REPORT

MEETING ON THE EARLY DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS

Tokyo, Japan
22-24 September 1988

Manila, Philippines
January 1989
REPORT

MEETING ON THE EARLY DIAGNOSIS
AND TREATMENT OF PNEUMOCONIOSIS

Convened by the
REGIONAL OFFICE FOR THE WESTERN PACIFIC
OF THE
WORLD HEALTH ORGANIZATION

Tokyo, Japan
22-24 September 1988

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NOTE

The views expressed in this report are those of the participants in the Meeting on the Early Diagnosis and Treatment of Pneumoconiosis and do not necessarily reflect the policies of the World Health Organization.

This report has been prepared by the Regional Office for the Western Pacific of the World Health Organization for governments of Member States in the Region and for the participants in the Meeting on the Early Diagnosis and Treatment of Pneumoconiosis held in Tokyo, Japan, from 22 to 24 September 1988.
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1. INTRODUCTION

1.1 Background

Pneumoconioses are common occupational diseases in most countries in the Western Pacific Region. The epidemiological surveys conducted in China and the Republic of Korea reported that the prevalence of different dust-borne lung diseases among workers exposed to dust varies from 3.5% to 8.3% going up to even 10% in some small-scale industries. There is no doubt that prevention of pneumoconiosis through the improvement of the working environment and control of dust is of paramount importance and much effort is still needed to improve the situation further. One should, however, pay more attention to improving the existing situation and finding new methods of early diagnosis and treatment of pneumoconioses.

The Western Pacific Advisory Committee on Health Research at its twelfth session held in Manila in March 1988, approved the recommendation of the Subcommittee on Research in Occupational Health, 14-16 December 1987, and recommended that research on occupational health should be included in the list of regional priorities for the health research programme. Among other problems, pneumoconioses or dust lung diseases, are a major concern.

The WHO has taken a lead in the research and development of early diagnosis and treatment of pneumoconiosis. It has cooperated with China, Japan and the Republic of Korea in developing a multicentre collaborative study on the use of bronchofibroscopy for the early detection of pneumoconiosis.

It is proposed to have a meeting on the early diagnosis and treatment of pneumoconiosis. This meeting will work out the modus operandi of collaboration between China, the Republic of Korea, Japan and WHO for developing research for the purpose mentioned above.

1.2 Objectives

1.2.1 To review the present status of etiopathology, early diagnosis and treatment of pneumoconiosis and related diseases and identify problems and future directions.

1.2.2 To formulate a protocol and workplan on collaborative research on early diagnosis and treatment of pneumoconiosis.

1.3 Participants

Four experts from China, three from Japan and four from the Republic of Korea and three temporary advisers attended the meeting (see Annex 1). In addition, members of the Small Airway Study group of Occupational Lung Diseases, Keio University (also listed in Annex 1), were present and also made technical contributions to the meeting.
1.4 Agenda

The programme of the meeting was organized with special emphasis on the review of the present status of studies on pneumoconiosis and the multicentre collaborative research project on early diagnosis and treatment of pneumoconiosis. On the second day, 23 September, the meeting was addressed by Dr Hiroshi Nakajima, Director-General of WHO (see Annex 2).

2. SUMMARY OF DAILY PROCEEDINGS

2.1 Thursday, 22 September

2.1.1 Working session

The meeting was opened by Dr Wang Liansheng, Regional Adviser in Organization of Medical Care, WHO/WPRO, Manila. Dr Wang welcomed the participants of the meeting and introduced the main objectives of the meeting: to develop a protocol to promote collaboration between China, Republic of Korea and Japan in research on pneumoconiosis.

2.1.2 Election of officers

The following officers were elected by acclamation:

Chairman - Dr Shozo Hashimoto, Keio University (Tokyo)
Vice-Chairmen - Dr Kyu Sang Cho, Catholic University Medical College (Seoul)
                  Dr Liu Shijie, Beijing Medical University (Beijing)
Rapporteur - Dr Stephen I. Rennard, WHO Temporary Adviser

2.1.3 Country report presentations

Dr Kyu Sang Cho began the scientific discussion summarizing the limits of the definition of pneumoconiosis. He pointed out that the disease can be histologically heterogenous and that the response to different dusts can be quite varied. Reasons for the varied response are that some dusts are very "fibrogenic", others can induce granuloma formation, while other dusts are relatively bland. Lack of understanding of the response to individual dusts and to the mixed exposures frequently encountered in industry raises serious problems in public health management. Specifically, it is not currently possible to define "tolerable"
exposure levels nor to plan programmes to alter industrial operations to limit exposures based on any definitive evidence of health benefit. Finally, Dr Kyu Sang Cho pointed out that little is known about other factors which can interact with dust exposure including other concurrent exposures, cigarette smoking, and individual subject variation. The need for early reliable diagnosis was felt to be crucially important.

Dr Im Goung Yun, Seoul, continued the discussion, pointing out that lack of early diagnostic criteria complicated the administration of workers' benefits programmes. These programmes must be based on a definition of disease severity and physical disability. The application of such programmes represented a tremendous social effort. He suggested that application of fibro-optic bronchoscopy may prove to be an accurate, reliable and sensitive diagnostic technique in dust-exposed patients. The current Korean pneumoconiosis classification system, as it is applied to industrial health workers management and administration of benefits was discussed by Dr Lee, Catholic Industrial Medical Centre. He pointed out that current methods may not use the best technology, especially since fibro-optic bronchoscopy is not included.

Dr Kyung Chung Chee, Seoul, presented data on pulmonary functions. The most frequently observed abnormalities in the most mild classifications of pneumoconiosis were consistently measures of small airways function. Flow volume loops, measurements of maximal mid-expiratory flow (MMEF), the maximal flow at 75% of vital capacity (Vmax 75), dynamic compliance (particularly at higher frequencies) and closing volumes, either the slope of the phase III or the transition of phase III to IV were all abnormal at early stages of pneumoconiosis.

Dr Toshiaki Higashi, Kitakyushu, discussed the Japanese classification of pneumoconiosis and compared it and the screening procedures used with the Korean system.

Dr Liu Shijie, Beijing, presented an overview of pneumoconiosis in China. He described the magnitude of the problem with around 10 000 000 workers exposed in large industrial operations and 80 000 000 exposed in small operations. At least 13 hazardous exposures are recognized and perhaps as many as 23 exist. While dust levels have been controlled mainly in large state-owned enterprises in the past, there is little scientific basis for the target dust levels chosen. He also presented an overview of pneumoconiosis research in China, which is conducted in 170 institutes throughout the country.
Dr Lu Shixuan, Beijing, reviewed the new classification system used in China since 1984. He compared it with the ILO classification system and described the development of new standard "boundary" X-rays. These films, combined with a "mid-range" standard film corresponding to the old ILO classification, were thought to allow for a more uniform application of a classification system.

Dr Hu Tianxi, Shanghai, summarized both animal and human studies of several possible therapeutic agents. These include: PVNO; piperaquine phosphate; hydroxypiperaquine phosphate; tetrandrine; and aluminum citrate. These agents appeared able to block collagen deposition in lung tissue and to improve histologic grades in animal studies. A beneficial effect was noted with PVIII in several human trials, although "rebound" when the drug was stopped was noted.

Dr Jiang Hui-Xin, Shanghai, presented a summary of animal and in vitro studies demonstrating that fibrogenic dusts have a toxic effect on macrophages and induce macrophage oxidant release. More fibrogenic dusts are more potent inducers of macrophage oxidant production suggesting that in vitro analysis may form a basis by which to gauge the hazards of a potential exposure. Therapeutic agents can also be tested by their ability to modify the in vitro system.

Dr Kohda, Tokyo, discussed the benefits of X-ray analysis. A representative of Shimazu Ltd. reviewed high voltage X-ray technology. A representative of Fuji films discussed X-ray film history and current capability. Although automated developing would help improve uniformity, it was felt to be expensive and not generally practical. Considering the wide geographical areas included in the protocol, Dr Lu suggested adopting a high temperature development technique.

Dr Cho then presented for discussion a draft protocol of collaborative research prepared by the secretariat based on research proposals of institutions. Dr Rennard and Dr Basset suggested that careful attention should be given in the analysis of the effects of smoking and of bronchitis. The members of the meeting generally accepted the major objectives, project areas and subjects for studies, responsibilities of each institution. The members agreed that each institution will develop and implement its individual project in conformity with the multicentre collaborative research project.

2.2 Friday, 23 September

Dr Shozo Hashimoto, Keio University, Tokyo, welcomed the delegates. He emphasised the importance of the problem of pneumoconiosis and hoped that the application of new technology, particularly fibro-optic bronchoscopy would be helpful in the management of pneumoconiosis.
Dr Hiroshi Nakajima, Director-General of WHO, made the keynote address reviewing the growing importance of pneumoconiosis, particularly in developing countries. He underscored the limitations in current understanding and methods. He emphasized the commitment of WHO to the goals of the meeting: developing methods for understanding the pathogenesis, establishing the early diagnosis and developing therapies for pneumoconiosis. These goals would permit both the management of individual affected workers and a rational programme of industrial hygiene. WHO is committed to the evaluation and application of new technologies, such as fibro-optic bronchoscopy, and of traditional remedies which hold considerable promise for the pneumoconioses.

Dr Liu Shijie, Beijing, gave an overview of the pneumoconioses in China. He discussed the importance of the problem from a public health and social point of view and reviewed both laboratory and epidemiological studies performed in the institutes engaged in pneumoconiosis research in China.

Dr Kyu Sang Cho, Seoul, reported on epidemiological studies of anthracite coal workers in Korea. He presented data showing considerable variations in the total dust, respirable dust, and silica content in mines in Korea. A chest X-ray study of 12700 workers revealed an increasing incidence of abnormalities with increasing exposure. Because of the marked variation in the quality of dust from mine to mine, particularly with regard to its biological potential, Dr Cho recommended that a “simple” recommendation of allowable dust of 2 mg or 4 mg per cubic meter, while perhaps acceptable for bituminous mines, may not be appropriate for Korean anthracite mines.

Dr James E. Gadek, Columbus, Ohio, reviewed concepts of lung damage caused by inflammation. He suggested that dust exposure could lead to activation of inflammatory cells in the lung. As a result, reactive oxidants which can injure lung cells and proteases which can injure the lung matrix are released. This sets the stage for loss of lung function. Dr Stephen I. Rennard, Omaha, Nebraska, reviewed mechanisms of repair in the lung which can lead to fibrosis. Following injury, fibroblasts are recruited and accumulate in the lung. Fibrosis results from incomplete repair in which the newly-recruited fibroblasts replace normal lung tissue. Measurement of the biochemical mediators which stimulate fibroblasts may have both diagnostic and prognostic value. Inhibition of these mediators may be a therapeutic strategy.
Dr Francois Nussot, Paris, France, presented an overview of the histological changes in the lung. The varying effects of asbestosis, silica, and pure coal were reviewed. The importance of early bronchiolar changes which can lead to either focal emphysema, fibrosis, or both, were discussed. She emphasized that "real" exposures were usually mixed and result in mixed histological features. Diagnostic techniques were also discussed. While open lung biopsy would be ideal to establish diagnosis, it was not felt necessary or practical in all cases. Bronchoalveolar lavage, particularly in combination with mineral analysis of the recovered fluid, was felt to be especially helpful.

2.3 Saturday, 24 September

2.3.1 Morning session

Dr Kyu Sang Cho chaired the meeting and reviewed the agenda. Dr Wang was invited to review the purpose of the draft protocol of collaborative research project and reiterated the purposes of the study.

Dr Cho opened the discussion on the proposed epidemiological study. It was felt to be beyond the focus of the project's emphasis on early diagnosis and treatment and beyond the resources currently available to the participating institutions. The epidemiological sub-objective was deleted therefore from the protocol, but Dr Liu Shijie requested that WHO give consideration to support of an epidemiological study on pneumoconiosis in the future.

The remainder of the protocol was discussed. Considerable attention was given to dissemination of technology and to the development of a training programme. Specifics of a programme will be developed by the Department of Radiology, Keio University. It was felt that every effort should be made to utilize similar methods and techniques at all participating institutions. Dr Gadek suggested that centres with established experience and expertise in various aspects could function as "core" facilities for reference and service as well as training for the duration of the protocol. It was felt that efforts to make study methods uniform were at crucial importance in acquiring comparable data at the various protocol institutions.

After discussion item by item, the protocol of collaborative research on early diagnosis and treatment was adopted.

2.3.2 Afternoon session

Dr Zhang Cuijuan, Beijing, demonstrated the recently adopted films used in the Chinese classification of pneumoconiosis. Dr Lu Shixuan pointed out that the films were prepared with low voltage technique. New films prepared with high voltage methods are in preparation. Dr Lu also emphasized that in this case the concept of a boundary was a "zone" rather than a "line".
Dr Mitsuru Tanaka, Keio University, presented studies using the ultrathin bronchoscope in selected patients. Abnormalities of pigmentation, dilatation, reddening and alterations in mucosal appearance were clearly demonstrated for patients with pneumoconioses. These changes were distinct from those associated with asthma, bronchiectasis, bronchiolitis, and cancer. The findings in bronchitis were noted to be inconsistent. It was observed that the ultrathin bronchoscope was fragile and must be handled with care.

Dr Toshiaki Higashi, University of Occupational and Environmental Health, presented a report on a study in Korea performed by the Small Airway Study Group of Occupational Lung Diseases. He reviewed the clinical features of patients studied by bronchoscopy, biopsy, and bronchoalveolar lavage. Dr Kawanami, Keio University, presented the available histological studies made by him in Korea and in China. Only 2 out of 6 specimens were completely satisfactory for analysis. Microscopic study, however, demonstrated abnormalities of the small airways including peribronchiolar fibrosis, pigment deposition and dilatation of the bronchiolar blood and lymphatic vessels. These findings correspond to the endoscopic observations of Dr Tanaka. Special stains for iron were able to distinguish at least two forms of pigmented lesions. Analysis of bronchoalveolar lavage specimens demonstrated birefringent dust particles and asbestos bodies.

Dr Kyu Sang Cho then closed the meeting thanking the participants and temporary advisers for its successful conclusion. He noted that regional and international cooperation on a shared health problem of the magnitude and complexity of pneumoconiosis represents a novel experience and carries with it a significant responsibility. He also thanked Drs Hashimoto and Tanaka of Keio University for hosting the meeting. Finally, Dr Cho thanked Dr Wang Liansheng and the WHO staff who assisted the meeting with a well-organized and an efficiently-run programme. Dr Tanaka on behalf of Keio University, thanked all participants. Dr Wang reiterated his thanks on behalf of Dr H. Nakajima and Dr S.T. Han to all the participants and hosts of the meeting.
3. PROTOCOL OF COLLABORATIVE RESEARCH ON EARLY DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS

(A) At the WHO Meeting on the Early Diagnosis and Treatment of Pneumoconiosis, held in Tokyo from 22 to 24 September 1988, the participants of the following institutions from the People's Republic of China, Japan and the Republic of Korea agreed to undertake joint research on early diagnosis and treatment of pneumoconiosis with support from WHO:

1. Institute of Occupational Medicine, Chinese Academy of Preventive Medicine;
2. School of Public Health, Beijing Medical University;
3. Shanghai Institute of Labour Hygiene and Occupational Diseases;
4. Department of Radiology, School of Medicine, Keio University Hospital and Small Airway Study Group of Occupational Lung Disease;
5. Catholic Industrial Medical Centre, Catholic Medical College, Seoul;

(B) Objectives

Overall objectives of the project are to improve existing methods and develop new ones for early diagnosis and treatment of pneumoconiosis with special reference to the application of bronchofibroscopy.

The sub-objectives are:

1. To improve chest X-ray diagnosis;
2. To improve pulmonary function tests;
3. To improve other patho-histological, clinical and biochemical methods;
4. To develop combined methods of diagnosis;
5. To study the therapeutic effects of tetrandrine and other drugs including traditional herbal medicine on silicosis at centres where such studies are possible.
(C) Project areas and responsibility of participant institutions:

Research projects over the next three-year period

1. All institutions will undertake the study on bronchofiberscopy.

1.1 Shanghai Institute of Labour Hygiene and Occupational Diseases:

1.1.1 The study on the early diagnosis (differential diagnosis) of pneumoconiosis by bronchofiberscope.
1.1.2 The early response of lung to mineral dusts.
1.1.3 The therapeutic effect of bronchoalveolar lavage and/or drugs, including Chinese herbal medicine, on silicosis.

1.2 Institute of Occupational Medicine, Chinese Academy of Preventive Medicine, Beijing:

1.2.1 Fibrobronchoscopy and differential diagnosis of pneumoconiosis

A. Fibrobronchoscopic biopsy in early diagnosis of pneumoconiosis.
B. Fibrobronchoscopic biopsy evaluation of early effects of mineral dusts.
C. Pulmonary-alveolar lavage in evaluation of organic dusts.

1.3 School of Public Health, Beijing Medical University:

A. Cytological study (morphological and functional) of cells recovered from BALF of patients with various pneumoconioses for early diagnosis.
B. Cytological changes of cells recovered from BALF in pneumoconiosis cases treated with aluminum citrate in early stages.

1.4 Department of Radiology, School of Medicine, Keio University Hospital:

A. To develop the study of ultrathin bronchoscopy.
B. To develop combined methods of diagnosis integrating BALF, Chest X-ray, CT scan, and histology.
C. Early diagnosis and treatment of pneumoconiosis with special reference to ultrathin bronchoscopy.
1.5 Catholic Industrial Medical Centre, Catholic University Medical College:

A. Early diagnosis and treatment of pneumoconiosis with special reference to the application of bronchoalveolar fiberscopy.

B. The application of pulmonary function test for the early diagnosis and treatment of pneumoconiosis.

(D) Training activities

The Department of Radiology, Keio University Hospital, will cooperate with other institutions in training personnel on application of bronchofiberscopy by conducting a fellowship programme and providing visiting consultants. A plan for a comprehensive training programme will be developed separately.

(E) Exchange of information

All institutions will periodically exchange information including progress reports, annual reports, papers and other publications concerning the research on pneumoconiosis.

Mutual visits of scientists and mutual attendance of scientific meetings and conferences will be highly recommended.

(F) Starting date

The whole collaborative research project started in January 1988. The first phase of the project is from January 1988 to December 1991.

(G) Input

The participants' institutions will make major efforts to mobilize all possible internal resources (including staff, facilities and funds) for undertaking the project activities.

It is proposed that WHO will continue to provide technical and financial support for this joint research project and to coordinate the research activities.

(H) Review and evaluation

The collaborative research project is the subject of periodic review by WHO Regional Office for the Western Pacific and by joint review meetings.

It is proposed that the second review meeting will be held in China in the latter part of 1989; the third will be held in the Republic of Korea in the latter part of 1990, subject to the availability of funds and the agreement of the governments concerned. A final review will be held in Manila in 1991.
The individual research project documents of each institution will be considered as part of this collaborative project document.

4. CONCLUSION AND RECOMMENDATIONS

4.1 The meeting noted that pneumoconiosis remains an important occupational health problem in China and the Republic of Korea. While further efforts should be made to strengthen the prevention and control of pneumoconiosis, its diagnosis and treatment, especially at its early stage, should also be improved.

The meeting members thanked WHO Regional Office for the Western Pacific for initiating action and giving support to China, Japan and the Republic of Korea in undertaking a collaborative study on early diagnosis and treatment of pneumoconiosis.

Since a protocol of collaborative research was formulated at the present meeting, it is recommended that WHO should continue to play its leading and coordinating role in this research and to provide technical and financial support to the implementation of planned activities.

4.2 Considering the resources and time constraints, the meeting recommended that the major efforts should be focused on application and use of bronchofibroscopy for early diagnosis and evaluation of effectiveness of treatment.

4.3 In order to promote the application of bronchofibroscopy, training of personnel is needed. It is recommended that WHO provide further support for the training activities, including consultancy and fellowships.

4.4 It is recommended that WHO Collaborating Centre for Research and Training in Diagnostic Endoscopy, Keio University, should continue to provide its technical expertise and to develop standard methods and record forms for the study of application of bronchofibroscopy to facilitate the comparable evaluation of the collaborative research.
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Annex 1

WPR/OCH/(3)/18/2

The following are members of the Small Airway Study Group of Occupational Lung Diseases, Keio University, who were present and contributed to the meeting: Dr Kazuo Shida, Dr Isao Okaraki, Dr Toshiaki Higashi, Dr Oichi Kawanami, Dr Susumu Yasuoka, Dr Chikao Torikata, Dr Eiichi Kohda, Dr Toshio Nakadate and Dr Norihiko Kohyama.
AGENDA

Thursday, 22 September

9:30 - 10:00 a.m. Registration

10:00 - 11:00 a.m. First working session

- Introduction of the objective of the meeting by Dr Wang Liansheng

- Opening remarks by Dr Shozo Hashimoto

- Election of officers: Chairman
  Co-chairman
  Rapporteur

- Adoption of the agenda

11:00 - 12:00 a.m. Discussions

Review the present status and identify the problems and future direction (Korea, China, Japan)

12:00 - 1:00 p.m. Lunch break

1:00 - 3:00 p.m. Review the present status .... (continued)

3:00 - 3:30 p.m. Coffee break

3:30 - 5:00 p.m. Discussion on the protocol of collaborative research (continued)
Annex 2
WPR/OCH/(3)/88.1

Friday, 23 September

1:00 - 1:30 p.m. Opening session
- Opening message by Dr Shozo Hashimoto
- Opening address by Dr H. Nakajima, Director-General of WHO

1:30 - 3:00 p.m. Presentations by:
1. Dr Liu Shijie (China)
2. Dr Kyu Sang Cho (Republic of Korea)

3:00 - 3:30 p.m. Coffee break

3:30 - 6:00 p.m. Continuation of presentations:
3. Dr James E. Gadek (WHO temporary adviser)
4. Dr Stephen I. Rennard (WHO temporary adviser)
5. Dr Francoise Basset (WHO temporary adviser)

Saturday, 24 September

10:00 - 12:00 a.m. Formulation of workplan of collaborative research

12:00 - 1:00 p.m. Lunch break

1:00 - 3:00 p.m. Finalization of protocol and workplan of collaborative research

3:00 - 3:20 p.m. Coffee break

3:20 - 5:00 p.m. Adoption of the summary report of the meeting
Adoption of the protocol and workplan

Closing remarks by:
Dr Shozo Hashimoto
Dr Wang Liansheng
It is a great pleasure for me to be present here today to join your meeting on the Early Diagnosis and Treatment of Pneumoconiosis. On behalf of the World Health Organization, I would like to extend my best regards to all of you and wish you every success in this meeting.

Occupational health is of particular significance in the Western Pacific Region, owing to the rapid development of industry and agriculture, which has led to the emergence of major health and safety problems for the working population.

Among other problems, pneumoconiosis, or dust lung diseases, are a major concern, as they are very common in most industrializing countries in the Western Pacific Region. The epidemiological surveys conducted in China and the Republic of Korea reported that the prevalence of different dust-borne diseases among workers exposed to dust varies from 3.5% to 8.3% rising even higher than 10% in some small-scale industries and mines. There is no doubt that the prevention of pneumoconiosis through the improvement of the working environment and the control of dust is of paramount importance, and much greater efforts will be called for. Generally speaking, knowledge and effective technologies for the control of dust are available in the world, subject to the availability of resources and the feasibility of using such technologies.

It is now time for WHO and Member States to give greater attention to the two other important aspects of the problem: diagnosis and treatment of pneumoconiosis. It is worth remembering, that any improvement in diagnosis, especially where there is early diagnosis and treatment, can contribute decisively to the prevention of complications and to the convalescence and rehabilitation of patients. It will finally protect workers' health and reduce the morbidity and mortality caused by pneumoconiosis. There are different technologies available for the early detection of pneumoconiosis, such as radiography...
Annex 3

(introduced by using the ILO International Classification of Radiographs of Pneumoconioses) lung function tests and biochemical tests. A recent important development for the early diagnosis of pneumoconiosis and other lung diseases is the introduction of bronchofiberscopy developed by Dr Mitsuru Tanaka, under the leadership of Dr Shozo Hashimoto. This technique permits the early detection of various patho-physiological changes in the peripheral airway which measures 2mm in diameter. Dr Tanaka is the head of the Fiberscopy Centre, Department of Radiology, School of Medicine, Keio University, a WHO Collaborating Centre for Research and Training in Diagnostic Endoscopy of Lung Diseases. In cooperation with this centre, WHO has already taken active steps to transfer this new diagnostic technology to China and the Republic of Korea.

With regard to the treatment of pneumoconiosis, no specific effective drug or method has been discovered. However, some studies are interesting and may provide clues. For example in China, Tilorone has been used in the treatment of experimental ratsilicosis and had a definite effect. Some Chinese traditional herbs, such as Mahonia Bealei Carr, have antisilicotic effect. However, we are still a long way from finding effective and safe drugs for the treatment of the different kinds of pneumoconiosis. Further efforts should be made for this purpose.

This is why WHO has sponsored this meeting, and invited experts from research institutions in China, Japan and the Republic of Korea to participate. The objectives are to review the present status of etiopathology, early diagnosis and treatment of pneumoconiosis and related diseases and identify problems and future directions, and to formulate a protocol and workplan on collaborative research in these three countries. We are very glad that Dr Francoise Basset from France and Dr James E. Gadek and Dr Stephen Rennard from the United States have also accepted our invitation to participate in this meeting and to share with us their knowledge and experience. On behalf of the World Health Organization, I would like to thank them for their valuable contribution to the meeting.

I see this meeting as the starting point of a long march against pneumoconiosis, which will be carried out by WHO and Member States of the Western Pacific Region. I believe that the collaboration of WHO, China, Japan and the Republic of Korea, will make this multicentre research project a successful one.

Thank you for your attention.
OPENING REMARKS BY DR WANG LI&SHENG
AT THE FIRST WORKING SESSION

Honourable Dr Shozo Hashimoto,
Colleagues and friends,

Please allow me to declare the Working Session of the WHO Meeting on the Early Diagnosis and Treatment of Pneumoconiosis open.

On behalf of Dr S.T. Han, Special Representative of the Director-General, World Health Organization and elected Regional Director of WHO Regional Office for the Western Pacific, I would like to extend to all of you, participants from China, Japan and the Republic of Korea as well as to the three temporary advisers, Dr Francoise Basset, Dr James Gadek and Dr Stephen I. Rennard warm welcome and best wishes for the success of the meeting. I would also like to express sincere thanks to Dr Hashimoto and Dr Tanaka for hosting this meeting in Japan.

Dr H. Nakajima, our Director-General, will do the opening of this meeting officially in the afternoon of Friday, 23 September. Today, I would only like to introduce the objectives of this WHO meeting.

Recently, WHO Regional Office for the Western Pacific has accorded great concern to the development of occupational health programme in this Region, and the prevention and control of pneumoconiosis is one of the priority areas of our programme. With WHO's support, some collaborative activities have already been started in China, Japan and the Republic of Korea. This meeting is aimed to further promote and strengthen the collaborative research in the field of pneumoconiosis among the three countries. The objectives are:

1. To review the present status of etiology, early diagnosis and treatment of pneumoconiosis and related diseases and identify problems and future directions; and

2. To formulate a protocol and workplan on collaborative research on pneumoconiosis.

I do believe, that through your efforts, this meeting will be a successful one.
AN OUTLINE OF RESEARCH PROJECT ON EARLY DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS
by
Shixuan Lu, M.D.

1. Present status

Pneumoconiosis remains to be an important problem in occupational diseases in China. Although remarkable progress is observed in many aspects of mining industry hygiene, some facets of present status of pneumoconiosis are uncertain, even obscure. It is understandable when many interruptions, either of long duration or short, are taken into consideration.

After 1980, important was the first step taken, which was the revision of 1963 Diagnostic Criteria of silicosis and asbestosis. Integrating the advanced experience of ILO system and other countries with the specific conditions of China, a Roentgenologic diagnostic criteria of pneumoconiosis was set up together with a set of 32 pieces of standard films. Hence, the diagnostic standards were unified into one and discrepancy of diagnosis was minimized. The second event is the promotion of a nationwide survey of pneumoconiosis, though a retrospective study of the diagnosed cases of selected factories and mines scattered all over the country, which will yield many figures which were uncertain or obscure in the past but are of great importance in elucidating the present condition as well as in setting up a future programme.

On the basis of experience gained in the past thirty years and the result of the survey in progress, following aspects of our programme may be considered.

2. Epidemiologic studies

2.1 Tuberculosis and pneumoconiosis

Management of pneumoconiosis complicated with tuberculosis is usually difficult and the result not satisfactory. Both diseases may be fairly common in certain mining districts, and co-existence of both diseases in a patient will be frequently found. Tuberculosis is to be considered in differential diagnosis and the prevalence and incidence of tuberculosis in that area may throw light on this problem. Morbidity and mortality of pneumoconiotic patients complicated with tuberculosis may illustrate the effect and result of preventive chemotherapy and therapeutic chemotherapy. Causes of therapeutic failure are of great reference value. Due consideration was paid to all these points in the nationwide survey which is in progress and will yield proper answers to all these points.

2.2 Causes of death of patients of pneumoconiosis

Causes of death of pneumoconiotic patients will be analyzed and should be checked against patients' death certificates, doctor's and hospital's records, autopsy records and statement of patient's family members. The analysis may reveal certain important facts which may be unknown or overlooked before death, thus, management of patients might be improved and living patients would be benefitted.
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2.3 Pneumoconiosis in ceramic workers

Workers in a ceramic factory had been followed regularly for more than 25 years. Accumulated data include dust measurement, clinical records and chest X-ray films. An analysis of these data will yield many valuable information about pneumoconiosis in ceramic workers, including prevalence, incidence, mortality, complications of pneumoconiosis (carcinoma, tuberculosis, c-v-diseases etc.) and late development of pneumoconiosis after removal from dust exposure. Examination of a series of chest films taken after first dust exposure up to present may reveal radiologic characteristics of pneumoconiosis in ceramic workers, the earliest changes and late development.

3. Early diagnosis of pneumoconiosis

Search for the evidences occurring prior to the appearance of X-ray shadows suggestive of pneumoconiotic in nature - there are several aspects to be considered.

3.1 Studies on BALF (broncho-alveolar-lavage-fluid)

Together with the advancement of fibrobronchoscopy, BALF has been studied widely in pulmonary interstial diseases. But in pneumoconiosis and other pulmonary changes induced by inhalation of organic dusts (cotton, tea, wood etc.), the study on BALF is only at its beginning. According to this context, studies on BALF should be carried out prior to the appearance of X-ray shadows suggestive of pneumoconiotic in nature.

Subjects: workers exposed to dusty of silica, coal and asbestos, without suggestive X-ray evidence of pneumoconiosis.

workers exposed to the same dusty, without definite X-ray evidence of pneumoconiosis of various stages (categories)

workers exposed to cotton, tea dust and wood dust.

Objects: analysis of the cellular components of BALF., Phagocytes, lymphocytes and subgroups, neutrophiles, etc. Analysis of OKT4 OKT8 in BALF and determination of their proportion. Lung biopsies taken in selected cases.

Determination of FIBRONECTIN in BALF.

Determination of enzymes in BALF., alpha-1 antitroisin, glycosidase, collagenase etc.

3.2 Studies... biochemistry

a. Determination of blood fibronectin concentration

b. Determination of blood superoxide dismutase
3.3 Comparative study on different kV technics (high, medium, low) in evaluation of early pneumoconiotic changes.

In this prospective study, individuals from different plants, and different posts, will be examined with 120, 100, 80 kV technics. Three films will be taken during each examination. Processed under same condition. Dust concentration and dosage are recorded during each examination.

Objects: Which technique can detect the earliest changes, and which technique is the best in illustrating the typical changes?

3.4 Studies on boundary films, especially the one between O+ and I stages.

Search for the boundary films is one of ILO's eight problems to be solved. Since boundary is arbitrary, it cannot be linear in nature. It might be zonal in appearance. Therefore, the boundary defined by two films (+ film and stage film) is more rational than the boundary defined by one film. This is what we do in setting up our STanard films. However, in practice as well as in reasoning, the boundary zone should be kept as narrow as possible. Hence, refinement of the quality of the films illustrating boundary (+ film and stage film) is always a challenge. Now the preparation of better films illustrating the boundary (+ film and stage film) is in progress.

3.5 Study in the mixed type of small opacities

For Chinese standard films, the small opacities are shown in accordance with p, q, r, s, t, u, but in practice, small opacities of mixed type is the one frequently encountered. There was a suggestion of recording mixed type small opacities with x, y, z, and ILO offers also a convenient way of recording such images. It is a common experience to see a film recorded as q/t by one person but t/q by another. Such confusion as well as mental process of estimating the proportion of small opacities would be very much minimized if there were standard films illustrating different stages of different mixed types of small opacities.

To prepare such standard films, it appears necessary to review carefully 1000 - 2000 or more chest films showing different combination of small opacities, then the frequent combinations will be known and corresponding standard films could be prepared.

4. Treatment of pneumoconiosis

Definition of therapeutic effect should be set up first.

Symptomatic improvement

Radiologic improvement: temporary lasting or permanent

Tetradrine administered through fibrobronchoscope for large opacity-administered at regular interval - examination of BALF may serve as monitor.

Preventive effect of tetradrine should be tested in human beings.
THE CHINESE 1986 ROENTGENODIAGNOSTIC CRITERIA OF PNEUMOCONIOSES AND STANDARD FILMS

Shixuan Lu, M.D., Maopo Ting, M.D., Cuijuan Zhang, M.D.
Institute of Occupational Medicine, Chinese Academy of Preventive Medicine, Beijing, PRC.

The last edition of Chinese Diagnostic Criteria of silicosis and asbestosis were issued in 1963. Twenty three years later, a new edition of Roentgenodiagnostic Criteria of Pneumoconioses ('1986 Chinese Criteria') was adopted and issued on Nov. 1st, 1986. Unlike the old 1963 edition which treated silicosis and asbestosis separately by preparing specific diagnostic criteria for each of them, the 1986 Chinese Criteria incorporating the advanced experience of II.O 1980 System with the specific conditions of China, cover the radiologic appearances seen in all prescribed types of pneumoconioses (12 in number) listed officially. It is implemental in clinical diagnosis of pneumoconioses and is also applicable in epidemiologic studies as well as for purposes of compensation. Although some modifications have been introduced to permit more detailed documentation of radiologic features, and a few alterations made to lessen the differences between the 1980 II.O Classification and the 1986 Chinese Criteria, the latter encompasses experiences gained in the past 30 years in China and retains the same basic principles included in the 1963 edition. This is well illustrated by the 1986 Standard Films (32 in number) which take precedence in classifying small opacities.

A. CHARACTERISTICS

1. Four Stages

Four major stages are kept at the same level as that of the 1963 edition, i.e. no pneumoconiosis (0, 0+), pneumoconiosis stage I (I, I+), pneumoconiosis stage II (II, II+), pneumoconiosis stage III (III, III+).

2. Profusion and extent are equally emphasized.

Assessment of profusion and extent is definitely defined.

a. Assessment of profusion must be an integrated consideration of the profusion in every zone of the lung.

b. A zone is considered affected only when 2/3 of its area is involved.

c. Extent implies the total number of affected zones.

d. Profusion in accordance to the category of majority of the affected zones is the basis for assessment.

e. When more than one category of profusion occur, the higher category in area not less than two zones is taken as the predominant value of assessment.
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3. Thirty-two pieces of standard films:
The standard films show the assuredly lower limit of each stage, not the mid category images shown by II.0 films. There are a series of 0, I, II; standard films which may be paired with the stage standard films, i.e., 0 and I, I and II, III and IV for comparison. Boundary of stage situates between stage film and (1) film.

4. Pleural plaque is considered as an adjunct evidence of asbestosis.

5. Two peculiar X ray features of grave outcome:
Longitudinal faint mottlings in peripheral parts of both upper zones and homogeneously hazy and patchy shadows over both upper zones are denoted as stage II, irrespective of the background profusion of small opacities. These progress rapidly to massive opacities usually in 2-4 years. They should be carefully followed.
In general, the 1986 Chinese Criteria is similar to the 1980 II.0 short classification.

B. SELECTION OF THE STANDARD FILMS:

These standard films (stage films and (1) films) were selected from thousands of chest films supplied by 20 local Diagnostic Panels of municipalities, provinces and autonomous regions. Their choice of films were based upon theREFERENCE FILMS WHICH HAD BEEN CHOSEN BY THE MEMBERS OF THE NATIONAL PANEL FROM A COLLECTION OF OVER 600 FILMS OF VARIOUS TYPES OF PNEUMOCONIOSES (13 TYPES, DUE TO: SILICA, ASBESTOS, COAL DUSTS, TALE, MICA, CEMENT, CARBON BLACK, KAOLIN, CERAMIC AND REFRUCTORY MATERIALS, AND MIXED DUSTS OF ELECTRIC WELDING, etc.)

Local Panels were asked to take their films of choice showing lower limit of each stage to sessions where films were discussed and compared in groups of 20 pieces each. The lower limit of each stage shown by each film was the main theme of discussion and comparison. The most illustrative one of each group was selected. The most illustrative ones were pooled together and reexamined by the session participants. They were representatives or chairmen of local Panels. The film showing slightest changes and being all agreed was considered as a candidate for standard film representing the assuredly lower limit of that stage. The films which were not all agreed were usually deficient in profusion or extent or both when compared with the candidate film for stage standard film. After repeated comparison, the one being very close to the candidate film was selected and was considered as candidate for (1) film.
of lower stage. All the candidate films were sent to the National Panel for final decision.

Members of National Panel were asked to join every session called for selection of candidate films that they could understand and weigh the pros and cons about each candidate film. They were responsible to choose the best one from candidate films for the standard film (of stage films and \( \cdot \) films). Thus, a standard film was usually selected from a couple of dozens of films and sometimes from hundreds of film.

In practice, using the standard stage film and \( \cdot \) film for comparison makes diagnosis easy and definite. The boundary of stage though arbitrary is between these two films. The inter-reader variability and intra-reader variability vary from 10%-15%.

The 1986 Chinese Criteria and standard films will be revised in 1992. The current standard films will be substituted by films made with 120 kV demonstrating better x-ray images.
The Chinese 1986 Roentgenodiagnostic Criteria of Pneumoconioses incorporating the advanced experience of ILO 1980 System with the specific conditions of China, is applicable to all types of pneumoconioses for compensatory purposes, clinical and epidemiologic studies. In classifying practice, the Standard Films take precedence. The characteristics are:

1. Four Stages of severity, coded as: 0, 0+, I, I+, II, II+, III, III+
2. Profusion and extent, equally emphasized in classifying small (rounded or irregular) opacities
3. Thirty-two pieces of Standard Films. Two for each Stage, one showing the assuredly lower boundary of each Stage, the other (+ film) being very close to the next Stage. These films are paired, e.g. 0+ and I, for use in comparison. Thus boundary of the Stage situates between these two films.
4. Pleural plague is considered as an adjunct evidence of asbestosis.
5. Two peculiar x-ray features are described and denoted as II+, because of the grave outcome and the rapid progression.

Processes of selection of standard films are described.
The 1986 Chinese Roentgenodiagnostic Criteria of Pneumoconioses

1963 Chinese Diagnostic Criteria: dealing with silicosis and asbestosis separately
No standard chest films

1986 Advanced experiences of 1980 ILO System + specific conditions of China
Covers all types of pneumoconioses
With a set of Standard Chest Films
Its applicability: clinical, epidemiological, for compensation

Characteristics:
1. Four stages: 0, O+
   I, I+
   II, II+
   III, III+

2. Profusion and extent, equally emphasized
   a. Integrated consideration of profusion in every zone
   b. Total number of affected zones
   c. 2/3 of zonal area affected
   d. Profusion of the majority of affected zones
   e. Higher category of profusion over an extent of not less than two zones

3. 32 pieces of Standard Films

4. Pleural plague, adjunct evidence

5. Two peculiar x-ray features of grave outcome, II+
Annex 5
Appendix 1
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National Panel -------- Reference films
600+ films of 18 types of pneumoconiosis:
- 32 -
32 films selected during first session
19 films selected during second session. Local Panels agreed with the selection.

Local Panels -------- Candidate films
Based upon reference films, from their collection of chest films
Selected films showing their lower limit of each stage, for sessions
Compared and discussed in groups of 20 pieces each. Main theme of discussion and comparison was the lower limit of each stage.
Most illustrative ones of each group was singled out and pooled together. Reexamined by session members (40-50)
All agreed ones showing the slightest lesion (= assuredly lower limit)
Candidate for standard films
Not all agreed ones, re-examined by session members
Candidate for (+) film of lower stage

National Panel ------ Final selection, decision
Responsibilities of National Panel Members

(+ ) film............arbitrary boundary.............stage film
Comparison of 1980 ILO Classification and 1986 Chinese Criteria

<table>
<thead>
<tr>
<th>Features</th>
<th>1980 ILO</th>
<th>1986 Chinese Criteria</th>
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<td>Zones</td>
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<td>1, II,</td>
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<td>III</td>
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<tr>
<td>Shape and type of opacity</td>
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<td>Small opacities</td>
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<td>rounded</td>
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<td>p, q, r,</td>
<td>p, q, r,</td>
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<td>profusion types</td>
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<tr>
<td>extent</td>
<td>s, t, u,</td>
<td>s, t, u,</td>
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<td>Large opacities</td>
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<tr>
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<td></td>
<td>B (&lt;r. u.)</td>
<td>diameter &gt; 20mm</td>
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<tr>
<td></td>
<td>C (&gt;r. u.)</td>
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Summary of the 1986 Chinese Radiodiagnostic Criteria

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<td>$&gt;4$</td>
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$\S$ for irregular small opacities only (for rounded small opacities, an area of 2 cm. diameter in each zone)

Size of small opacities:

- Rounded: p, q, r (same as ILO)
- Irregular: s, t, u (same as ILO)

Size of large opacities: 2 times 1 cm.
Comparison of results classified by 1980 ILO and 1968 Chinese Criteria

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<th>ILO Classification</th>
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<td>III</td>
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<td>84</td>
</tr>
<tr>
<td>Total</td>
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</table>

The 1986 Roentgenodiagnostic Criteria of Pneumoconioses and Principles of Management

The 1986 roentgenodiagnostic criteria of pneumoconioses are applicable to all prescribed types of pneumoconoses.

1. Principles of Diagnosis

Radiologic examination of the chest is the basic approach used in the diagnosis and classification of pneumoconioses. Correct interpretation of the radiographic manifestations of pneumoconioses depends on a reliable, detailed occupational history followed by the careful scrutinization of standard size PA chest film of optimal technical quality. To understand any progression of the disease previous chest films are invaluable and should be sought and reviewed. Data of dust exposure monitoring is exceptionally important confirmatory information and if available, should be consulted.

In addition to the roentgenologic diagnosis and classification of X-ray manifestations, the clinical diagnosis of pneumoconioses includes the early recognition of any complications and reasonable exclusion of other diseases which may give rise to similar X-ray appearances. Evaluation of the compensatory status is achieved by an integrated study of the history, symptoms and signs, physical examination, routine laboratory examination, and if indicated, examination of the sputum and pulmonary function testing.

2. Criteria for diagnosis and "stages" of pneumoconiosis:

2.1 No pneumoconiosis (Code 0)
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a. 0: No roentgenologic manifestations of pneumoconiosis.

b. 0*: With roentgenologic manifestations of pneumoconiosis up to the lower limit of "stage I".

2.2 Pneumoconiosis "stage I"

a. I: Small rounded opacities, with profusion of category 1, and extent of one area in at least two zones of the lung, the diameter of each area being not less than 2 cm.

For small irregular opacities, with profusion of category 1, and extent of not less than two zones of the lung.

b. I*: Small opacities, either rounded or irregular, with profusion of category 1, and extent of more than four zones of the lung; or with profusion of category 2, and extent of up to four zones of the lung.

2.3 Pneumoconiosis "stage II" (Code II)

a. II: Small rounded or irregular opacities, with profusion of category 2, and extent of more than four zones of the lung; or with profusion of category 3, and extent up to four zones of the lung.

b. II*: Small rounded or irregular opacities of profusion category 3, and extent of more than four zones of the lung; or with a large opacity less than 2 cm in length and 1 cm in width.

2.4 Pneumoconiosis "stage III" (Code III)
a. III: With a large opacity of not less than 2 cm in length and not less than 1 cm in width.

b. III: With a large opacity or opacities of total area larger than the right upper zone of the lung.

3. Principles of management

The purposes of management of pneumoconioses is to delay the progress of the disease, to alleviate the symptoms, to adopt effective measures to prevent and treat the complications. Hence, the combined measures of management should include medical treatment, an educational program for personal hygiene, a well scheduled life pattern, appropriately individualized exercise and rehabilitation, and preventive measures for pulmonary tuberculosis.

4. Evaluation of working ability

The working ability of a patient is ascertained in accordance with his/her stage of pneumoconiosis and status of functional compensation.

5. Medical surveillance

A pre-employment physical examination including radiological examination must be carried out before dust exposure for any worker, and must be repeated periodically during work exposure.

The aim of the periodic physical examination during exposure is to
discover new cases of pneumoconiosis or to follow the progress of old cases.
Examinations are carried out at intervals decided by the local office of health. Generally, the interval between two examinations is 1 year (maximum 2 years) for heavy exposure, 2-3 yrs for light exposure, and 3-5 yrs in special circumstances such as migratory workers, intermittent exposure or where facilities are not available.

6. Contra-indications for dust exposure

   a. Any severe chronic diseases of the upper respiratory tract.

   b. Active tuberculosis of lung, or any extra-pulmonary active tuberculosis. Severe chronic pulmonary disease.

   c. Diseases of the pleura, thoracic cage, or vertebra, markedly influencing pulmonary function.

   d. Severe cardio-vascular disease.
Appendix A

Explanatory Notes of 1986 Roentgenodiagnostic Criteria for Pneumoconioses

A.1 Zonal division of the lung

The lungs are divided into six zones: upper, middle, and lower; right and left; by horizontal lines drawn at one-third and two-thirds of the vertical distance between lung apices and domes of the diaphragm.

A.2 Small opacities

Small opacities are opacities with either length or width less than 1 cm.

A.2.1 Rounded small opacities

Rounded or nearly round in shape, with regular or irregular margin, divided into three types according to the diameter:

p: diameter up to approximately 1.5 mm;

q: diameter approximately 1.5-3 mm;

r: diameter approximately 3-10 mm.

A.2.2 Irregular small opacities

A series of definite linear opacities of variable width, length, and shape,
may be discrete, or may be disorderly, interwoven as a network, or in some cases as a honeycomb. These opacities may be subdivided into three types according to width:

s: width up to approximately 1.5 mm;

t: width approximately 1.5-3 mm;

u: width approximately 3-10 mm.

A.3 Profusion of small opacities

Profusion is defined as the quantity or concentration of opacities in a limited area, i.e. a zone of the lung and is graded into three categories. Profusion is assessed by comparison with the standard roentgenographs which illustrate those three categories.

A.3.1 Profusion of rounded small opacities

a. Category 1: small rounded opacities definitely present but few in number, no obscuration of pulmonary vasculature. (in case of "p", there should be about 10 p-opacities in an area of 2 cm in diameter).

b. Category 2: Numerous small rounded opacities, normal pulmonary vasculature is still visible.

c. Category 3: Very numerous small rounded opacities, the normal pulmonary vasculature is totally or partly obscured.
A.3.2 Profusion of small irregular opacities

a. **Category 1:** Small irregular opacities, the normal pulmonary vasculature is still visible.

b. **Category 2:** Numerous small irregular opacities, the normal pulmonary vasculature is usually partly obscured.

c. **Category 3:** Very numerous small irregular opacities, the normal pulmonary vasculature is usually totally obscured.

A.4 Assessment of profusion and extent

a. Assessment of profusion must be an integrated consideration of the profusion of small opacities in every zone of the lung.

b. A zone is considered affected only when two-thirds of its area is involved.

c. Extent implies the total number of affected zones.

d. Profusion according to category of the majority of the affected zones is the primary basis for assessment of profusion.

e. When more than one category is involved, the higher category of profusion over an extent of not less than two zones is taken as the predominant value of the assessment.

A.5 Large opacities
These are opacities with the broadest diameter more than 1 cm.

A.6 Large opacities less than 2 cm in length and 1 cm in width as required for "stage III", include the following:

a. Aggregation of small opacities, but not an evenly dense massive shadow.

b. Large shadow, but slightly less than 2 cm in length and 1 cm in width.

c. Definite shadows, in appearance longitudinal, faint mottling in peripheral parts of both upper zones, or homogenous, hazy and patchy shadows over both upper zones.

A.7 Pleural abnormalities

Pleural abnormalities, such as adhesions, thickening or calcification, may occur in pneumoconiosis. Significant abnormalities should be denoted by symbols.

A plaque is a circumscribed pleural thickening more than 5 mm in thickness.

If asbestos-related irregular opacities can be classified as 0+, and circumscribed chest wall plaques can also be detected bilaterally, then the case can be classified as stage I instead of 0+. If the small irregular opacities are classified as 1+, and the plaque is big enough to involve and blur part of the cardiac and diaphragm, then the classification should be stage II. Similarly, in the case of II+, where no large opacities are observed, but an extensive plaque involves the cardiac border and make part of the cardiac border
shaggy, this should be classified as stage III.

A.8 The $0^+$, $I^+$, $II^+$, $III^+$.

These supplement each respective stage, (namely 0, I, II, III) and are designed to record the progress of the disease. The plus signs do not indicate stages.

A.9 Complications of pneumoconioses

a. Pulmonary tuberculosis: implying active tuberculous lesions only, such as exudative, proliferative, aseous, cavitary, bronchial and lymphatic or hematogenic dissemination. Fibrosed and calcified tuberculous lesions are not considered to be complications.

b. Pulmonary emphysema and pulmonary heart disease:

Diagnosed according to the criteria set up for these diseases by the Unit of Respiratory Diseases of the Chinese Medical Association (CMA).

c. Acute and chronic inflammatory diseases of the lung.

d. Chronic bronchitis; bronchiectasis.

e. Spontaneous pneumothorax.

f. Carcinomas such as asbestos-related carcinoma of the lung, mesothelioma.
of the pleura.

A.10 Symbols

a. bu--bullae

b. ca--cancer of the lung or pleura

c. cp--cor pulmonale

d. cv--cavity

e. ef--effusion

f. em--definite emphysema

g. es--eggshell calcification of hilar or mediastinal lymph nodes.

h. pc--pleural calcification

i. pt--pleural thickening

j. px--pneumothorax

k. rp--rheumatoid pneumoconiosis

l. tb--active tuberculosis
A.11 Standard radiographs

A set of 32 standard roentgenographs bears the same importance as the text in the assessment of chest films. During assessment of the shape and profusion of small opacities, the standard radiographs must be consulted.
Criteria and grades for excellence of technical quality in chest radiographs

B.1 Criteria

a. Film must be of adequate size to include the whole thoracic cage, from the lung apices to just below the costophrenic angles.

b. Areas directly exposed to X-ray must be black in color, and deep enough to obscure the shadow of a finger behind the film when held up to the light.

c. The clavico-sternal joints must be symmetrical on both sides; the scapulae brought forward, casting shadows outside the bony cage.

d. The intervertebral spaces of the thoracic spine must be just visible through the mediastinal structures, and T₅-T₆ easily seen.

e. Pulmonary vasculature must be shown in the greatest detail.

f. Different densities of bones, muscles, and soft tissues must be clearly demonstrated.

B.2 Grades

a. Good--no technical defect.

b. Acceptable--with no technical defect likely to impair classification of the pneumoconiosis.
c. Poor—with some technical defect but still acceptable for classification purposes.

d. Unacceptable—can not be used for diagnosis of pneumoconiosis.

If the technical quality is not of grade a, some comments should be made about the defects.
Appendix C

Technological conditions of X-ray examination

D. 1 Equipment

D. 1.1 Capacity X-ray generator should have a minimum capacity of 200 mA, and must be full-wave rectified.

D. 1.2 X-ray tube
   a. rotating anode tube
   b. focal spot, 1-2 mm²
   c. power 20-40 KW

D. 1.3 Intensifying screen
   a. medium speed, calcium tungstate
   b. discrimination, not less than 7 line-pairs/mm

D. 1.4 X-ray film
   a. medium sensitivity, Model I-II
   b. Contrast, greater than 2.5
   c. foggy, less than 0.2
   d. blue based
   e. size, 12"X15"; 14"X14"

D. 1.5 Electric power
   a. adequate capacity and stability
   b. special transformer, independent of others
   c. potential drop not exceeding 5%

D. 2 Technology of exposure

D. 2.1 Adjustment of the conditions
   Previous films should be reviewed in order to decide whether the conditions should be modified.
D.2.2 Positioning of the body

a. P-A view is the standard position, oblique views are used if necessary.

b. Position of the subject should be upright and straight, with the anterior wall of the chest in close contact with the cassette. Scapulae should be brought forward to avoid their shadows falling in the lung field. Shoulders are kept in the natural position and should not be elevated during positioning.

c. It is good practice to have the subject try positioning several times before actual exposure. The subject must be fully cooperative. Subject is asked to hold his/her breath at the end of full inspiration during exposure. This can be better done if the subject breathes deeper than usual several times before full inspiration.

d. The X-ray tube is adjusted so that the central beam passes through the level of T5 perpendicular to the cassette.

D.2.3 Focal spot-film distance: 1.8 m.

D.2.4 Exposure time

a. High energy X-ray generator; not to exceed 0.06".

b. Low energy X-ray generator; if the subject is cooperative, the exposure time can be prolonged, up to the limit of 0.1".
D.2.5 Selection of KV value

a. Selection formula: KV = thickness of chest in centimeters \( \times 2 \) + empirical constant

b. Thickness of chest is measured during quiet breathing.

c. Films made with adequate KV value will clearly show both T4 and the bronchial shadow at the same level.

d. High KV: within the capacity of the X-ray tube, 80-95 KV could be used. Higher than 95 KV may be tried.

D.2.6 Amount of exposure

a. This is expressed as mAs, in direct proportion to the darkness of the film.

b. The exposure is adjusted according to the contrast shown on the film. In the case of high contrast, KV value can be increased, and mAs lowered; if the contrast is low, mAs should be increased and the exposure time should not exceed 0.1".

D.3 Dark room processing

a. Developer is made according to the formula given by the film factory. Temperature of the developer is kept at 18-20 degrees centigrade. (High temperature developing process may be tried,
if conditions permit. Developing time depends on how long the developer has been prepared and how frequently it has been used.

b. Fixing process must last long enough. The time required is about twice the time for complete developing.

c. Rinsing must be thorough, in running water for about half an hour. When the ambient temperature is high, it is not appropriate to rinse too long.

d. Films should not be exposed to red light for too long in the dark room. otherwise, the films may appear foggy.

e. Intensifying screen must be cleaned regularly. Those stained by mould spots or age should be replaced. The cassette must be in close contact with the screen and closed tightly.

D.4 Additional

a. Tomography: whenever indicated.

b. Indirect radiography: When available, 100 mm indirect radiographic equipment can be used for screening purposes during regular surveys.
Standard films for the roentgenodiagnosis of pneumoconiosis

E.1 Standard films and the text of Criteria for Roentgenodiagnosis of Pneumoconioses.

The 1986 Criteria for Roentgenologic Diagnosis of Pneumoconioses is composed of two parts, the text and the standard films. Both parts are equally effective. For assessing the profusion of small opacities, the standard films take precedence over the text.

E.2 Copyright of the standard films

Standard films are one of the Official Standards. Copyright of the Standard films belongs to the Chinese Ministry of Public Health (CMOPH).

E.3 Application of Standard Films

The Complete Set of Standard Films has been put on trial and found to be a valid presentation of the roentgenologic manifestation of pneumoconioses. The set encompasses all prescribed types of pneumoconioses.

E.4 Diagnostic criteria of Standard Films

Standard Films are arranged according to the minimal diagnostic criteria for each Stage. These limits clearly define shape, size, and profusion. The Standard Films clearly demonstrate the appearance of the lower limits of stage I.

E.5 Profusion denoted on the Standard Films

Profusion denoted on each Standard Film is a comprehensive description of both the profusion and the
extent. Since profusion of small opacities is closely related with the extent, they are to be considered concurrently during assessment of profusion. Profusion and extent should not be considered separately, nor be put together in any combination arithmetically.

E.6 Distribution of the Standard Films

Before distribution, copies of Standard Films are examined, approved, numbered, and stamped by the National Panel of Pneumoconioses.
Notes on the correct application of Diagnostic Criteria

F.1 Scope of application

The Criteria are applicable to all prescribed types of pneumoconioses, namely all pneumoconioses registered in the "Official List of Occupational Diseases".

F.2 Text and Standard Films

These Criteria are compiled in two parts, text and Standard Films, which complement each other; hence, both are to be comprehensively used during actual application.

F.3 Functions of the Provincial Panels

The panels for diagnosis of pneumoconiosis in the provinces, cities, and autonomous regions play important roles in the correct application of the Criteria. Their functions include: arranging scientific activities and controlling the accuracy of the diagnoses of pneumoconioses; assembling data, experience and suggestions; and making final decisions on diagnoses.
THE EARLY RESPONSE OF LUNG TISSUE TO MINERAL DUSTS

by

Dr Hui-Xin Jiang

Inhaled mineral dusts damage the lung by inducing an inflammatory reaction that leads to fibrosis of pulmonary tissue. The development of mineral dust-induced fibrosis involves pulmonary alveolar macrophage and its alteration of biological characteristics at early stage.

Several of our earlier studies have demonstrated that the proportion of total lipid and phospholipid increased in pulmonary tissue of human pneumoconiosis. The results indicate that the lipid content of lung tissue differed in the different lung diseases.

It is noteworthy that some toxic oxygen metabolites, superoxide anion, etc. can be released from phagocytic cells during stimulation of membrane by mineral dusts. These active oxygen species are highly reactive and damage biological material in numerous ways, including lipid peroxidation, leading to membrane damage.

In this paper, luminol-dependent chemiluminescence (CL) assay was used. It provides an indirect but sensitive and rapid measurement of oxygen and radical production by phagocytosis of the cell.

Four samples of respirable mineral dusts were used in experimental study. The results suggest that all the dusts induced some degree of CL, but the level varied and was dependent on the dust type. In addition, six samples of macrophage recovered by BAL from patients with pneumoconiosis (silicosis and asbestosis) and other lung diseases were determined. The similar results are also obtained in human samples. The patients with pneumoconiosis or who were exposed to mineral dusts showed a higher CL responses than those who with other lung disease non-exposed mineral dusts.
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It might be explained that macrophage activated by vivo by mineral dusts can produce greater CL in vitro than unstimulated macrophage and these mineral dusts most likely to cause fibrotic lung diseases would be that most efficient in causing the release of oxygen radicals from phagocytic cell.

The active oxygen radicals can be inactivated by a number of biological compounds, such as vitamin E, SOD, B-carotene and some Chinese traditional drugs.

At this time, the analysis of cells recovered by BAL such as the number and type of cells recovered, the analysis of recovered extracellular molecules, such as the total protein, albumin immunoglobulins, total lipid phospholipid, interleukin I, II and fibronectin, etc. are also determined.
A REVIEW ON THERAPEUTIC EFFECTS OF DRUGS ON SILICOSIS

by

Dr Hu Tian-Xi and Dr Li Quan-Lu

The therapeutic effects of some drugs on silicosis have been studied for 20 years in our Institute. The drugs are PVNO (1967), piperaquine phosphate (1974), hydroxyl piperaquine phosphate (1978), bixbenzyl-iso-quinoline tetrandrine (1976), and aluminium preparation (1975).

The experimental studies showed that PVNO can prevent erythrocyte from hemolysis induced by quartz, inhibited silicotic fibrosis and even cause regression of silicotic lesions in rats. The therapeutic effects are increasing with molecular weights of PVNO. As PVNO is administered intratracheally, silicotic fibrosis can be inhibited for a long period. Pulmonary clearance was studied using non-invasive magnetic measurements at chest of hamsters and the results showed it can be affected by quartz and improved by PVNO. Added combination therapy with PVNO and tetrandrine showed more effective in comparison to monotherapy with PVNO or tetrandrine. The experimental studies on the effects of piperaquine phosphate, hydroxyl piperaquine phosphate, and aluminium preparation also showed an obvious inhibition of silicotic fibrosis, especially tetrandrine, in rats, dogs, or monkeys.

All of the drugs were used to treat silicosis patients. Clinical investigation showed after PVNO aerosol inhalation, the symptoms such as cough, chest pain, and dyspnea improved, X-ray films demonstrated progression of silicotic fibrosis inhibited, especially in acute cases, tbc-complication and fatality rate decreased and without apparent evidence of side effects. As piperaquine phosphate, hydroxyl piperaquine phosphate and tetrandrine were used to treat silicosis patients, a dramatic appearance of regression were observed on X-ray films. Massive fibrosis opacities diminished both in density and size in duration of treatment. Some evidences of side effects were observed and it recovered as drugs withdraw.
Based on research work mentioned above, a new programme of treatment on silicosis is suggested. Patients would be lavage with PVNO or aluminium preparation so that drugs can enter alveoli and reach the sites of silicotic lesions directly. In this way, the free dust in lungs can be removed by both lavage and improved clearance. During or after application of PVNO or aluminium preparation, the patients should be treated with one of the other three drugs. By combined application of drugs, it may be more successful to remain the therapeutic effects.
Clinical Investigations

Endoscopic Observation of Peripheral Airway Lesions

Mitsuru Tanaka, M.D., F.C.C.P.;* Oichi Kawanami, M.D., F.C.C.P.;† Masaru Satoh, M.D.; Kazuhiro Yamaguchi, M.D.; Yasumasa Okada, M.D.; and Fumihiro Yamasawa, M.D.

Peripheral airways of 2 mm or less in diameter were observed in 142 patients by means of an ultrathin bronchofiberscope measuring 1.8 mm in outside diameter. On the basis of the observed and photographed endoscopic findings, an endoscopic classification of peripheral airway lesions was proposed. The endoscopic findings showed changes in the bronchial wall consisting of reddening, pallor, absence of mucosal luster, edema, engorgement of blood vessels, irregular mucosal surface, and elevated mucosa. In the lumen, stenosis, obstruction, ectasis, and deformation due to pressure were recognized, in addition to excessive secretion and pigmentation as morphologic abnormalities or abnormal findings at bifurcation.

Peripheral airways measuring 2 mm or less in diameter are called a silent zone, and lesions in this area are often left untreated until they become marked. This is probably because accurate objective evaluation of functional disorders is difficult compared with that of subjective and objective symptoms. There are many factors and diseases which are considered to inflict damage on peripheral airways. Among these, smoking and air pollution are now social issues, and new causative factors are likely to arise in the future.

Since usual therapies for chronic nonspecific pulmonary disease are not necessarily effective for peripheral airway disease, it may be useful to determine accurately the pathologic characteristics of peripheral airway disease from both the diagnostic and therapeutic standpoints. As compared with conventional diagnostic methods, endoscopy of peripheral airways, which enables direct observation of the mucosa in the peripheral airways, is considered a fundamental and highly useful method for analyzing the pathologic characteristics of peripheral airway lesions. We have developed a new very thin bronchofiberscope measuring 1.8 mm in outer diameter (Olympus BF-1.8T), which allows for the first time observation and photography of peripheral airways of 2 mm or less to a clinically satisfactory extent. Examination was carried out with the BF-1.8T in 142 patients who were diagnosed as having peripheral airway lesions. On the basis of endoscopic findings obtained from such observations and photography, an endoscopic classification for peripheral airway lesions was attempted.

Material and Methods

Subjects (Table 1)

The subjects were 142 patients: 93 males and 49 females. Their ages ranged from 15 to 84 years, with a mean of 53.9 years. There were five normal subjects who were evaluated as having no complaint, no smoking history, and no abnormal findings on auscultation, chest roentgenography, spirometry or arterial blood gas analyses. These five normal patients were males ranging in age from 16 to 56 years, with a mean of 37.4 years. The main diseases of the other

Table 1—Diagnosis in 142 Subjects

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects</td>
<td>5</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>22</td>
</tr>
<tr>
<td>Bronchial asthma due to air pollution</td>
<td>22</td>
</tr>
<tr>
<td>Bronchiolitis</td>
<td>15</td>
</tr>
<tr>
<td>Bloody sputum</td>
<td>10</td>
</tr>
<tr>
<td>Interstitial pneumonia</td>
<td>9</td>
</tr>
<tr>
<td>Chronic pulmonary emphysema</td>
<td>8</td>
</tr>
<tr>
<td>Bronchial asthma</td>
<td>8</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>4</td>
</tr>
<tr>
<td>Chronic bronchitis</td>
<td>4</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>4</td>
</tr>
<tr>
<td>Pneumocooniosis</td>
<td>3</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>2</td>
</tr>
<tr>
<td>Pulmonary tuberculosis</td>
<td>2</td>
</tr>
<tr>
<td>Others</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>142</td>
</tr>
</tbody>
</table>
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Table 2—Specifications of the BF-1.8T

<table>
<thead>
<tr>
<th></th>
<th>BF-1.8T</th>
<th>BF type 10 (conventional bronchofiberscope)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total length</td>
<td>1350 mm</td>
<td>760 mm</td>
</tr>
<tr>
<td>Effective length</td>
<td>1150 mm</td>
<td>550 mm</td>
</tr>
<tr>
<td>Tip part diameter</td>
<td>1.8 mm</td>
<td>5.3 mm</td>
</tr>
<tr>
<td>Angle deflection</td>
<td>none</td>
<td>up 160°</td>
</tr>
<tr>
<td>Angle of visual field</td>
<td>75°</td>
<td>100°</td>
</tr>
<tr>
<td>Visible range</td>
<td>2-20 mm</td>
<td>3-50 mm</td>
</tr>
</tbody>
</table>

and those of the BF type 10 are compared in Table 2. The BF-1.8T has an outer diameter of 1.8 mm, which is about one third that of the conventional bronchofiberscope. Its image guide is composed of 3,000 glass fibers. In contrast, the conventional bronchofiberscope has about 10,000 glass fibers.

In contrast to the conventional bronchofiberscope with an observation range of 3-50 mm, the BF-1.8T brings the focal point more forward to obtain an observation range of 2-20 mm so that the approach of the lens surface to the subject does not interfere with observation and photography of peripheral airways. In addition, the tip of the BF-1.8T is rounded so as not to injure the mucosal surface of the peripheral airways.

At present, endoscopy of peripheral airways is mainly aimed at viewing inflammatory changes, whereas ordinary bronchofiberscopy is mainly aimed at observing changes in lung cancer, with observation of inflammatory changes a minor purpose. Therefore, changes in color tone of the bronchial mucosa are very important in endoscopy of peripheral airways. As natural a color tone as possible should be achieved, taking into consideration the intensity and kind of light as well as other factors that affect the color tone. We took still photographs with an SC-16-3 endoscopic camera with a xenon light source of the CLX-F type, and regulated the color tone by using test charts. In particular, characteristic features of the BF-1.8T were determined by using test charts, with special reference to changes in color tone due to the distance between the subject and the lens surface.

Method of Examination

Any bronchofiberscope with a channel for biopsy forceps insertion that allows a 1.8 mm BF-1.8T to pass through can be used with the BF-1.8T. We use BF type IT 10 (Olympus) and BF type IT 10R (Olympus), which have a wide channel and are easy to handle.

Before examination, sufficient laryngeal anesthesia (with 2 percent lidocaine spray) is performed to prevent coughing of the bronchial mucosa, hemoptysis and the coughing reflex. An intramuscular injection of 0.5 mg atropine sulfate is given 15-15 minutes before insertion of the bronchofiberscope into the trachea.

A conventional bronchofiberscope is inserted into the trachea with the patient in the supine position and wedged into the bronchus. Then, under monitoring by X-ray television, the BF-1.8T is slowly inserted from a channel of the bronchofiberscope up to a target site determined by preliminary roentgenography, with care not to injure the bronchial mucosa. The BF-1.8T is extended to thinner airways, making use of changes in the inner diameter of the bronchus respiration.

Even if secretions are so voluminous as to interfere with observation, aspiration is avoided in order to observe the bronchial mucosa under natural conditions. If observation is difficult, secretions between the tip of the BF-1.8T and the subject are removed, taking advantage of the characteristic feature that close-approach observation is possible within a range of 2-20 mm. Namely, the target area is observed while having the patient repeat respiration, or waving the tip of the BF-1.8T toward the right and left. With repeated trials, the

subjects were bronchiectasis in 22, bronchial asthma occurring in the area with air pollution in 22, bronchiolitis in 15, bloody sputum in ten, interstitial pneumonia in nine, chronic pulmonary emphysema in eight, bronchial asthma in eight, sarcoidosis in four, chronic bronchitis in four, lung cancer in four, pneumoconiosis in three, filariasis in two, and others.

All of the subjects underwent chest roentgenography, bronchoscopy, selective alveolobronchography, spirometry and P(A-a)O₂. All had lung biopsies except 45 subjects of whom five were normal, 22 had bronchiectasis, ten had bloody sputum and eight had bronchial asthma. Among the biopsied cases, 85 underwent transbronchial lung biopsy (TBLB) and 12 had lung biopsy by thoracotomy.

Characteristic Features of BF-1.8T (Table 2)

Bronchofiberscopes such as the BF type 10 (Olympus) are now used widely in clinics. These conventional bronchofiberscopes, measuring about 5-3 mm in outer diameter, allow observation and photography as deep as the subsegmental bronchi (4th order bronchi). The BF-1.8T, which enables observation of bronchi 2 mm or less in diameter, has an observation range of 2-20 mm, on the basis of morphometric theoretic values for airway dimensions. Although still uncertain, it probably allows observation and photography down to the 10th level of bronchial branches, taking into account the angle of bronchial bifurcation, static changes in bronchial diameter due to inflammation, and changes in the diameter of bronchi in inspiration and expiration for insertion (Fig 1). The performance and specification of the BF-1.8T

![Figure 1](image-url)
Table 3—Endoscopic Classification of Peripheral Airway Lesions

<table>
<thead>
<tr>
<th>a. Abnormal organic changes of the bronchial wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Reddening</td>
</tr>
<tr>
<td>2. Pallor</td>
</tr>
<tr>
<td>3. Absence of mucosal surface luster</td>
</tr>
<tr>
<td>4. Swelling (edema)</td>
</tr>
<tr>
<td>5. Engorgement of blood vessels</td>
</tr>
<tr>
<td>6. Irregular mucosal surface</td>
</tr>
<tr>
<td>7. Elevated mucosa</td>
</tr>
<tr>
<td>b. Endobronchial abnormalities</td>
</tr>
<tr>
<td>1. Stenosis</td>
</tr>
<tr>
<td>2. Obstruction</td>
</tr>
<tr>
<td>3. Ectasis</td>
</tr>
<tr>
<td>4. Compression</td>
</tr>
<tr>
<td>5. Abnormal findings at bifurcation</td>
</tr>
<tr>
<td>c. Abnormal substances in the bronchial lumen</td>
</tr>
<tr>
<td>1. Secretion</td>
</tr>
<tr>
<td>2. Pigmentation</td>
</tr>
</tbody>
</table>

knack of handling it is easily obtained. There have so far been several cases of difficult observation, but observation was nevertheless possible in all our cases.

At the time of observation, lung biopsy was undertaken via TBLB and roentgenography with a BF-1.8T. In order to carry out the lung biopsy as nearly as possible at the same site where the BF-1.8T was inserted, a roentgenogram of the site was taken. Using the roentgenogram for reference, the lung biopsy was carried out under fluororontgenographic observation.

RESULTS

Endoscopic Findings in Peripheral Airways (Table 3)

Endoscopic findings in 142 patients who were considered to have peripheral airway lesions were observed and photographed, and the results analyzed to prepare an endoscopic classification of peripheral airways.

When endoscopic findings in peripheral airways were compared with those of central airways, it was found that more findings were obtained in central airways and that all findings in peripheral airways were also obtained in central airways, except for pigmentation. However, even when findings in peripheral airways were identical to those obtained in central airways, the details were different in many cases.

Representative endoscopic findings in peripheral airways were: reddening, pallor, absence of mucosal luster, swelling (edema), engorgement of blood vessels, irregular mucosal surface, and elevated mucosa as abnormal changes in the wall; stenosis, obstruction, ectasis and compression, and abnormal findings at bifurcation as endobronchial abnormalities; and excessive secretion and pigmentation as abnormal substances in the bronchial lumen.

Compared endoscopic findings with pathologic findings in peripheral airways, reddening in endoscopic findings corresponded well to the changes of accumulation of neutrophils in the bronchial lumen, and congestion of vessels in the tissue, but pallor and absence of mucosal surface luster did not correspond to pathologic findings.

Ectasis was of different types such as in case 2, 3 and without changes of bronchial wall.

Pigmentation was anthracosis, and the color tone of the bronchial mucosa was black, blue, green and other colors.

CASE REPORTS

CASE 1

The patient was a 21-year-old man with no history of smoking, with normal pulmonary function test results, and with normal results of TBLB. Endoscopy of peripheral airways revealed no reddening or pigmentation, and no abnormality was found in the bronchial mucosa (Fig 2).

CASE 2

The patient was a 74-year-old man whose bronchogram showed ectatic airways with some radiotransparent areas in the lumen at B in the peripheral region. In this area, marked changes in the surface structure of the bronchial wall, which had not been found with ectatic airways, were observed (Fig 3).

CASE 3

The patient was a 51-year-old man whose bronchogram showed moniliform ectasis in the lower right lobe. The endoscopic findings (in the peripheral region) in peripheral airways were irregular mucosal surface, absence of mucosal surface luster, pigmentation

FIGURE 2. Endoscopic view of bronchioles of less than 2 mm in diameter in a healthy 21-year-old man.

FIGURE 3. The BF-1.8T discloses marked changes in the surface structure of the bronchiolar mucosa.
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Figure 4. The BF-1.8T reveals irregular mucosa surface, absence of mucosa surface luster, pigmentation and engorgement of veins at level of bronchioles less than 1.8 mm in diameter.

Figure 5. Macrophages with dark and brownish pigments densely scatter over the connective tissue of small bronchioles and lymph vessel (original magnification, ×200).

and engorgement of veins (Fig 4). TBLB showed accumulation of histiocytes phagocytophing carbon particles (Fig 5). The case was diagnosed as pneumonia by the occupational history and lung biopsy.

Case 4

The patient was a 20-year-old woman with bronchial asthma who was living in an area of severe air pollution. Bronchography revealed stenosis, pigmentation, excessive secretion, and absence of mucosal luster (Fig 6). TBLB showed anthracosis and the presence of giant cells and epithelioid cells in the bronchiolar wall, suggesting anthracotic granuloma formation.

From the present illness, clinical laboratory test findings, results of respiratory function test, and the results of lung biopsy, this case seemed to have morphologic conditions that are quite different from those in conventional cases of bronchial asthma occurring in areas of air pollution.

Case 5

The patient was a 20-year-old woman with sarcoidosis whose chest x-ray film showed swollen hilar lymph nodes in both lungs and disseminated granular shadows in the entire lung field. The endoscopic findings in peripheral airways were stenosis, small polypoid lesions, and elevated mucosa (Fig 7). TBLB showed the lesion protruding from the wall of the bronchiol toward the lumen.

The case was diagnosed as sarcoidosis by chest roentgenograms, high level of serum ACE, and lung biopsy.

Case 6

The patient was a 67-year-old woman with bronchiolitis whose chest x-ray film showed disseminated granular shadows in both lungs. Selective alveolography disclosed obstruction, dilatation and lack of bronchiolar bifurcation. The endoscopic findings in peripheral airways were stenosis, reddenning, and irregular mucosal surface (Fig 8). Bronchiol biopsy specimen from the proximal portion

Figure 6. The BF-1.8T reveals pigmentation and absence of mucosal surface luster at level of bronchioles.
FIGURE 8. The BF-1.8T discloses stenosis, reddening, and irregular mucosal surface at level of bronchioles less than 2 mm in diameter.

to the lesion observed by the endoscope showed marked changes of acute bronchiolitis.

Lung biopsy showed conditions in accordance with bronchiolitis. These findings along with the clinical laboratory test findings, led to a diagnosis of bronchiolitis.

DISCUSSION

Bronchioles are anatomically different from central airways in many ways: 1) there is no cartilage and hardly any bronchial glands; 2) the muscular tunic is well developed as compared with lumen; 3) there are only a few ciliated cells and hardly any goblet cells. Because of these differences, there should be some differences between endoscopic findings in peripheral airways and those in central airways. Therefore, it seems to be important to produce an endoscopic classification of peripheral airway lesions that is separate from the existing classification of bronchoscopic findings of large airway lesions.

In case 1, the comparison of the disease with other conditions in one of the normal subjects helps in recognizing abnormalities.

In case 3, a marked deposit of carbon powder was observed in the peripheral airway in pneumoconiosis. This condition, which is considered to be characteristic of pneumoconiosis, seems useful for early detection of this disease.

In case 4, there are endoscopic findings which have not been manifested in conventional cases of bronchial asthma. Taking these findings into consideration along with the histopathologic findings, the condition is considered to be a result of air pollution. This suggests that endoscopy can be a useful procedure for studying air pollution.

Case 5 is interesting in considering the morbidity of the peripheral airway in sarcoidosis when the endoscopic findings are taken along with the histopathologic findings. In such a case, a diffuse micronodular shadow in the lung field, endoscopy reveals vascular swelling, micronodular elevation, etc, but in a case showing no micronodular shadow in the lung field, the endoscopic findings are different.

In case 6 with bronchiolitis, endoscopy revealed inflammation in the bronchioli and was useful in understanding the severity of inflammation, etc. The significance of endoscopic examination thus seems to be great in diagnosing bronchiolitis.

Endoscopic findings in peripheral airways differ from those in central airways as follows: 1) an accordion pattern of alternating reddish mucosal surface and whitish cartilage rings is observed in central airways; particularly in the trachea; peripheral airways exhibit a similar accordion pattern of smooth muscles, despite there being no cartilage rings in peripheral airways; 2) longitudinal mucosal folds, common in segmental or subsegmental bronchi, cannot be found in peripheral airways; 3) in patients who smoke, inhaled substances are deposited in submucosal areas in peripheral airways, unlike in central airways, causing marked pigmentation; 4) changes in the structure of the bronchial mucosal surface associated with dilatation of peripheral airways, as found in case 2, are not observed in central airways; 5) pigmentation due to dust is more markedly observed in peripheral airways than in central airways.

Endoscopy of peripheral airways is still at the starting point. Fundamental studies with a large number of patients are required to support the analysis of endoscopic findings.

CONCLUSION

Endoscopy with our bronchofiberscope, which allows observation and photography of peripheral airways of 2 mm or less, was performed in 142 patients. Special attention was paid to obtaining as natural a color tone of the bronchial mucosa as possible.

An endoscopic classification of peripheral airway lesions was proposed, and ordinary endoscopic findings of the peripheral airway and specific findings from patients with peripheral airway lesions were investigated, as an initial step in determining an analysis of endoscopic findings.

REFERENCES

Annex 8

WPR/OCH/(3)/88.5

14 Weibel ER. Morphometry of the human lung. Berlin: Springer, 1953
1. Overview of pneumoconiosis and coal mines in Korea

1.1 Prevalence of pneumoconiosis in Korean coal miners

1) Situation of dust exposure related diseases in work related diseases (from the research of St. Catholic University)

[The result of special health examination for work related diseases]
Total No. of plants; 5,273,
Total No. of workers examined; 305,349
Total No. of yields; 8,660 (Prevalence rate=8,660/305,349=2.8%)

<table>
<thead>
<tr>
<th>Type of hazard (Type of work)</th>
<th>Subjects examined</th>
<th>yields</th>
<th>prevalence rate(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>dust</td>
<td>135,698</td>
<td>4,959</td>
<td>3.7</td>
</tr>
<tr>
<td>vibration</td>
<td>5,242</td>
<td>36</td>
<td>0.7</td>
</tr>
<tr>
<td>noise</td>
<td>169,639</td>
<td>3,509</td>
<td>2.1</td>
</tr>
<tr>
<td>heavy load</td>
<td>827</td>
<td>10</td>
<td>1.2</td>
</tr>
<tr>
<td>organic solvent</td>
<td>25,799</td>
<td>77</td>
<td>0.3</td>
</tr>
<tr>
<td>special chemical substance</td>
<td>9,410</td>
<td>10</td>
<td>0.1</td>
</tr>
<tr>
<td>lead</td>
<td>11,053</td>
<td>57</td>
<td>0.5</td>
</tr>
<tr>
<td>others</td>
<td>5,180</td>
<td>2</td>
<td>0.03</td>
</tr>
</tbody>
</table>

% of Pneumoconiosis in total yields: 4,959/8,660=57.3%
2) Dust concentration and prevalence of pneumoconiosis in Korean coalminers (extracts from (1) "Im Goung Yun, Young Lim and Young Zun Kim; The epidemiological study on dust concentration and prevalence of pneumoconiosis in Korean coalminers. Korean J. Occup. Health, 27(1), pp27-37, 1988" and (2) "Kyu Sang Cho, Im Goung Yun, Seung Han Lee and Jung Man Kim; A study on progression of pneumoconiosis among Korean coalface workers with special reference to anthracite exposure, Catholic Industrial Medical Center, Catholic Medical College, Seoul, 1985")

<table>
<thead>
<tr>
<th>Tab. 1</th>
<th>Distribution of the subject according to the kind of underground Work(1984)</th>
</tr>
</thead>
<tbody>
<tr>
<td>total number of subject</td>
<td>coalface workers (%)</td>
</tr>
<tr>
<td>240,000</td>
<td>12,796(53.3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tab. 2</th>
<th>Dust concentration in the coalface and tunnel drill according to the scale of collieries(1984)</th>
</tr>
</thead>
<tbody>
<tr>
<td>work place</td>
<td>dust</td>
</tr>
<tr>
<td></td>
<td>respirable</td>
</tr>
<tr>
<td>&lt;500</td>
<td>2.54±1.43</td>
</tr>
<tr>
<td>500</td>
<td>3.80±1.86</td>
</tr>
<tr>
<td>siO₂ (%)</td>
<td>1.97±1.38</td>
</tr>
<tr>
<td>&lt;500</td>
<td>0.97±0.92</td>
</tr>
<tr>
<td>500</td>
<td>5.94±1.31</td>
</tr>
</tbody>
</table>
Tab. 3 Prevalence rate of pneumoconiosis according to the scale of collieries (1984)

<table>
<thead>
<tr>
<th>Category</th>
<th>0/0</th>
<th>0/1</th>
<th>1/0-1/2</th>
<th>2/1-2/3</th>
<th>3/2-3/+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subj.</td>
<td>case</td>
<td>%</td>
<td>case</td>
<td>%</td>
<td>case</td>
<td>%</td>
</tr>
<tr>
<td>CF3,052</td>
<td>2,596</td>
<td>85.5</td>
<td>144</td>
<td>4.7</td>
<td>218</td>
<td>7.1</td>
</tr>
<tr>
<td>TD1,136</td>
<td>858</td>
<td>75.5</td>
<td>100</td>
<td>8.8</td>
<td>106</td>
<td>9.3</td>
</tr>
<tr>
<td>OT2,008</td>
<td>1,721</td>
<td>85.7</td>
<td>107</td>
<td>5.3</td>
<td>109</td>
<td>5.4</td>
</tr>
<tr>
<td>subtotal</td>
<td>6,196</td>
<td>5,174</td>
<td>351</td>
<td>433</td>
<td>151</td>
<td>87</td>
</tr>
<tr>
<td>500&lt;</td>
<td>CF9,744</td>
<td>8,342</td>
<td>85.6</td>
<td>526</td>
<td>5.4</td>
<td>676</td>
</tr>
<tr>
<td>TD2,408</td>
<td>2,085</td>
<td>86.6</td>
<td>119</td>
<td>4.9</td>
<td>140</td>
<td>5.8</td>
</tr>
<tr>
<td>OT5,652</td>
<td>5,095</td>
<td>90.1</td>
<td>273</td>
<td>4.8</td>
<td>214</td>
<td>3.8</td>
</tr>
<tr>
<td>subtotal</td>
<td>17,804</td>
<td>15,522</td>
<td>918</td>
<td>1,303</td>
<td>238</td>
<td>96</td>
</tr>
<tr>
<td>total</td>
<td>24,000</td>
<td>20,696</td>
<td>86.2</td>
<td>1,269</td>
<td>5.3</td>
<td>1,463</td>
</tr>
</tbody>
</table>

CF: coalface worker **: P<0.01
TD: tunnel driller
OT: others

Tab. 4 Prevalence rate of pneumoconiosis according to the duration of the dust exposure (1984)

<table>
<thead>
<tr>
<th>Duration</th>
<th>coalface worker</th>
<th>tunnel driller</th>
<th>others</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subj.</td>
<td>case</td>
<td>%</td>
<td>case</td>
<td>%</td>
</tr>
<tr>
<td>&lt;4</td>
<td>6,684</td>
<td>146</td>
<td>2.2</td>
<td>1,507</td>
</tr>
<tr>
<td>5-9</td>
<td>3,472</td>
<td>639</td>
<td>18.4</td>
<td>1,165</td>
</tr>
<tr>
<td>10-14</td>
<td>1,794</td>
<td>640</td>
<td>35.7</td>
<td>564</td>
</tr>
<tr>
<td>15-19</td>
<td>616</td>
<td>305</td>
<td>49.5</td>
<td>206</td>
</tr>
<tr>
<td>20</td>
<td>230</td>
<td>119</td>
<td>51.7</td>
<td>102</td>
</tr>
<tr>
<td>total</td>
<td>12,796</td>
<td>1,849</td>
<td>14.4</td>
<td>3,544</td>
</tr>
</tbody>
</table>

**: P<0.01

Tab. 5 Prevalence rate of pneumoconiosis according to the age and kind of underground work (1984)

<table>
<thead>
<tr>
<th>Age</th>
<th>CF</th>
<th>31-40</th>
<th>41-50</th>
<th>51&lt;</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>subj</td>
<td>case</td>
<td>%</td>
<td>case</td>
<td>%</td>
<td>case</td>
</tr>
<tr>
<td>&lt;30</td>
<td>3,871</td>
<td>3,816</td>
<td>98.6</td>
<td>34</td>
<td>0.9</td>
</tr>
<tr>
<td>31-40</td>
<td>5,257</td>
<td>4,758</td>
<td>90.5</td>
<td>230</td>
<td>4.4</td>
</tr>
<tr>
<td>41-50</td>
<td>3,362</td>
<td>2,205</td>
<td>65.6</td>
<td>363</td>
<td>10.3</td>
</tr>
<tr>
<td>51&lt;</td>
<td>306</td>
<td>158</td>
<td>51.6</td>
<td>44</td>
<td>14.1</td>
</tr>
<tr>
<td>TD</td>
<td>935</td>
<td>913</td>
<td>97.6</td>
<td>16</td>
<td>1.7</td>
</tr>
<tr>
<td>&lt;30</td>
<td>1,546</td>
<td>1,358</td>
<td>87.8</td>
<td>91</td>
<td>5.9</td>
</tr>
<tr>
<td>31-40</td>
<td>940</td>
<td>600</td>
<td>63.8</td>
<td>96</td>
<td>10.2</td>
</tr>
<tr>
<td>41-50</td>
<td>123</td>
<td>59</td>
<td>48.0</td>
<td>16</td>
<td>13.0</td>
</tr>
<tr>
<td>OT</td>
<td>1,944</td>
<td>1,927</td>
<td>99.1</td>
<td>14</td>
<td>0.7</td>
</tr>
<tr>
<td>&lt;30</td>
<td>2,765</td>
<td>2,611</td>
<td>94.4</td>
<td>90</td>
<td>3.2</td>
</tr>
<tr>
<td>31-40</td>
<td>2,523</td>
<td>2,009</td>
<td>79.6</td>
<td>215</td>
<td>8.5</td>
</tr>
<tr>
<td>51&lt;</td>
<td>428</td>
<td>269</td>
<td>62.9</td>
<td>61</td>
<td>14.3</td>
</tr>
<tr>
<td>total</td>
<td>24,000</td>
<td>20,683</td>
<td>86.2</td>
<td>1,270</td>
<td>5.3</td>
</tr>
</tbody>
</table>

CF: coalface work TD: tunnel drill OT: others **: P<0.01
### Tab. 6 Prevalence rate of pneumoconiosis according to the respirable dust level in coal face (1984)

<table>
<thead>
<tr>
<th>Category</th>
<th>D.I.D (%)</th>
<th>0 / 0</th>
<th>0 / 1</th>
<th>1 / 0-1 / 2</th>
<th>2 / 1-2 / 3</th>
<th>3 / 2-3 / +</th>
<th>subtotal</th>
<th>total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>low (2 mg/l)</td>
<td>(84.5)</td>
<td>2,590</td>
<td>195</td>
<td>224</td>
<td>36</td>
<td>19</td>
<td>474</td>
<td>3,064</td>
</tr>
<tr>
<td>middle (2.4 mg/l)</td>
<td>(84.4)</td>
<td>3,465</td>
<td>222</td>
<td>316</td>
<td>70</td>
<td>32</td>
<td>640</td>
<td>4,105</td>
</tr>
<tr>
<td>high (4 mg/l)</td>
<td>(86.8)</td>
<td>4,882</td>
<td>253</td>
<td>354</td>
<td>104</td>
<td>34</td>
<td>745</td>
<td>5,272</td>
</tr>
<tr>
<td>total</td>
<td></td>
<td>10,937</td>
<td>670</td>
<td>894</td>
<td>210</td>
<td>85</td>
<td>1,859</td>
<td>12,796</td>
</tr>
</tbody>
</table>

### Tab. 7 Prevalence rate of pneumoconiosis according to the free silica level in coal face (1984)

<table>
<thead>
<tr>
<th>Category</th>
<th>D.I.D (%)</th>
<th>0 / 0</th>
<th>0 / 1</th>
<th>1 / 0-1 / 2</th>
<th>2 / 1-2 / 3</th>
<th>3 / 2-3 / +</th>
<th>subtotal</th>
<th>total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>low (&lt;1%)</td>
<td>(83.6)</td>
<td>3,153</td>
<td>155</td>
<td>273</td>
<td>76</td>
<td>26</td>
<td>530</td>
<td>3,683</td>
</tr>
<tr>
<td>middle (1.2%)</td>
<td>(86.1)</td>
<td>7,077</td>
<td>434</td>
<td>551</td>
<td>112</td>
<td>45</td>
<td>1,142</td>
<td>8,219</td>
</tr>
<tr>
<td>high (2%)</td>
<td>(79.1)</td>
<td>707</td>
<td>81</td>
<td>70</td>
<td>22</td>
<td>14</td>
<td>187</td>
<td>894</td>
</tr>
<tr>
<td>total</td>
<td></td>
<td>10,937</td>
<td>670</td>
<td>894</td>
<td>210</td>
<td>85</td>
<td>1,859</td>
<td>12,796</td>
</tr>
</tbody>
</table>

### Tab. 8 Exposure period in the development of simple pneumoconiosis in years

<table>
<thead>
<tr>
<th>Exposure period</th>
<th>Category</th>
<th>0 / 0</th>
<th>0 / 1</th>
<th>1 / 0</th>
<th>1 / 1</th>
<th>1 / 2</th>
<th>2 / 1</th>
<th>2 / 2</th>
<th>2 / 3</th>
<th>3 / 2</th>
<th>3 / 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>range</td>
<td></td>
<td>0-29</td>
<td>0-29</td>
<td>1-29</td>
<td>2-28</td>
<td>5-25</td>
<td>5-24</td>
<td>5-26</td>
<td>6-22</td>
<td>8-22</td>
<td>7-22</td>
</tr>
<tr>
<td>lower 5 percentile</td>
<td></td>
<td>1.0</td>
<td>3.5</td>
<td>4.5</td>
<td>5.1</td>
<td>7.1</td>
<td>6.4</td>
<td>7.1</td>
<td>8.1</td>
<td>10.2</td>
<td>9.1</td>
</tr>
<tr>
<td>median</td>
<td></td>
<td>3.5</td>
<td>9.2</td>
<td>10.3</td>
<td>11.4</td>
<td>11.1</td>
<td>12.5</td>
<td>13.3</td>
<td>15.2</td>
<td>14.2</td>
<td></td>
</tr>
</tbody>
</table>
### Table 1. The respirable dust concentration and free silica content in Korean collieries.

<table>
<thead>
<tr>
<th>Scale of collieries (No. of workers)</th>
<th>&lt; 500</th>
<th>500 ≤</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of mines</td>
<td>11</td>
<td>9</td>
<td>20</td>
</tr>
<tr>
<td>Respirable dust (mg/m³)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± S.D.</td>
<td>2.54 ± 1.43</td>
<td>3.80 ± 1.86</td>
<td>3.07 ± 1.78</td>
</tr>
<tr>
<td>Range</td>
<td>0.70 - 8.30</td>
<td>1.40 - 7.50</td>
<td>0.70 - 8.30</td>
</tr>
<tr>
<td>SiO₂(%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± S.D.</td>
<td>1.97 ± 1.38</td>
<td>0.97 ± 0.92</td>
<td>1.62 ± 1.30</td>
</tr>
<tr>
<td>Range</td>
<td>0.10 - 6.35</td>
<td>0.03 - 3.07</td>
<td>0.03 ± 6.35</td>
</tr>
<tr>
<td>Fixed Carbon(%)**</td>
<td>46.2 - 59.3</td>
<td>51.9 - 79.1</td>
<td>46.2 - 79.1</td>
</tr>
<tr>
<td>Moisture(%)**</td>
<td>4.3 - 4.6</td>
<td>3.2 - 3.9</td>
<td>3.2 - 4.6</td>
</tr>
</tbody>
</table>

* Total dust (mg/m³): 9.13 ± 4.74 (Range 2.15 - 29.43)

** Data cited from a report by the Korea Coal Corp.

### Table 2. The number of collieries classified according the respirable dust concentration and silica content.

<table>
<thead>
<tr>
<th>Range* (mg/m³)</th>
<th>No.</th>
<th>%</th>
<th>Range† (mg/m³)</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2</td>
<td>7</td>
<td>35</td>
<td>&lt; 1</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>2 - 4</td>
<td>7</td>
<td>35</td>
<td>1 - 2</td>
<td>13</td>
<td>65</td>
</tr>
<tr>
<td>4 &lt;</td>
<td>6</td>
<td>30</td>
<td>2 &lt;</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>

* 0.70 - 8.30 mg/m³  † 0.03 - 6.35%
1.2 Background concerning Korean coal mines (1984):

1) Use
   a) electric power plants; 25% of total generation of electricity
   b) home heating; major fuel

2) Supply of anthracite*
   a) Domestic production; 21 million ton/year
   b) Import; 804 thousand ton

* coal mines in Korea are only for anthracite, and major use is for home heating.

2. Result of spot sampling in an anthracite mine.
   Name of mine: Moonkyung colliery (開慶鉱業所) of Daesung Consolidated Coal mining Co., LTD (大成炭業開発株式会社)

Profile of the Moonkyung colliery
   Sort of coal; anthracite for house heating
   Reserve; 31,352,000 M/T
   Annual production; 860,000 M/T
   No. of employee; 1,200 (400/shift, 8hrs/day including 1hr rest for lunch, underground during 9hrs, Saturday and Sunday are holidays)
2.1 Respirable dust concentration by Laser Dust Monitor (LD-1, Shibata Scientific Industry) at some points in underground

<table>
<thead>
<tr>
<th>Place sampled</th>
<th>No. of points</th>
<th>Concentration (mg/cubic m)</th>
<th>max</th>
<th>min</th>
</tr>
</thead>
<tbody>
<tr>
<td>shafts and levels</td>
<td>6</td>
<td></td>
<td>2.70</td>
<td>0.80</td>
</tr>
<tr>
<td>near the conveyer</td>
<td>4</td>
<td></td>
<td>4.70(a)</td>
<td>2.00</td>
</tr>
<tr>
<td>beneath the quarry</td>
<td>2</td>
<td></td>
<td>7.60(b)</td>
<td>5.50</td>
</tr>
<tr>
<td>workface (resting)</td>
<td>4</td>
<td></td>
<td>1.90(c)</td>
<td>1.20</td>
</tr>
</tbody>
</table>

(a), (b), (c): Photographs (a), (b), (c) for reference, respectively
2.2 Photographs of particles sampled in Moonkyung mine with the cascade impactor (With scanning electron microscope and the energy disperse X-ray analyser)

Photograph 2.1. Crude particles (step No.2)
Photograph 2.2. Medium size particles (step No. 5)
Photograph 2.3. Fine particles (step No. 8)
3. The clinical examinations on pneumoconiosis patients

3.1 Study subjects and list of examination performed on each subject

(July, 1988)

<table>
<thead>
<tr>
<th>ID No.</th>
<th>Age</th>
<th>Duration of exposure (yrs)</th>
<th>Year of first exposure (yrs)</th>
<th>Examinations</th>
</tr>
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<td>KP-1</td>
<td>61</td>
<td>13</td>
<td>1966 (22)*</td>
<td>○</td>
</tr>
<tr>
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<td>50</td>
<td>19</td>
<td>1964 (24)</td>
<td>○</td>
</tr>
<tr>
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<td>55</td>
<td>32</td>
<td>1950 (38)</td>
<td>○</td>
</tr>
<tr>
<td>KP-4</td>
<td>54</td>
<td>20</td>
<td>1965 (23)</td>
<td>○</td>
</tr>
<tr>
<td>KP-5</td>
<td>60</td>
<td>6</td>
<td>1977 (11)</td>
<td>○</td>
</tr>
<tr>
<td>KP-6</td>
<td>52</td>
<td>11</td>
<td>1960 (28)</td>
<td>○</td>
</tr>
<tr>
<td>KP-7</td>
<td>48</td>
<td>10</td>
<td>1970 (18)</td>
<td>○</td>
</tr>
<tr>
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<td>72</td>
<td>35</td>
<td>1944 (44)</td>
<td></td>
</tr>
<tr>
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<td>56</td>
<td>20</td>
<td>1961 (27)</td>
<td></td>
</tr>
<tr>
<td>Q-3</td>
<td>32</td>
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<td>16</td>
<td>1961 (27)</td>
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<td>50</td>
<td>14</td>
<td>1967 (21)</td>
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<tr>
<td>Q-6</td>
<td>56</td>
<td>7</td>
<td>1959 (29)</td>
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<td>Q-7</td>
<td>53</td>
<td>10</td>
<td>1964 (24)</td>
<td></td>
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<tr>
<td>Q-8</td>
<td>57</td>
<td>18</td>
<td>1962 (26)</td>
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</tr>
<tr>
<td>Q-9</td>
<td>59</td>
<td>17</td>
<td>1966 (22)</td>
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</tr>
<tr>
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<td>1976 (22)</td>
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</tr>
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<td>16</td>
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<td>11</td>
<td>1960 (28)</td>
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<td>Q-23</td>
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<td>14</td>
<td>1958 (30)</td>
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<td>Q-24</td>
<td>53</td>
<td>12</td>
<td>1969 (19)</td>
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<td>10</td>
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<td>Q-26</td>
<td>36</td>
<td>13</td>
<td>1972 (16)</td>
<td></td>
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<td>Q-27</td>
<td>57</td>
<td>23</td>
<td>1964 (24)</td>
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<td>Q-28</td>
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<td>1951 (37)</td>
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<td>Q-29</td>
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<td>12</td>
<td>1957 (31)</td>
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<td>49</td>
<td>10</td>
<td>1976 (12)</td>
<td></td>
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<tr>
<td>Q-32</td>
<td>57</td>
<td>11</td>
<td>1958 (30)</td>
<td></td>
</tr>
</tbody>
</table>

All the subject are male, *; Latent period from first exposure (yrs)
### 3.2 Demographic characteristics and history of exposure on selected 7 cases of pneumoconiosis.

<table>
<thead>
<tr>
<th>ID No. of Subjects</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>History of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>KP-1 韩 永年</td>
<td>60</td>
<td>163.3</td>
<td>48.0</td>
<td>coal face (1966-1979;13years)</td>
</tr>
<tr>
<td>KP-2 韩 良年</td>
<td>52</td>
<td>158.9</td>
<td>45.0</td>
<td>underground safety stuff of coal mine(1964-1972;8years) coal face (1972-1983;11years)</td>
</tr>
<tr>
<td>KP-3 李 镭年</td>
<td>55</td>
<td>167.9</td>
<td>59.0</td>
<td>drilling of coal mine (1950-1982;32years)</td>
</tr>
<tr>
<td>KP-4 崔 寅年</td>
<td>54</td>
<td>160.0</td>
<td>47.0</td>
<td>coal cutter (coal face) (1965-1985;20years)</td>
</tr>
<tr>
<td>KP-5 朴 敷年</td>
<td>60</td>
<td>158.5</td>
<td>54.0</td>
<td>stone destroy (in coal mine?) (1977-1983;6years)</td>
</tr>
<tr>
<td>KP-6 張 求年</td>
<td>52</td>
<td>162.0</td>
<td>49.0</td>
<td>coal cutter (coal face) (1960-1971;11years)</td>
</tr>
<tr>
<td>KP-7 欧 求年</td>
<td>48</td>
<td>162.0</td>
<td>56.0</td>
<td>silica mine, work face (1970-1980;10years)</td>
</tr>
</tbody>
</table>

**Appendix. List of examinations performed in this study (nation of examiner)**

1. Bronchofiberscopy (Korea, Japan)
2. Chest rentogenographic diagnosis and interpretation of pneumoconiosis according to ILO Classification, 1980. (Korea, Japan)
3. Pulmonary function test (Korea)
   - List of indices of pulmonary function tests including demographic data for calculation of predicted values by the bodyplethysmograph
   - Age, Height(cm), Weight(kg), VC(L), FVC(L), RV(L), FRC(L), TLC(L), FEV1.0(L), FEV1.0%(%), RV/TLC(%), PFR(L/sec), V75(L/sec), V50(L/sec), V25(L/sec), Raw(cmH20/L/sec), Cst(L/cmH20)
4. Trans bronchial lung biopsy(TBLB), pathological diagnosis (Japan)
5. Broncho alveolar lavage(BAL), cell diagnosis (Korea, Japan)
6. Respiratory symptoms used standardized questionnaire (Korea, Japan)
7. Electrocardiograph (Korea, Japan)
8. Spot sampling of dusts in underground air of a coal mine (Japan)
### 3.3 Past history of respiratory diseases, respiratory symptoms, smoking, and alcohol consumption on selected 7 cases of pneumoconiosis.

<table>
<thead>
<tr>
<th>ID No. of Subjects</th>
<th>KP-1</th>
<th>KP-2</th>
<th>KP-3</th>
<th>KP-4</th>
<th>KP-5</th>
<th>KP-6</th>
<th>KP-7</th>
</tr>
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<td><strong>Past Illness</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) Pulmonary tuberculosis</td>
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<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
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<tr>
<td>(2) Pleuritis</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>(3) Chronic bronchitis</td>
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<td>YES</td>
<td>-</td>
<td>YES</td>
<td>-</td>
<td>-</td>
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<tr>
<td>(4) Bronchiectasia</td>
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<td>-</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
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<td>YES</td>
<td>-</td>
<td>YES</td>
<td>-</td>
<td>-</td>
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<tr>
<td>(6) Emphysema</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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<td>-</td>
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<tr>
<td>(7) Cardiac disease</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>(8) Other chest illness</td>
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<td>-</td>
<td>YES</td>
<td>YES</td>
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<td><strong>Pulmonary Symptoms</strong></td>
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<td>(1) Breathlessness</td>
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<td>V</td>
<td>IV</td>
<td>V</td>
<td>V</td>
<td>V</td>
<td>V</td>
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<td>(2) Cough</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>1) Cough on getting up in winter</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>2) Cough like this, ≥ 5 days/week</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>1') Cough in winter, day or night</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
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<tr>
<td>2') Cough ≥ 7 times, ≥ 5 days/week</td>
<td>YES</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
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<tr>
<td>3) Most days for ≥ 3 months/yr</td>
<td>YES</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
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<tr>
<td>(3) Phlegm</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) Phlegm on getting up in winter</td>
<td>NO</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>2) Phlegm like this, ≥ 5 days/week</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
</tr>
<tr>
<td>1') Phlegm in winter, day or night</td>
<td>-</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>2') Phlegm ≥ 2 times, ≥ 5 days/week</td>
<td>-</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>3) Most days for ≥ 3 months/yr</td>
<td>-</td>
<td>YES</td>
<td>-</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
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<tr>
<td>(4) Palpitation</td>
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<td>Palpitation at light movement</td>
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<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) Have you ever smoke?</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>2) Are you a current smoker?</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>*3) When did you quit? (yrs ago)</td>
<td>3</td>
<td>1</td>
<td>-</td>
<td>5</td>
<td>7</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Why did you quit?</td>
<td>B</td>
<td>B</td>
<td>-</td>
<td>B</td>
<td>B</td>
<td>B</td>
<td>A</td>
</tr>
<tr>
<td>4) When did you start? (yrs old)</td>
<td>25</td>
<td>20</td>
<td>-</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>22</td>
</tr>
<tr>
<td>5) How many? (pieces/day)</td>
<td>5-14</td>
<td>15-24</td>
<td>-</td>
<td>5-14</td>
<td>5-14</td>
<td>5-14</td>
<td>25</td>
</tr>
<tr>
<td>(6) Alcohol consumption</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) Do you drink it habitually?</td>
<td>YES</td>
<td>YES</td>
<td>NO</td>
<td>×</td>
<td>NO</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>2) When did you start? (yrs old)</td>
<td>25</td>
<td>20</td>
<td>-</td>
<td>×</td>
<td>-</td>
<td>25</td>
<td>21</td>
</tr>
<tr>
<td>3) How much/day? (ml as alcohol)</td>
<td>×</td>
<td>60</td>
<td>-</td>
<td>×</td>
<td>-</td>
<td>-</td>
<td>×</td>
</tr>
</tbody>
</table>

* ×: no adequate statement, -: answered "NO" or need not to answer
* Why you quitted smoking? A: own will, B: due to diseases, doctor's advice C: other reasons.
INTRODUCTION

There are many papers reporting on pneumoconiosis risks in coal mines. However, the overwhelming majority of them do not include quantitative data on levels of exposure to or concentrations of dust.

After reviewing the articles published on the relation between exposure to coal dust and pneumoconiosis, the WHO Study Group on "Health-Based Occupational Exposure Limits for Selected Mineral Dusts" (Silica, Coal) in 1984 concluded that the hazard associated with a given exposure to coal-mine dust is affected by a number of factors which, by themselves or in combination, may alter the risk in the order of magnitude. These factors include the rank of coal (or its carbon content), the proportion of free silica in the dust, proportions of other non-coal minerals present, and the particle size distribution of the dust. The Study Group therefore concluded that no single figure could be chosen validly as a exposure limit.

The study group tentatively recommended 0.5 to 4.0 mg/m³ of coal dust concentration as health-based exposure limit. The range of limit varies widely depending on the composition of coal.

The dust concentrations established as health-based exposure limits by the Study Group were defined based on average working-life amounting
to about 560,000 hours (8 hr shift, 40 hr working week, 35 years working life).

The limits are expected to ensure that risks of developing massive fibrosis during such a working life do not exceed two in a thousand. The figures quoted refer to concentration of respirable dust containing less than 7% free silica. When the silica content exceeds 7%, then the exposure limit should follow the recommendations for silica dust.

Anthracite is widely used in Korean households as a major source of energy. It is mined by the traditional method and the anthracite mining has always been the most dangerous of the Korean industries with respect to both the safety and health of workers.

This study was carried out for the purpose of disclosing radiographic progression of coal workers' pneumoconiosis of Korean coal-face workers in the course of employment in underground anthracite mines, and the results were compared with the incidence rates shown by other studies and the Health-based limits which were tentatively set by the WHO Study Group.

This is a report of the study preliminary report was presented at the Pittsburgh Conference, supported by IDRC research grant, recommending a practical basis for the establishment of environmental and managerial preventive measures for coal workers' pneumoconiosis in anthracite mines in Korea.

METHOD OF STUDY

Dust measurement: For all the 20 collieries of this study, five sample points of caves in individual collieries were selected by random
sampling method. At each sample cave, two workers were asked to wear a personal air sampler (DuPont P4000) each during their total shift-work hours. The pump will draw 1.7 liter per minute of air through a 10 mm nylon cyclone. Of the two samples collected at each sample cave, one was used for free silica analysis and the other one for the estimation of the amount of dust inhaled. Free silica analysis was carried out by X-ray diffraction method and airborne dust concentration was measured by Piezo-balance.

**Duration of dust exposure and other information:** Each coal-face worker was interviewed to note the dust exposure period in each colliery and also his previous experience at the time of chest X-ray examination. The age of workers and the area of the mine where they had worked previously were noted at the same time.

**Chest X-ray examination:** Chest X-rays were taken on 12,796 coal-face workers who were employed in the above collieries. X-ray with rotating anode tube (500 mA. 60KVP) was applied at the distance of 1.5 m. 14 x 14" size films were employed in this study. Two specialists, who had more than 20 years of reading experience participated in the reading of the films. The stages of pneumoconiosis were classified according to the ILO/UC International Classification of Radiographs of Pneumoconiosis (1971).

**RESULT**

**Coal dust exposure**

As shown in Table I the mean concentration of the total dust in air at working areas was 9.13 ± 4.74 mg/m³ (2.15 - 29.43 mg/m³) and the mean
concentration of respirable dust was $3.07 \pm 1.78 \text{ mg/m}^3$ ($0.70 - 8.30 \text{ mg/m}^3$).

The mean content of silica in coal dust was $1.62 \pm 1.30\%$ ($0.03 - 6.35\%$).

The distribution of individual collieries by respirable coal dust concentration and silica content was shown in Table II. By airborne dust concentration, 7 collieries (35\%) had a dust concentration of under 2 mg/m$^3$ (TLV of ACGIH) and in 13 collieries the concentration exceeded 2 mg/m$^3$. Six collieries among them had over 4 mg/m$^3$. As for silica content, 4 collieries had a content of under 1\% and 13 collieries had a range of 1 to 2\%, and at the other three collieries the contents exceeded 2\%.

The selected collieries were divided into two groups, based on the number of workers in collieries with more than 500 workers and ones having less than 500 workers. The mean respirable dust concentration and silica content of coal dust were in large-scale collieries, $2.54 \pm 1.43 \text{ mg/m}^3$ ($0.70 - 8.30 \text{ mg/m}^3$) and $1.97 \pm 1.38\%$ ($0.10 - 6.35\%$) respectively and $3.80 \pm 1.86 \text{ mg/m}^3$ ($1.40 - 7.50 \text{ mg/m}^3$), $0.97 \pm 0.92\%$ ($0.03 - 3.07\%$) in small-scale collieries. But the difference in dust concentration between the two groups was not significant statistically.

Prevalence of pneumoconiosis

The prevalence of each category of pneumoconiosis among coal-face workers was shown in Table IV. Of the total number of 12,796 coal-face workers, 1,189 cases showed simple pneumoconiosis, showing the overall prevalence rate of 9.3\%. The number of pneumoconiosis with large opacities which was not shown in the table was very small, accounting only for 13 cases or 0.01\% of the total number of coal-face workers.
Among the various categories of pneumoconiosis, category 1 was most frequent, accounting for 7.0% of the total number of coal-face workers, and was followed by category 2 and 3 in descending order.

Meanwhile, the prevalence of pneumoconiosis increased in the course of dust exposure until 20-24 years. However, category 2 reached the highest value in 15-19 years.

The decline may be partly attributed to the fact that coal-face workers retire rather early, because the age of retirement was customarily set at 48-53 yrs for underground mining workers in this country.

The overall prevalence of subcategory 0/1 or suspect cases was 5.2%. It was also noted that subcategory 0/1 showed the increase of the prevalence rate up to 15-19 years of dust exposure as well.

**Duration of dust exposure required for the occurrence of pneumoconiosis**

The distribution of each category of pneumoconiosis in the course of dust exposure was shown in Table V, and the range, the years corresponding to 5 percentile of pneumoconiosis distribution and the median were summarized in Table VI.

Generally speaking, the progression of pneumoconiosis showed a close association with the duration of dust exposure. Subcategory 0/1 was first observed within one year of dust exposure, subcategory 1/1 within three years, subcategory 2/2 within five years and subcategory 3/3 within eight years. The occurrence of lower 5-percentile of pneumoconiosis was noted at 3.5 years in subcategory 0/1, at 5.1 years in subcategory 1/1, at 7.1 years in subcategory 2/2 and at 9.1 years in subcategory 3/3. The median was 9.2 years in subcategory 0/1, 11.4
years in subcategory 1/1, 13.3 years in subcategory 2/2 and 14.2 years in subcategory 3/3.

In view of the findings on these relations between coal dust exposure and health effect among the Korean underground workers, the TLV of 2 mg/m$^3$ of ACGIH or 4 mg/m$^3$ of the West Germany seems too high and the tentative level of 0.5 mg/m$^3$, which has advocated by the WHO Study Group, may be more reasonable particularly with regard to anthracite dust.
Table I. The mean concentration of coal dust and silica content in the 20 Korean Anthracite mines

<table>
<thead>
<tr>
<th></th>
<th>Total dust (mg/m³)</th>
<th>Respirable dust (mg/m³)</th>
<th>SiO₂ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± S.D.</td>
<td>9.13 ± 4.74</td>
<td>3.07 ± 1.78</td>
<td>1.62 ± 1.30</td>
</tr>
<tr>
<td>Range</td>
<td>(2.15 - 29.43)</td>
<td>(0.70 - 8.30)</td>
<td>(0.03 - 6.35)</td>
</tr>
</tbody>
</table>

Table II. Distribution of dust concentration and silica content by number of collieries

<table>
<thead>
<tr>
<th>Respirable dust (mg/m³)</th>
<th>SiO₂ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range*</td>
<td>No</td>
</tr>
<tr>
<td>&lt; 2</td>
<td>7</td>
</tr>
<tr>
<td>2-4</td>
<td>7</td>
</tr>
<tr>
<td>&gt; 4</td>
<td>6</td>
</tr>
</tbody>
</table>

* 0.70 - 8.30 mg/m³  
⁺ 0.03 - 6.35%
Table III. Respirable dust concentration and free silica content by area and size of collieries

<table>
<thead>
<tr>
<th>Area</th>
<th>Size of mine (No. of workers)</th>
<th>&lt; 500</th>
<th>500 &lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of mines</td>
<td>Changsong Wongwol</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>Respirable dust (mg/m³)</td>
<td></td>
<td>3.67 ± 2.04</td>
<td>2.13 ± 1.37</td>
</tr>
<tr>
<td>Mean ± S.D.</td>
<td></td>
<td>1.06 - 8.30</td>
<td>0.70 - 6.55</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>0.70 - 8.30</td>
<td>1.40 - 7.50</td>
</tr>
<tr>
<td>SiO₂ (%)</td>
<td></td>
<td>1.73 ± 1.58</td>
<td>1.44 ± 0.86</td>
</tr>
<tr>
<td>Mean ± S.D.</td>
<td></td>
<td>0.04 - 6.35</td>
<td>0.03 - 3.10</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>0.10 - 6.35</td>
<td>0.03 - 3.07</td>
</tr>
<tr>
<td>Fixed Carbon (%)</td>
<td></td>
<td>51.9 – 79.1</td>
<td>46.2 – 59.3</td>
</tr>
<tr>
<td>Moisture (%)</td>
<td></td>
<td>3.2 – 3.9</td>
<td>4.3 – 4.6</td>
</tr>
</tbody>
</table>

* data sited from a report by the Korea Coal Corp.
Table IV. Prevalence of pneumoconiosis classified according to the duration of dust exposure

<table>
<thead>
<tr>
<th>X-ray category</th>
<th>0/0</th>
<th>0/1</th>
<th>Subtotal</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>Subtotal</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>years of dust exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0- 4</td>
<td>6,538(97.8)</td>
<td>97( 1.5)</td>
<td>6,635(99.3)</td>
<td>49( 0.7)</td>
<td></td>
<td></td>
<td>49( 0.7)</td>
<td>6,684(100.0)</td>
</tr>
<tr>
<td>5- 9</td>
<td>2,833(81.6)</td>
<td>281( 8.1)</td>
<td>3,114(89.7)</td>
<td>308( 8.9)</td>
<td>44( 1.3)</td>
<td>6( 0.2)</td>
<td>358(10.3)</td>
<td>3,472(100.0)</td>
</tr>
<tr>
<td>10-14</td>
<td>1,144(63.8)</td>
<td>196(10.9)</td>
<td>1,340(74.7)</td>
<td>322(17.9)</td>
<td>94( 5.2)</td>
<td>28( 2.1)</td>
<td>454(25.3)</td>
<td>1,794(100.0)</td>
</tr>
<tr>
<td>15-19</td>
<td>311(50.5)</td>
<td>71(11.5)</td>
<td>382(62.0)</td>
<td>152(24.7)</td>
<td>54( 8.8)</td>
<td>28( 4.5)</td>
<td>234(38.0)</td>
<td>616(100.0)</td>
</tr>
<tr>
<td>20-24</td>
<td>94(46.1)</td>
<td>23(11.3)</td>
<td>117(57.4)</td>
<td>58(28.4)</td>
<td>16( 7.8)</td>
<td>13( 6.4)</td>
<td>87(42.6)</td>
<td>204(100.0)</td>
</tr>
<tr>
<td>25-</td>
<td>17(65.4)</td>
<td>2( 7.7)</td>
<td>19(73.1)</td>
<td>5(29.2)</td>
<td>2( 7.7)</td>
<td></td>
<td>7(26.9)</td>
<td>26(100.0)</td>
</tr>
<tr>
<td>Total</td>
<td>10,937(85.5)</td>
<td>670( 5.2)</td>
<td>11,607(90.7)</td>
<td>894(7.0)</td>
<td>210(1.6)</td>
<td>85(0.7)</td>
<td>1,189(9.3)</td>
<td>12,796(100.0)</td>
</tr>
</tbody>
</table>

Percentage in parentheses
Table V. Distribution of pneumoconiosis patients classified according to the duration of dust exposure

<table>
<thead>
<tr>
<th>Year of dust exposure</th>
<th>X-ray category</th>
<th>0</th>
<th>Sub-total</th>
<th>1</th>
<th>Sub-total</th>
<th>2</th>
<th>Sub-total</th>
<th>3</th>
<th>Sub-total</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0/0</td>
<td>1/0</td>
<td>1/1</td>
<td>1/2</td>
<td></td>
<td>2/1</td>
<td>2/2</td>
<td>2/3</td>
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<td></td>
</tr>
<tr>
<td>0</td>
<td>540</td>
<td>3</td>
<td>543</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>543</td>
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<tr>
<td>1</td>
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</tr>
<tr>
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<td>21</td>
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<td>747</td>
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<tr>
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<td>563</td>
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<td>52</td>
<td>15</td>
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<td>3</td>
<td>14</td>
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</tr>
<tr>
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<td>263</td>
<td>41</td>
<td>304</td>
<td>16</td>
<td>38</td>
<td>9</td>
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<td>2</td>
<td>13</td>
<td>15</td>
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<tr>
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<td>17</td>
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<td>3</td>
<td>13</td>
<td>23</td>
</tr>
<tr>
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<td>174</td>
<td>20</td>
<td>42</td>
<td>1</td>
<td>63</td>
<td>-</td>
<td>15</td>
<td>3</td>
</tr>
<tr>
<td>14</td>
<td>130</td>
<td>27</td>
<td>157</td>
<td>16</td>
<td>44</td>
<td>6</td>
<td>66</td>
<td>3</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>15-19</td>
<td>311</td>
<td>71</td>
<td>382</td>
<td>36</td>
<td>103</td>
<td>13</td>
<td>152</td>
<td>9</td>
<td>30</td>
<td>15</td>
</tr>
<tr>
<td>20-24</td>
<td>94</td>
<td>23</td>
<td>117</td>
<td>15</td>
<td>36</td>
<td>7</td>
<td>58</td>
<td>1</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>25-</td>
<td>17</td>
<td>2</td>
<td>19</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>5</td>
<td>-</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>10,937</td>
<td>670</td>
<td>11,607</td>
<td>286</td>
<td>533</td>
<td>75</td>
<td>894</td>
<td>29</td>
<td>123</td>
<td>58</td>
</tr>
</tbody>
</table>
Table VI. Duration of dust exposure (years) required for the occurrence of pneumoconiosis

<table>
<thead>
<tr>
<th>X-ray category</th>
<th>0/1</th>
<th>1/0</th>
<th>1/1</th>
<th>1/2</th>
<th>2/1</th>
<th>2/2</th>
<th>2/3</th>
<th>3/2</th>
<th>3/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Years of dust exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>0-29</td>
<td>1-29</td>
<td>3-28</td>
<td>5-25</td>
<td>5-24</td>
<td>5-26</td>
<td>6-22</td>
<td>8-22</td>
<td>7-22</td>
</tr>
<tr>
<td>Lower 5th percentile</td>
<td>3.5</td>
<td>4.5</td>
<td>5.1</td>
<td>7.1</td>
<td>6.4</td>
<td>7.1</td>
<td>8.1</td>
<td>10.2</td>
<td>9.1</td>
</tr>
<tr>
<td>Median</td>
<td>9.2</td>
<td>10.3</td>
<td>11.4</td>
<td>11.1</td>
<td>12.5</td>
<td>13.3</td>
<td>13.3</td>
<td>15.2</td>
<td>14.2</td>
</tr>
</tbody>
</table>
Dust Concentration and Silica Content in the 20 Korean Anthracite Mines

<table>
<thead>
<tr>
<th>Mine No</th>
<th>Total dust (mg/m³)</th>
<th>Respirable dust (mg/m³)</th>
<th>SiO₂ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11.93±2.13 (9.83-14.84)</td>
<td>5.10±1.21 (3.60-6.42)</td>
<td>0.17±0.18 (0.02-0.4)</td>
</tr>
<tr>
<td>2</td>
<td>11.49±3.38 (7.69-15.28)</td>
<td>4.60±1.33 (3.44-6.23)</td>
<td>1.90±1.28 (0.7-3.2)</td>
</tr>
<tr>
<td>3</td>
<td>14.86±3.25 (11.37-18.35)</td>
<td>5.36±1.61 (4.00-7.50)</td>
<td>0.20±0.14 (0.08-0.4)</td>
</tr>
<tr>
<td>4</td>
<td>14.52±1.74 (14.14-16.77)</td>
<td>3.55±0.52 (2.90-4.15)</td>
<td>1.30±0.57 (0.8-2.1)</td>
</tr>
<tr>
<td>5</td>
<td>19.99±9.66 (9.82-29.43)</td>
<td>6.18±2.27 (3.90-8.30)</td>
<td>1.98±1.20 (0.7-3.0)</td>
</tr>
<tr>
<td>6</td>
<td>9.37±3.40 (5.46-12.93)</td>
<td>4.79±1.63 (2.92-6.50)</td>
<td>0.95±0.58 (0.4-1.1)</td>
</tr>
<tr>
<td>7</td>
<td>5.96±1.69 (4.52-8.40)</td>
<td>1.88±0.26 (1.59-2.18)</td>
<td>1.32±0.10 (0.4-2.3)</td>
</tr>
<tr>
<td>8</td>
<td>12.02±5.77 (6.28-18.85)</td>
<td>4.20±2.39 (1.95-6.55)</td>
<td>1.47±1.62 (0.03-2.9)</td>
</tr>
<tr>
<td>9</td>
<td>6.72±3.71 (3.04-11.13)</td>
<td>1.21±0.61 (0.75-2.09)</td>
<td>1.08±0.73 (0.4-1.8)</td>
</tr>
<tr>
<td>10</td>
<td>7.46±3.01 (3.80-10.46)</td>
<td>1.64±0.99 (0.70-2.70)</td>
<td>1.38±1.08 (0.1-2.5)</td>
</tr>
<tr>
<td>11</td>
<td>8.30±3.10 (3.88-10.47)</td>
<td>2.44±0.81 (1.71-3.30)</td>
<td>1.65±0.66 (1.0-2.4)</td>
</tr>
<tr>
<td>12</td>
<td>7.56±1.62 (5.57-9.20)</td>
<td>1.33±0.58 (0.75-1.90)</td>
<td>1.89±0.94 (1.1-3.1)</td>
</tr>
<tr>
<td>13</td>
<td>5.24±2.67 (2.15-8.35)</td>
<td>2.24±0.75 (1.40-2.94)</td>
<td>1.58±1.02 (0.6-2.6)</td>
</tr>
<tr>
<td>14</td>
<td>9.75±4.73 (4.70-14.60)</td>
<td>3.88±2.64 (1.40-6.60)</td>
<td>0.08±0.04 (0.04-0.1)</td>
</tr>
<tr>
<td>15</td>
<td>5.15±0.79 (4.10-6.00)</td>
<td>1.38±0.17 (1.15-1.54)</td>
<td>1.89±1.04 (0.46-2.93)</td>
</tr>
<tr>
<td>16</td>
<td>5.36±0.68 (4.64-6.02)</td>
<td>1.38±0.23 (1.06-1.60)</td>
<td>2.41±0.46 (2.08-3.07)</td>
</tr>
<tr>
<td>17</td>
<td>6.55±0.86 (5.54-7.64)</td>
<td>2.39±0.53 (1.90-3.07)</td>
<td>4.89±1.51 (3.27-6.35)</td>
</tr>
<tr>
<td>18</td>
<td>5.65±0.64 (4.97-6.25)</td>
<td>1.82±0.27 (1.55-2.17)</td>
<td>3.28±0.56 (2.76-4.08)</td>
</tr>
<tr>
<td>19</td>
<td>5.22±0.20 (4.98-5.41)</td>
<td>2.28±0.66 (1.60-3.07)</td>
<td>1.48±0.39 (0.96-1.84)</td>
</tr>
<tr>
<td>20</td>
<td>5.35±0.30 (4.97-5.68)</td>
<td>2.14±0.44 (1.70-2.70)</td>
<td>1.17±0.38 (0.74-1.65)</td>
</tr>
<tr>
<td>Total</td>
<td>9.13±4.74 (2.15-29.43)</td>
<td>3.07±1.78 (0.70-8.30)</td>
<td>1.62±1.30 (0.03-6.35)</td>
</tr>
</tbody>
</table>
THE EARLY DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS  
(Summary Paper)  
by  
Professor Kyu Sang Cho  

1. Pneumoconiosis is defined by WHO as accumulation of dust in the lungs and tissue reactions to its presence. From the pathological point of view, pneumoconiosis may be divided for the sake of convenience into collagenous and non-collagenous forms.  

Non-collagenous pneumoconiosis is caused by non-fibrogenic dust. Stanosis and barytosis caused by pure dust of tin oxide and barium sulphate are examples. Collagenous pneumoconiosis may be caused by fibrogenic dust or by altered tissue response to non-fibrogenic dust. In some cases, collagenous pneumoconiosis, such as silicosis and asbestosis, is caused by fibrogenic dust, whereas complicated coal workers' pneumoconiosis or progressive massive fibrosis is an altered tissue response to a relatively non-fibrogenic dust. Practically, however, the distinction between collagenous and non-collagenous pneumoconiosis is difficult. Continued exposure to the same dust, such as coal dust, may cause transition from a non-collagenous to a collagenous form. Furthermore, exposure to a single kind of dust is now becoming less common, and exposures to mixed dust having different degrees of fibrogenic potential may result in pneumoconiosis ranging from the non-collagenous to the collagenous forms.  

In addition, there are occupational chronic pulmonary diseases which, although they develop from the inhalation of dust, are excluded from pneumoconiosis because it is not known whether or not the particles accumulate in the lung. Byssinosis, berylliosis and farmer's lung are examples of potentially disabling occupational chronic lung diseases. These etiologic components of dust have sensitised the pulmonary or bronchial tissue so that the lung tissue responses, the inflammation tends to be granulomatous; and if the bronchial tissue responds, there is apt to be bronchial constriction.
Exposures to the inhalation of noxious materials in certain industries are associated with an increased risk of mortality from carcinoma of the respiratory tract. Examples of such materials are radioactive ores, asbestos, and chromates.

Occupational exposure to dust may be also one factor among several more important ones in the etiology of chronic bronchitis. At present, there is no sufficient evidence that chronic bronchitis may be considered an occupational lung disease of workers exposed to dust. Bronchofibroscopic study will give knowledge of this field.

2. Radiographic findings of pneumoconiosis are the most sensitive indicator of health impairment arising from exposure to silica or coal-mine dust. There have been reports of dust-induced impairment of lung function in the absence of radiological signs, but interpretation of these findings remains controversial.

Some authors have suggested that there may be a relationship between pneumoconiosis and cancer of the lung, but this view has been also challenged, and there is no consensus among the experts. Bronchofibroscopic study may support these studies in the future.

3. Health-based exposure limits have been expressed in time-weighted average concentrations. In real working conditions, the concentrations of airborne particles vary greatly in the average value. It has been suggested that the clearance capacity of the respiratory system may have a definite limit, and that when this limit is exceeded, increased dust retention cannot be prevented.

Therefore, bronchofibroscopic research is needed to identify the extent to which fluctuations in concentrations around a time-weighted average may be acceptable. This will provide the data required to determine short-term limits for airborne dust levels which are consistent with recommendations designed to restrict the long-term health effects of dusts.

4. Some countries attempt to control the development of pneumoconiosis by restricting the individual's cumulative exposure to dust (Concentration x time) by limiting the period of work under conditions in which high concentrations of dust are not controllable by technical measures. However, in view of the uncertainty discussed above about the clearance rates of dust from the lung, and the unknown relationship between dust deposition and the development of pneumoconiosis, there remains some doubt about the validity of this approach. In the absence of results demonstrating that it is safe, this strategy is regarded as questionable. Bronchofibroscopic study may be useful for clarifying this question.
5. Operational exposure limits in a number of countries have different values for quartz, tridymite, and cristobalite. Laboratory evidence indicates a different level of fibrogenicity for these crystalline forms of silica, but the problem is a virtual absence of convincing epidemiological data to support this. Published reports claiming to have found differences in the attack rate of silicosis, or its progression, in workers exposed to quartz, tridymite or cristobalite, have not provided quantitative exposure limits. It is desirable that bronchofibroscopic studies be carried out to test the hypothesis.

6. Finally, it should be studied how the presence of accompanying elements in the rocks (e.g. iron, aluminium) affects the fibrogenicity of silica, as well as how coal itself interacts with rocks and modifies the effect. Epidemiological studies have demonstrated large differences between the risk of developing coal workers' pneumoconiosis at different coal-mines with similar concentrations of airborne dust. Empirical observations suggest that such differences may also exist in mines other than coal-mines. Research is recommended to try to elucidate these apparent interactions, using both epidemiological and bronchofibroscopic methods.

7. Little is known about the differences between individuals in susceptibility to the development of pneumoconiosis in dust exposure.

It is recognized that the health problems related to exposure to mineral dust are not limited to pneumoconiosis, but also involve chronic nonspecific respiratory diseases; cases connected with bronchial obstruction need particular attention. It is recommended, therefore, that attention be given to bronchiolar observation.
STUDY ON THE EARLY DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS
(Summary Paper)

by

Dr Seung Han Lee

The legal basis for the safety and health as well as the compensation of occupational diseases and injuries was introduced for the first time in Korea with the enactment of the Labour Standard Act. However, the Act was never fully practised, as far as health matters were concerned, until the end of 1950's, when a government-owned coalmine started its own pneumoconiosis compensation scheme along the legal lines. The scheme grew into the preventive services against pneumoconiosis in later years in this mine.

Then, the programme spread gradually into the surrounding mines and was adopted by the Government as a national model when it started the nationwide Workers Compensation scheme in 1964 with the enactment of the Industrial Accident Compensation Insurance Act.

The pneumoconiosis control scheme, including compensation aspects, is rather similar between Korea and Japan, with rather wider differences in compensation aspects and narrower differences in preventive aspects. For example, organic dust pneumoconiosis is compensable in Korea as a type of pneumoconiosis, the disability benefits are given to all pneumoconiosis patients as far as they show respiratory disability and irrespective of the necessity of medical treatments, and the respiratory disability is rated much higher in pneumoconiosis than in other occupational diseases and injuries. Subcategory 0/1 is also medically compensable, if it complicates pneumoconiosis. In other words, the tendency to extend the scale and scope of compensation is far greater for pneumoconiosis than for other conditions.

Therefore, the pathogenesis and progression of all forms of pneumoconiosis are a matter of great interest both to occupational physicians and policymakers.
Generally speaking, pneumoconiosis may be regarded as an intraluminal and/or interstitial reticuloendothelial response to the accumulation of overloading inorganic dust in the lung. When the terminal elements of the lung are overloaded with respirable dusts, dusts accumulate, macrophages and fibroblasts are mobilized, reticulin and/or collagen fibers are formed, and the fibrous tissue mass with focal ischemic necrosis produce nodules. The rapidity and magnitude as well as the site of such tissue responses depend largely on the composition of dust and the dose and length of dust exposure. Although the pathologic changes of pneumoconiosis are customarily classified into three types, namely, asbestosis type, silicosis type and coal worker's pneumoconiosis type, further variations may be expected, particularly in the case of mixed dust pneumoconioses. Although radiography is valuable in the clinical diagnosis of pneumoconiosis, it may not be sensitive enough to reflect pathologic changes of an earlier stage. Tanaka bronchoalveolar fiberscopy is hoped to clarify the extent of discrepancies between radiography and pathology. The study results seem a matter of great interest not only to physicians but also to policy makers.

The most disabling stage of pneumoconiosis is usually observed in its conglomerate form and progressive massive fibrosis. These forms are a matter of particular interest, because it also shortens the life of patients. Although theories are postulated in many ways to explain the mechanism involved in the development of these forms of pneumoconiosis, they seem still not definitive. Tanaka bronchoalveolar fiberscopy may also be useful in this field of study.

In pneumoconiosis also, functional changes are likely to precede the gross morphological changes which may be observed through radiography. However, restrictive impairments are more difficult to assess than obstructive impairments by routine lung function tests. In view of the facts that many tests are applied to pneumoconiosis patients in practice, the comparative study with pathological findings obtained by the simultaneous bronchoalveolar fiberscopy may help clarify these points.

Also, simple pneumoconiosis is known to affect little the respiratory impairment and the life span of pneumoconiosis patient. If it is affected, it is usually due to emphysema complicating pneumoconiosis. Tanaka bronchoalveolar fiberscopy may help confirm diagnosis of such cases.
APPLICATION OF PULMONARY FUNCTION TEST
FOR THE EARLY DIAGNOSIS OF PNEUMOCONIOSIS
(Summary Paper)

by

Dr Kyung Chung Chee

In the Republic of Korea, anthracite has been traditionally in wide use both at home and in industry and, therefore, anthracite mining has always been the major source 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. Accordingly, the control of pneumoconiosis has always been the matter of interest both to the industry and to the general public. Lung function testing has a wide area of application and an important place in diagnosis as well as in the clinical management and disability evaluation of pneumoconiosis patients.

Coal workers' pneumoconiosis (CWP) is a condition caused by the inhalation and retention of particles of coal dust in those regions of the lung concerned with gas exchange, and the tissue's reaction to its presence. Before there can be a reaction to inhaled dust, the dust must be deposited and retained, at least for long enough to allow the lung parenchyma to react. Thus, it is only those particles that are deposited in the alveolar respiratory bronchioles that are capable of inducing pneumoconiosis. In general such particles will be between 0.5 and 5 μm, and the particles of larger size will be deposited in the nose or tracheobronchial system and hence removed by the mucociliary escalator. For these reasons the pathological changes induced by pneumoconiosis are observed almost entirely in the alveoli and respiratory bronchioles.

In the early stages, characteristic pathological lesion of CWP is the coal macula. This is characterized pathologically by the accumulation of dust around the respiratory bronchioles, a little surrounding fibrosis, and some dilatation of the respiratory bronchioles. This latter lesion is commonly referred to as focal emphysema. In approximately 10-20% of subjects with simple CWP, the disease is complicated by the development of large aggregates of fibrous tissue, usually in the upper lobe. This condition is known as complicated pneumoconiosis or progressive massive fibrosis (PMF). Simple pneumoconiosis produces relatively little ventilatory impairment, whereas PMF may lead to severe restriction of ventilatory capacity.
Simple CWP in miners from the anthracite region is characterized by the histological findings of both mainly the presence of coal maculas, which are typical of simple CWP and smaller numbers of silicotic nodules which are typical of silicosis. As the coal macula and silicotic nodule are both found in relation to the respiratory bronchiole, they could potentially compressor constrict the lumen of the bronchiole.

Since the essential pathological lesion of CWP is the coal macula with its attendant focal emphysema, the physiological abnormalities might be detected by the measurements associated with emphysema. These might include an increase in $R_l$ and alterations in lung mechanics, such as a high static 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 could demonstrate a reduced compliance and elevated pulmonary recoil pressure and coefficient. Studies in coal miners with simple bituminous or anthracite CWP have demonstrated the physiological evidence of small airway narrowing and slight changes in lung mechanics.

As the earliest changes of pneumoconiosis occur in the respiratory bronchioles, detection of functional impairment in the smallest airways of the lung is technically difficult and the standard tests of ventilatory capacity, such as estimation of airway resistance and the various derivatives of forced expiratory volume maneuver, are of limited use for this.

In healthy subjects, the peripheral airways, namely those airways from the twelfth generation down and whose lumen is less than 2 mm in diameter, are responsible for only about 10% of the total airway resistance. For this reason, determination of the total airway resistance might be unlikely to detect small airways disease. Forced vital capacity (FVC) has been the most widely accepted single test from which one-second forced expiratory volume (FEV$_{1.0}$) and expiratory flows such as maximal mid-explanatory flow (MMF) have been calculated. However, these parameters are insensitive in the detection of small airways disease because, though these are influenced by limitation of flow by dynamic compression in all the airways, the influence of flow in the larger airways predominates, and expiratory flow of the initial 25% of expired volume during the performance of the FVC maneuver appears to be effort-dependent which is influenced mainly by the subject's effort. Hence the standard methods of assessing ventilatory capacity are of little help in the detection of distal airways disease. Some of the newer techniques widely used to assess the function of the smaller airways at present are frequency dependence of compliance and measurements of closing volume, residual volume and flow-volume curve.
The characteristic abnormalities on the chest radiograph as well as a history of exposure to coal dust have an important place in the diagnosis of CWP. However, the chest radiograph is faced to some problems for the detection of abnormalities of small airways caused by coal dust. Some miners have demonstrated the physiological evidence of small airway obstruction and slight changes in lung mechanics in the absence of abnormalities on the chest radiograph. It has been assumed that the bronchofibroscopic examination and bronchoalveolar lavage might identify the early pathological lesion of pneumoconiosis and provide a valuable information about the early effects of dusts.

Bronchofibroscopic examination, bronchoalveolar lavage and lung function tests used to assess the function of the smallest airways, such as frequency dependence of compliance, flow-volume curve, closing volume and residual volume, will be carried out on pneumoconiosis patients who are difficult to detect early by the conventional chest radiograph for the purpose of early diagnosis of pneumoconiosis. The specific objectives are to detect the early pathological lesion of pneumoconiosis by bronchofibroscopic examination and bronchoalveolar lavage, to detect the functional abnormalities in the smallest airways by lung function tests, and to compare the pathological lesion with the function abnormalities.
APPLICATION OF TANAKA FIBROSCOPY IN THE EARLY DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS
(Summary Paper)

by

Dr Im Goung Yun

The first report on pneumoconiosis in the Korean coal mines appeared in 1954. The number of pneumoconiosis patients steadily increased ever since and dust work has spread from underground coal mines to the various manufacturing industries. Very recently, a case of pneumoconiosis which was contracted by a housewife after residence of eight years in the vicinity of a coal storage plant was reported and drew great public attention.

Nowadays, the number of pneumoconiosis patients amounts to 12,000 in Korea. Of those patients, 1,300 are placed under medical care in thirteen hospitals owing to various medical reasons. Catholic University Industrial Medical Center has carried out detailed medical examinations on 20,525 dust workers almost exclusively during the past twenty-two years from 1966 to 1988. The Center has also taken care of 2,351 pneumoconiosis patients medically from the very beginning of the national pneumoconiosis control programme, of which 2,154 patients were discharged successfully and the remaining 334 patients died in the course of hospitalization.

The diagnosis of pneumoconiosis usually depends on the findings of the investigation in occupational histories, of chest radiographs and physical examinations. However, we are often faced with difficulties in the detection of early changes of the lung, which was brought by dust exposure, with those old tools.

Also, the patient who shows minimal radiographic changes of 0/1 subcategory of pneumoconiosis and probably with more severe pathological changes of lung tissue is excluded from benefits under the current Workers Compensation scheme in Korea. It follows, therefore, that, even if these patients develop complications, such as pulmonary tuberculosis, pneumothorax, tuberculous pleuritis, chronic bronchitis, bronchiectasis, etc., they are unable to enjoy the various benefits prescribed in the Industrial Accident Compensation Insurance Act and the Pneumoconiosis Patient Protection Law.
The minimal abnormal findings on chest X-ray films do not necessarily mean the minimal abnormal changes in the pathological study. It is usually considered that abnormalities on radiographs appear only after a certain stage of pathological development. Naturally, many cases of pneumoconiosis patients with less severe changes of lung tissue will be overlooked under the current diagnostic procedures. It is expected, therefore, that the conventional method supplemented with fibroscopic study of small airways will enhance greatly the efficacy of detecting pneumoconiosis patients.

The cellular study of smaller airways, which may be made possible by bronchoalveolar lavage with Tanaka fibroscope, will also facilitate the diagnosis of pneumoconiosis at an earlier study.

The Tanaka bronchoalveolar fibroscopy combined with bronchoalveolar lavage study will be particularly valuable in the research and the development of diagnostic method of pneumoconiosis in the following patients: (1) apparently healthy workers with long-term dust exposure, (2) dust workers with impairment of pulmonary functions, regardless of findings on chest X-ray, and (3) pulmonary tuberculosis patients with long-term dust exposure, regardless of the presence of pneumoconiosis findings.
The topic of this presentation is limited to occupational lung diseases resulting from exposure to mineral dusts. Involvement of the small airways, and its progressive or late consequences will be especially considered.

Depending on the nature of the inhaled particles, different types of pathological reactions can be observed: schematically, concentric hyaline nodules result from inhalation of silica, diffuse interstitial fibrosis may result from inhalation of asbestos and granulomatous inflammation can be related to inhaled beryllium or talc. All the preceding particles are classified as "fibrogenic". Another class of particles, considered as "inert", typically induces only small dust deposits and collections of dust-laden macrophages in the vicinity of bronchioles and blood vessels, with no minimal surrounding fibrotic reaction.

However a clear-cut distinction between fibrogenic and inert particles is often difficult to assess, and in many occupations, workers are exposed to mixed dusts, including mainly coal, iron and quartz particles. Coal workers' pneumoconiosis is probably the most frequent disease induced by mixed dusts. One of us (M G) had the opportunity to study lung samples in a large population (> 1000) of such patients over a period of fifteen years. From this study, it appears that the severity of the lesions largely depends on the amount of silica in the mixture of dusts, and on the duration of exposure. In early lesions, dust deposits are located close to bronchioles and vessels and are often associated with mild bifrosis. In some patients, more severe lesions or more advanced stages are characterized by focally distributed interstitial fibrosis.

With or without associated fibrosis, bronchiolar changes are often present and show different patterns:

- first: bronchioles can be dilated (leading progressively to Gough's focal emphysema),

- secondly: bronchiolar stenosis can occur and be followed by obliteration and destruction of the bronchioles and fibrotic scars developing along the bronchiole-vascular axes,

- thirdly: these two types of changes can also be associated in variable proportions.

In conclusion, bronchiolar changes appear to play an important role in some occupational diseases, and they need to be better known and earlier recognized, especially to prevent additional harmful exposures.
Fibrosis is the local accumulation of fibroblasts and the extracellular connective tissue produced by these cells. It is the frequent result of inflammatory conditions. When the fibrosis disrupts normal tissue function, it can have important physiologic consequences. As a result, significant disease can result. It is now becoming clear that fibrosis results from the activity of specific biochemical mediators.

These mediators are released following injury. In the lung, it is thought that the alveolar macrophage plays a central role in the release of such mediators. These mediators can serve to recruit fibroblasts, and probably epithelial cells, to sites of injury. These cells can then become attached and oriented at the sites of injury where they can proliferate. The newly accumulated cells can then produce connective tissue matrix restoring tissue integrity. The process is completed by remodeling which restores the tissue to its original histologic architecture. Fibrosis results when the process is incomplete. That is, remodeling does not successfully occur and an accumulation of fibroblasts and the connective tissue matrix produced by these cells replaces normal tissue structures.

The alveolar macrophage is the normal resident mononuclear phagocyte of the lower respiratory tract. It possesses the capability of mediating all phases of the fibrotic process. Moreover, activation of macrophages is consistently observed in fibrotic lung disease. Considerable evidence is accumulating that these macrophage-derived mediators are responsible, at least in part, for the fibrotic process in lung diseases.

Two mediators have been studied in detail. These include fibronectin, a 440,000 dalton glycoprotein, and the alveolar macrophage-derived growth factor (AMDGF), a 30,000 dalton peptide. Macrophage-derived fibronectin is a potent chemotactic factor for fibroblasts. It can cause the migration of fibroblasts to
sites of inflammation and injury within the lung. Fibronectin is also an attachment factor for fibroblasts. It can mediate the attachment of fibroblasts by binding to specific receptors on the fibroblast cell surface and, in addition, by binding to components of the extracellular matrix. Under normal circumstances, fibronectin binds to a specific site on extracellular collagen fibers. Fibronectin is also capable of binding to a specific site on polymerized fibrin and to a specific site on the C1q component of complement. As a result, fibronectin can bind to the fibrin which polymerizes at sites of inflammation and plasma exudation. It can then serve as a nidus for fibroblast attachment and subsequent growth. Similarly, by binding to the C1q component of complement which in turn binds to immune complexes, fibronectin can serve as a nidus for fibroblast accumulation at abnormal sites in tissues where immune complexes have been deposited.

While fibroblast attachment to components of the extracellular milieu is an absolute requirement for cell growth, it is not sufficient. Fibroblasts must, in addition, be stimulated by growth factors. Optimal fibroblast growth requires the presence of two classes of growth factors termed competence factors and progression factors. The alveolar macrophage is capable of producing at least two of each of these factors. Fibronectin can function as a competence factor. Alveolar macrophage-derived growth factor (which is synonymous with the insulin-like growth factor: IGF-1A) can function as a progression factor. Together, fibronectin and AMDGF are capable of stimulating maximal fibroblast growth.

It is likely that factors other than fibronectin and the alveolar macrophage-derived growth factor can also mediate fibroblast recruitment attachment and proliferation. A large number of fibroblast chemotactic factors which may be present at sites of inflammation have been described. These include LTB4, activated complement components, interleukin 1, transforming growth factor beta, the platelet-derived growth factor (PDGF), integrated matrix components including collagen
fragments, elastin fragments and fibrin fragments. In addition, factors released by lymphocytes can recruit fibroblasts. Similarly, fibroblast growth can be stimulated by a variety of factors. The platelet-derived growth factor can function as a competence factor. Interferon gamma has been suggested, under some circumstances, to function as a progression factor. Thus, in in vivo circumstances, fibroblast accumulation may result from a variety of factors. Evidence supporting a role for fibronectin and AMDGF comes from studies of patients with interstitial lung diseases of idiopathic origin. In these conditions, macrophages produce increased amounts of both mediators in a significant number of individuals. Those individuals whose macrophages are producing both factors, when followed for two years, demonstrated considerable deterioration in lung function. Those individuals whose macrophages were producing only fibronectin or only AMDGF or neither did not show deterioration when followed for two years.

A role for these factors in dust-induced diseases is not established but is likely. In vitro studies, animal studies and human studies suggest that these factors will be released by alveolar macrophages in individuals exposed to mineral dusts.

The identification of increased release of these mediators may be helpful in identifying those patients who have had a significant exposure to dusts. It may also be a means of establishing prognosis in specific cases. Finally, the ability to block the release of these mediators with drugs offer a therapeutic strategy for the treatment of individuals exposed to dusts.

Future questions in the area of mineral dust-induced lung disease include: 1) defining the specific mediators released in response to various kinds of dust exposure; 2) the use of the analysis of macrophage activation for the prognosis of individuals who are dust-exposed; 3) the use of mediator analysis to develop and evaluate therapeutic strategies.