

OCT 13 1972

MECHANISM OF AIR-FLOW OBSTRUCTION IN COAL MINERS

N. LeRoy Lapp, M. D., F.A.C.P. and Anthony Seaton, M. D.

Presented by

N. LeRoy Lapp, M. D.

Tri-State Regional Meeting

American College of Chest Physicians

October 13 and 14, 1972

Charleston, West Virginia

cc. 252

## Introduction

Coal workers' pneumoconiosis (CWP) is a condition caused by 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 accumulation of dust around the first and second order respiratory bronchioles, a little surrounding fibrosis, and some dilatation of the respiratory bronchioles. This latter is referred to as focal emphysema. Simple pneumoconiosis produces relatively little ventilatory impairment detectable by the standard spirometric tests unless accompanied by the presence of chronic obstructive airway disease. This latter is often a consequence of factors other than deposition of coal dust.

Most studies of the mechanical properties of the lungs have been performed on miners who were seeking medical care or industrial compensation and thus suffer from the selection biases often found in such populations.

The present study was designed to investigate the mechanical properties of the lungs in working miners in order to determine what abnormalities result from simple coal workers' pneumoconiosis itself. In particular, it was hoped to determine what the mechanism is that leads to air-flow obstruction among coal miners.

## Subjects and Methods

Twenty-five working bituminous coal miners were studied. All 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. Fourteen were lifelong nonsmokers. Of the 11

ex-smokers, only two had smoked the equivalent of one pack daily for 20 years, the rest less and none within the last 10 years. None had obstructive airway disease as defined by an  $FEV_{1.0}/FVC$  ratio of less than 70 percent.

Six males in the same age group served as the control group. All were lifelong nonsmokers and none suffered from cardiopulmonary disease.

Spirometry, lung volumes, and airway resistance were measured using an electronic spirometer and a constant volume plethysmograph. Flow versus volume curves of the forced vital capacity maneuver were recorded from the electronic spirometer, and pressure volume curves of the lungs under static and dynamic conditions were recorded by means of an esophageal balloon catheter--pneumotachograph system.

### Results

The miners differed appreciably from the control subjects only in a slightly lower ratio of  $FEV_{1.0}$  to FVC (mean, 76 versus 81 percent).

The first slide shows the mean values for maximal expiratory flows versus the lung volumes, plotted as percent TLC, at which they occurred for the controls and the miners. It is evident that miners as a group were unable to achieve flows as high as the control subjects at comparable lung volumes throughout the vital capacity. These differences were significant for all values save peak flow.

Maximal expiratory flows developed during the forced vital capacity maneuver reflect a complex interplay among such factors as upstream airway resistance, dynamic compression of airways, and lung elastic recoil. In

order to better understand the interplay of these factors, we analyzed the pressure, flow and volume relationships in these subjects.

Seventeen of the 25 miners demonstrated a fall in dynamic compliance at faster respiratory frequencies. This phenomenon has been shown to occur in subjects with abnormally elevated upstream airway resistance even when standard tests of ventilatory function were not abnormal. None of the controls demonstrated this phenomenon.

The next slide shows mean values for maximal expiratory flows versus lung volumes at which they occurred for the controls and the miners grouped according to whether or not they demonstrated frequency dependence of dynamic compliance. It is evident that both groups of miners achieved lower flows than the controls but that little difference existed between them.

The next slide shows the pressure versus volume curves for the controls and the miners grouped according to whether or not they demonstrated frequency dependence of dynamic compliance. It is evident that the miners whose compliance fell had a mean pressure volume curve that was identical to that of the controls, while the mean curve for the miners whose compliance did not fall was shifted upward, demonstrating less lung recoil pressure at comparable lung volumes.

In the final slide we have plotted maximal expiratory flow versus lung recoil pressure. This method of analysis, developed by Mead and associates, demonstrates the role of loss of lung elastic recoil in causing reduced maximal expiratory flow. Thus if reduced lung recoil were the sole cause of reduced flows the curve should follow that of the control

subjects. Indeed, it can be seen that this is the case for the miners with no fall in dynamic compliance. On the other hand, if the values fall to the right of and below the control curve, then increased resistance of the upstream airways rather than loss of lung recoil is the mechanism accounting for the reduced expiratory flows. That this is the case for the miners who demonstrated a fall in dynamic compliance is also evident on this slide.

#### Summary and Conclusions

We have demonstrated that working miners with category 2/1 or greater simple CWP have decreased expiratory flows when compared with age matched controls. In the majority, 17 of 25, this appears to be the result of an abnormal increase in upstream airway resistance. In the remaining 8, the reduced flow appears to be a consequence of reduced lung recoil pressure. Increased upstream airway resistance could result from a bronchiolitis in the region of the terminal and first or second order respiratory bronchiole, the part of the lungs where coal dust accumulates in coal workers' pneumoconiosis. Focal emphysema, an abnormal dilatation and disruption of the tissues surrounding the terminal and respiratory bronchioles in simple coal workers' pneumoconiosis, could account for the reduced lung recoil pressure. It appears that both mechanisms can be found among underground coal miners as a cause of air-flow obstruction.

Recent studies employing this kind of analysis in working miners with category 0 and category 1 chest radiographs lead us to conclude that both mechanisms of air-flow obstruction are related to the presence of pneumoconiosis and not simply a nonspecific effect of underground exposure.