.50	1.50
9	16.55	18.36	1.81
10	24.32	26.50	2.18
11	28.03	38.50	10.47
12	20.77	31.00	10.23
13	13.81	14.45	0.64
14	11.76	18.74	6.98
15	15.85	20.30	4.45
16	13.06	19.86	6.80
Average	17.11	21.89	
SD	7.26	7.96	

[a] The *CV/CV%* measured at 8 months minus that measured at 1.5 months.

and/or parenchymal lung disease. The changes observed were costophrenic angle blunting in the absence of pleural effusion (Nos. 4 and 23), lingular scarring (No. 17), and possible increase of the interstitial markings (No. 11).

DISCUSSION

This study demonstrates a high incidence of airway obstruction in the absence of restrictive defects after an acute 5-month exposure to chrysotile asbestos. The data given in Table 7 show that 17 of 23 men (74%) had airflow obstruction demonstrated at either the initial or follow-up study. All but two of these had their obstruction documented by more than one test. Also documented in Table 7 are the interrelationships among the presence of airway obstruction, the intensity of asbestos exposure, and the incidence of other potential causes of obstruction in this group, namely, cigarette smoking and intrinsic pulmonary disease. In six (35%) of those men with obstruction no possible cause other than asbestos exposure could be identified.

Laboratory investigation by Burrows *et al.* (1977) and clinical observations suggest that less than 20 pack-years of smoking would rarely give rise to airflow obstruction. Using this criterion, 12 (71%) of the subjects in this study would be left with asbestos exposure as the probable major determinant of their airway obstruction. No obvious correlation exists between the intensity of asbestos exposure and the occurrence of obstruction (Table 7). However, it must be recognized that the estimates of intensity of exposure are very crude. Finally, since the

TABLE 6
CARBON MONOXIDE DIFFUSING CAPACITY AND PULMONARY COMPLIANCE MEASUREMENTS IN 16 MEN WITH RECENT ASBESTOS EXPOSURE

Subject No.	D_LCO[a] (ml CO/min/mmHg)	D_LCO (% pred)	*Cst* (cc/cm H_2O)	$Cdyn_{15}$ (cc/cm H_2O)	$Cdyn_{15}/Cst$ (%)	$Cdyn_{60}$ (cc/cm H_2O)	$Cdyn_{60}/Cst$ (%)
1	46.2	138	384.6	227.7	59	214.6	56
2	40.2	119	263.6	206.9	78	192.0	73
3	37.7	155	295.1	208.4	71	185.2	63
4	40.5	190	237.9	201.9	85	151.3	64
5	45.2	132	344.5[b]	228.5	66	194.0	56
6	36.8	106	337.1[b]	226.3	67	195.7	58
7	38.3	106	230.8	167.9	73	133.0	58
8	36.7	127	340.9	194.8	57	*	*
9	48.5	179	234.4	180.8	77	170.9	73
10	37.1	128	358.3	232.4	65	173.4	48
11	24.8	84	344.9	258.6	74	202.5	59
12	46.7	153	312.5	200.2	64	240.8	77
13	25.9	81	178.7	184.8	103	155.0	86
14	39.8	124	260.3	207.2	80	150.9	58
15	35.4	123	315.9	189.8	60	194.5	62
16	48.4	176	234.8	239.8	102	189.8	81
Average	39.3	133	292.1	209.8	74	186.9	64
SD	6.8	31	55.6	23.4	11	30.2	10

[a] Abbreviations used: D_LCO, diffusing capacity of carbon monoxide; *Cst*, static pulmonary compliance; $Cdyn_{15}$, pulmonary compliance at 15 breaths/min; $Cdyn_{60}$, pulmonary compliance at 60 breaths/min.

[b] Inadequate tracing to measure *Cst* and *Cqst* is reported.

An asterisk ( ) indicates inadequate tracing to measure results.

TABLE 7

AIRFLOW OBSTRUCTION IN RELATION TO SMOKING HISTORY, PULMONARY DISEASE, AND INTENSITY OF ASBESTOS EXPOSURE IN 23 MEN

	Smoking history (pack-years)	Active lung disease	Exposure history	$FEV_1/FVC\%$	$FEF_{25-75}/FVC\%$	$CV/VC\%$	*Cdyn.*	Hypoxemia	Airflow obstruction
1	0	–	H[a]	N[b](N)[c]	N(N)	N(N)	+[d]	–	Yes
2	0	Asthma	H	N(N)	↓[b](↓)	N(N)	–	–	Yes
3	0	–	H	N(N)	N(N)	N(↑[b])	–	++[e]	Yes
4	8	–	M	↓(↓)	↓(↓)	↑(↑)	–	–	Yes
5	0	–	H	N(N)	N(N)	N(N)	+	++	Yes
6	4	–	M	N(N)	N(N)	N(↑)	+	++	Yes
7	0	–	L	N(N)	N(N)	↑(N)	+	++	Yes
8	22	Bronchitis	H	N(N)	↓(↓)	↑(↑)	+	++	Yes
9	0	–	H	N(N)	↓(↓)	N(N)	–	–	Yes
10	37	Bronchitis	H	N(N)	↓(↓)	↑(↑)	+	–	Yes
11	37	Bronchitis	H	↓(↓)	↓(↓)	↑(↑)	+	++	Yes
12	Ex-sm[f]; 10	–	H	N(N)	↓(↓)	N(↑)	–	–	Yes
13	Ex-sm; 5	–	H	N(N)	N(N)	N(N)	–	–	No
14	Ex-sm; 1	–	L	N(N)	N(N)	N(↑)	+	–	Yes
15	0	–	H	N(N)	↓(↓)	N(N)	+	++	Yes
16	Ex-sm; 6	–	H	N(N)	N(N)	N(N)	–	–	No
17	17	–	L	↓	↓	*[g]	*	++	Yes
18	10	–	M	N	N	N	*	++	No
19	10	Bronchitis	L	N	↓	↑	*	–	Yes
20	Ex-sm; 2	–	L	N	↓	N	*	++	Yes
21	10	–	M	N	N	N	*	–	No
22	0	–	L	N	N	N	*	–	No
23	4	–	M	N	N	N	*	–	No

[a] Abbreviations used: H, heavy asbestos exposure, M, moderate; L, light.

[b] Abbreviations used: N, normal result; ↑ and ↓, indicate results diagnostic of airflow obstruction.

[c] The parentheses contain results of the follow-up study in the 16 returning men.

[d] A plus (+) indicates frequency dependence of pulmonary compliance was documented.

[e] Two pluses (++) indicate that PaO_2 was abnormal at rest or fell below 5 mmHg after exercise.

[f] Ex-sm, ex-smoker.

[g] An asterisk (*) indicates study not completed.

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pulmonary function test abnormalities observed most commonly were in the measurements of the $FEF_{25-75\%}$, closing volume, and frequency dependence of compliance, it seems likely that the site of airflow obstruction in this group is in the small peripheral airways.

The finding of airflow obstruction is consistent with other published data. Jodoin *et al.* (1971) reported on the early effects of asbestos exposure on lung function and state that their findings support the possibility that asbestos dust is another cause of obstructive disease of the small airways. Becklake *et al.* (1976), in reviewing the published pulmonary function data obtained during the evaluation of patients with asbestosis, have suggested airflow obstruction may occur in some cases. Decreased airway conductance has been documented by Ostrow and Cherniack (1973) in interstitial lung diseases of other etiologies. Hourihane and McCaughey (1966) reported that the basic lesion in asbestosis is a peribronchial fibrosis which obliterates surrounding alveoli. These clinical and pathological data can be correlated with the findings of Peress *et al.* (1976), suggesting that chrysotile asbestos dust, representative of environmental dust, had the small airways as one of the target sites as based upon closing volume data. Although other investigators (Murphy *et al.*, 1972) have shown in a study population of shipyard workers exposed to low concentrations of asbestos fiber that airflow obstruction was no more common than a matched control population, it is not clear what a higher dose would do.

Of the 10 men who had either arterial hypoxemia at rest or an exercise-induced fall in arterial PaO_2, all but one had airflow obstruction. Furthermore, the D_LCO was normal in all subjects in whom it was measured. Thus, there is no evidence that a defect in alveolar capillary gas diffusion played a role in the gas exchange abnormalities observed.

The absence of any evidence of a restrictive defect in this group of subjects is not surprising when one considers the short interval between exposure and study. Such defects occur when there is pathologic and/or radiologic evidence of fibrosis. The few radiographic abnormalities noted in this study could not be related to the asbestos exposure.

Since the exposure encountered by these men probably exceeded OSHA standard, we cannot predict the effects of an acute exposure that is less than the standard level. The OSHA standard is, however, intended as a protection against asbestosis and not necessarily malignancy. We would expect that these men may risk development of neoplastic disease in the future. We conclude that acute exposure to chrysotile asbestos dust causes airflow obstruction as early as 1.5 months after exposure.

ACKNOWLEDGMENTS

The authors wish to express their appreciation to Dr. John Armstrong, M.D., Associate Professor of Radiology, University of Utah College of Medicine, Salt Lake City, Utah for interpreting the chest radiographs; Alan G. Barbour, M.D., Bureau of Disease Prevention, Utah State Division of Health for his referral of the workers studied; Mr. Jeff Throckmorton, Public Health Engineer, Utah State Division of Health for conducting the air sampling at the construction site; Ms. Angela Devito for her technical assistance; and Ms. Barbara Parker and Ms. Polly Detton for typing the manuscript.

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