UPPER LOBE CHANGES AND EXPOSURE TO ASBESTOS

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ABSTRACT

Asbestosis is classically a disease which mainly affects the lower lobes. However, it has been known for many years that sometimes the upper lobes are affected and indeed severely so, even if this is a rare occurrence. There have been discussions on whether these lesions are indeed due to asbestos or some other disease. In particular, many cases have been labelled tuberculosis and treated as such.

In the county of Uppsala, Sweden, we have been interested in the asbestos problem for more than a decade and have now collected more than 1500 persons with asbestos-related pleural and/or parenchymal disease which we follow. Most of these have had comparatively light exposure. Among those workers, 40 patients have been seen with typical upper lobe changes.

These 40 persons have had intermittent exposure for in the mean 25 years (2 to 46 years). The latency time from first exposure was in the mean 34 years with a range of 5 to 51 years. The upper lobe is sometimes severely affected with a pleural thickening of up to 40 mm (mean 21 mm) and an obvious shrinkage of the lobe with the hilus drawn upwards and the trachea deviating to the affected side. In 4 patients (10 percent) the left upper lobe only was affected, in 21 the right, and in 15 both sides. The disease often progresses fairly rapidly within 5 to 10 years and is in almost all cases part of a general pleuroparenchymal disease. Hence, the lung function is severely affected. VC was in the mean 62% of predicted and TLC 69%.

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OCCUPATIONAL SILICOSIS AMONG WORKERS IN AN ORE MILL, THAILAND

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BACKGROUND

In July, 1986, the chest disease hospital in Nonthaburi reported a death to the divisions of occupational health and epidimiology of the ministry of public health. The death occurred in a 27 year-old female worker with a diagnosis of silicosis. The patient had a long history of dyspnea treated at numerous clinics. Two weeks prior to her death she was admitted to the Nakorn patom provincial hospital with fever, dyspnea, cough, and crepitation in both lungs. Her chest X-ray was abnormal with marked reticulo-nodular infiltration. The index case worked in a mill in Nakorn patom province, located 60 kilometers southwest of Bangkok.

The factory ground minerals such as quartz, feldspar, flint, talcum, clay, dolomite, and phosphate, as seen here before the crushing process. The process of grinding very fine granules 300 mesh of these ores.

The effects of silicosis, the chronic fibrosis of the lungs produced by prolonged and extensive exposure to free crystalline silica, have been recognized for centuries. Pulmonary disease produced by dust is mentioned by Agricola in his Treatise on Mining and is described in stonecutters by Van Diemerbroeck Ramazzini.[1] Clinical evidences of previous exposure to free silica in old mines, abandoned quarries, and ancient flint tools and weapons were demonstrated.[2] Silicosis is caused by the inhalation and retention of dust containing silica in occupations such as mining, tunnelling, quarrying, stone dressing, sandblasting, fettling, boiler scaling, and in pottery, ceramics and brick manufacture.[3] Symptoms of silicosis cases are increasing dyspnea, non productive cough and chest pain, progressing to compensatory emphysema and cor pulmonaly.[4] There was a report of silicosis among miners which the prevalence of 19.48% (5,366/27,553) together with pulmonary tuberculosis of 13.83% (742/5,366).

In order to carry out the investigation of this fatal case, and to search for other possible cases, meetings were held with officials from various other goverment agencies, including the ministry of interior and ministry of industry.

The objectives of our investigation were (1) to establish whether or not there was an epidemic of silicosis among the workers in the factory; (2) to find the epidemiological distribution of silicosis; (3) to identify risk factors for silicosis disease; and (4) to develop appropriate preventive and control measures.

METHODS

The methods included (1) describing the epidemiology of the problem; (2) a case-control study for risk factors for disease; and (3) studying the environment of the factory.

To describe the problem, we interviewed all 80 workers in the plant with questionnaires about work histories and history of illness, and performed physical examinations included chest auscultation, chest percussion, and measurement of chest expansion. All 80 workers received posteroanterior chest radiographs. All 80 workers underwent pulmonary function testing. Capro's prediction equation was used to find predicted values.[5] The predicted values were corrected with 0.85 for non-caucasian people.

A case of silicosis was defined as any worker in plant whose chest radiograph change by ILO-1980 international classification of pneumoconiosis at 1/1 profusion of lung parenchyma.[6] Four controls for each case in the case-control study were selected by simple random sampling from non-case workers in the mill.

The environmental survey included a walk-through of the plant. We measured dust concentrations at various sites in the plant. We also observed the workers performing their jobs, and we attached portable dust concentration measuring devices top selected workers.

RESULTS

The mill was divided into three plants. Plant number one ground feldspar and phosphate. Plant number two usually ground quartz and flint, and on occasion would grind feldspar and phosphate. Plant number three ground clay, only. Of the total of 80 workers in the plant, we found 10 workers or 12.5% with silicosis,including the fatal index case. This index case was the only worker to have died in the previous year, for a mortallity rate of 1.2 percent, and a case-fatality rate of 10 percent.

Chest radiographs revealed the small opacities of parenchymal profusion of 1/1 at 20%, 2/1 at 30%, 3/3 at 50%. Most of the sizes and shapes of such abnormalities were p and q. One case had large capacity catagorized as "B".

Pulmonary function tests were normal in five, or half of the cases. The five cases with abnormal pulmonary function generally had a restrictive ventilatory defect. Clinical symptoms and sign in the case included dyspnea, chest pain,

chronic cough, restriction of lung expansion, and decreasing breath sounds.

The mean age of the 10 cases was 34.9 years with a range from 34 to 49 years. The duration of working in the plant averaged 8 years before our study,with a range of 1 to 14 years of exposure.

The attack rates for silicosis by job category were highest in two housekeepers whose job was to sweep up the dust which accumulated in the working areas. The second highest attack rate was in ore-grinders at rate of 33% (7/21).

Attack rate by job location were highest in plant number two, where quartz and flint were ground. The attack rate was 3.5%. This is the plant where the fatal case worked. The attack rate was 10.7% in plant number one, where feldspar and phosphate were ground. There were no cases in plant number three, where clay was ground. There were no cases in office workers, but there were two cases among foremen, who were exposed to dust in all the plant areas.

For the environmental inspection, we tested the mineral content of the ores used in the plant. Silica is the mineral ingredient of greatest hazard. Quartz and sand had the highest silica content, at 45.7%.

We found no engineering dust control measures in use; no hoods and no protective dust enclosures. A few workers tried to avoid breathing the hazardous dust with cloths over their face, but this is very ineffective. Our inspection revealed very hazardous levels of dust being created by the grinding process. No occupational health services were provided for workers in the plant, as required by regulations.

The Thai ministry of interior's maximum permissible level of respirable dust in workplace air is 5 mg/cubic meter. We measured an average concentration of respirable dust in the breathing zones of ten workers of 437.7 mg/cubic meter of air, 87 times the permissible level.

In the case-control study, the workers with a duration of exposure of greater than 5 years had odds ratio of 16 times the likelihood of being a case than workers with less exposure.

The case-control study also found that workers in the grinding and packing areas of the mill had an odds ratio of 12 times the likelihood of being a case than workers in other areas of the plant.

When it was analyzed by the measured amounts of dust in various work areas, we found that workers in areas with dust exceeding permissible exposure limit had an odds ratio of 11 times the likelihood of having silicosis, compared to workers in areas with dust within regulation limits.

When we measured amounts of silica contained in the dusts, we found that workers in areas with dust containing 33% or more of silica had an odds ratio of eight times the likelihood of having silicosis than workers in areas with dust containing less than 33% silica.

DISCUSSIONS AND RECOMMENDATIONS

This investigation revealed 12.5% of silicosis prevalence rate with one dead case. It is a quite severe situation. To prevent workers from exposure to silica is among the highest priorities in protecting the health of the workers. Silicosis is not reversible. If one gets the disease, one will be affected for the rest of one's life. Thus, this epidemiological study aimed ultimately to such prevention, as one definition of epidemiology is the study of distribution and determinants of the disease. The classical process consists of examining a series of variables to ascertain causation including age, sex, socioeconomic status and other. Since it is known that silica causes silicosis, but there are several major difficulties involved in attempting to do this, which were difficulties in the accurate determination of exposed dose, difficulties in the accurate determination of the health effects and difficulties in dealing with competing variables such as cigarette smoking and host susceptibility.[7]

As the epidemiological data show that the housekeepers and the ore-grinders were the highest risk group to develop silicosis. This group should be firstly provided preventive measures, if there were any constraints.

At the conclusion of the investigation, we gave essential health education lectures to all the workers about the hazards of silica and how to avoid it by using NIOSH-approved masks. We advised the owners how to make engineering changes that would make the workplace safer. The report was provided to the ministry of industry, which closed down the factory for 6 months because of the health dangers. A joint meeting of the ministries of industry, interior, and public health was held with many owners and operators of ore and stone grinding plants throughout Thailand to tell the plant operators about the dangers of silica, and how to prevent workers from developing silicosis.

This plant was reopened after making recommended changes in workplace practices, including (1) building covers and hoods over grinding machines to prevent escape of dust, (2) providing approved masks for exposed workers, (3) offering medical care for workers, and (4) tranferring those with lung problems to safer parts of the plant.

This plant was inspected about six months after the original investigation. Dust levels were found to be below permissible limits and workers were wearing approved masks.

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SILICOSIS AND LUNG CANCER: PRELIMINARY RESULTS FROM THE CALIFORNIA SILICOSIS REGISTRY

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ABSTRACT

Exposure to silica (SiO_2) causes silicosis and silicotuberculosis; now there is new concern about its carcinogenicity (see IARC Monograph on Silica, 1987). The California Silicosis Registry was created to examine the mortality of silicotics (individuals with high SiO_2 exposures) among state Workers' Compensation Appeals Board claimants. The authors used proportionate mortality ratios (PMRs) to assess the risks among 212 while male silicotics who filed claims between 1945 and 1963 and were followed until 1984. The PMRs (and 95% confidence intervals [$Cl_{95\%}$]) indicated that silicotics had excesses of cancer of the lung (PMR=2.22; $Cl_{95\%}$=1.30, 3.37), tuberculosis (PMR=9.75; $Cl_{95\%}$=6.58, 13.92), and nonmalignant respiratory diseases (PMR=4.18; $Cl_{95\%}$=3.10, 5.51). Removing tuberculosis deaths from the analysis produced a lung cancer PMR of 2.53. There was no excess risk for gastrointestinal or lymphatic cancers. These preliminary findings from the Registry suggest that silicotics in California have a significant respiratory cancer risk. This risk is consistent with findings from many international studies and strongly suggests that silicosis acts as a precursor lesion for lung cancer.

BACKGROUND

Since the 1930s there has been unsubstantiated conjecture that silicosis predisposes to cancer of many sites. It is established that silica exposure causes silicosis, silicotuberculosis, cor pulmonale, and perhaps nephritis. However, that silica may be a carcinogen or that silicosis may increase the risk of bronchogenic cancer (Goldsmith et al., 1982; Goldsmith et al., 1986; IARC, 1987) has been seriously considered only in the past seven years and challenges a great deal of expert medical opinion (see Heppleston, 1985; Selikoff, 1978; Ziskind et al., 1976).

The following is a summary of the scientific evidence of silica's carcinogenicity and the relationship between silicosis and respiratory cancer:

1. Very recent evidence shows that silica produces mutations and premalignant hyperplasia *in vivo* (Hesterberg et al.; 1986, Gibson et al., 1986; Saffiotti, 1986).

2. Since 1980 silica (as Min-U-Sil) has been shown to be a pulmonary carcinogen in five lifetime rat studies using both intratracheal and inhalation methods (Holland et al., 1983; Groth et al., 1986; Dagle et al., 1986; Wagner et al., 1980; Muhle et al., 1989). Muhle et al.'s research on rats shows that quartz is a carcinogen at a level of 1 mg/m^3. Although the tumorigenic results were not replicated in hamsters (Holland et al., 1983; Saffiotti, 1986), this is the first time that pulmonary tumors were induced using inhalation (Dagle et al., 1986; Muhle et al., 1989; Holland et al., 1986), paralleling the human route of exposure.

3. Workers in dusty trades with known high exposure to crystalline silica, including sandblasting, firebrick manufacture, ceramics, granite and stone work, and tunneling have statistically significant excess mortality from lung cancer (reviewed in Goldsmith et al., 1986; IARC, 1987; Lynge et al., 1986). Smoking was not controlled in most of the studies, however its lack of adjustment cannot completely explain the high lung cancer risk ratios (Axelson, 1978, Fletcher and Ades, 1984). By way of contrast, Goldsmith and Guidotti (1986) presented evidence that smoking may be synergistic with silica exposure in the risk of lung cancer.

4. Paralleling the lymphatic malignancies induced by silica in animals (Wagner et al., 1980), there are several studies indicating an elevated risk in humans (Brown et al., 1986; Kurppa et al., 1986; Redmond et al., 1981; Mirer et al., 1986). In addition there are several studies indicating excess mortality from stomach cancer among workers in which silica exposure predominated (reviewed by Greenberg, 1986).

5. Similar to the observations of pulmonary tumors adjacent to silicotic nodules in rats, lung cancer excesses have been reported consistently in studies of silicotics (reviewed by Goldsmith et al., 1988). The risk measures range from 1.4 to 5.9 and have been reported from Sweden (Westerholm, 1980), Ontario (Finkelstein

et al., 1982, 1987), Switzerland (Schuler and Ruttner, 1986), Austria (Neuberger et al., 1986), Finland (Gudbergsson et al., 1984; Kurppa et al., 1986), Italy (Forastiere et al., 1986; Rubino et al., 1985; Zambon et al., 1986), U.S. (Steenland and Beaumont, 1986), and Japan (Chiyotani, 1984).

EXPERIMENTAL DESIGN AND METHODS

Background of This Study

In 1986 and 1989 we received funding from the California Thoracic Society and the Centers for Disease Control, respectively, to create the California Silicosis Registry. Its purpose is to provide a registry of all claims for silicosis (and claims for other occupational pulmonary disease) within California, and to provide a basis for testing the association beween silicosis and cancer. This paper will describe the proportional mortality ratio (PMR) findings from a partial and preliminary examination of the registry.

Methods

Patients having claims for silicosis diagnosed in California who were state residents from January 1, 1946 to December 31, 1980 are being identified. We are using the files of the California Workers' Compensation Appeals Board (WCAB). The following data are being extracted from the archived records of WCAB: name, address, sex, Social Security number, date of birth, name and address of primary physician, date of claim, type of injury alleged, and employer's name and address. There are three types of controls being selected (excluding fatal injuries): one, matched for age (± 2 years), year of claim, region and sex; second, the next noncase in the files after a lung disease claim is found; the third control is a random selection of one claim from every 12 boxes of files (approximately 1 per 400 sample). These cases of silicosis and controls constitute the California Silicosis Registry.

All subjects are being traced up to December 31, 1984 to determine their vital status. Using California Department of Motor Vehicles (DMV) records, tracing is now being carried out to determine last known address if alive. Deceased subjects are being identified and information extracted from the California Vital Statistics Section of the Department of Health Services.

We used a proportionate mortality ratio (PMR) analysis program developed by Maizlish (1986) to provide a preliminary assessment of mortality between silicosis and respiratory disease claims whose death certificates (up to 1984) could be abstracted from California vital records. Expected values are from U.S. white male rates according to the 8th revision of the International Classification of Diseases. The major advantage is that this approach uses all the deaths obtained in the registry to date and provides a useful indication of any excesses relevant to the hypotheses under evaluation in this study.

RESULTS

This paper will examine only those data from the San Francisco district in a preliminary follow-up. All claims listing either silicosis, silicotuberculosis, anthracosilicosis, pneumoconiosis, dust diseases of the lung, or any lung illness (not accidents) are being extracted from the files of WCAB.

Table I shows the number of WCAB files examined, the number of lung disease claims found and the proportion of cases found per 1,000 records. These preliminary results indicate that about 5 of every 1,000 files reviewed contains a WCAB claim for occupational respiratory disease. As of May, 1988, over 130,000 individual files had been reviewed, and nearly 700 pneumoconiosis claims (plus controls) have been extracted. For this study we focused only on white males because fewer than 5% of the deaths occurred among nonwhites or females.

Tables II and III presents PMRs for white male cases dying from nonmalignant and malignant diseases, respectively. As expected, the cases have a striking excess mortality from tuberculosis (TB), a frequent complication of silicosis (PMR = 9.75; 95% CI = 6.58, 13.92). There is a significant deficit of mortality for all circulatory diseases, PMR = 0.60; 95% CI = 0.47, 0.75. Nonmalignant respiratory disease mortality showed the expected pattern with claimants having a clear excess PMR of 4.18; 95% CI = 3.10, 5.51. Cirrhosis of the liver showed a nonsignificant PMR of 1.39. Accidental deaths among lung disease claimants had a borderline significant deficit PMR of 0.42, and there was no excess mortality for suicide.

Table III indicates that the silicosis claimants have a PMR of 1.07 for all malignant neoplasms. In contrast to the recent findings of Finkelstein et al. (1987), there was no excess of cancer of the gastrointestinal tract among cases (PMR = 0.91). An excess risk for lymphopoeitic cancers was not born out among these pneumoconiotics, PMR = 0.90. There was also a PMR of 2.32 for pancreatic cancer (based on only 5 deaths).

Malignant neoplasms of the respiratory system were significantly elevated for lung disease claimants (PMR = 2.16; 95% CI = 1.37, 3.24). For cancer of the trachea, bronchus and lung, the pneumoconiotics had a PMR of 2.22; 95% CI = 1.39, 3.37. Removing the deaths from tuberculosis, produced a PMR of 2.53 for pulmonary cancer among silicotics.

DISCUSSION

In this preliminary PMR analysis (with the known weaknesses of PMR data acknowledged), respiratory disease claims showed an expected striking excess risk for TB and nonmalignant pulmonary disease mortality. In addition, we demonstrated a significant doubling of respiratory system cancer among claims for occupational lung disease (cases). Because we could not adjust for smoking, this finding can only be considered preliminary. However, these findings support the association between silicosis and lung cancer mortality risk described by Goldsmith et al. (1988), and the consistency of this association suggests that silicosis may act as a "precursor lesion" in the risk of pulmonary cancer (Goldsmith et al., 1983). The nature of the association will become clearer as the research on the California Silicosis Registry progresses.

Table I

Number of Files Examined, Number and Proportion of Claims for Lung Disease Found from the San Francisco Office of WCAB, 1945 to 1965

Years of claim	# Files Reviewed	Claims found	Proportion/1,000
1945-49*	27,242	174	6.4
1951-55	33,321	122	3.7
1956-60	21,931	112	5.1
1962-65	18,776	57	3.0
TOTAL	101,270	465	4.6

* 1950 and 1961 are missing from current files

Table II

PMR Findings from the California Silicosis Registry: Claims of Pneumoconiosis from 1945 to 1963 Followed until 1984 (Nonmalignant Diseases Among White Males; N = 171)

CAUSE OF DEATH	OBS	EXP	PMR	95% CON INT
Tuberculosis	30	3.1	9.75	6.58, 13.92
All Circulatory Diseases	71	119.2	0.60	0.47, 0.75
All Respiratory Diseases	50	12.0	4.18	3.10, 5.51
Cirrhosis of Liver	5	3.6	1.39	0.44, 3.20
All Accidents	4	9.5	0.42	0.11, 1.07
Suicide	0	3.4	000	--- ---

Table III

PMR Findings from the California Silicosis Registry: Claims of Pneumoconiosis from 1945 to 1963 Followed until 1984 (Malignant Neoplasms Among White Males; N = 41)

CAUSE OF DEATH	OBS	EXP	PMR	95% CON INT
All Malignant Neoplasms (MN)	41	38.4	1.07	0.77, 1.45
MN of Digestive Tract	12	13.2	0.91	0.47, 1.59
MN of Pancreas	5	2.2	2.32	0.74, 5.42
MN of Respiratory System	23	10.6	2.16	1.37, 3.24
MN of Lung	22	9.9	2.22	1.39, 3.37
MN of Lymphopoietic System	3	3.3	0.90	0.18, 2.65

CONCLUSION

This preliminary assessment of the proportionate mortality risk for silicosis claims has confirmed the finding of an elevated risk for pulmonary cancer. Because this finding is preliminary and because it only represents a minority of cases expected to be in the registry when finished, some caution is needed in drawing definitive conclusions. Spoecifically, adjustment for smoking is needed, and industry-specific risks should be calculated in order to see if the risk differs according to whether there is confounding from asbestos (in construction and shipbuilding), from pyrolosis products and metal fumes (in metallurgical industries), or from radon (in mining).

In the United States there are over 3,000,000 workers exposed to silica (Frazier and Sundin, 1986) and over 100,000 patients suffering from silicosis (Wegman, 1983). In spite of the limitations of this study, several activities can and should now be undertaken:

1. Physicians who now care for silicosis patients must become aware of the consistent findings of excess lung cancer risk and transmit this information to their patients.
2. Occupational health agencies, including NIOSH, OSHA, and other public health agencies, should reexamine the current standards for silica (0.1 mg/m^3 TWA) in order to factor in the consequences of its being labelled as a probable human carcinogen by IARC (1987).
3. Additional research should now be undertaken among silica-exposed workers in order to prevent new cases of fibrotic lung disease, and to assess their risk for cancer in the absence of silicosis.

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OCCUPATIONAL ASTHMA FROM MADRAS: SOUTH INDIA

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48 cases of asthma of occupational origin were studied from industrial workers of Madras City over a period of two years. They were all from Ambathur Industry, Guindy Industrial Estate and Manali Chemical Industries. The maximum age group was 20–29 years (52%). Cotton dust, flower mills and chemicals formed 62%, 30% and 8% respectively. Lung function tests showed obstructive type of ventilatory defects. Prevention and drug therapy are discussed.

INTRODUCTION

Asthma of occupational origin is of importance, *medically* because new techniques have resulted in increased knowledge of physiopathological mechanisms involved, *occupationally* because of introduction of new chemicals.

Asthma is defined as a reversible reduction in the diameter of the bronchi which by muscular contraction hinders the passage of air. Occupational asthma is brought about by a single or repeated exposure to an active substance present in the working environment.

MATERIALS AND METHODS

About 70 doctors refer to the cases of asthma for investigation and treatment from E.S.I. Dispensaries, Madras. Cases were admitted and detailed history and clinical examinations were done—Atopy type of job, work environment and treatment already had. Blood count investigations, blood and sputum eosinophilia, chest X-ray and lung function tests were done. Intradermal skin tests were also done in the majority of the cases. Treatment with bronchodilators—oral, parenteral, aerosols were given. Desensitization of the allergen was also done. Of the total hospital admission cases, 10% were asthmatic; 10% were occupational.

Table I

Age	Male	Female	Total	%
20–29	10	15	25	52%
30–39	15	2	17	27%
40–49	6	—	6	13%
	31	17	48	

Of the 48 cases 52% were in the age group of 20–29 years, 27% were in the age of 30–39 years.

Occupations involved are tabled as:

Table II

	No.	Percentage
Cotton Dust	30	62%
Flower Mills	14	30%
Chemicals (Isocyanates)	4	8%

62% were working in cotton mills and export cotton industries.

Table III

Symptoms	No.	Percentage
Dyspnea	40	83%
Cough	18	100%
Wheezing	48	100%

Table IV

INVESTIGATION: Showed Blood and Sputum Eosinophilia

—Leukocytosis	24	50%
—Blood—Eosinophilia	48	100%
—Sputum Eosinophilia	48	100%
—Chest X-rays		
Over inflated	40	83%
Pneumothorax	2	4%
Normal	6	13%

Lung function tests were done because chest X-ray findings were not specific of asthma, and to assess the severity of respiratory impairments. It was observed that the obstructive type of ventilatory defects were due to narrowing of lung airways. The FEV_1/FVC ratio expresses the slowed expiration. Normal is 75%. Its reduction in airflow obstruction percentage results of lung function tests in cotton, flower mills and chemicals are shown in tables before and 10 minutes after solbutanol inhalation.

Table V

LFT	Cotton		Flower Mill		ChemicaL	
	B	A	B	A	B	A
FVC	1.2	1.25	1.65	1.75	2.10	2.5
FEV_1	0.67	0.67	0.90	0.90	1.65	1.75
FEV_1/FVC	57%	57	49%	59%	72	73
PER	2.2	3.3	2.3	3.4	2.4	3.6
FMEFR	0.4	0.5	0.4	0.4	0.8	0.4

Lung function tests which were done before and after exposure showed decrement of 20% predicted values in all the cases.

Treatment

Elimination of the causative agents from the environments is most ideal. Desensitization with cotton dust, flower mill dust is under trial. Routine bronchodilators with tablets, injections or aerosols, steroids and chromolynsodium are also being tried.

Occupational asthma is really a problem in our industrial country. Identifying the allergen in the working environment, removal of the causative agent and better healthier conditions of the industrial worker will augment the industrial output and the country will certainly prosper.

MANAGEMENT OF ASTHMA

Occupational asthma can be prevented by reduction of air-dusts in the workplaces by exhaust ventilation and hoods or enclosures. Reallocation to different works, though advised, is difficult because of the special training and the job skills they require. In the manufacturing process proteolytic enzymes may be added to change the allergen. Encapsulation into inert compounds and substitution for chemical powders by paste or solutions can be the ideal way of preventing occupational asthma.

Drugs used only for symptomatic relief of the attacks are:

1. Sympathomimatic (adrenergic) Epinephrine .5 ml. of 1:1000 by subcutaneously Ephedrine—50 mg. Orciprenaline sulphate—tab.10 mg. t.d.s. .5ml I.M. or I.V. drops.

2. Beta—adrenergic drugs—advantage it orally gives; sustained action, sympathomimatic side—effects are absent. Hence replaced sympathomimatic drugs. SALBUTAMOL —4 mg. tab. twice daily. Terbetaline sulphate 2.5 mg. tab.

3. Xanthenis are widely used: effective

Amino Phylline	.25 gm.	10 ml. I.V. twice daily
Theophylline	50 mg.	$\frac{\chi}{\chi}$ I.M. or I.V. twice daily
Etophyline	150 mg.	χ

4. Anticholinergic—Atropine sulphate given orally or in drip—Not used because of drying up of mucous secretion and further aggrevating bronchospasm.

5. Mast-cell Stabilizers—CROMOLYN SODIUM can be given for prophylaxis.

6. Corticosterods—oral prednisolone 30–60 mg. t.d.s. betamethazone .5 mg tab.

   4 mg/ml.—I.V. drip; Dexametha Zone 4 mg/ml. can be given; found to be less effective compared with Betamethazone studies; not effective for immediate reactions.

7. Inhalation—AEROSOL.

   Adrenaline by nebulizers or intermittent positive Breathing device (IPBD) inhalation.

   —SALBUTAMOL—100 mcg/per metered dose.
   —TERBETALINE—0.25 mg/per metered dose.
   —BETAMETHAZONE—50 mgg/inhalation
   —CROMOLYN SODIUM—inhalation.

8. ANTIBIOTICS, OPIATES, SEDATIVES, TRANQUILIZERS, EXPECTORANTS MUCOLYTIC AGENTS, I.V. FLUIDS can be used if needed.

9. Desensitization for the specific allergen can also be tried.

STATUS ASTHMATICUS: can be due to respiratory infections, respiratory depressants of early withdrawal of stands. Blood gas analysis, PH may help in addition to chest X-ray—Oxygen inhalations, adequate hydration, expectorants, antibiotics and physiotherapy will help these patients remarkably.

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LUNG MECHANICS IN ANTHRACITE COAL WORKERS' PNEUMOCONIOSIS

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INTRODUCTION

Anthracite has been traditionally in wide use both at home and in industry in Korea and, therefore, anthracite mining has always been one of the major sources of pneumoconiosis. The prevalence rate of pneumoconiosis in anthracite mines reaches at present 14.5% among coalface workers and even 24.3% among rock drillers. Coal mine dust from anthracite mines may have higher concentrations of silica than that found in bituminous coal mines. Morgan[1] reported the existence of higher prevalence rates for both simple and complicated coal workers' pneumoconiosis (CWP). The essential pathological lesion of CWP is the coal macula with its attendant focal emphysema and a little surrounding fibrosis. CWP in miners from the anthracite region is associated mainly with the presence of coal macula but also with smaller number of silicotic nodule.[2] As these lesions involve only the respiratory bronchiole, they could potentially compress or constrict the lumen of the bronchiole. Studies in coal miners with simple bituminous CWP have demonstrated small airway obstruction and slight changes in lung mechanics. However, these physiologic evidences are not detected in coal miners with simple anthracite CWP. The present study was planned to investigate the physiologic evidence of small airway obstruction and significant abnormalities of lung mechanics in anthracite coal miners with CWP.

SUBJECTS AND METHODS

The subjects of this study comprised 178 anthracite coal miners with CWP and 25 normal subjects as a control group. Miners with significant heart or lung disease were excluded. Miners with CWP were divided into two groups. The first group consisted of 148 miners with suspected or simple CWP. This group was selected on the basis of an absence of significant impairment of ventilatory capacity as defined by a $FEV_{1.0}$ to FVC ratio of not less than 70%. The second group consisted of 30 miners with progressive massive fibrosis (PMF). Many of these subjects had airway obstruction ($FEV_{1.0}$/FVC <70%).

Spirometry was performed using a waterless electronic spirometer attached to Autobox system (model CS-828 FC, Chest Co., Japan). Lung volumes and airway resistance were measured in a constant pressure (flow displacement type) both plethysmograph of Autobox system by the methods of DuBois et al.[3] and Woitowitz and Buchheim.[4]

Compliance was obtained as the ratio of the change in volume to the change in transpulmonary pressure. Transpulmonary pressure was obtained by electronically subtracting mouth pressure from esophageal pressure, which was measured by a 10 cm later balloon attached by polyethylene tube to a pressure transducer. The tip of the balloon was positioned approximately 40–45 cm from the nares. Static compliance was measured from the linear portion, just above FRC, of the expiratory limb of the pressure-volume curve obtained over the full VC range. Pulmonary recoil pressure at TLC (Pst TLC) was also obtained from this curve. The coefficient of retraction was calculated by dividing Pst TLC by TLC. Dynamic compliance was measured at respiratory rates of approximately 30 (0.5 Hz), 42 (0.7 Hz), 54 (0.9 Hz) and 72 (1.2 Hz) breaths/min, and calculated by dividing the change in volume by the corresponding change in transpulmonary pressure at points of zero flow over five respiratory cycles and taking the mean value. Frequency dependence of compliance was defined as a fall in Cdyn/initial Cdyn to less than 80%. All measurements were performed in the sitting position.

RESULTS AND DISCUSSION

The subjects with category 3 and PMF were significantly older than those with control and suspected CWP. The subjects with PMF had significantly worked longer underground than those with suspected CWP. (Table I)

Lung function datums of the subjects are illustrated in Table II. No significant differences were found for all measurements among the groups with control and each category of simple CWP. However, RV tended to be rather higher than predicted in all categories of simple CWP. Mean values of $FEV_{1.0}$ and $FEV_{1.0}$ /FVC were abnormally reduced in all categories of PMF, and those of RV and RV/TLC were significantly lower in PMF than in simple CWP. These findings might be related to the airway obstruction.

Mean results of lung mechanics of the subjects are recorded in Table III. Static compliance (Cst), specific compliance (SCst), pulmonary recoil pressure at TLC (Pst TLC) and coefficient of retraction in individual subjects are illustrated in Figures 1, 2, 3 and 4. In suspected or simple CWP, mean values of airway resistance, Cst, Pst TLC and coefficient of retraction were normal. There were no significant differences in these measurements between control group and CWP group. Static compliance was not related to the category of simple CWP and also, no relationship was observed between specific compliance and increasing

Table I

Characteristics of the Subjects by Radiographic Finding of Coal Workers' Pneumoconiosis

(Mean±SD)

Radiographic finding	No. of subjects	Age (yr)	Height (cm)	Weight (kg)	BSA (m^2)	Cigarettes (pack yr)	Time underground (yr)
Controls	25	41.0 ±11.3	168.4 ±5.5	59.8 ±6.8	1.68 ±0.11	17.9 ±12.3(19)	
Suspected	24	43.9 ±6.6	165.2 ±5.0	60.6 ±6.4	1.66 ±0.09	13.6 ±6.6(19)	14.4 ±6.3
SRO	124	45.5 ±7.2	165.2 ±5.5	59.9 ±6.9	1.66 ±0.10	15.3 ±9.7(105)	16.4 ±6.2
Category 1	66	44.9 ±7.5	164.8 ±5.7	59.9 ±6.4	1.65 ±0.10	15.0 ±7.7(56)	15.9 ±6.3
2	41	45.1 ±7.0	165.1 ±5.2	60.3 ±8.0	1.66 ±0.11	16.5 ±11.2(35)	16.3 ±6.1
3	17	48.9 ±5.5	166.7 ±5.4	59.1 ±6.0	1.66 ±0.09	13.7 ±7.3(12)	19.0 ±5.0
LO	30	51.3 ±5.4	164.4 ±6.0	57.8 ±8.0	1.63 ±0.13	9.3 ±10.7(12)	18.2 ±6.4

BSA: body surface area, SRO: small rounded opacity, LO: large opacity, Pack yr: packs of cigarettes per day×smoking years. Parentheses indicated the number of current smoker.

Table II

Lung Function Data in Controls and Miners with Coal Workers' Pneumoconiosis

Radiographic finding	No. of subjects	VC (% pred.)	$FEV_{1.0}$ (% pred.)	$FEV_{1.0}$/FVC(%)	FRC (% pred.)	RV (% pred.)	TLC (% pred.)	RV/TLC (%)	RV/TLC (% pred.)
Controls	25	107.9 ±9.1	100.8 ±11.1	83.7 ±5.6	104.3 ±9.1	129.2 ±25.8	106.4 ±9.1	30.7 ±6.5	107.0 ±15.8
Suspected	24	109.1 ±11.6	100.0 ±10.5	80.6 ±5.9	110.3 ±18.5	139.2 ±32.4	111.4 ±15.4	32.1 ±6.2	109.3 ±18.9
SRO	124	107.7 ±12.5	99.0 ±12.3	79.6 ±5.7	110.1 ±13.3	137.8 ±27.1	107.9 ±10.2	32.7 ±5.7	109.3 ±18.2
Category 1	66	109.6 ±11.6	100.5 ±12.0	79.9 ±5.9	110.6 ±12.1	138.4 ±27.7	109.2 ±9.3	32.2 ±5.8	108.3 ±18.9
2	41	106.3 ±12.8	96.1 ±11.5	78.3 ±5.3	110.1 ±15.5	136.6 ±27.0	106.5 ±11.6	32.6 ±5.3	109.7 ±17.2
3	17	103.9 ±14.7	100.4 ±14.5	81.8 ±5.4	108.3 ±12.4	138.2 ±25.9	106.3 ±9.8	35.0 ±6.1	112.0 ±18.9
LO	30	91.2 ±19.3	66.7 ±25.2	62.5 ±15.9	109.8 ±18.1	159.6 ±43.1	103.6 ±14.7	41.8 ±9.3	128.9 ±27.2
Category A	12	93.1 ±21.2	73.0 ±23.4	66.2 ±13.6	106.5 ±23.3	157.7 ±52.3	104.1 ±17.4	40.4 ±10.0	126.4 ±31.3
B	12	91.8 ±18.2	70.4 ±28.8	64.4 ±15.5	111.9 ±13.6	159.1 ±38.1	104.1 ±12.7	41.7 ±8.8	130.6 ±25.8
C	6	86.1 ±20.0	47.0 ±8.9	51.8 ±18.5	112.5 ±16.1	164.5 ±39.1	101.6 ±15.1	44.7 ±9.6	130.6 ±25.1

Table III

Lung Mechanics in Controls and Miners with Coal Workers' Pneumoconiosis

Radiographic finding	Raw ($cm H_2O/l/sec$)	MBC (% pred.)	Cst ($l/cm H_2O$)	SCst ($l/cm H_2O/l$)	Cdyn ($l/cm H_2O$)	Pst TLC ($cm H_2O$)	Coeff. retract. ($cm H_2O/l$)
Controls	1.16 ±0.24		0.222 ±0.050	0.062 ±0.020	0.186 ±0.053	29.19 ±7.12	4.850 ±1.210
Suspected	1.32 ±0.46	112.5 ±11.5	0.244 ±0.061	0.067 ±0.011	0.187 ±0.058	26.93 ±7.11	4.517 ±1.419
SRO	1.25 ±0.29	107.3 ±14.5	0.229 ±0.069	0.062 ±0.016	0.179 ±0.054	30.30 ±11.51	5.177 ±2.211
Category 1	1.25 ±0.29	109.6 ±14.9	0.239 ±0.067	0.064 ±0.015	0.182 ±0.049	27.52 ±8.79	4.627 ±1.613
2	1.27 ±0.34	104.0 ±13.3	0.225 ±0.072	0.061 ±0.017	0.180 ±0.058	31.97 ±13.57	5.558 ±2.694
3	1.20 ±0.18	106.1 ±14.7	0.201 ±0.060	0.054 ±0.015	0.161 ±0.062	37.03 ±12.55	6.395 ±2.381
LO	2.96 ±2.35	76.7 ±26.6	0.185 ±0.080	0.046 ±0.020	0.132 ±0.068	34.65 ±13.77	6.510 ±3.447
Categery A	2.88 ±2.82	81.2 ±22.5	0.195 ±0.089	0.051 ±0.021	0.136 ±0.056	28.97 ±14.22	5.591 ±3.886
B	2.66 ±2.07	81.3 ±31.5	0.214 ±0.088	0.051 ±0.016	0.160 ±0.078	34.06 ±12.90	6.216 ±3.215
C	3.74 ±2.06	52.9 ±9.5	0.106 ±0.025	0.027 ±0.068	0.071 ±0.016	47.20 ±4.76	8.935 ±1.915

Raw: airway resistance, MBC: maximal breathing capacity, Cst: static compliance, SCst: specific compliance (Cst/FRC), Cdyn: dynamic compliance during quiet breathing (23 breaths/min), Pst TLC: static pulmonary recoil pressure at TLC, Coeff. retract: coefficient of retraction (Pst TLC/TLC)

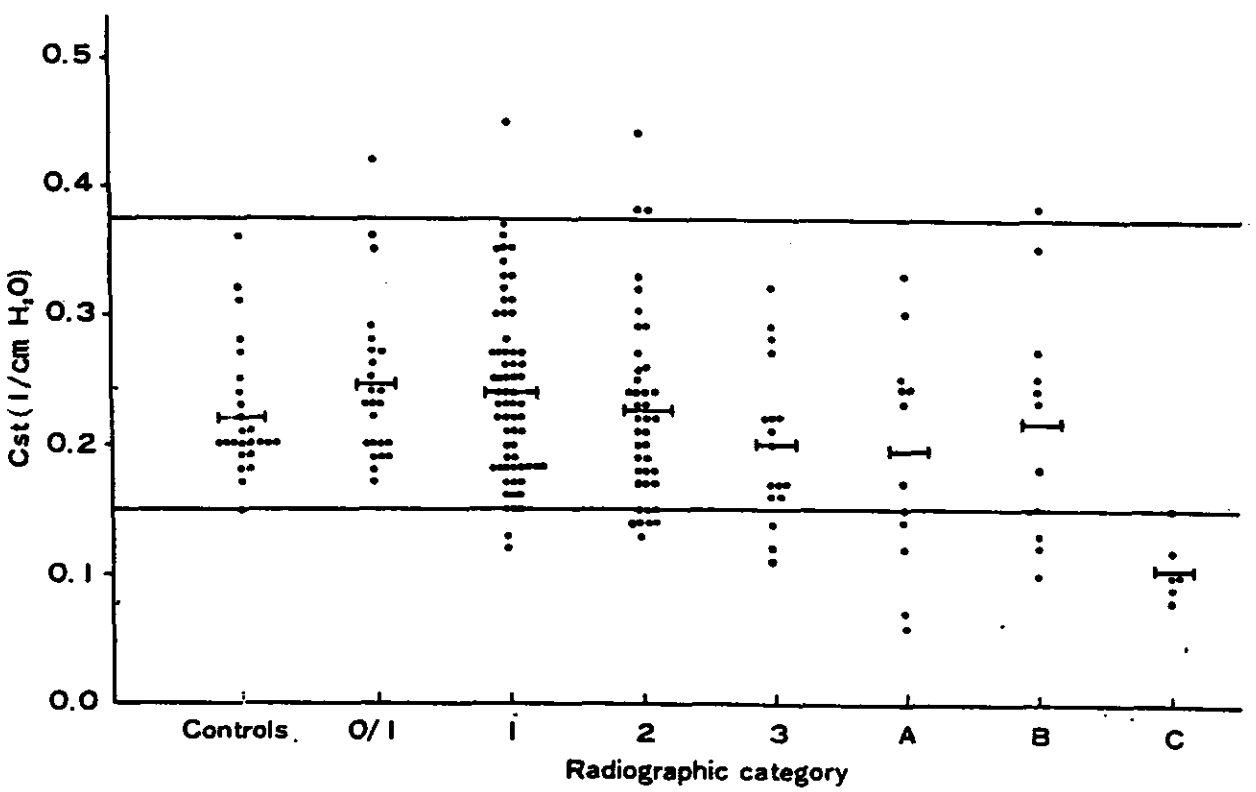

Figure 1. Static compliance in controls and miners with different radiographic categories of coal workers' pneumoconiosis (CWP). Horizontal line represented the normal range and horizontal bar represented the mean value.

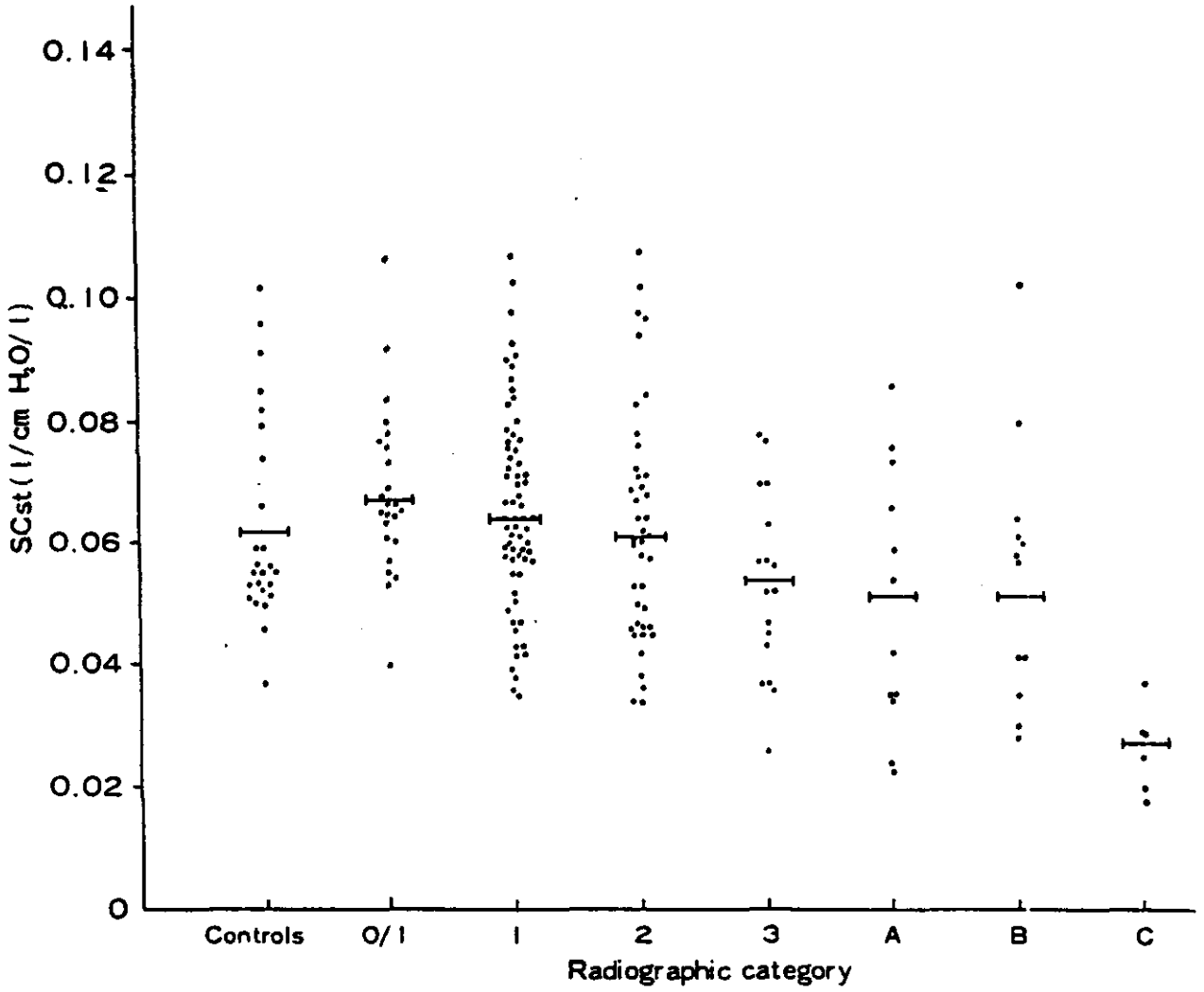

Figure 2. Specific compliance in controls and miners with different radiographic categories of CWP. Horizontal bar represented the mean value.

category of simple CWP. Dynamic compliance at quiet breathing tended to be somewhat lower than static compliance in all categories. Mean coefficient of retraction and number of individuals with higher coefficient tended to increase with increasing category. Subjects with abnormally high coefficient showed 9.1%, 14.6% and 41.2% in category 1, 2 and 3, and those with abnormally low coefficient 22.7%, 14.6% and 0% in category 1, 2 and 3. Pst TLC had the same tendency as coefficient.

In PMF, airway resistance was abnormally increased and mean values of Cst, Pst TLC and coefficient of retraction were normal except category C. A large number of miners fell outside the normal range of Cst and most of these showed values lower than normal, and this was most marked in miners with category C. Dynamic compliance at quiet breathing was lower than static compliance, as in suspected or simple CWP. Similarly, in category C, 5 and 4 of 6 miners showed values of Pst TLC and coefficient higher than normal.

Since the essential pathological lesion of CWP is the coal macula with its attendant focal emphysema and a little surrounding fibrosis, the physiological abnormalities might be detected by the measurements associated with emphysema. These might include an increase in RV and alterations in lung mechanics, such as a high compliance and a low pulmonary recoil pressure and coefficient of retraction. However, if significant interstitial fibrosis were present in the coal macula, these latter changes could be masked and demonstrate a low compliance and a high pulmonary recoil pressure and coefficient.

In a detailed study of lung mechanics in CWP, Ferris and Frank[5] found compliance to be somewhat reduced in subject with complicated CWP. In the subjects with no or simple CWP, there was a wide scatter of compliance value. Seaton et al.[6] studied the lung mechanics in bituminous miners with CWP. In the subject with simple CWP, static compliance was mostly in the normal range, whereas it was

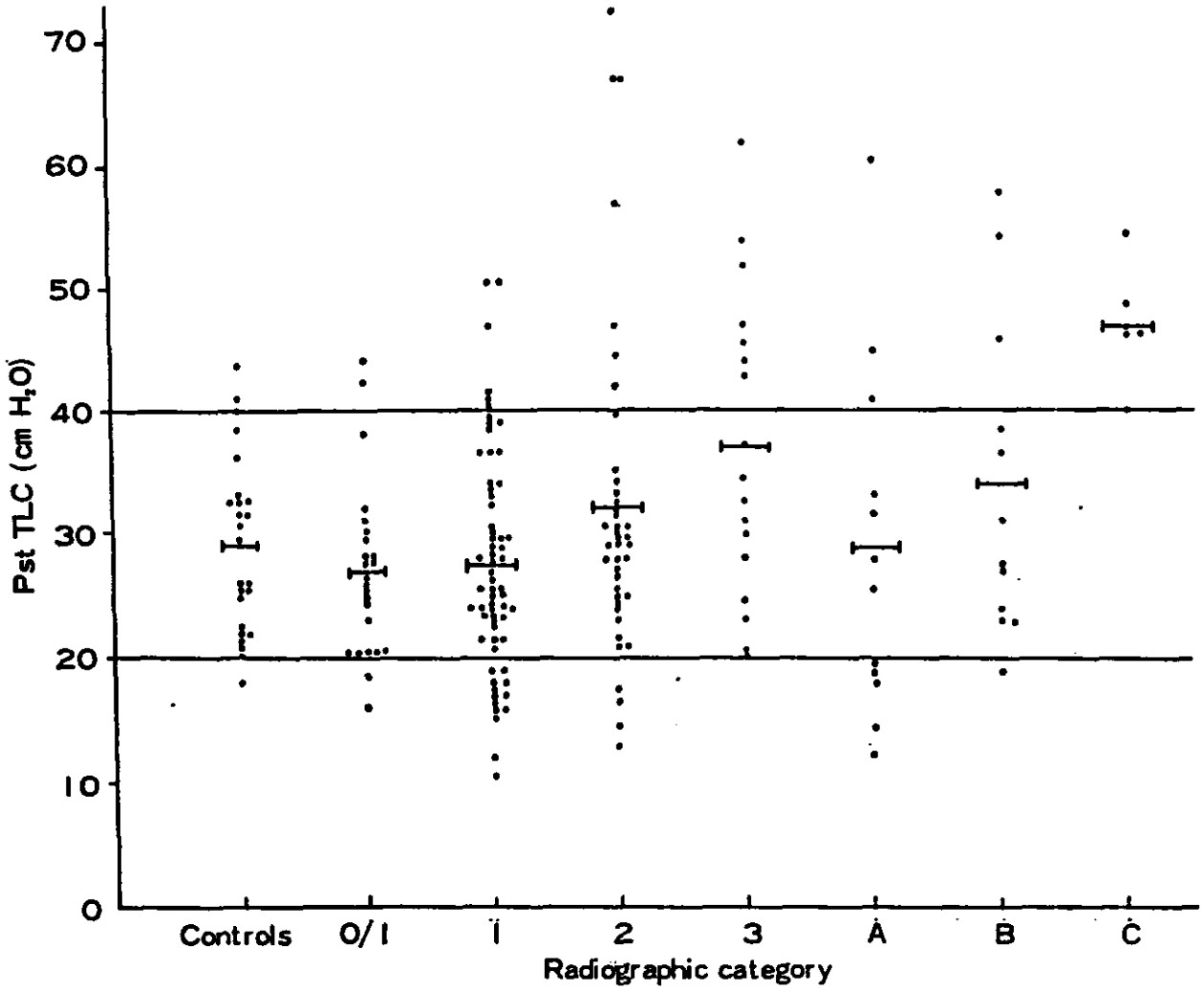

Figure 3. Static pulmonary recoil pressure at total lung capacity in controls and miners with different radiographic categories of CWP. Horizontal line represented the normal range and horizontal bar represented the mean value.

often reduced in complicated CWP. The coefficient of retraction was normal or reduced in most subjects except those with category B and C PMF. They noted that so far as simple CWP was concerned, abnormalities reflected emphysema rather than fibrosis and in severe PMF, changes suggesting fibrosis tended to predominate. Murphy et al.[7] studied a group of 20 miners with simple anthracite CWP and found no significant alterations in lung mechanics. They pointed out that the absence of decreased maximum expiratory flow, a normal coefficient, and a normal specific compliance made significant emphysema most unlikely.

Our findings studied anthracite miners with CWP were similar to the results obtained by the above authors and found a negative correlation between coefficient of retraction and RV, FRC and RV/TLC in miners with suspected or simple CWP. As these measurements correlated with the pathological demonstration of emphysema,[8] a low coefficient may well also be a guide to the presence of emphysema.

If interstitial fibrosis were the predominant change in miners, they should have demonstrated reduced compliance and elevated coefficient and pulmonary recoil pressure. Among the subjects with simple CWP, the number of individuals with abnormally reduced coefficient tended to be more than those with elevated coefficient in category 1 and individuals with elevated coefficient were more than those with reduced coefficient. Among the subjects with PMF, especially category C, most of them had reduced compliance and elevated coefficient. These findings suggested that in simple anthracite CWP, the abnormalities tended to reflect focal emphysema rather than fibrosis in category 1 and fibrosis rather than emphysema in category 3, and in severe PMF, fibrosis tended to predominate.

As the coal macula and the silicotic nodule which are characteristic histological findings of anthracitic CWP are found in relation to the respiratory bronchiole, they could potentially compress or constrict the lumen of the bronchiole.

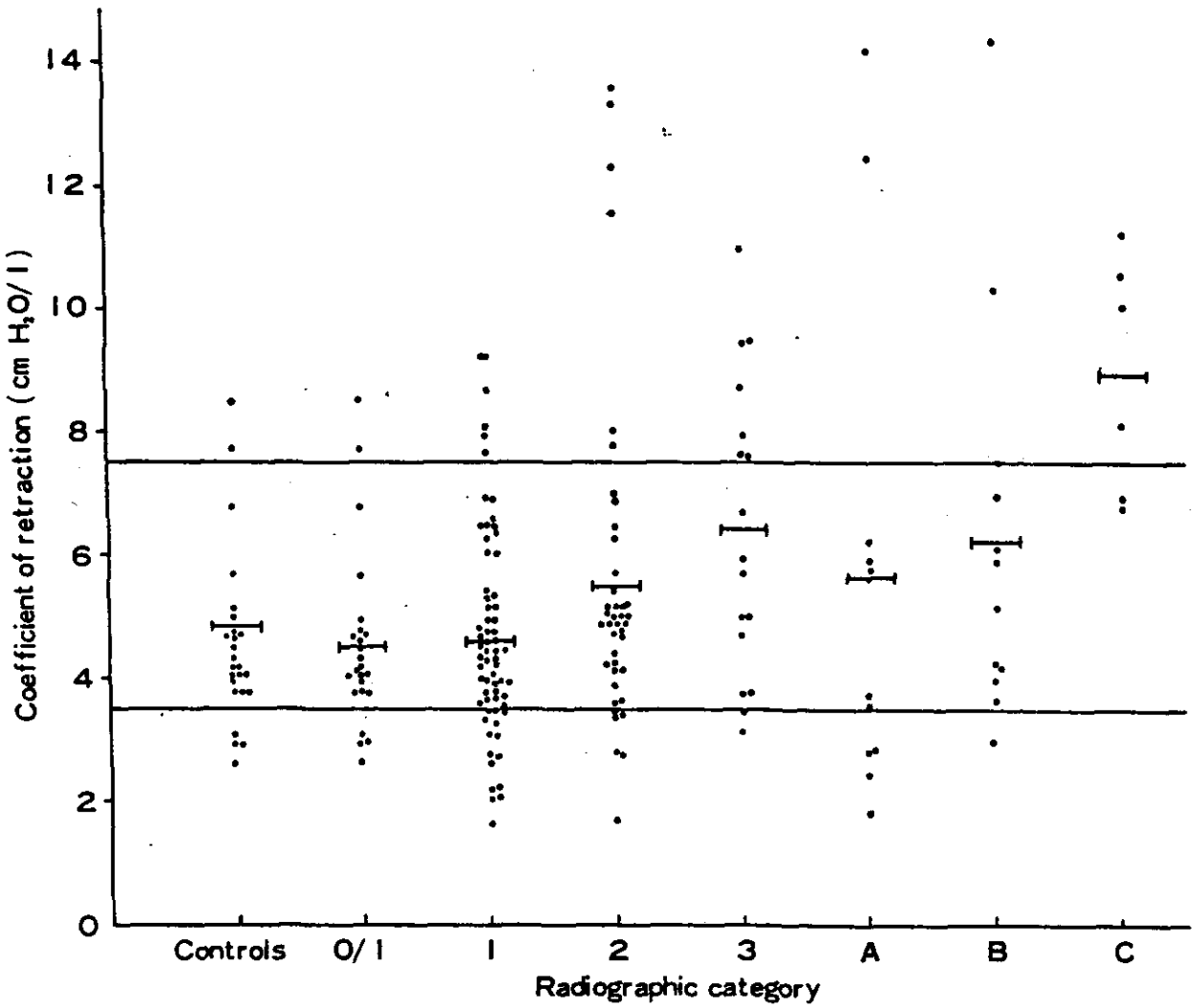

Figure 4. Coefficient of retraction in controls and miners with different radiographic categories of CWP. Horizontal line represented the normal range and horizontal bar represented the mean value.

The technique available to assess the presence of abnormal resistance to airflow through the small airways is the measurement of compliance at increasing respiratory rates.[9] It has been assumed that regional differences in elastic properties sufficient to cause a detectable fall in dynamic compliance at rapid respiratory rates should result in an abnormal static compliance curve. Thus, if a patient has normal pulmonary resistance, spirometry and static pressure-volume curve, a fall in dynamic compliance at faster rates of respiration (frequency dependence of compliance) is most probably due to peripheral airways obstruction. Seaton et al.[6] and Lapp and Seaton[10] noted that 17 of the 25 working bituminous miners with simple CWP demonstrated frequency dependence of compliance, and that this finding did not relate to bronchitis and type of opacity. However, Murphy et al.[7] were unable to detect significant changes in either small airway narrowing and lung mechanics in miners with simple anthracite CWP.

In our study, miners with simple anthracite CWP showed a fall (less than 80%) in dynamic compliance at faster rates of respiration, compared to those with control and suspected CWP. There was no relationship between a change in dynamic compliance at faster rates of respiration and category of simple CWP. (Table IV. Figure 5) In 96 suspected or simple CWP miners with normal spirometry and static pressure-volume curve, the frequency dependence of compliance was observed in 46.9% at 55 breaths/min and in 62.5% at 76 breaths/min. This finding appeared to be the result of increased resistance to airflow in small peripheral airways but was not related to the category of opacity. (Table V)

SUMMARY

The mechanical properties of the lungs were studied on 25 normal subjects, 148 anthracite miners with suspected or simple coal workers' pneumoconiosis (CWP) and 30 miners with progressive massive fibrosis (PMF). The mean values of

Table IV

Dynamic Compliance at Different Respiratory Rates in Controls and Coal Workers' Pneumoconiosis Miners with Normal Pressure-Volume Curve

(l/cm H_2O)

Radiographic category	No. of subjects	Respiratory rate (breaths/min)				
		23	31	43	55	76
Controls	21	0.185±0.054	0.176±0.055 (94.5±8.1)	0.169±0.057 (90.6±9.3)	0.161±0.052 (86.4±8.2)	0.151±0.051 (81.1±9.2)
Suspected	17	0.181±0.040	0.167±0.038 (92.5±8.5)	0.161±0.039 (89.4±15.9)	0.156±0.034 (87.2±15.9)	0.144±0.035 (80.2±16.1)
SRO	79	0.179±0.040	0.163±0.037 (91.4±11.7)	0.157±0.040 (87.8±13.7)	0.145±0.039 (81.2±15.8)	0.124±0.037 (69.4±15.8)
Category 1	46	0.179±0.038	0.165±0.037 (92.2±11.5)	0.159±0.039 (88.4±13.6)	0.142±0.038 (79.5±16.7)	0.120±0.033 (67.6±15.5)
2	26	0.178±0.041	0.159±0.034 (90.7±11.0)	0.154±0.038 (87.9±14.6)	0.147±0.039 (83.4±14.8)	0.126±0.040 (73.4±18.3)
3	7	0.187±0.050	0.166±0.047 (89.4±17.2)	0.157±0.052 (83.5±10.7)	0.156±0.047 (83.5±14.7)	0.137±0.049 (73.4±18.3)

Parentheses indicated the dynamic compliance expressed as a percentage of the initial (23 breaths/min) dynamic compliance.

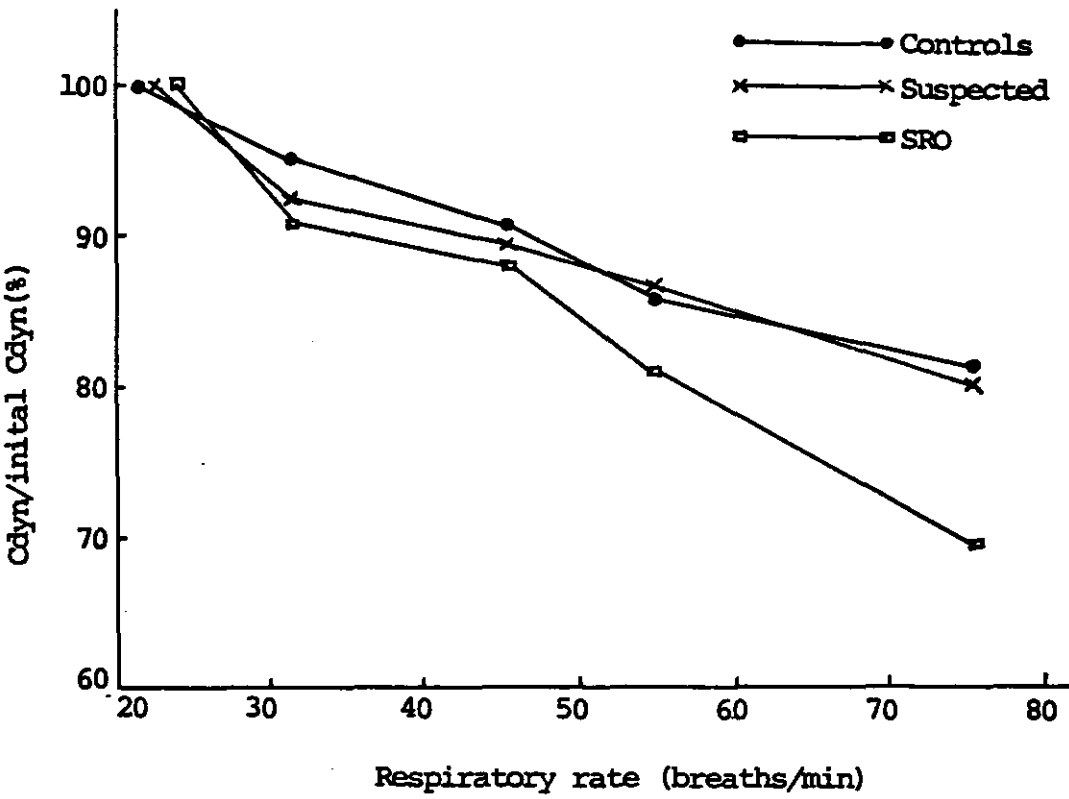

Figure 5. Dynamic compliance with different respiratory rates expressed as a percentage of the dynamic compliance during quiet breathing in controls and suspected or simple CWP miners with normal pressure-volume curve.

Table V

Number of Subjects with Frequency Dependence of Dynamic Compliance in Different Respiratory Rates by the Category of Coal Workers' Pneumoconiosis

Radiographic category	No. of subjects	Respiratory rate (breaths/min)			
		31	43	55	76
Controls	21	—	2 (9.5)	3(14.3)	5(23.8)
Category 0/1	17	1 (5.9)	6(35.3)	7(41.2)	10(58.6)
1	46	6(13.0)	9(19.6)	24(52.2)	32(69.6)
2	26	3(11.5)	8(30.8)	11(42.3)	14(53.8)
3	7	2(28.6)	2(28.6)	3(42.9)	4(57.1)
Subtotal	96	12(12.5)	25(26.0)	45(46.9)	60(62.5)

Frequency dependence of dynamic compliance was defined as a fall in Cdyn/initial Cdyn to less than 80 % (Seaton *et al.*, 1972a). Parentheses inidicated the percentage.

static compliance, pulmonary recoil pressure at total lung capacity and coefficient of retraction were normal in all categories of CWP. The static compliance was within normal range in most miners with suspected or simple CWP, whereas it was often reduced in miners with PMF, particularly in category C. The number of miners with higher coefficient of retraction tended to increase in parallel with the progression of pneumoconiosis in terms of the category of opacity. The coefficient of retraction was mostly elevated in miners with category C among PMF. In simple CWP, the abnormalities of lung functions concerned with gas exchange tended to reflect focal emphysema rather than fibrosis in category 1, and fibrosis rather than focal emphysema in category 3. In severe PMF (especially category C), fibrosis tended to predominate. In 96 suspected or simple CWP miners with normal spirometry and static pressure-volume curve, the frequency dependence of compliance was observed in 45 miners (46.9%) at 55 breaths/min and in 60 miners (62.5%) at 76 breaths/min. This finding appeared to be the result of increased resistance to airflow in small peripheral airways.

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