LUNG MECHANICS IN COAL WORKERS' PNEUMOCONIOSIS

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Coal workers' pneumoconiosis (CWP) is a condition caused by the inhalation and deposition of coal dust in the regions of the lungs concerned with gas exchange. In the early stages, it is characterized pathologically by the accumulation of dust around the first and second order respiratory bronchioles, a little surrounding fibrosis, and some dilatation of the respiratory bronchioles. This latter lesion is referred to as focal emphysema.[1] In approximately 20% of subjects with simple CWP, large aggregates of fibrous tissue develop, usually in the upper lobes.[2] When this change occurs, the disease is then known as complicated pneumoconiosis or progressive massive fibrosis (PMF). Simple pneumoconiosis produces relatively little ventilatory impairment, whereas the complicated form may lead to severe restriction of ventilatory capacity and *cor pulmonale*.[3]

Dyspnea occurs frequently in coal miners and is often related to the presence of chronic obstructive airway disease that is detectable by standard spirometric tests. There is generally poor correlation, however, between this symptom and the radiological category of simple pneumoconiosis. A somewhat better correlation exists between dyspnea and the presence of complicated pneumoconiosis.[4] Studies of gas exchange may demonstrate abnormalities among miners with simple CWP,[3] and there is evidence that an increased residual volume may occur in miners with simple CWP even in the absence of large airway obstruction.[5]

Most studies of the mechanical properties of the lungs have been performed on miners who are seeking medical care or industrial compensation and thus suffer from selection biases often found in such populations.[6, 7] The present study was designed to investigate the mechanical properties of the lungs in working coal miners in order to determine what abnormalities result from simple CWP. In particular, we hoped to determine whether the elastic properties of the lung might be altered by either the small amount of fibrosis or the focal emphysema that are found in this condition. An attempt was also made to determine whether resistance in the small airways was increased in the absence of significant large airway obstruction.

SUBJECTS AND METHODS *

Twenty-five working bituminous coal miners were studied. They were selected from three mines located in southwestern Pennsylvania and northern West Virginia. The mines were among the 31 selected as part of an epidemiological study conducted by personnel of the Appalachian Laboratory for Occupational Respiratory Diseases to determine the prevalence and progression of coal workers' pneumoconiosis in the United States. All the participants had radiological evidence of at least category 2/1 or greater simple pneumoconiosis of either the pinhead (p) or micronodular (q) type of small, regular opacities.[8] All were either nonsmokers or had not smoked for the last ten years. Of the 11 exsmokers, two had been heavy (more than 20 pack-years), five had been moderate (10 to 20 pack-years), and four had been light (less than ten pack-years) smokers. Fourteen were lifelong nonsmokers. None had a one-second forced expiratory volume to forced vital capacity (FEV_1/FVC) ratio of less than 70%. Their ages ranged from 42 to 65, with a mean of 55 years.

Six males in the same age range (41 to 61, mean 54 years) served as the control group. They were members of the teaching staff of the medical school. All were nonsmokers, and none suffered from cardiopulmonary disease. Each of the control subjects underwent the same tests of lung mechanics as the coal miners.

Spirometry was performed utilizing a waterless, high fidelity spirometer and recording system described previously.[9] Flow-volume (FV) curves of three forced vital capacity (FVC) maneuvers were recorded, following two practice trials. The following measurements, corrected to body temperature pressure saturated (BTPS), were obtained from the largest of the FV curves: forced vital capacity (FVC), one-second forced expiratory volume (FEV_1), peak flow (PF), maximal expiratory flows at 25% of FVC exhaled ($\dot{V}_{max\ 25\%}$), at 50% FVC exhaled ($\dot{V}_{max\ 50\%}$), and at 75% FVC exhaled ($\dot{V}_{max\ 75\%}$).

Lung volumes and airway resistance were measured in a constant volume plethysmograph by the methods of Dubois and colleagues.[10, 11] The normal values for total lung capacity (TLC) were those predicted by the short formulae of Needham and colleagues.[12] Esophageal pressure was measured by a 10 cm latex balloon attached by polyethylene tubing to a pressure transducer (Statham PM131TC). The balloon was positioned in the lower third of the esophagus, with the tip approximately 42 cm from the nares.[13] Pressure at the mouth was electronically subtracted from the esophageal pressure to obtain transpulmonary pressure.

Pressure-volume curves were photographically recorded as X-Y plots on an Electronics for Medicine † multi-channel recorder. Lung compliance was measured during static conditions from the linear portion, just above the resting end-expiratory volume, of the expiratory limb of the pressure-volume curve obtained over the full vital capacity. Lung recoil pressure at total lung capacity was also obtained from this curve.

Dynamic compliance was measured at respiratory frequencies of approximately 15, 30, and 60 breaths per minute. Care was taken that the tidal volume remained relatively constant and that there was no change in end-expiratory volume at the faster frequencies. The subjects inspired maximally before each maneuver. Transpulmonary pressure was recorded as noted above. Volume was obtained by electrical integration of airflow at the mouth via a Fleisch No. 3 heated pneumotachograph connected to an appropriate

* Mention of commercial concerns or products does not constitute endorsement by the United States Public Health Service.

† White Plains, N.Y.

pressure transducer (Statham PM97). The transducer recording transpulmonary pressure showed a linear response over the range from 0 to 30 Hz with no appreciable phase shift. The pneumotachograph-pressure transducer used to record airflow at the mouth, and by electrical integration volume, showed a linear response from 0 to 15 Hz with no appreciable phase shift. Dynamic compliance was calculated from the change in volume, divided by the corresponding change in transpulmonary pressure at points of zero flow over the course of at least eight successive respiratory cycles for each breathing frequency. The results reported are the mean of both inspiratory and expiratory values. Oscillations in the transpulmonary pressure tracing due to cardiac impulses were smoothed out by eye where necessary before measurement. All tests for statistical reliability were made using the 95% confidence interval.

TABLE 1

CHARACTERISTICS OF THE SUBJECTS

	Controls	Miners
Number of subjects	6	25
Age (years)	54 (2.8) *	55 (1.0)
Height (cm)	174 (4.0)	175 (2.0)
Years underground (years)	0	33 (1.6)
Cigarette smoking (pack-years)	0	13 (1.9)
Total lung capacity (% predicted)	99 (4.5)	94 (2.2)
$FEV_1/FVC \times 100$ (%) †	81 (1.6)	76 (1.2)
Residual volume (% predicted)	72 (10.6)	76 (4.6)
Airway resistance (cm $H_2O \times$ liters$^{-1} \times$ second^{-1})	1.3 (0.2)	1.7 (0.1)
Lung recoil at TLC (cm H_2O)	25 (3.3)	27 (1.2)
Static compliance (liters $\times$ cm H_2O^{-1})	0.27 (0.02)	0.24 (0.01)
Dynamic compliance (liters $\times$ cm H_2O^{-1})		
at 15 breaths per minute	0.20 (0.04)	0.24 (0.01)
at 30 breaths per minute	0.21 (0.03)	0.19 (0.01)
at 60 breaths per minute	0.20 (0.03)	0.16 (0.01)

* Standard error of the mean.
† FEV_1=one second forced expiratory volume; FVC=forced vital capacity.

RESULTS

The mean values for age, height, years worked underground, cigarette smoking, lung volumes, and mechanics for the controls and the miners are shown in TABLE 1. The miners had a slightly lower total lung capacity and FEV_1/FVC ratio when compared to the controls who were the same age and height. The miners also demonstrated a slightly increased mean residual volume and airway resistance compared to the controls. Static compliance was somewhat higher among the controls than the miners, whereas lung recoil pressure at TLC was higher among the miners than the controls. The miners tended to show a decrease in dynamic compliance at faster respiratory frequencies, whereas the controls showed no change.

The mean values for maximal expiratory flows, static recoil pressure ($P_{L\,(St)}$), and the lung volumes at which these occurred are presented in TABLE 2. In order to correct for differences among subjects, lung volumes are expressed as % of observed total lung capacity (% TLC).

FIGURE 1 shows the mean values for maximal expiratory flows ($\dot{V}_{max}$) versus the lung volumes (% TLC) at which they occurred. As a group, the miners were unable to achieve flows as high as the control subjects at comparable lung volumes. These differences were significant for all except peak flow.

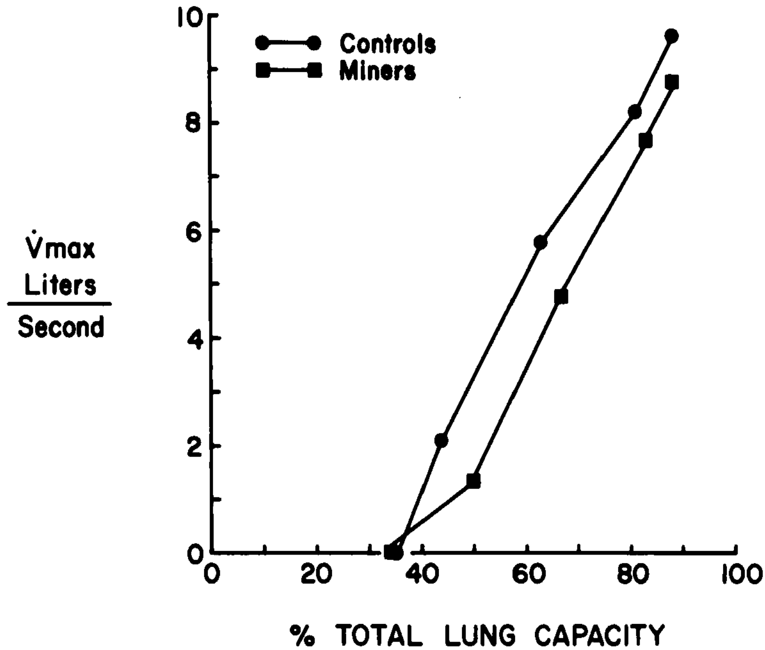

FIGURE 1. Flow-volume curves for controls and miners. Mean values for maximal expiratory flows ($\dot{V}_{max}$) at peak and at 25, 50, and 75% of FVC exhaled are plotted versus lung volumes (% TLC) at which they occurred.

FIGURE 2 shows the mean pressure-volume curves for the controls and the miners. The curve for the miners demonstrates a slight reduction in static recoil pressure at comparable lung volumes. In FIGURE 3, $\dot{V}_{max}$ is plotted versus static recoil pressure ($P_{L\,(St)}$). This maneuver corrects $\dot{V}_{max}$ for changes in $P_{L\,(St)}$ and demonstrates the role of airway obstruction in reducing $\dot{V}_{max}$. When compared by this analysis to control subjects, miners appear to have reduced $\dot{V}_{max}$ largely because of airway obstruction.

In order better to understand the complex interplay of such factors as bronchitis, the varying types of pneumoconiosis, and the possible role of small airway obstruction, the group of coal miners was analyzed in terms of these factors. Pressure, flow, and volume relationships for the controls com-

TABLE 2

PRESSURE-FLOW-VOLUME DATA IN CONTROLS AND MINERS

Subjects	Peak Flow (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 25% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 50% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 75% (Liters/Sec)	Volume (% TLC)
Controls	9.6	88	8.3	81	5.8	63	2.1	44
(SEE) *	(0.7)	(2.3)	(0.4)	(0.4)	(0.5)	(0.9)	(0.4)	(1.4)
Miners	8.8	88	7.7	83	4.8	67	1.4	50
(SEE)	(0.4)	(1.0)	(0.3)	(0.4)	(0.3)	(0.6)	(0.1)	(1.0)

	Transpulmonary Pressure									
	2 cm H_2O		4 cm H_2O		6 cm H_2O		8 cm H_2O		10 cm H_2O	
Subjects	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)
Controls	0.3	35	1.7	40	4.4	54	5.8	65	7.3	73
(SEE)	(0.1)	(4.2)	(1.0)	(5.1)	(2.1)	(5.3)	(2.7)	(5.1)	(3.3)	(4.3)
Miners	0.4	39	1.4	50	2.8	57	4.8	73	6.6	76
(SEE)	(0.2)	(2.3)	(0.3)	(1.8)	(0.4)	(2.6)	(0.5)	(3.8)	(0.5)	(1.9)

* Standard error of the estimate.

pared to the miners with and without symptoms of bronchitis are shown in TABLE 3. Both miners with and without symptoms of bronchitis achieved a lower mean $\dot{V}_{max}$ at all lung volumes than the controls (FIGURE 4). These differences were significant for all $\dot{V}_{max}$ save peak flow. Mean $\dot{V}_{max}$ was not significantly different between the miners with symptoms of bronchitis and those without symptoms.

The pressure-volume curve (FIGURE 5) for the miners with symptoms of bronchitis showed some tendency for a reduction in static recoil pressure, whereas the curve for the miners without bronchitic symptoms tended to

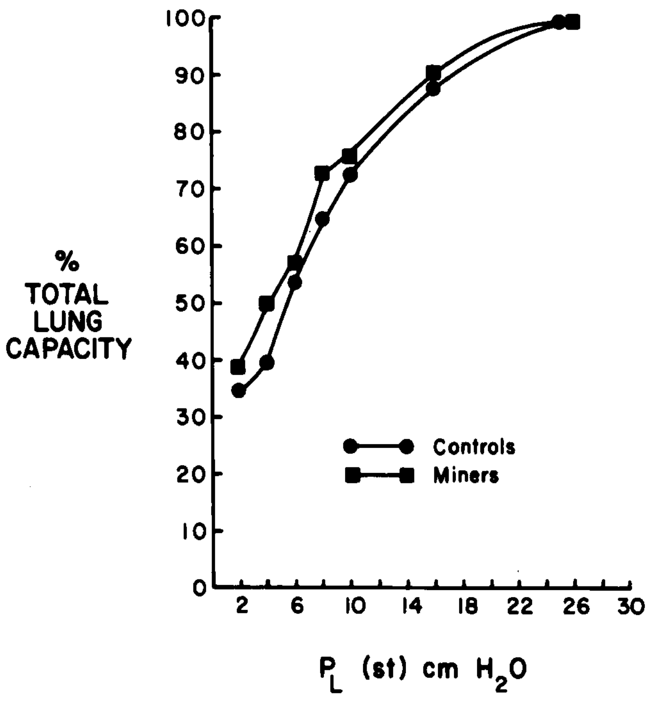

FIGURE 2. Pressure-volume curves for controls and miners. Mean values for static recoil pressure ($P_{L(st)}$) are plotted versus lung volumes (% TLC) at which they occurred.

follow that for the control subjects. The plot of $\dot{V}_{max}$ versus $P_{L(St)}$ (FIGURE 6), that corrects for loss of static recoil pressure, indicates that the miners without symptoms of bronchitis had the lowest $\dot{V}_{max}$. Miners with bronchitic symptoms had a mean $\dot{V}_{max}$ at all lung volumes that was intermediate between the controls and miners without symptoms.

The mean values for $\dot{V}_{max}$, lung volume (% TLC), and $P_{L(St)}$ for the control subjects and the miners grouped according to whether they demonstrated a fall in dynamic compliance (C dyn) with faster respiratory frequencies are shown in TABLE 4. Seventeen of the miners had C dyn that fell, eight dem-

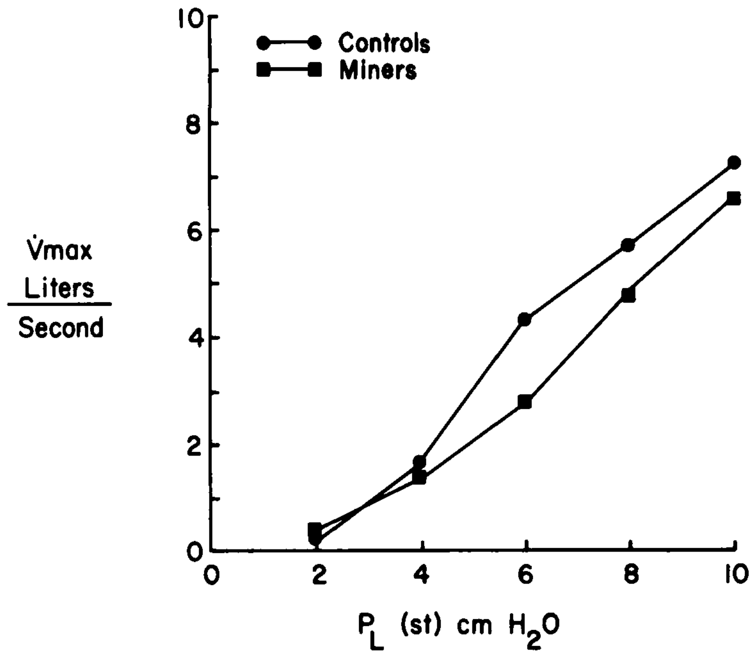

FIGURE 3. Pressure-flow curves for controls and miners. Mean values for maximal expiratory flows ($\dot{V}_{max}$) are plotted versus static recoil pressures ($P_{L(st)}$) at which they occurred.

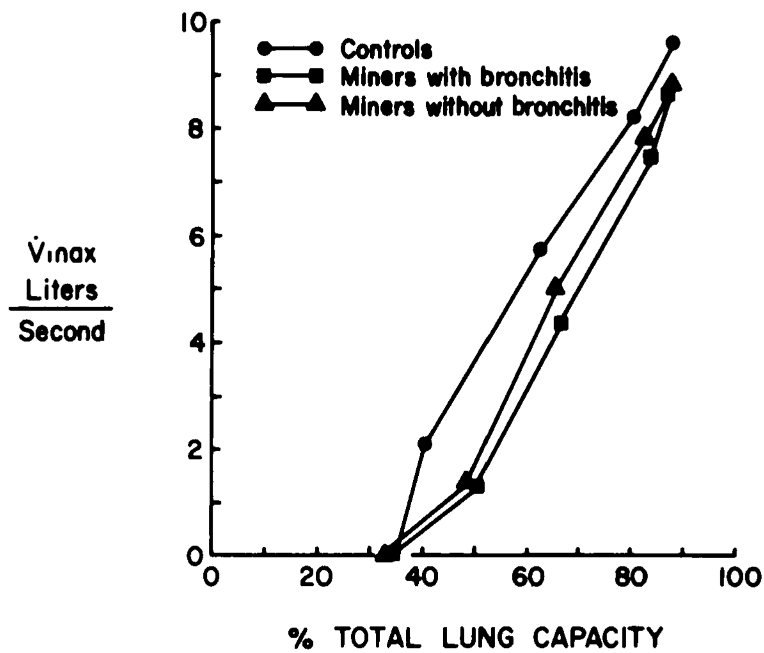

FIGURE 4. Flow-volume curves for controls, miners without bronchitis and miners with bronchitis. Mean values for maximal expiratory flows ($\dot{V}_{max}$) at peak and at 25, 50, and 75% of FVC exhaled are plotted versus lung volumes (% TLC) at which they occurred.

TABLE 3

PRESSURE-FLOW-VOLUME DATA IN CONTROLS, MINERS WITH BRONCHITIS, AND MINERS WITHOUT BRONCHITIS

Subjects	Peak Flow (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 25% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 50% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 75% (Liters/Sec)	Volume (% TLC)
Controls	9.6	88	8.3	81	5.8	63	2.1	44
(SEE) *	(1.8)	(5.7)	(1.1)	(1.0)	(1.2)	(2.3)	(1.0)	(3.5)
Miners with bronchitis	8.9	88	7.9	83	5.0	66	1.4	49
(SEE)	(2.1)	(4.4)	(1.8)	(1.6)	(1.5)	(3.0)	(0.7)	(4.4)
Miners without bronchitis	8.7	87	7.5	84	4.4	67	1.3	51
(SEE)	(1.7)	(6.1)	(1.3)	(2.1)	(1.6)	(3.6)	(0.5)	(5.8)

	Transpulmonary Pressure									
	2 cm H_2O		4 cm H_2O		6 cm H_2O		8 cm H_2O		10 cm H_2O	
Subjects	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)
Controls	0.3	35	1.7	40	4.4	54	5.8	65	7.3	73
(SEE)	(0.1)	(4.2)	(1.0)	(5.1)	(2.1)	(5.3)	(2.7)	(5.1)	(3.3)	(4.3)
Miners with bronchitis	0.4	44	1.8	53	3.0	60	5.4	71	6.7	76
(SEE)	(0.2)	(0.6)	(0.6)	(2.9)	(0.8)	(4.9)	(0.7)	(3.7)	(0.5)	(2.3)
Miners without bronchitis	0.4	38	1.4	48	2.6	55	4.5	65	6.6	75
(SEE)	(0.2)	(3.2)	(0.4)	(2.1)	(0.5)	(2.8)	(0.6)	(2.5)	(0.7)	(2.4)

* Standard error of the estimate.

TABLE 4

PRESSURE-FLOW-VOLUME DATA IN CONTROLS, MINERS WITH FALL IN C DYN, AND MINERS WITH NO FALL IN C DYN

Subjects	Peak Flow (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 25% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 50% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 75% (Liters/Sec)	Volume (% TLC)
Controls	9.6	88	8.3	81	5.8	63	2.1	44
(SEE) *	(1.8)	(5.7)	(1.1)	(1.0)	(1.2)	(2.3)	(1.0)	(3.5)
Miners with fall in C dyn	8.9	87	7.8	83	5.1	66	1.4	49
(SEE)	(0.5)	(1.3)	(0.3)	(0.5)	(0.4)	(0.8)	(0.2)	(1.3)
Miners with no fall in C dyn	8.6	88	7.4	84	4.2	68	1.3	51
(SEE)	(0.8)	(1.6)	(0.7)	(0.6)	(0.4)	(1.0)	(0.2)	(1.6)

	Transpulmonary Pressure									
	2 cm H_2O		4 cm H_2O		6 cm H_2O		8 cm H_2O		10 cm H_2O	
Subjects	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)	Flow (Liters/Sec)	Volume (% TLC)
Controls	0.3	35	1.7	40	4.4	54	5.8	65	7.3	73
(SEE)	(0.1)	(4.2)	(1.0)	(5.1)	(2.1)	(5.3)	(2.7)	(5.1)	(3.3)	(4.3)
Miners with fall in C dyn	0.2	36	1.1	46	2.2	53	4.2	63	6.2	74
(SEE)	(0.03)	(2.7)	(0.4)	(1.9)	(0.5)	(2.9)	(0.5)	(2.3)	(0.7)	(2.1)
Miners with no fall in C dyn	0.7	44	2.0	55	3.8	64	6.0	75	6.9	80
(SEE)	(0.3)	(2.7)	(0.5)	(2.2)	(0.8)	(3.7)	(0.9)	(2.9)	(1.1)	(2.8)

* Standard error of the estimate.

onstrated no appreciable fall, and none of the six controls showed any significant fall in C dyn. Both groups of miners achieved a significantly lower mean $\dot{V}_{max}$, except for peak flow, than did the control subjects at all comparable lung volumes (FIGURE 7). Interestingly, the miners with no fall in C dyn had a lower mean $\dot{V}_{max}$ than the miners with a fall in C dyn. Differences in $\dot{V}_{max}$ between the miners with and without a fall in C dyn were not significant.

The pressure-volume plot of these data (FIGURE 8) shows that the miners with no fall in C dyn had a reduced mean $P_{L(St)}$, whereas the miners with a fall in C dyn had a normal $P_{L(St)}$ at lung volumes comparable to the

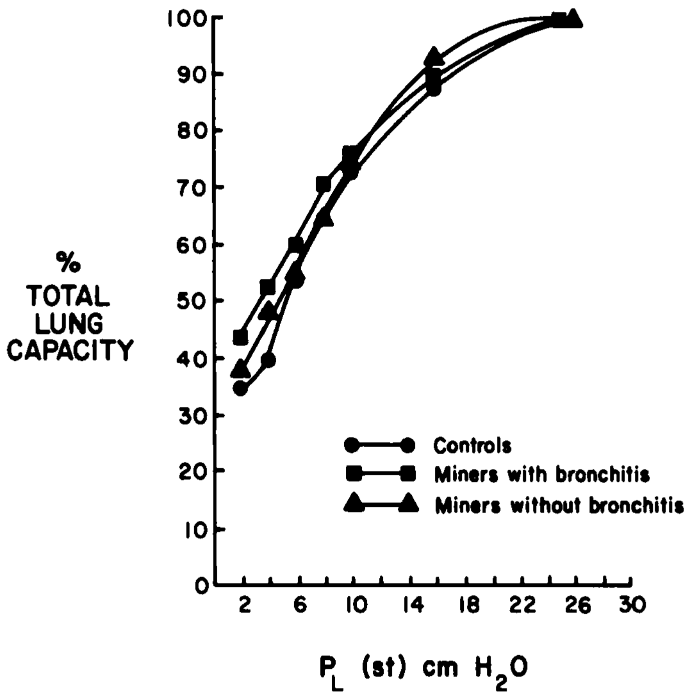

FIGURE 5. Pressure-volume curves for controls, miners without bronchitis and miners with bronchitis. Mean values for static recoil pressures ($P_{L(St)}$) are plotted versus lung volumes (% TLC) at which they occurred.

control subjects. The plot of $\dot{V}_{max}$ versus $P_{L(St)}$ (FIGURE 9), that corrects for loss of recoil pressure, shows that the miners with a fall in C dyn achieved a lower mean $\dot{V}_{max}$ than either the miners with no fall or the control subjects.

Lastly, the miners were grouped according to the type of small opacities present on their chest radiographs. Ten had predominantly pinhead (p), and 15 had predominantly micronodular (q) opacities. The mean $\dot{V}_{max}$, $P_{L(St)}$, and lung volumes (% TLC) at which these occurred for the controls, miners with p opacities, and miners with q opacities are listed in TABLE 5. The miners with either p or q opacities achieved a mean $\dot{V}_{max}$ that was

TABLE 5

PRESSURE-FLOW-VOLUME DATA IN CONTROLS, MINERS WITH "p" OPACITIES, AND MINERS WITH "q" OPACITIES

Subjects	Peak Flow (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 25% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 50% (Liters/Sec)	Volume (% TLC)	$\dot{V}_{max}$ 75% (Liters/Sec)	Volume (% TLC)
Controls	9.6	88	8.3	81	5.8	63	2.1	44
(SEE) *	(1.8)	(5.7)	(1.1)	(1.0)	(1.2)	(2.3)	(1.0)	(3.5)
Miners with p opacities	8.6	86	8.0	83	5.1	67	1.6	50
(SEE)	(0.5)	(1.9)	(0.4)	(0.6)	(0.6)	(1.0)	(0.3)	(1.6)
Miners with q opacities	8.9	88	7.5	83	4.6	67	1.2	50
(SEE)	(0.6)	(1.1)	(0.4)	(0.5)	(0.3)	(0.9)	(0.1)	(1.3)

Transpulmonary Pressure

Subjects	2 cm H_2O Flow (Liters/Sec)	2 cm H_2O Volume (% TLC)	4 cm H_2O Flow (Liters/Sec)	4 cm H_2O Volume (% TLC)	6 cm H_2O Flow (Liters/Sec)	6 cm H_2O Volume (% TLC)	8 cm H_2O Flow (Liters/Sec)	8 cm H_2O Volume (% TLC)	10 cm H_2O Flow (Liters/Sec)	10 cm H_2O Volume (% TLC)
Controls	0.3	35	1.7	40	4.4	54	5.8	65	7.3	73
(SEE)	(0.1)	(4.2)	(1.0)	(5.1)	(2.1)	(5.3)	(2.7)	(5.1)	(3.3)	(4.3)
Miners with p opacities	0.3	37	1.8	48	3.0	57	5.0	68	7.1	76
(SEE)	(0.1)	(3.6)	(0.5)	(2.9)	(0.7)	(3.8)	(0.7)	(2.9)	(0.4)	(2.3)
Miners with q opacities	0.5	42	1.3	51	2.6	56	4.6	67	6.3	76
(SEE)	(0.3)	(2.8)	(0.4)	(2.4)	(0.5)	(3.5)	(0.6)	(3.0)	(0.7)	(2.6)

* Standard error of the estimate.

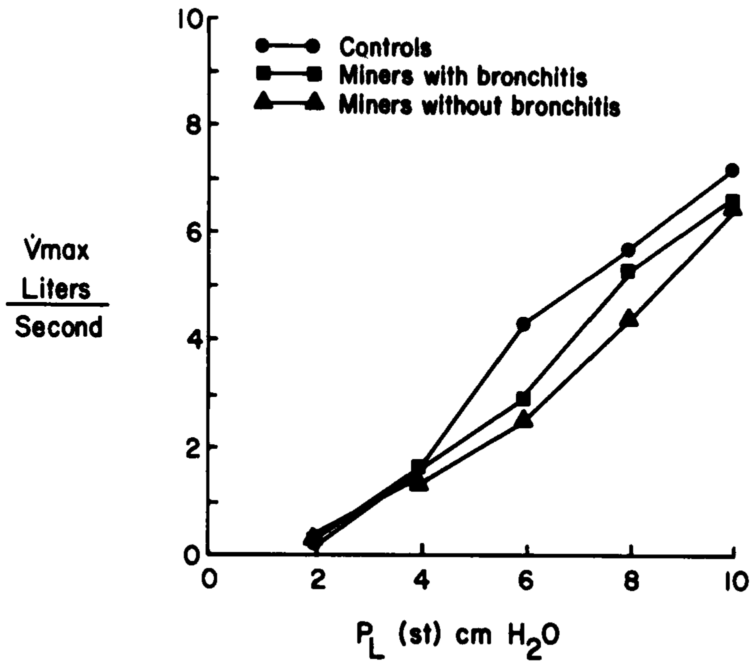

FIGURE 6. Pressure-flow curves for controls, miners without bronchitis and miners with bronchitis. Mean values for maximal expiratory flows ($\dot{V}_{max}$) are plotted versus static recoil pressures ($P_{L\ (st)}$) at which they occurred.

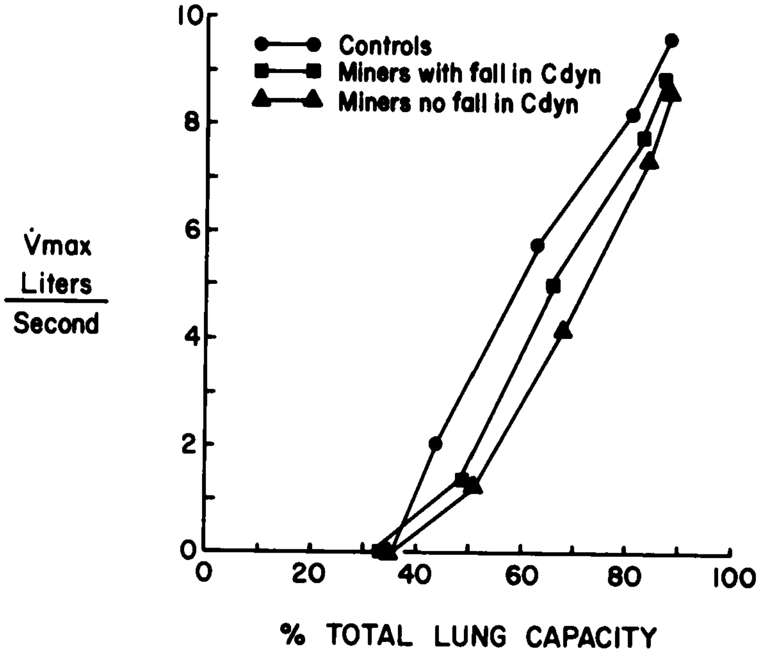

FIGURE 7. Flow-volume curves for controls, miners with no fall in C dyn and miners with fall in C dyn. Mean values for maximal expiratory flows ($\dot{V}_{max}$) at peak and at 25, 50, and 75% of FVC exhaled are plotted versus lung volumes (% TLC) at which they occurred.

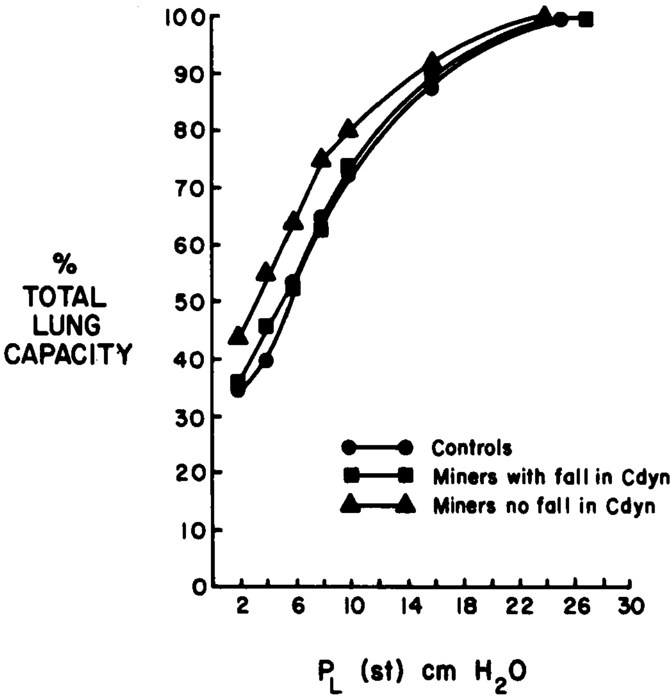

FIGURE 8. Pressure-volume curves for controls, miners with no fall in C dyn, and miners with fall in C dyn. Mean values for static recoil pressures ($P_{L\,(st)}$) are plotted versus lung volumes (% TLC) at which they occurred.

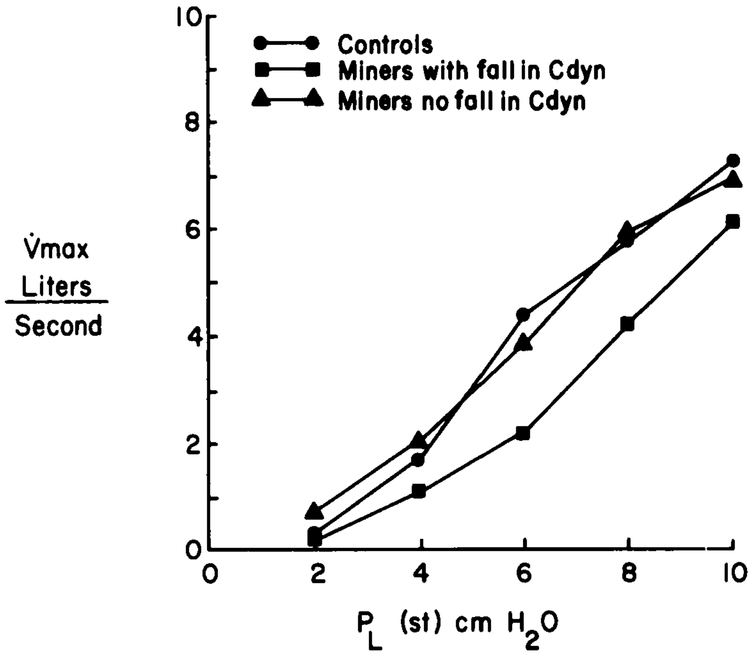

FIGURE 9. Pressure-flow curves for controls, miners with no fall in C dyn and miners with fall in C dyn. Mean values for maximal expiratory flows ($\dot{V}_{max}$) are plotted versus static recoil pressures ($P_{L\,(st)}$) at which they occurred.

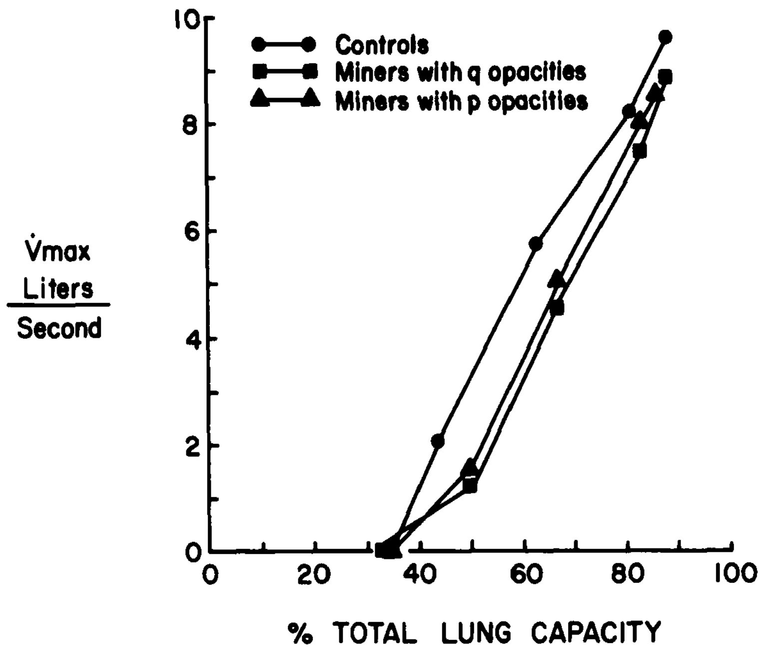

FIGURE 10. Flow-volume curves for controls, miners with pinhead (p), and miners with micronodular (q) opacities. Mean values for maximal expiratory flows ($\dot{V}_{max}$) at peak and at 25, 50, and 75% FVC exhaled are plotted versus lung volumes (% TLC) at which they occurred.

significantly lower than the controls at comparable lung volumes for all except peak flow (FIGURE 10). Miners with p opacities tended to have the lower $\dot{V}_{max}$, although the difference between them and miners with q opacities was not significant. Both the miners with p and those with q opacities showed slight reduction in mean $P_{L(st)}$ at comparable lung volumes when compared with the control group (FIGURE 11). When $\dot{V}_{max}$ was plotted versus $P_{L(st)}$ (FIGURE 12), thus correcting for loss in recoil pressure, the miners with q opacities achieved lower flows than either miners with p opacities or the control subjects.

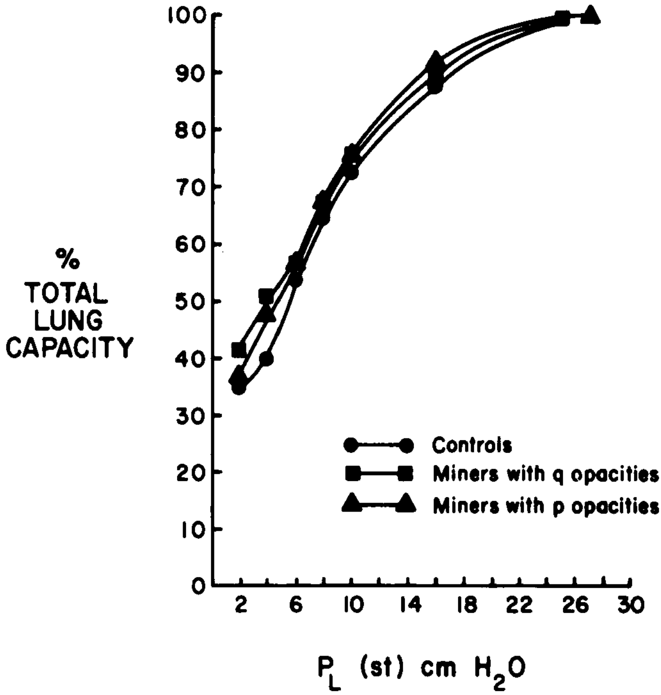

FIGURE 11. Pressure-volume curves for controls, miners with pinhead (p), and miners with micronodular (q) opacities. Mean values for static recoil pressures ($P_{L(st)}$), are plotted versus lung volumes (% TLC) at which they occurred.

DISCUSSION

Previous studies have demonstrated that underground bituminous coal miners have lower ventilatory capacity than nonminers, especially after age 50.[14-16] The relationships between ventilatory function and category of pneumoconiosis, years underground, smoking habits, and respiratory symptoms of miners are complex.[4, 15-18] Among nonworking and symptomatic miners, there is generally good correlation between standard spirometric tests of ventilatory function, respiratory symptoms, and smoking habits, whereas the association between these factors and category of pneumoconiosis is often poor.[3] Among working miners, there tends to be better correlation between category of pneumoconiosis and lung function if the ratio of observed to predicted residual volume is used.[5] Selection factors operating in the populations studied probably account for these differences.

Pemberton [14] showed that working miners had a lower FEV_1 than urban or rural factory workers of comparable ages, even though cigarette consumption among the miners was less than in either group of factory workers. Hyatt and coworkers [17] studied a random sample of working and retired

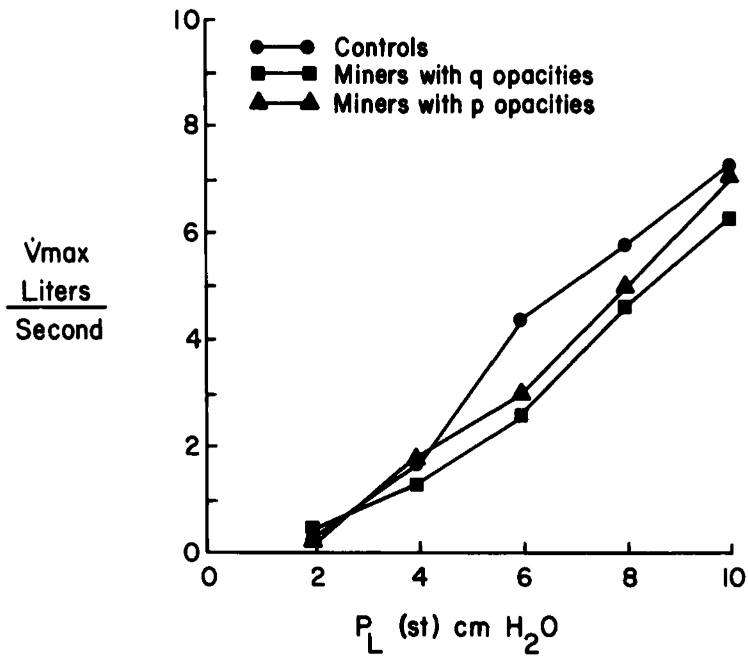

FIGURE 12. Pressure-flow curves for controls, miners with pinhead (p), and miners with micronodular (q) opacities. Mean values for maximal expiratory flows ($\dot{V}_{max}$) are plotted versus static recoil pressures ($P_{L\,(st)}$) at which they occurred.

miners in southern West Virginia aged 45 to 55 years. They found that maximal midexpiratory flow (average flow over the middle half of the forced vital capacity) among miners was reduced and that it related in a complex fashion to years of underground exposure, category of pneumoconiosis, and cigarette consumption. Higgins and associates [15] demonstrated a lower FEV_1 in miners and exminers with 30 years or more of underground experience than in nonminers from the same communities in northern West Virginia. Enterline [16] was able to show that the FEV_1 in miners was lower than among nonminers in one community in southern West Virginia but not in another community in central West Virginia.

In studying pulmonary mechanics in British coal miners, many of whom were disabled or seeking compensation, Leathart[6] found a slight decrease in dynamic compliance, normal inspiratory airway resistance, and some increase in the nonelastic work of breathing with increasing age and time spent underground. Ferris and Frank[7] reported an investigation of 25 symptomatic Appalachian bituminous coal miners, two of whom had complicated pneumoconiosis and the remainder, simple pneumoconiosis. In the miners with normal pulmonary airflow resistance, little change in dynamic compliance occurred at increased respiratory frequencies. Dynamic compliance did fall as the rate of respiration increased in the miners with elevated pulmonary airflow resistance. These investigators were unable to find any consistent differences in static recoil pressure at TLC between their subjects, whether grouped according to disability or radiographic category of CWP.

We have previously reported the tendency for unobstructed but symptomatic miners with simple CWP to manifest an increase in static compliance, a decrease in the coefficient of retraction (static recoil pressure at TLC divided by TLC), and a ratio of dynamic to static compliance that is at the lower limits of normal.[3]

The mechanisms by which reduction in ventilatory function develops among miners are not well understood. Resistance measurements predominantly reflect the pressure-flow behavior of large intrathoracic and extrathoracic airways in their resting, noncompressed state. Maximal expiratory flows ($\dot{V}_{max}$) developed during the FVC maneuver reflect a complex interplay among such factors as airway geometry, dynamic compression of airways, and lung elastic recoil.[19] By analyzing $\dot{V}_{max}$ in terms of the lung volumes and static recoil pressures at which these occurred and the overall pressure-volume behavior of the lungs, it is possible to look at the role that these factors play in accounting for decreases in $\dot{V}_{max}$.

The miners as a group had slightly reduced lung elastic recoil (FIGURE 2) but this reduction did not alone account for their reduced $\dot{V}_{max}$, or their curve of $\dot{V}_{max}$ versus $P_{L\ (St)}$ (FIGURE 3) would have closely followed that of the control subjects. The fact that it did not suggests either a change in airways geometry or the presence of dynamic compression of the airways. The fact that 17 of the 25 miners demonstrated frequency dependence of compliance in the absence of large airway obstruction and with little loss of lung elastic recoil suggests obstruction in small peripheral airways. Such a pattern has been reported by Woolcock and colleagues[20] in subjects with early bronchitis and in asthmatics in remission. This change is not likely to be an effect of age, because it did not occur in any of the control subjects. Although symptoms of bronchitis were present in six of the miners whose C dyn was frequency-dependent and thus could account for this finding, no obvious cause other than CWP was evident for the frequency dependence of C dyn in the remaining 11 miners.

The relationship of reduced $\dot{V}_{max}$ and frequency dependency of C dyn to type of radiographic opacity in simple CWP is also complex. Several investigators[21-23] have demonstrated that miners with p opacities have lower carbon monoxide diffusing capacity (transfer factor) than miners with q opacities. The reasons for these differences in diffusing capacity are not clear. Cotes and coworkers[22] postulated that the p type of opacity might be associated with an increase in the tissue of the lung parenchyma. Being thus in an unstable state, the lung might progress to either focal emphysema or interstitial fibrosis or both, giving rise to characteristic changes in lung function.

Our data do not appear to support the abovementioned concept. If miners with p opacities had predominantly increased interstitial fibrosis, the lung elastic recoil should have been increased, rather than slightly decreased, as compared to the controls. On the other hand, if focal emphysema were the predominant change in miners with p opacities, they should have demonstrated reduced lung elastic recoil and maximal expiratory flows that were not appreciably lower than the controls when plotted versus static recoil pressure. Our miners with p opacities did not clearly demonstrate either pattern of change in lung function. Furthermore, by the same criteria, it appears that miners with q opacities demonstrated the changes indicative of focal emphysema more than miners with p opacities.

A more likely explanation for our findings is that both miners with p and with q opacities had increased resistance to airflow in small peripheral airways. This conclusion is supported by the fact that six of the ten miners with p opacities and 11 of the 15 miners with q opacities demonstrated frequency dependency of dynamic compliance. Mechanical factors *per se*, at least in terms of lung recoil, frequency dependence of dynamic compliance, and maximal flow versus static recoil pressure, do not offer a ready explanation for the difference in diffusing capacity (transfer factor) between miners with p and those with q opacities.

The exact site of expiratory flow limitation in obstructive airway disease remains a subject for speculation. Macklem and Mead [24] partitioned the resistance of the intrathoracic airways into central and peripheral components. The peripheral component included the resistance of airways from the alveoli to approximately the ninth to twelfth generation. The central component included the resistance of airways from this level to the level of the intrathoracic trachea. That significant disease involving the peripheral airways could occur with little alteration in total airway resistance or standard spirometric tests of ventilatory function was predicted by these authors and confirmed by Hogg and associates.[25]

Since the coal macule is situated around the first and second order respiratory bronchiole, it might thus produce a significant effect on the peripheral resistance in the "quiet zone" of the lungs.[26] The patterns of disturbance in lung mechanics reported herein are what might be expected, bearing in mind the site and the pathological features of the coal macule.

Further studies are necessary to ascertain whether the changes reported are solely a consequence of simple CWP or are a less specific effect of working in coal mines. It is possible that the lesion causing frequency dependence of C dyn and reduced $\dot{V}_{max}$ is situated in small terminal bronchioles rather than in respiratory bronchioles and represents a bronchiolitis related to nonspecific irritation caused by coal dust or noxious fumes, rather than CWP itself.

SUMMARY

Lung volumes and mechanics of respiration were investigated in 25 working, nonsmoking, underground bituminous coal miners who demonstrated category 2 or 3 simple pneumoconiosis and also in six nonsmoking controls of comparable age. Analysis of pressure, flow, and volume relationships demonstrated that miners as a group achieved lower maximal expiratory flows than the controls at comparable lung volumes and pressures. Although chronic

bronchitis was present in 10 of the 25 miners, it did not account for these findings. Reduction in dynamic compliance (C dyn) at faster rates of respiration, a finding that occurred in 17 of the 25 miners despite the absence of significant obstruction of large airways, appeared to be the result of increased resistance in small, peripheral airways rather than reduction in lung recoil (driving) pressure. These alterations in lung mechanics did not appear to be closely related to type of opacity (either pinhead or micronodular) seen on the chest radiographs of the miners.

ACKNOWLEDGMENTS

The authors are indebted to Dr. Robert E. Hyatt of the Mayo Clinic and to Dr. W. K. C. Morgan for valuable suggestions. Mr. Robert Reger performed statistical analyses. Mr. Bruce Shipe and Mr. Allen Harding provided technical assistance. Mrs. Lynne Morris typed the manuscript.

REFERENCES

1. HEPPLESTON, A. G. 1953. The pathological anatomy of simple pneumokoniosis in coal workers. J. Path. Bact. **66:** 235.
2. MORGAN, W. K. C. 1968. The prevalence of coal workers' pneumoconiosis. Am. Rev. Resp. Dis. **98:** 306.
3. LAPP, N. L. & A. SEATON. 1971. Pulmonary function. *In* Pulmonary Reactions to Coal Dust; A Review of U.S. Experience. M. Key, L. Kerr & M. Bundy, Eds.: 153–177. Academic Press. New York, N.Y.
4. LAINHART, W. S. 1969. Prevalence of coal miners' pneumoconiosis in Appalachian bituminous coal miners. *In* Pneumoconiosis in Appalachian Bituminous Coal Miners. W. S. Lainhart, H. N. Doyle, P. E. Enterline, A. Henschel & M. A. Kendrick, Eds.: 31–60. U.S. Govt. Printing Office. Washington, D.C.
5. MORGAN, W. K. C., D. B. BURGESS, N. L. LAPP & A. SEATON. 1971. Hyperinflation of the lungs in coal miners. Thorax **26:** 585.
6. LEATHART, G. L. 1959. The mechanical properties of the lung in pneumoconiosis of coal miners. Brit. J. Ind. Med. **16:** 153.
7. FERRIS, B. & N. R. FRANK. 1962. Pulmonary function in coal miners. J. Occup. Med. **4:** 274.
8. UICC COMMITTEE. 1970. UICC/Cincinnati classification of the radiographic appearances of pneumoconioses; a cooperative study. Chest **58:** 57–67.
9. HANKINSON, J. L. & N. L. LAPP. 1970. Time-pulse generator for flow-volume curves. J. Appl. Physiol. **29:** 109.
10. DUBOIS, A. B., S. Y. BOTELHO, G. N. BEDELL, R. MARSHALL & J. H. COMROE. 1956. A rapid plethysmographic method for measuring thoracic gas volume: a comparison with nitrogen washout method for measuring functional residual capacity in normal subjects. J. Clin. Invest. **35:** 322.
11. DUBOIS, A. B., S. Y. BOTELHO & J. H. COMROE. 1956. A new method for measuring airway resistance in man using a body plethysmograph. J. Clin. Invest. **35:** 327.
12. NEEDHAM, C. D., M. C. ROGAN & I. MCDONALD. 1954. Normal standards for lung volumes, intrapulmonary gas mixing and maximum breathing capacity. Thorax **9:** 313.
13. MILIC-EMILI, J., J. MEAD & J. M. TURNER. 1964. Topography of esophageal pressure as a function of posture in man. J. Appl. Physiol. **19:** 212.
14. PEMBERTON, J. 1956. Chronic bronchitis, emphysema, and bronchial spasm in bituminous coal workers. Arch. Ind. Health **13:** 529.

15. HIGGINS, I. T. T., M. W. HIGGINS, M. D. LOCKSHIN & N. CANALE. 1968. Chronic respiratory disease in mining communities in Marion County, West Virginia. Brit. J. Ind. Med. **25:** 165.
16. ENTERLINE, P. E. 1969. Prevalence of respiratory symptoms in two Appalachian communities. *In* Pneumoconiosis in Appalachian Bituminous Coal Miners. W. S. Lainhart, H. N. Doyle, P. E. Enterline, A. Henschel & M. A. Kendrick, Eds.: 61–73. U.S. Govt. Printing Office. Washington, D.C.
17. HYATT, R. E., A. D. KISTIN & T. K. MAHAN. 1964. Respiratory disease in southern West Virginia coal miners. Am. Rev. Resp. Dis. **89:** 387.
18. HENSCHEL, A. 1969. Ventilatory function and work capacity in Appalachian bituminous coal miners and miners and non-miners in two West Virginia communities. *In* Pneumoconiosis in Appalachian Bituminous Coal Miners. W. S. Lainhart, H. N. Doyle, P. E. Enterline, A. Henschel & M. A. Kendrick, Eds.: 77–111. U.S. Govt. Printing Office. Washington, D.C.
19. LAPP, N. L. & R. E. HYATT. 1970. Evaluation of lung mechanics for epidemiological studies. Bull. Physio-Pathol. Resp. **6:** 595.
20. WOOLCOCK, A. J., N. J. VINCENT & P. T. MACKLEM. 1969. Frequency dependence of compliance as a test for obstruction in the small airways. J. Clin. Invest. **48:** 1097.
21. LYONS, J. P., W. G. CLARKE, A. M. HALL & J. E. COTES. 1967. Transfer factor (diffusing capacity) for the lung in simple pneumoconiosis of coal workers. Brit. Med. J. **4:** 772.
22. COTES, J. E., C. N. DEIVANAYAGAM & G. B. FIELD. 1970. Relation between types of simple pneumoconiosis (p, m, n) and lung function. Third Intern. Conf. Inhaled Particles. London, England.
23. SEATON, A., N. L. LAPP & W. K. C. MORGAN. 1972. The relationship of pulmonary impairment in simple coal workers' pneumoconiosis to the type of radiographic opacity. Brit. J. Ind. Med. **29:** 50.
24. MACKLEM, P. T. & J. MEAD. 1967. Resistance of central and peripheral airways measured by a retrograde catheter. J. Appl. Physiol. **22:** 395.
25. HOGG, J. G., P. T. MACKLEM & W. M. THURLBECK. 1968. Site and nature of airway obstruction in chronic obstructive lung disease. New Eng. J. Med. **278:** 1355.
26. MEAD, J. 1970. The lung's "quiet zone." New Eng. J. Med. **282:** 1318.