GALLIUM-67 EMISSION TOMOGRAPHY IN ASBESTOSIS

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ABSTRACT

Asbestosis and interstitial collagen vascular lung diseases are characterized by a combination of alveolitis and fibrosis with an early alveolitis/or bronchiolar component usually preceeding the fibrotic stage. The severity of alveolitis is not well documented by chest X-ray or pulmonary function testing except for the abnormal diffusion capacity being a relatively early manifestation. Imaging with gallium-67 is useful in delineating the inflammatory component of interstitial lung disease but mild diffuse disease may escape detection. To determine the added advantage of emission tomography (SPECT) we have studied 31 asbestosis patients with both planar and SPECT methods. Planar images of 500k counts were acquired 48 hours after injection of 6010 mCi Gallium-67 Citrate. SPECT involved 64 views of the thorax over a 360 transaxial rotation. Reconstruction provided image slices in multiple body planes. Abnormal pulmonary Gallium-67 Citrate activity was present in 20/31 planar scans (65%) and in 27/31 (87%) SPECT studies compared to our established normals. Planar abnormalities were randomly patchy while SPECT imaging showed predominant lower lobe involvement SPECT correlated well with clinical disease estimates, less so with Pulmonary Function Tests (Diffusing Capacity), and poorly with chest X-ray findings. SPECT documents more accurately and earlier the alveolitis component of asbestosis. This may be beneficial in evaluating further experimental chemoprevention treatment programs utilizing the retinoid compounds.

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PULMONARY FIBROSIS CAUSED BY SYNTHETIC TEXTILE FIBRES?

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INTRODUCTION

In 1975, Pimentel et al.[1] described respiratory disease caused by synthetic textile fibres. Since then, to the authors' knowledge, no further reports on this have been published. We have now seen three patients with pulmonary fibrosis, probably due to this cause.

PATIENTS

Case 1. A 52-year old woman was referred because of pulmonary infiltrates on her chest X-ray and a dry cough combined with increasing dyspnoea. She had previously been healthy and had never smoked. Chest X-ray showed a reticular pattern with some confluent areas in the upper lobes (Figure 1). The tuberculine test was negative, as were tests for auto-antibodies of various kinds. Routine blood tests, including erythrocyte sedimentation rate, were completely normal.

The patient started working in a textile shop fifteen years before admission. She measured and cut cloth that was mainly synthetic (acrylic fabrics, polyesters, imitation leather etc) and occasionally also glass fibre, but she was not otherwise exposed to silica or other dust. The work was very dusty.

Figure 1. (Case 1). Diffuse pulmonary fibrosis, more marked apically and at the periphery.

In the first years the patients was only slightly irritated by this dust, but she then developed an irritating dry cough which was much worse in the afternoons of working days. After 12 years she had to stop, as she found it too disturbing. She also noticed that during weekends and prolonged absence from work, she was very much better, and was not bothered by perfumes and other strong smells which on weekdays gave her coughing attacks.

Pulmonary function tests showed lung volumes within low normal values. The compliance was abnormal and the elastic recoil remarkably high (max 45 cm H_2O). The nitrogen washout curve was abnormal, with a steep alveolar plateau. A needle biopsy (Tru-Cut^R) of the right lung showed a late stage of interstitial fibrosis with considerable alveolar thickening (Figure 2). In polarized light, there were multiple small foreign bodies within the fibrotic areas.

The patient has been followed for seven years after the biopsy. She was awarded workmens compensation and retired from her work. There has been a slow progression of her fibrosis with an increasing dyspnoea, despite the fact that she is no longer in contact with textile dust.

Case 2. A 66-year old woman was seen because of dyspnoea and pulmonary infiltrates on chest X-ray (Figure 3). 40 years earlier she started working in a textile shop with very similar working conditions as case 1. Routine blood tests were normal. Lung function test showed a slight restrictive disease. Bronchoscopy revealed small hyperplastic infiltrates in many places in the bronchial tree. Biopsy showed granulation tissue, histiocytes, multinuclear giant cells, and foreign bodies which looked like small fibres. The changes were diagnosed as inflammation due to foreign body inhalation.

Case 3. A 47-year old woman presented with bilateral pulmonary fibrosis clinically and radiologically. She had a restrictive lung disease with a lung function about 75% of predicted. Like the former two patients, this patient had been working with textiles and particularly with synthetic textiles, which she had cut and measured. An open lung biopsy revealed the same type of change as in case 1 with diffuse pulmonary fibrosis, foreign body granulomas and foreign bodies.

DISCUSSION

The histories of the patients strongly suggest that the pulmonary fibrosis was caused by the exposure to synthetic fibres, and this is supported by the biopsies. Unfortunately, as the foreign bodies were in minuscule pieces, it was not possible to analyze them. Glass fibres are unlikely to reach

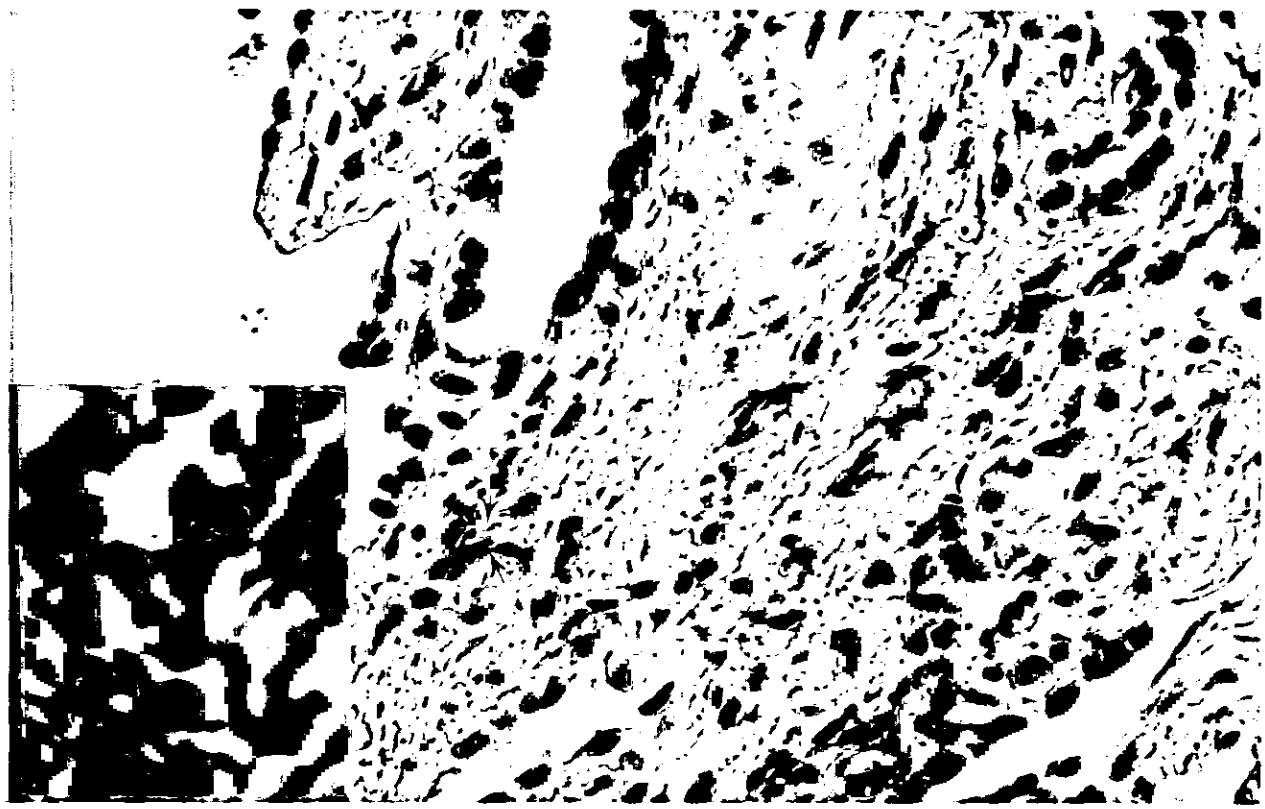

Figure 2. (Case 1). Lung biopsy. Interstitial fibrosis with marked thickening of alveolar septa. A macrophage (arrows and inserted detail) contains birefringent foreign material.

Figure 3. (Case 2). Pulmonary fibrosis.

the alveoli because of their size, and no fibrogenic effect of these fibres in humans has been described. We believe that the small foreign bodies were in fact small pieces of synthetic fibres, but so far this remains unproven.

Synthetic textile fibres consist of a number of different substances. Some of them are polyesters which are difficult to degrade biologically. The lung disease radiologically and microscopically is very similar to that described from polyvinyl chloride.[2,3] This type of agent is also very resistant to biological degradation, and it is quite possible that inhaled small particles of this type, with a great resistance against biological enzymes, in selected patients can cause inflammatory reactions of foreign body type, leading to pulmonary fibrosis.

Thus, synthetic fibres seem to be one more possible cause for pulmonary fibrosis. No doubt, there are many other exogenic agents that can also be fibrogenic when inhaled, and a thorough occupational history of any patient with "idiopathic" pulmonary fibrosis is mandatory.

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THE DISTRIBUTION OF LYMPHOCYTES AND LYMPHOCYTE PHENOTYPE SUGGESTS SILICO-PROTEINOSIS IS NOT A SYSTEMIC DISEASE

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ABSTRACT

In order to study the immunologic features of silico-proteinosis, we produced this illness in specific pathogen free Fisher 344 rats by providing an exposure of 10 mg/m^3 of free crystalline silica for 3 months (6 hrs./day, 5 days/wk.). Cell suspensions from minced spleen and collagenase-digested lung of the silica-exposed and non-exposed rats were assessed for differences (mean ± S.E.M.) in total cells and lymphocytes recovered, as well as lymphocyte subpopulations, using FTIC-conjugated monoclonal antibodies and flow cytometry.

The total cells recovered (x10^6) from the minced spleen of silica exposed (n=5) and non-exposed (n=5) rats were not significantly different (146±19 v. 173±50). Neither was there a significant difference in lymphocyte subpopulations distribution (% T helper [31±3 v. 27±2], % T non-helper [18±3 v. 16±2], T helper:non-helper ratio [1.8±0.1 v. 1.8±0.1], and % B cells [46±4 v. 53±2]) in the silica and non-exposed rats, respectively.

In the collagenase-digested lungs of silica exposed (n=5) and non-exposed (n=5) rats, total cells (x10^6) (37±8 v. 28±6), total lymphocytes (x10^6) (18±4 v. 18±2), % B cells (21±1 v. 27±3), and T helper:non-helper ratio (1.0±0.3 v. 0.65±0.1) were not significantly different. The % lymphocytes (50±2 v. 79±4), % T helper 17±1 v. 28±2), and % T non-helper (20±3 v. 44±3) in silica-exposed and non-exposed, respectively, were significantly different.

Systemic alterations in lymphocyte number and subpopulations were not apparent in the rats with silico-proteinosis. Abnormalities in the number and distribution of lymphocytes appear localized to the lung.

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COMPUTED RADIOGRAPHY UTILIZING SCANNING LASER STIMULATED LUMINESCENCE AND ITS APPLICATION FOR CLASSIFICATION OF PNEUMOCONIOSES

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BASIC CONFIGURATION OF THE CR SYSTEM

A new computed radiographic system based on new concepts and the latest computer technologies has been developed. The system eliminates the drawbacks of conventional radiography by combining digital image processing and the Imaging Plate as an X-ray sensor.[1]

Figure 1 shows a basic block diagram of the system which is called Fuji Computed Radiography (FCR). The Computed Radiography (CR) system consists of five major units:

1. X-ray image sensor which temporarily stores the X-ray energy pattern. It is called "Imaging Plate" in this system.
2. Image reader which converts the latent image on the Imaging Plate into digital time-series signals.

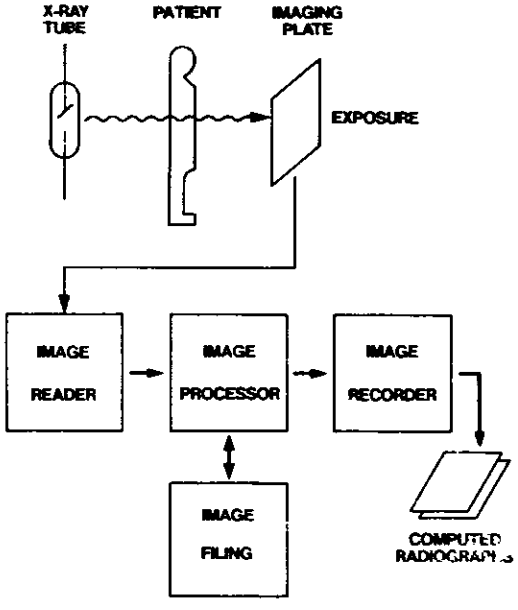


Figure 1. Basic block diagram of the CR system.

3. Image processor which manipulates the image digitally to provide radiographs the high diagnostic quality.
4. Image recorder which records the processed signals on film by scanning laser beam.
4. Image recorder which records the processed signals on film by scanning laser beam.
5. Image filing which has made it possible to store radiographic image digitally by use of reversible or irreversible data compression technique.

The basic principle of the CR system is illustrated in Figure 2. "Imaging Plate (IP)" is a flexible plate of 1mm or less thickness coated with fine photostimulable phosphor crystals using an organic binder. It can be used to obtain radiographs in exactly the same way as screen-film combination is used in conventional radiography.

After the X-ray image is stored on the Imaging Plate, it is scanned with the helium-neon laser beam to produce the photo-stimulable luminescence radiation.[2] The laser beam is deflected by a scanning mirror while the Imaging Plate is traversed to form an orthogonal scan. The luminescence radiation is collected through a light guide into photomultiplier tube and it is converted into electrical signals. This mechanism is called "Scanning-Laser-Stimulated-Luminescence." The Imaging Plate can be used repeatedly after erasing the residual energy on the plate with light.

AUTOMATIC ADJUSTMENT OF READING SENSITIVITY

The dynamic Range of IP is wide and linear over 10^4: 1 of the X-ray dose. In order to utilize this exposure range, we adopted an automatic adjustment of reading sensitivity and gain by histogram analysis. The basic idea is shown in Figure 3. IP is first scanned roughly by weak laser beam and only a minor portion of the energy stored therein is read out in order to prepare the histogram of the images stored.

The histogram is analyzed by combining it with the data concerning anatomic region and radiographic technique to compute the exposure level and exposure range of stored image, thereby pre-set PMT sensitivity and the amplifier gain at the time the stored images are read out. This method will always offer the radiographs with optimum density.

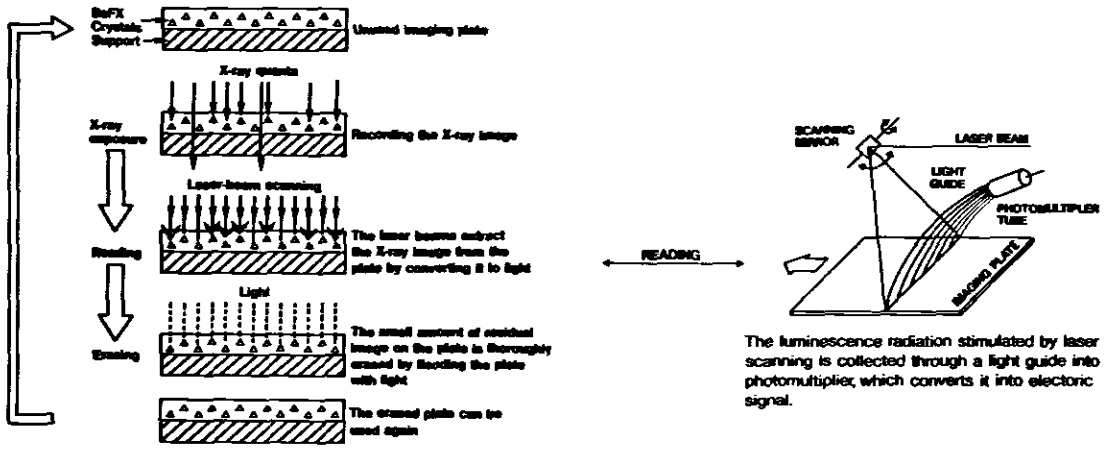


Figure 2. Principle involved in image recording, reading and erasing.

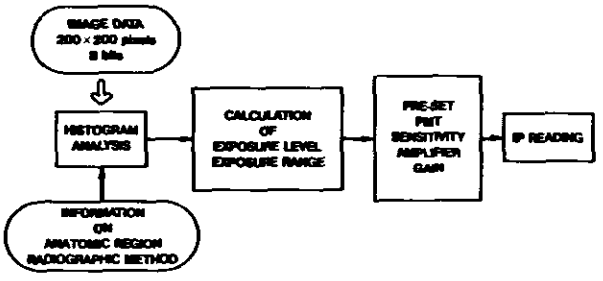


Figure 3. Basic principle of automatic IP reading.

This will be better understood by the operating characteristics curves shown in Figure 4. The first quadrant shows the characteristics of IP. The second quadrant shows the relationship between the input signals corresponding to the PSL radiation and the output digital signals of IMAGE READER. As mentioned above, the condition of reading sensitivity and dynamic range are automatically adjusted to the exposure conditions. For example, CASE A and B (quadrant I in Figure 4) represent higher exposure level and lower exposure level respectively. In this example, both cases have the same exposure range. These cases are read using different conditions (such as CONDITION A and B respectively) so that the output digital image signals are normalized and occupy the same range. The third quadrant shows the relationship between the input signals and output signals of IMAGE PROCESSOR. The digital image processing are conducted at this stage.

Finally, the fourth quadrant shows a sum of characteristics in the form of tonal reproduction curve, i.e., film-density versus X-ray exposure. In the screen-film system, only one reproduction curve is available since the sensitivity and dynamic range of the screen-film combination is fixed. In the CR system, however, arbitrary reproduction curves are possible because of the sensitivity and dynamic range adjustment mechanism of the computer. Therefore, the image reproduction range of the CR system is much wider and flexible, compared with the conventional screen-film system.

Figure 5 shows the example of dosage reduction by automatic adjustment of reading sensitivity mechanism. The exposure conditions of the radiograph (A) were 80kVp and 15mR skin dosage, which were equal to those of conventional screen film system such as Fuj Hi-STD screen and Fuji RX film combination. On the other hand, The skin dosage to obtain the radiograph (B) was 3.8mR, that is 1/4 of conventional dosage.

DIGITAL IMAGE ENHANCEMENT

In order to obtain the sufficient image contrast, the digital image processing,[3] particularly image enhancement tech-

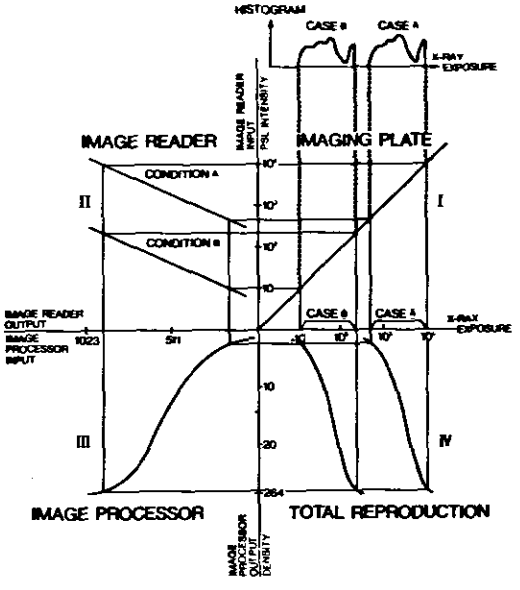
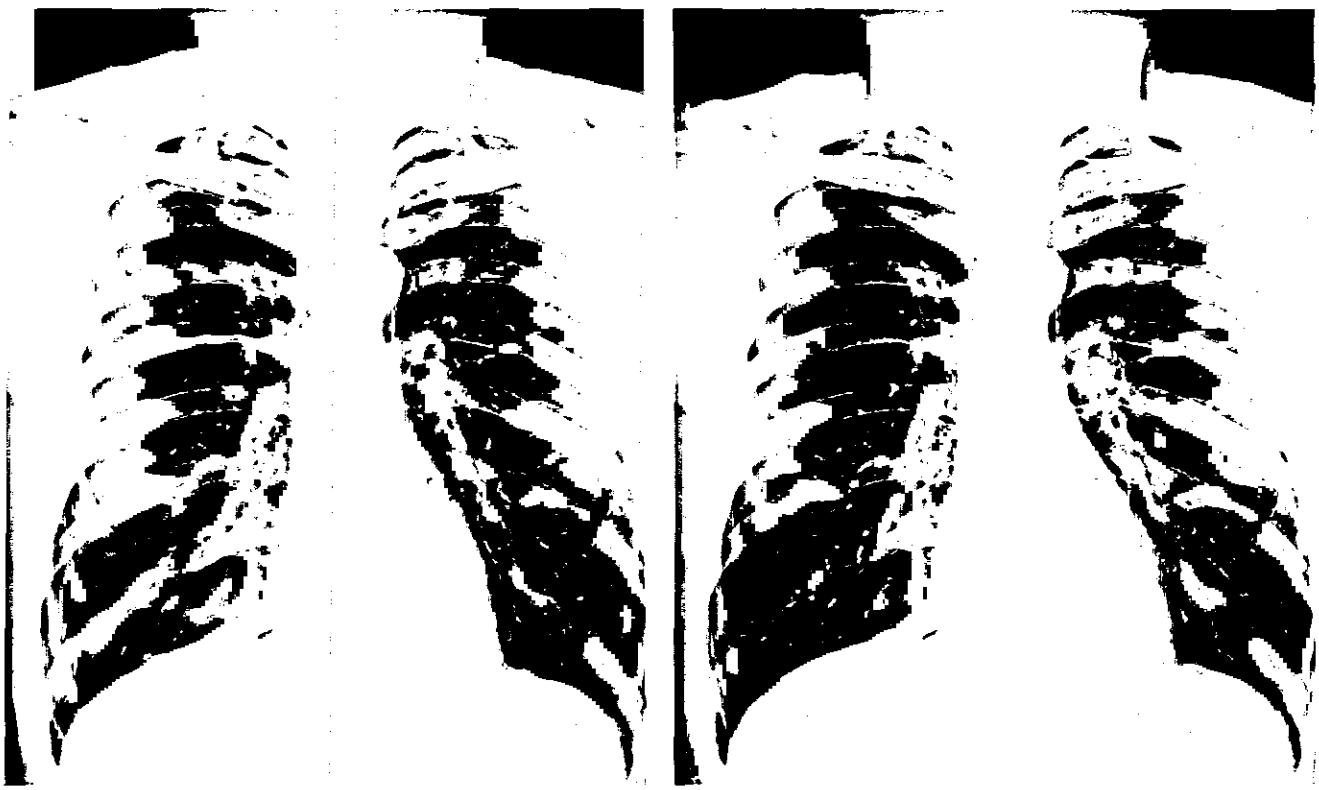


Figure 4. Operating characteristics of the CR system.



(A) (B)

Figure 5. (A) Example of standard exposure level 80kVp, 15mR).
(B) Example of lower exposure level (80kVp, 3 8mR)

niques is applied adequately. Image enhancement techniques may roughly be classified into two, that is tonal conversion and spatial frequency modification. The former freely realizes the non-linear gradation by table-looking algorithm, which is equivalent to varying the film characteristic curve in conventional radiography and may be illustrated in Figure 6.

As the latter technique, we employed the so-called "unsharp masking" technique shown in Figure 7. S_o and S_p are the original and processed image data, respectively. S_{us} is the blurred version of the original image data and K is the weighting factor for determining the magnitude of enhancement. The dashed line shows the frequency content of the difference between the original and blurred image.

By varying the degree of blurring for S_{us} and the weighting factor K, it is possible to control the frequency to be emphasized and the degree of such an emphasis.

The advantages of image enhancement techniques are demonstrated in the following radiographs. Figure 8 shows the chest radiographs of a 67-year-old man with the rounded opacities of pneumoconioses (P 1/1) the radiograph (A) was made by conventional screen-film system and the radiograph (B) was made by the CR system at the same exposure condition. Figure 9 shows the chest radiographs of

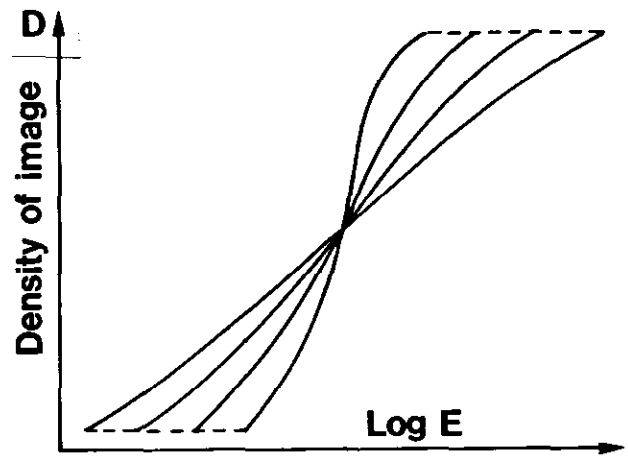


Figure 6. Tornal conversion.

a 69-year-old-man with the irregular opacities of pneumoconioses (S 2/2). Similar to Figure 8, the radiograph (A) and (B) were made by conventional screen-film system and the CR system respectively.

It may appear that both the rounded and the irregular opacities of pneumoconioses are seen much more clearly in the CR radiograph than in the conventional radiograph.

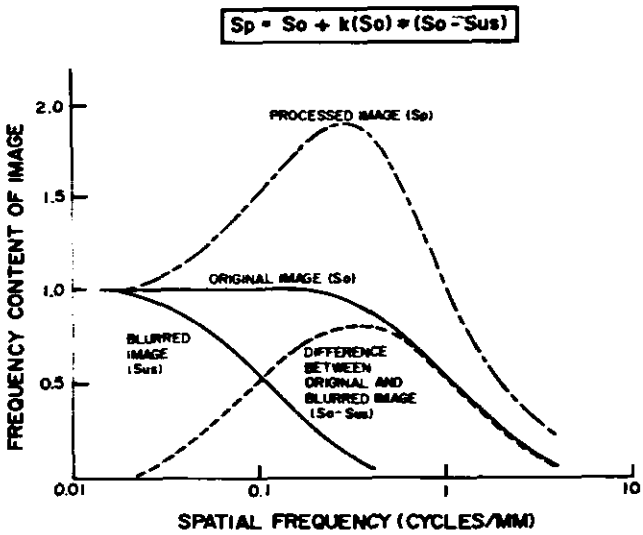


Figure 7. Spatial frequency modification.

FEATURES OF THE CR SYSTEM

The CR system is a new digital radiographic system which has possibilities in replacing the conventional screen-film system, in the field of the diagnosis of pneumoconioses, too.

The features of the system are summerized as follows:

1. Reduced dosage requirement.
2. Clear and wide latitude X-ray image suitable for accurate diagnosis.
3. Easy normalization of image quality by computer image processing.
4. Possible computer aided diagnosis such as computer classification of pneumoconioses.

These are very good features for the diagnosis of pneumoconioses.

On the other hand, computer aided diagnosis of pneumoconioses has been studied by several groups since the 1970's, and are intensively studied now with the advance of computer technologies. Some excellent results[4-6] are presented in this conference; the VIIth International Pneumoconioses Conference (1988). In the near future, the good combination of the CR system which has the good features and the new type algorithm of computer classification of pneumoconioses will be developed and utilized in hospitals.

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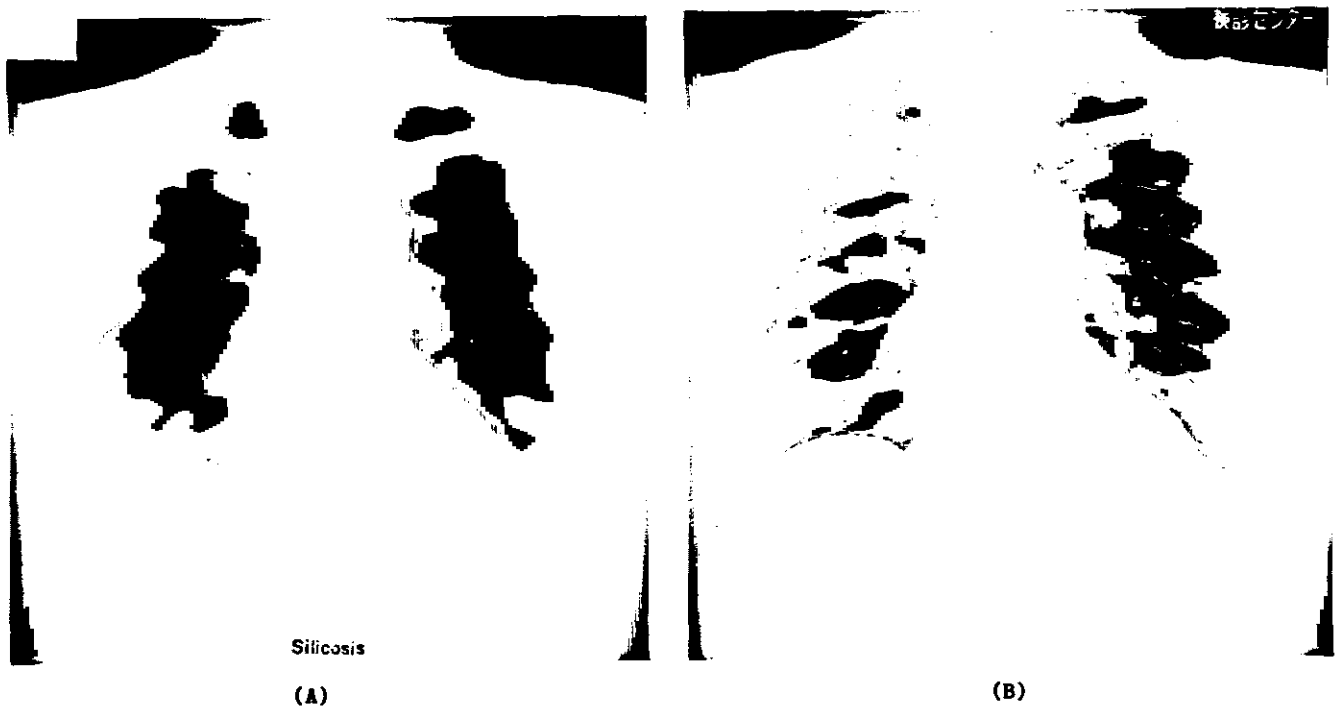

Figure 8. Chest radiographs of 67-year-old-man with the rounded opacities of pneumoconioses (1/1). (A) Example of conventional screen-film radiograph. (B) Example of computed radiograph by the Cr system.

Figure 9. Chest radiographs of 69-year-old man with the irregular opacities of pneumoconioses (S 2/2). (A) Example of conventional screen-film radiograph. (B) Example of computed radiograph by the CR system.

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ASBESTOS DISEASE IN COMMERCIAL ROOFERS: RADIOLOGIC SIGNS

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Commercial roofers are mainly employed in constructing large flat roofs on relatively large buildings. There are two major processes involved: the removal, or "tear off," of an old roof prior to partial or complete replacement and the laying down of a new roof. Removal is often conducted down to the felt or insulation barrier. Initial tear-off involves use of a scratching machine, which has metal ribbed disks to break up the initial pitch gravel layer down to the felt and/or insulation. This is followed by a "power broom machine," which sweeps the gravel-pitch debris into wind rows on the roof. The gravel pitch is then shoveled by hand into small waste carts and transported to the side of the building for discarding via an enclosed chute to ground level. While loading the gravel pitch, the roof is scraped with shovels and other hand tools to assure that the surface is prepared for subsequent installation of a four-ply felt layer plus a top pitch-gravel layer. Removal of an entire roof entails using power claws and roof cutters to remove all layers, including insulation layers which may contain asbestos.

MATERIALS AND METHODS

Local 3 of the Roofers International Union represents 453 active and retired roofers. It represents approximately 30 percent of commercial roofers in the Boston metropolitan area. In 1987, the union sponsored a medical group survey program for members. The program provided a physical examination, chest X-rays, spirometry and detailed occupational and medical histories. The program was offered on a voluntary basis to any active or retired member with 10 or more years of membership in the union. Data was collected in June 1987. Chest X-rays of all 69 participants were read by a radiologist with extensive experience with pneumoconioses (R.G.). The radiologist had no knowledge of individuals' exposure history, other than their employment as a roofer. Chest X-ray interpretation was reported according to the ILO/UC 1980 Classification of Radiographs of Pneumoconioses.[1] For the purposes of this study, a profusion grade of 1/0 or higher on the ILO scale was considered evidence of interstitial parenchymal fibrosis. Notations of pleural thickening, diaphragmatic pleural plaques, pleural calcification of the diaphragm or chest wall were all considered evidence of pleural disease.

The recorded occupational history would not allow characterization of exposure to asbestos; therefore, we used length of union membership as a surrogate measure. Union membership records were available for all 69 participants.

RESULTS

The age distribution of the examined population was different from the membership as a whole (Table I). Two hundred and seventy-one, or 60% of the total membership, were members of the roofers for 10 or more years. Of these 271, 25% were examined in the survey. The members examined comprised more subjects in the 45–64 year age grouping, and less in the 25–34 year age group, than the union as a whole. The mean age of the examined population was 52 years, and mean number of years as a roofer was 29. The vast majority had more than ten years in the trade, with 74 percent having more than 20 years at the time of examination (Table II). Forty-six individuals, 67 percent of the population, had radiologic evidence of pleural disease (Table III). The mean age of this group was 54 years and mean years of membership 29 years. There was a significant relationship between years as a roofer and prevalence of pleural disease. Eighty percent of roofers with 20 or more years in the trade had evidence of pleural abnormalities, ($x^2 = 4.7$, $p < .05$).

In contrast, parenchymal abnormalities were uncommon, with only two (3 percent) individuals having radiologic evidence of fibrosis (Table IV). Both individuals with parenchymal fibrosis had more than 20 years membership in the union. There was no relationship between smoking status and the presence of pleural abnormalities (Table V).

DISCUSSION

This cross-sectional survey found that 67 percent of roofers who participated had radiologic abnormalities characteristic of asbestos-related pleural disease. The prevalence of pleural abnormalities increased with years of union membership, i.e., duration of exposure and time since first exposure. These data support the conclusion that roofers, like other members of the construction trades, have sustained significant occupational exposure to asbestos and are therefore at risk for developing asbestos-related disease.

Eighty percent of roofers with 20 or more years of union membership had evidence of pleural disease, with two (2.5 percent) having evidence of parenchymal fibrosis (asbestosis).

The rate of pleural abnormalities in this population of roofers is substantially higher than those found in unexposed populations.[2] However, cross-sectional studies of other construc-

Table I

Age Distribution of Union Membership and Roofers Examined

Age (Years)	Percentage of Union Union Membership (N = 288)	Percentage of Population Examined (N = 69)
0-24	3	0
25-34	23	4
35-44	17	16
45-64	42	71
65+	15	9
TOTAL	100	100

Table II

Population Examined by Years of Union Membership

Years of Membership	N	Percentage
≤10	1	1
11-20	17	25
21-30	20	29
30+	31	45
	69	100

Table III

Prevalence of Pleural Abnormalities

Years as Roofer	Number Examined	N	Percentage
≤10	1	1	-
11-20	17	8	47
20+	51	37	80
TOTAL	69	46	67

$X^2 = 4.7$, $p < .05$ for prevalence < 20 years vs. > 20 years.

Table IV
Prevalence of Interstitial Opacities

Years as Roofer	Number Examined	N	Percentage
≤10	1	0	0
11-20	17	0	0
20+	51	2	4
TOTAL	69	2	3

Table V
Pleural Abnormalities and Smoking Status

	Number Examined	N	Percentage
Current	30	16	53
Ex-	30	23	77
Non-	9	7	78
TOTAL	69	46	67

$X^2 = 3.25$, $p > 0.05$

tion trades workers have revealed high prevalences of asbestos-related abnormalities, particularly pleural abnormalities. Baker et al. found pleural abnormalities in 70 percent of sheet metal workers who had 30 or more years of employment.[3] Sprince et al. found pleural thickening in 31 percent of a group of actual and retired pipefitters with more than 20 years since first exposure who attended a voluntary medical screening.[4] Michaels et al. reported that sheet metal workers with 20 or more years of union membership had a prevalence of 29 percent of X-ray abnormalities characteristic of pleural and/or parenchymal disease due to asbestos.[5] The differences in reported prevalences among construction workers in these studies is likely to result from differences in exposure among populations studied and differences in X-ray interpretation. Nevertheless, it is clear that asbestos-related abnormalities, predominantly pleural diseases, are prevalent in these populations.

This study is limited in two respects. First, relatively small numbers (i.e. 25% of eligible workers) limits accurate estimation of prevalence. Second, the program was voluntary and it is therefore possible that disease status or exposure influenced participation.

The age distribution of participants differed from the union as a whole. However, reported prevalences of pleural and parenchymal abnormalities actually may be underestimates in the group with more than 10 years of exposure, since many non-participants were of retirement age in this group.

Benign pleural abnormalities resulting from exposure to asbestos include parietal pleural plaques along the chest wall and diaphragm, pleural calcification of plaques, diffuse visceral pleural thickening and rounded atelectasis. In addition, pleural effusions in the absence of malignancy have been described in asbestos-exposed workers, usually early in their careers.[6] All of these conditions (as well as the malignant manifestations of asbestos exposure) may be seen in roofers. Our data suggest that pleural abnormalities are extremely common in roofers with more than 20 years of work.

Our findings emphasize the need for medical surveillance of all asbestos-exposed construction workers. Our findings should also alert clinical practitioners to the type of radiologic abnormalities commonly seen among commercial roofers. Furthermore, while the OSHA standard mandates medical surveillance for currently exposed workers,[7] our data suggest that workers with past exposure and retirees be included in such programs. In addition, since the potential for on-going asbestos exposure exists during old roof removal, appropriate control of exposure needs to be rigorously implemented.

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POSSIBLE OCCUPATIONAL PULMONARY ALVEOLAR PROTEINOSIS (PAP)

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ABSTRACT

This is a case of a 39 year old man with pulmonary alveolar proteinosis (PAP) which appeared at least temporarily, related to work place exposures. In July of 1985 the patient began to work for a company which manufactures mobile homes. Although his job was restricted to mechanical tasks such as installing doors and windows, he was exposed to glue and laminate vapors. There was a fiberglass shop adjacent to his work station. After working there for approximately six weeks he developed acute chest pain and shortness of breath initially diagnosed as pneumonia. Subsequent workup and bronchoalveolar lavage confirmed the diagnosis of PAP. He underwent additional therapeutic lavages, and has been feeling well since.

We were able to locate chest X-rays from 1983 which demonstrate interstitial markings. It is also true that the patient's symptoms persisted past the sessation of occupational exposures until the lavage was performed. On the other hand there is literature which suggests that hydrocarbons and other chemicals are associated with 50% or more of PAP cases.

The poster presentation will include a short personal and occupational history. Copies of serial chest X-rays and slides of lavage cytology will also be shown. Our patient was treated unsuccessfully by a number of physicians. It is important to maintain a high index of suspicion among occupational physicians who are caring for workers exposed to hazardous airborne substances despite their relative rarity of this condition.

No Paper provided.

SUBPLEURAL CURVILINEAR SHADOW IN INTERSTITIAL PULMONARY DISEASES

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Chest radiographic findings in pulmonary asbestosis have been studied,[1,2] and pulmonary asbestosis is mainly diagnosed by irregular opacities on chest radiographs.[3] However, computed tomographic (CT) findings have not been studied to the same extent as radiographic findings. In our previous study,[4] we revealed a line parallel to the inner chest wall in the lung on high-resolution CT alone, and we called it subpleural curvilinear shadow (SCLS, Figure 1).

In this study, to analyze the significance of the SCLS in pulmonary asbestosis, we studied the prevalence rate of the SCLS in several interstitial pulmonary diseases and in asbestos workers with normal chest roentgenograms.

MATERIALS (Table I)

Twenty-two patients with pulmonary asbestosis (ASBESTOSIS) were examined. They were 21 men and a woman ranged in age from 40 to 64 years (mean; 53 years) and ranged in duration from the first exposure to asbestos from 15 to 43 years (mean; 30 years). By ILO classification, 12 of ASBESTOSIS had category 1 (1/0, 1/1, 1/2), 7 had category 2 (2/1, 2/2, 2/3), and 3 had category 3 (3/2, 3/3, 3/+) disease. For the controls, 33 patients with idiopathic pulmonary fibrosis (IPF), 23 patients with interstitial pneumonia due to collagen vascular diseases (CVD), and 102 patients with lung cancer admitted to our hospital during the last one year (LC) were examined. The IPF group consisted of 29 men and 4 women ranged in age from 49 to 78 years (mean; 66 years). The CVD group included 11 patients with rheumatoid arthritis (7 men and 4 women), 6 with progressive systemic sclerosis (a man and 5 women), 3 women with polymyositis or dermatomyositis, 2 women with mixed connective tissue disease, and a woman with systemic lupus erythmatosus. They were ranged in age from

Table I
Characteristics of the Materials

	n (male)	age (range) [yrs]	exposure (range) [yrs]
ASBESTOSIS	22 (21)	53 (40-64)	30 (15-43)
IPF	33 (29)	66 (49-78)	-
CVD	23 (8)	55 (35-83)	-
L C	102 (83)	63 (32-82)	-
WORKERS	22 (18)	49 (35-61)	27 (14-43)

Note-- ASBESTOSIS; patients with pulmonary asbestosis
IPF; patients with idiopathic pulmonary fibrosis
CVD; patients with interstitial pneumonia due to collagen vascular diseases
LC; patients with lung cancer
WORKERS; current asbestos workers with normal chest roentgenograms
exposure; duration from the first exposure to asbestos dust

Figure 1. Subpleural curvilinear shadow (SCLS).

35 to 83 years (mean; 55 years). The LC group consisted of 83 men and 19 women ranged in age from 32 to 82 years (mean; 63 years).

Twenty-two current asbestos workers (WORKERS) with normal chest roentgenograms (ILO category 0) were also examined. They were 18 men and 4 women ranged in age from 35 to 61 years (mean; 49 years) and ranged in duration of exposure to asbestos from 14 to 43 years (mean; 27 years).

METHODS

A GE CT/T 8800 whole-body scanner (General Electric Medical Systems, Milwaukee, Wis.) was used for this study. High-resolution CT scanning (target reconstruction of the bone detail) was performed in all cases. The scan time was 9.6 seconds and section thicknesses were 1.5 mm or 2 mm. The window level was set at −550 and the window width at 2000 Hounsfield units. In determining the presence of SCLS, an SCLS shorter than 1 cm in length was excluded.

In the first study, the prevalence rates of SCLS were compared among several interstitial pulmonary diseases (ASBESTOSIS, IPF and CVD) and non-interstitial pulmonary disease (LC). In LC, the lung regions without tumor were examined.

In the second study, following the evaluation of the SCLS prevalence rate in WORKERS, sex, age, duration of asbestos

exposure, and smoking habit were compared between WORKERS with and without SCLS.

In the third study, CT findings including SCLS and honey comb shadow (HC) were compared with ILO classification in both ASBESTOSIS and WORKERS. CT findings were classified to five types (Figure 2) according to our previous study;[4]; type 0 has no SCLS and HC, type I has SCLS without other pulmonary abnormality or with abnormality located only peripherally, type II has HC located in the subpleural zone, type III has HC spreading intermediate between types II and IV, and type IV has HC spreading to the hilar region.

RESULTS

In the first study (Table II), we observed SCLS in 17 (77%) of 22 cases with pulmonary asbestosis. SCLS was also detected 7 (21%) of 33 patients with IPF and 9 (39%) of 23 patients with CVD. But in LC, SCLS was not detected. The SCLS prevalence rate in ASBESTOSIS was statistically higher than in IPP and in CVD ($p < 0.001$).

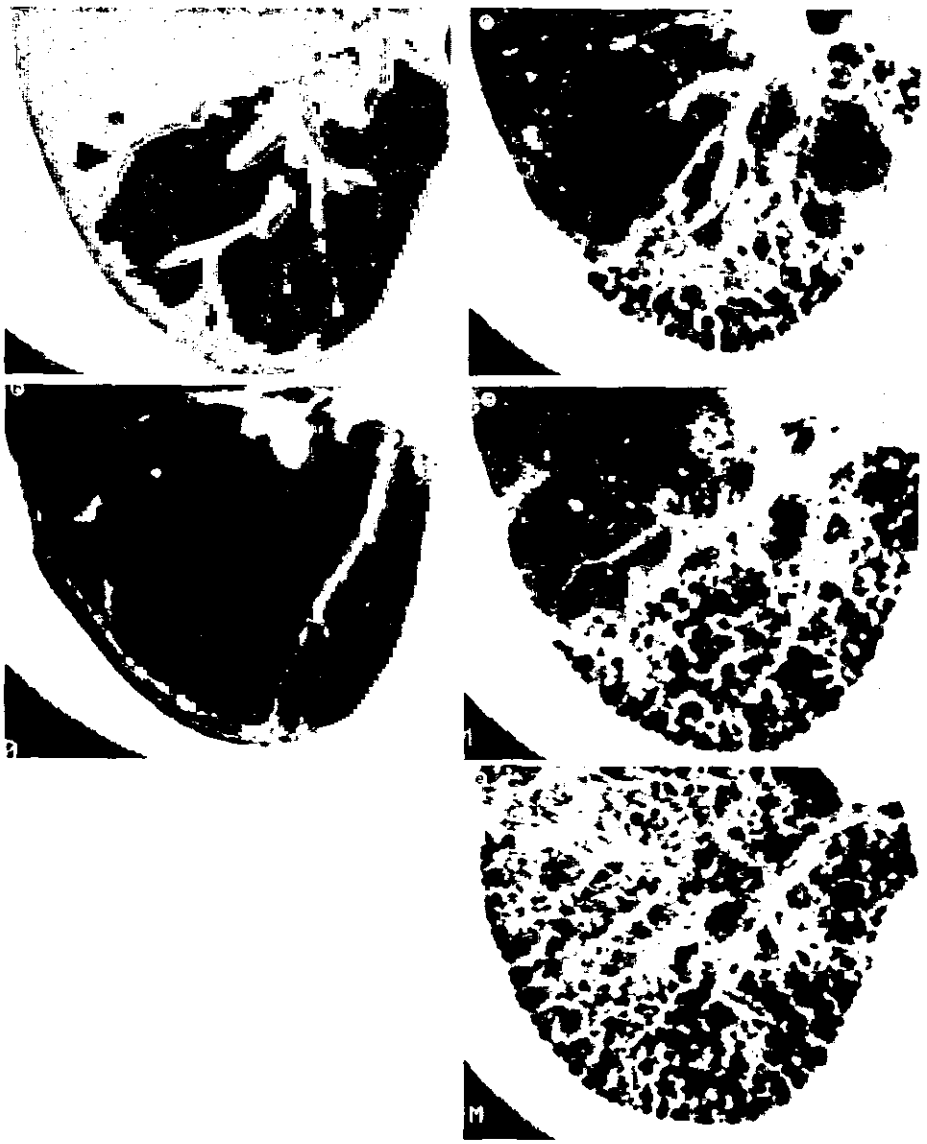

Figure 2. CT classification.
- a: type 0; no SCLS and no honey comb shadow (HC)
- b: type I; SCLS without other pulmonary abnormality or with abnormality located only peripherally
- c: type II; HC located in the subpleural zone
- d: type III; HC spreading intermediate between types II and IV
- e: type IV; HC spreading to the hilar region

In the second study (Table III), we observed SCLS in 7 (33%) of 22 cases in spite of their normal chest roentgenograms. And there were statistically no differences between WORKERS with and without SCLS in sex, age, duration of asbestos exposure, and smoking habit.

In the last study (Table IV), the relationship between ILO and CT classification in both ASBESTOSIS and WORKERS were evaluated. Of the 3 patients with ILO classification category 3 and CT classification type IV, 2 patients without SCLS had wide spreading HC from their lower lobes to their apices. Excluding these 2 severe cases, the SCLS prevalence rates were increasing in proportion to ILO classification.

DISCUSSION

In our previous study,[4] radiologic-pathologic correlation was described in one postmortem lung sample. The patient was a 55-year-old man with pulmonary asbestosis (ILO category 1). He had the SCLS in the right lower lobe. He died of respiratory failure 3 months later, and an autopsy was performed. It was made clear that the structure of SCLS was consisted of peribronchiolar fibrotic thickening with anthracosis joined by flattening and collapse of the alveoli with fibrosis. In other words, SCLS had a histologic pattern of fibrosing bronchiolo-alveolitis. This histologic finding is characteristic of pulmonary asbestosis, especially fibrosing bronchiolitis is known to be initial change of pulmonary asbestosis.

Following our previous study, several studies of high-resolution CT findings about pulmonary asbestosis were reported. Friedman et al. made use of SCLS for one of the criteria to diagnose pulmonary asbestosis.[7] Aberle et al. also reported SCLS (they called it "curvilinear subpleural line") was observed in 28% of 29 patients with pulmonary asbestosis.[5]

In the first study, we were able to observe SCLS not only in pulmonary asbestosis (ASBESTOSIS) but also in idiopathic pulmonary fibrosis (IPF) and interstitial pneumonia due to collagen vascular diseases (CVD). But no SCLS were detected in lung cancer (LC). From this results, the SCLS on high-resolution CT seemed one of the findings of interstitial pulmonary diseases. However, the prevalence rate of SCLS was markedly higher in ASBESTOSIS than in IPF and in CVD, and so the SCLS was supposed to be a more characteristic finding in ASBESTOSIS.

In the second study, we observed similar SCLS in one-third of current asbestos workers (WORKERS). They had been exposed to asbestos dust during 27 years in average but they had no findings of pulmonary asbestosis on their chest roentgenograms. And in the next study the backgrounds were compared between WORKERS with and without SCLS. But there were no differences between them. The cause of appearance of SCLS seemed not only the length of duration of asbestos exposure or smoking habit, but also individual constitution of each person and so on.

In the last study including ASBESTOSIS and WORKERS, the SCLS prevalence rates were increasing in proportion to ILO classification. These results supported the hypotheses that the SCLS was one of the common findings in pulmonary asbestosis and that the SCLS shown in asbestos workers with normal chest roentgenograms was an early change of pulmonary asbestosis. In our histologic survey,[4] the structure of SCLS was also compatible to the initial change of pulmonary asbestosis.

From the results mentioned above, we concluded that SCLS on high-resolution CT scan was useful for early detection of pulmonary asbestosis in asbestos workers.

Table II

Prevalence Rate of SCLS in Several Interstitial Pulmonary Diseases

	n	SCLS (%)
ASBESTOSIS	22	17 (77)
IPF	33	7 (21)*
CVD	23	9 (39)*
L C	102	0 (0)

Note-- * p<0.001 against ASBESTOSIS

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Table III

Comparison of Background between Asbestos Workers with and without SCLS

	n	male (%)	age	exposure	smoker(%)
SCLS(-)	15	12 (80)	49 ±5	27 ±7	9 (60)
SCLS(+)	7	6 (86)	49 ±8	28 ±9	6 (86)

Note-- smoker; both current smokers and exsmokers

Table IV

Relationship between ILO and CT Classification in Patients with Pulmonary Asbestosis and Asbestos Workers

CT classification	ILO classification 0	1	2	3	total
type 0	14 (0)	1 (0)			15 (0)
I	7 (7)	6 (6)			13 (13)
II	1 (0)	4 (2)	4 (4)		9 (6)
III		1 (1)	3 (3)		4 (4)
IV				3 (1)	3 (1)
total	22 (7)	12 (9)	7 (7)	3 (1)	44 (24)

Note-- Numbers in parentheses indicate patients with SCLS

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DO SILICON-OXYGEN RADICALS PLAY A ROLE IN THE QUARTZ-INDUCED HEMOLYSIS AND FIBROGENICITY?

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INTRODUCTION

In an earlier communication[1] from our laboratory it was reported that mechanical crushing of coal and quartz under normal air atmosphere generates of free radicals on the particle surfaces, and that these radicals decay with time, hence pointing to a higher toxicity of fresh dusts in relationship to pneumoconiosis and silicosis. More recently Fubini et al.[2] have also reported the detection by electron spin resonance (ESR) of the formation of SiO_3^- and $Si_2O_5^-$-type of radicals from quartz particles crushed under atmospheric conditions. In agreement with earlier ESR studies on single crystals of quartz crushed under high vacuum ($\sim 10^{-10}$ torr)[3] and subsequent exposure to air,[3] and to other gases,[4] these radicals were identified[2] as being formed by the homolytic cleavage of the Si-O-Si bonds and the reactions of the $Si^•$ and $SiO^•$ radical with atmosphere. Fubini et al.[2] also suggested that these radicals might be involved in the mechanism of the fibrotic action by silica, either by transforming the particle surface into a selective oxidating agent or as an initiator of a sequence of reactions leading to fibrosis. Earlier Gabor and Anca[5] had reported that lipid peroxidation caused by free radicals on the silica surface might be involved in the red blood cell membrane damage. Thus far, however, no parallel cytotoxicity, fibrogenicity, and free radical studies on a given quartz dust sample have been reported, except for some earlier work from our laboratory.[1,6,7] We now present more recent results obtained from parallel cytotoxicity, fibrogenicity, and free radical measurements on a freshly made quartz dust. The dust's free radical content was measured using ESR spectroscopy while its cytotoxicity potential was estimated via hemolysis. Hemolysis was employed as the toxicity test because it is a widely used method for estimating the potential of a dust for disrupting the cell membrane.[8] The fibrotic potential was followed by measuring the dust-induced lipid peroxidation, using linoleic acid as a model lipid. As discussed below, the results obtained suggest new clues to the mechanism of the quartz-related cytotoxicity and fibrogenicity.

MATERIALS AND METHODS

Reagents

Crystalline quartz particles with a size range of 0.2–2.5 mm were obtained from the Generic Respirable Dust Technology Center, Pennsylvania State University, University Park, Pennsylvania. These particles were crushed in air to obtain quartz dust samples with particle sizes smaller than 20 microns. We chose to work with a dust with mixed particle sizes, rather than a specific range, as an effort to simulate the mining atmosphere. An agate mortar-pestle arrangement was used for the crushing and grinding because of the close similarity of the structure of agate to that of quartz. Diethylenetriaminepentaacetic acid (DETAPAC) were purchased from Sigma. All other chemicals were purchased from Fisher or Aldrich.

Hemolysis Experiments

Hemolytic activity of silica was measured, following an establishsd procedure,[9] as the amount of hemoglobin released from a 2% suspension of sheep erythrocytes after incubation with 10 mg of silica dust for one hour at 37°C. The hemoglobin release was estimatsd via the absorbance at 540 nm using a Gioford spectrophotometer. The procedure was calibrated by substituting the silica dust by a phosphate buffer solution as a negative control (background) and 0.5% Triton-X-100 as a positive control (100% hemolysis). The percentage of hemolysis was calculated as follows:

$$\% \text{ Hemolysis} = (I_{silica} - I_{neg}) / (I_{pos} - I_{neg})$$

where I_{silica} is ths absorbance after incubation with the silica dust, while I_{neg} and I_{pos} are those with buffer only and 0.5% Triton-X-100, respectively.

Lipid Peroxidation Measurements

Peroxidation of the polyunsaturated lipid linoleic acid (cis-9-cis-12-octadecadienoic acid) by freshly ground or aged silica was monitored using a fluorescence method[10] with minor modifications. The reaction mixture in a total volume of 0.5 ml contained freshly ground or aged silica and 20 µl of 0.52 mM linoleic acid emulsion in 95% ethanol in HEPES buffer (pH 7.4) with calcium and glucose. The mixture was heated for one hour in a shaking water bath at 37°C. This procedure was followed by the addition and mixing of 0.5 ml of 3% sodium dodecyl sulfate and then of 2.0 ml 0.1 N HCl, 0.3 ml 10% phosphotungstic acid and 1.0 ml 0.7% 2-thiobarbituric acid. The mixture was then heated for 30 min at 95–100°C and the reactive substance formed was extracted with 5 ml 1-butanol after cooling. The extraction was then centrifuged at 3000 rpm for one minute and the fluorescence of the butanol layer was measured using a

515 nm excitation and 555 nm emission, with a Perkin-Elmer fluorospectrophotometer (Model MPG-36). Malondialdehyde standards were prepared from 1,1,3,3,-tetramethoxypropane to obtain a calibration curve, which was used for calculating the amounts of malondialdehyde produced.

ESR Measurements

ESR spectroscopy was used for identifying the crushing-induced silicon-oxygen radicals, and to follow their concentration as described elsewhere.[1,6,7] The ESR measurements were made with a Bruker ER 200D spectrometer operating at X-band (~9.5 GHz) frequencies, and 100 kHz magnetic field modulation. The magnetic field was calibrated with a self-tracking NMR gaussmeter (Bruker, model ERO35M). The microwave frequency was measured with a Hewlett-Packard, Model 5340A, digital frequency counter. All ESR measurements were made at room temperature.

RESULTS AND DISCUSSION

Figure 1 shows a typical, room temperature, ESR spectrum of freshly ground quartz particles. The spectrum is not identical but similar to those reported earlier for the measurements made at room temperature and ambient air environment.[1,2] Here we focused on the major species, characterized by g = 2.0015, and assigned to a combination of silicon-oxygen radicals.[1,2] To correlate the radical content with hemolysis, it was necsssary to control the radical concentration. The first method used for this was thermal annealing. Thus the free radical concentration was measured via ESR (at room temperature) after thermal annealing from 50° to 800°C for 30 minutes at each temperature. Figure 2 shows the change in the radical concentration on thermal treatment (Plot A) and the corresponding hemolysis msasurements (Plot B). The data for the samples heated above 300°C show that while the free radical content decreases sharply with the heat treatment above 300°C, the hemolytic potential remains virtually unchanged for heating up to 550°C, and starts to decrease on further heating only. It, thus, follows that there is little, if any, direct correlation between the concentration of the free radicals and the hemolytic potential of the dust samples.

Second, measurements of both the radical concentration and the hemolytic potential were made at several time intervals after the dust preparation. Figure 3 shows the time dependence of the free radical concentration on storing the dust in air after grinding (Plot A) and the hemolysis induced by the same sample (Plot B). It is seen that while the radical concentration decreases with a half-life of about one and a half day, in agreement with our earlier studies,[1,5,7] the hemolytic potential does not change noticeably over at least two weeks, again showing that the grinding-induced radicals on the quartz particles do not play any direct role in the mechanism of ths hemolysis by quartz particles.

As the third method for controlling the radical concentration, some freshly ground quartz particles were boiled in a phosphate buffer for about 30 minutes. ESR measurements on thess samples showed that their radical concentration decreased to about 10%, while their hemolytic activity decreased to almost zero. In order to find if this decrease was related to the silicon-oxygen radicals, experiments were

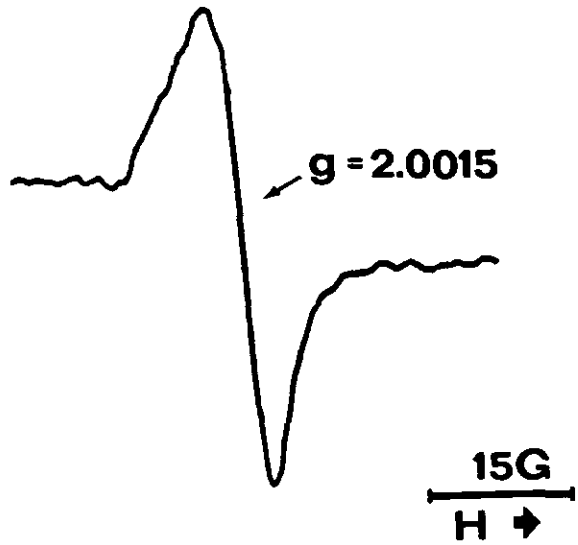

Figure 1. A typical, room temperature, ESR spectrum of freshly ground quartz particles.

conducted as an attempt to restore the hemolytic activity. It was found that while an exposure to a phosphate buffer or a $KMnO_4$ solution (a strong oxidant) did not restore the hemolytic activity, the addition of DETAPAC, a strong metal chelate, to the incubation medium restored the activity to about 60%. Thus, ths boiling-induced reduction in the silica's hemolytic potential cannot be attributed to the loss of the radicals on boiling, since it is unlikely that the addition of DETAPAC could restore the silicon-oxygen radicals. These results seem to indicate that the attachment of metal ions to the particle surfaces causes the loss in the hemolytic activity by quenching certain reactive (surface) sites. This conclusion is not unprecedented since earlier hemolysis studies[8] have shown that the presence of metal ions such as Al^{3+} causes a significant decrease in the quartz dust's hemolytic potential. We indeed confirmed that addition of Al^{3+}, Cu^{2+}, or Fe^{2+} ions, at about 1.0 mM concentration, to the incubation medium results in the loss of the hemolytic activity. The new result obtained here is that the subsequent addition of DETAPAC restores it, implying that the metal ions were only loosely bonded to the silica surface.

The above results are consistent with an earlier suggestion[8] that surface silanol (SiOH) groups play a key role in the mechanism of hemolysis by quartz particles. Metal ions are expected to be bonded via the surface silanol (SiOH) groups by replacing the H^+ ions, thus reducing the number of silanol groups responsible for red blood cell membrane damage.[8] Infrared studies on heated silica-gel[11] and silica surfaces[12] demonstrated that silanol groups are formed on the silica surface, and that these moieties are annealed only if silica is heated to higher than 700°C.[11,13]. Since the present work shows that the hemolytic activity of silica decreases markedly on heating to 700°C (Figure 2), the role of the silanol groups in the hemolysis by silica seems fairly well establishsd. This finding is consistent with an earlier

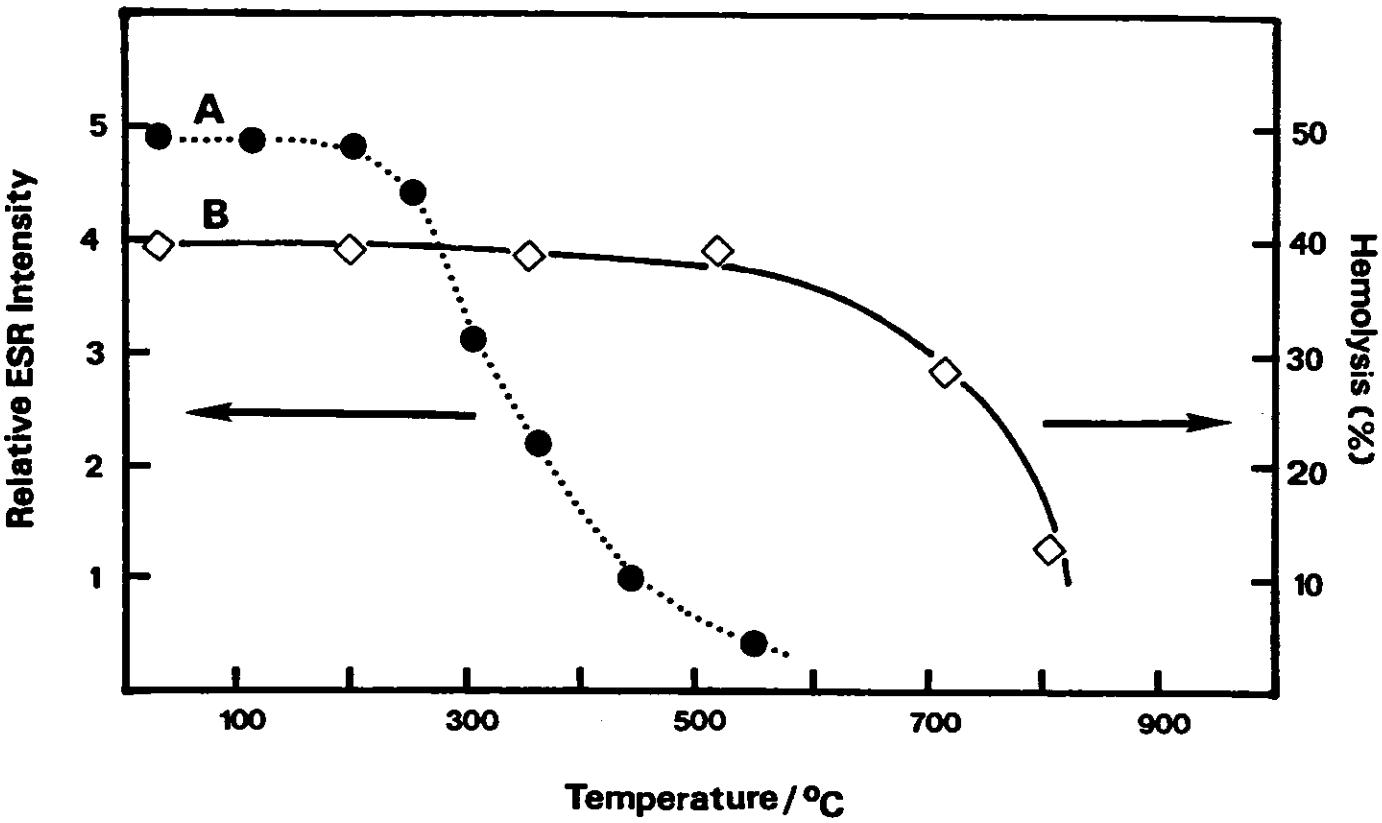

Figure 2. The effect of heating on the ESR intensity of the grinding-induced silicon-oxygen radicals, plot A(•), and silica-induced hemolysis by the same samples, plot B (◇).

report[8] of the reduction in the silica toxicity by Al^{3+} and polyvinyl-pyridine-N-oxide (PVPNO).

For obtaining further clues to the mechanism of silica's fibrogenicity, we investigated the possible relationship between silicon-oxygen radicals on fresh dust particles and the dust's lipid peroxidation potential by parallel measurements of the time dependence of radical content by ESR and of the silica-induced lipid peroxidation using linoleic acid as a model lipid.[10] Figure 4 shows the time dependence of the lipid peroxidation. It is seen that the ability of freshly ground silica to peroxidize a lipid decreases on storage, since the rate of silica-induced lipid peroxidation declined markedly over the first 48 hours after grinding and remained fairly constant thereafter. The similarity of the time dependence of the lipid peroxidation (Figure 4) with the decay behavior of the silicon-oxygen radicals (Figure 3, plot A) indicates that these radicals might be directly or indirectly involved in ths silica-induced lipid peroxidation, which may result in a progressive degeneration of the membrane structure and eventual loss of membrane activity[14]

In conclusion, this work shows that the fracture-induced silicon-oxygen radicals are not directly involved in the mechanism of the erythrocyte hemolysis by quartz. This is consistant with earlier reports which suggest that dust-induced hemolysis and lipid peroxidation proceed via independent mechanisms.[15,16] Thus the hypothesis[5] that lipid peroxidation caused by free radicals on the silica surface might be directly involved in the erythrocyte membrane damage does not seem likely. However, these radicals might be directly or indirectly involved in an oxidative-type chain reaction leading to macrophage membrane perturbation through lipid peroxidation and eventual fibrosis as noted earlier.[2,6,7] It is interesting to note that fibrotic action, as a result of failed phagocytosis, was suggestsd to be due to the perturbation of macrophage membrane and the consequent release of a macrophage fibrotic factor.[17] Recent ESR studies have shown that silica particles release •OH radicals in the presence of exogenous H_2O_2[18] and even without it,[7] and that the amount of the •OH radicals formed decreases with the "aging" of the quartz dust.[6,7]. Thus it is suggested that the •OH radical related mechanism of fibrogenesis by silica might be a fruitful new approach to understanding the pathogenesis of the silica-induced lung injury.

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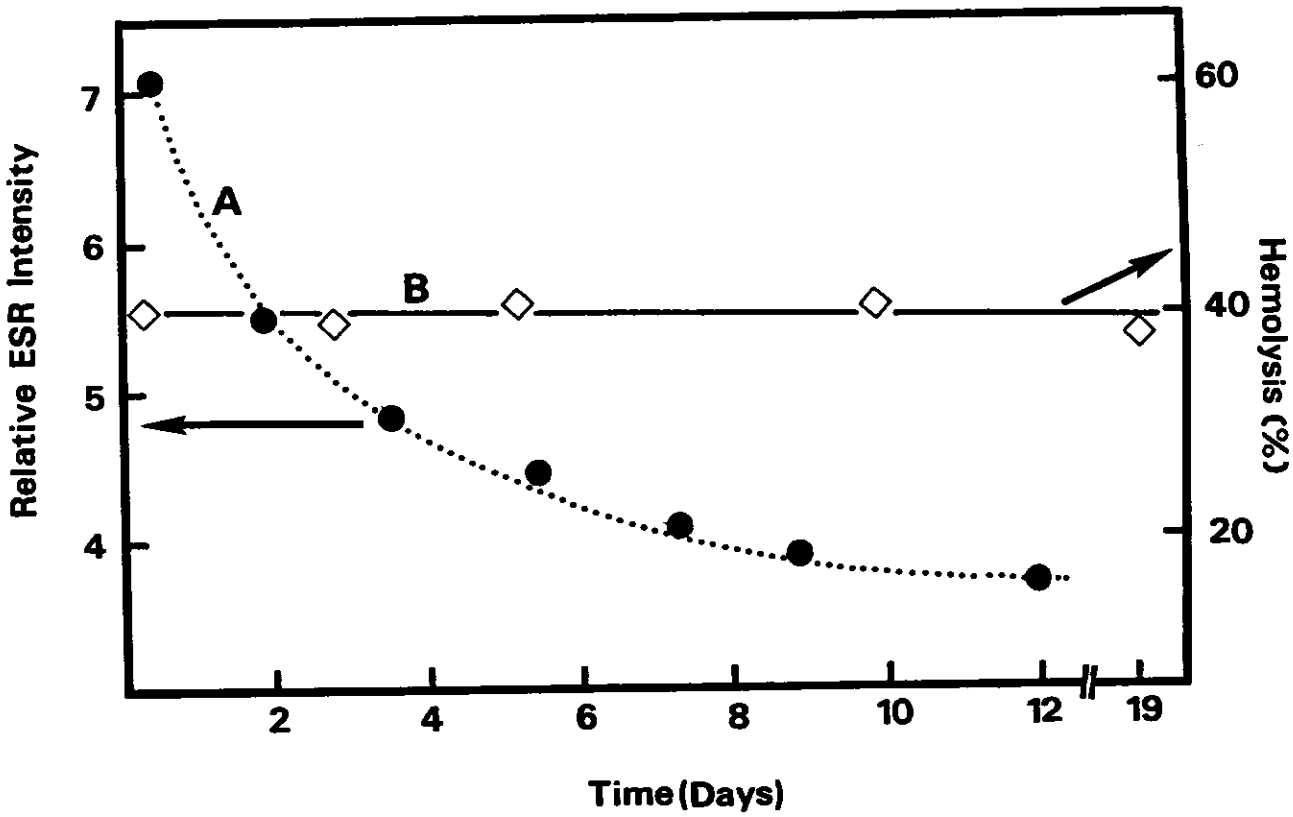

Figure 3. Time dependence of the ESR intensity of the silicon-oxygen radicals, plot A (•), on storing in air, and the hemolysis, plot B (◇), by the same sample.

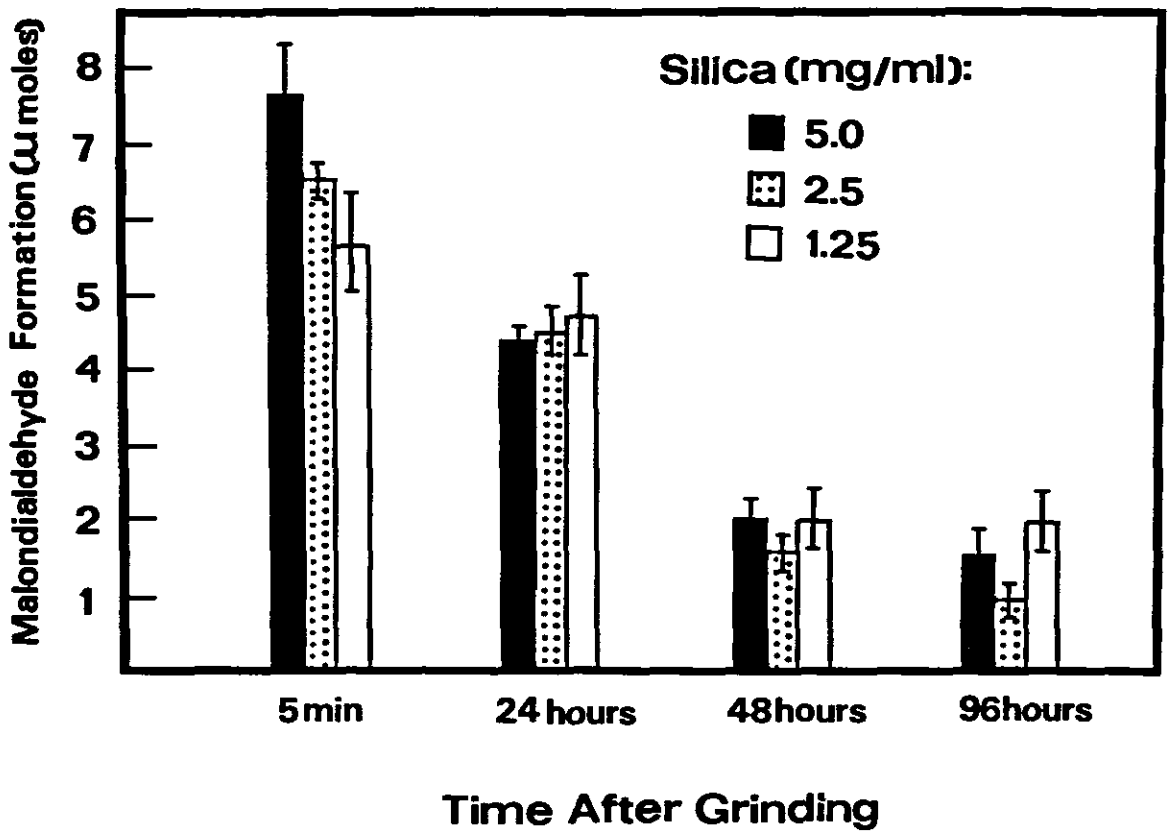

Figure 4. Effect of "aging" of the quartz dust on the rate of peroxidation of linoleic acid.

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HISTOCOMPATIBILITY ANTIGENS IN A POPULATION-BASED SILICOSIS SERIES

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ABSTRACT

Individual susceptibility to silicosis is suggested by the lack of a uniform dose-response relation and by the presence of immunologic epiphenomena, such as increased antibody levels and associated diseases which reflect altered immune regulation. Human leukocyte antigens (HLA) are linked with immune response capability and might indicate possible genetic susceptibility to silicosis. We identified 49 silicotic subjects from chest radiographs in a population-based study in Leadville, CO. They were interviewed for symptoms and occupational history and gave a blood specimen for HLA-A, -B, -DR, and -DQ typing, as well as antinuclear antibody, immune complexes, immunoglobulins, and rheumatoid factor. Silicotic subjects had double the prevalence of B44 (45%) of the reference population and had triple the prevalence of A29 (20%), both of which were statistically significant when corrected for the number of comparisons made. Notably, no perturbations in D-region antigen frequencies were detected, B44-positive subjects were older at diagnosis and had less dyspnea than other subjects. A29-positive subjects were more likely to have abnormal levels of IgA and had higher levels of immune complexes. This study is the first to find significant HLA antigen excesses among a series of silicotic cases and extends earlier hypotheses in the literature which were based on groups of antigens of which B44 and A29 are components. The prognostic or screening value of HLA typing in silica-exposed workers requires further evaluation and confirmation in other population-based studies.

No Paper provided.

HARDROCK MINING EXPOSURES AFFECT SMOKERS AND NONSMOKERS DIFFERENTLY: RESULTS OF A COMMUNITY PREVALENCE STUDY

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ABSTRACT

The physiologic consequences of occupational dust exposure, their relation to smoking, and their reversibility with cessation of exposure remain controversial. To address these questions, we studied a random sample of male residents of Leadville, CO, when the major employer, a hardrock mine, had been closed for 5–11 months. Subjects were interviewed for respiratory symptoms and occupational history, underwent plethysmographic measurements of lung volume and airflow, and performed a single-breath diffusing capacity procedure. Dyspnea was the only respiratory symptom exacerbated by mining exposures. Cumulative dust exposure, estimated with historical respirable dust measurements for mining job titles, weighted by time at the job, was associated with decreases in maximal expiratory flow rates, when controlled for smoking, age, and height. However, determinations of plethysmographic lung volume which allowed calculation of isovolume flow rates indicated that dust effects differed in neversmokers and smokers. In neversmokers, dust exposure was associated with decreased lung volume, increased flow rates, and increased DLCO/V_A. In smokers, dust exposure was associated with increased lung volume, lower flow rates, and lower DLCO/$_A$, than that accounted for by smoking. We suggest that hardrock mining exposures result in irreversible pulmonary function changes of airflow limitation in smokers and of a restrictive nature in neversmokers.

No Paper provided.

BLOOD LYMPHOCYTE RESPONSE TO BERYLLIUM AS A WORKPLACE SCREENING TEST FOR SUBCLINICAL BERYLLIUM DISEASE

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ABSTRACT

Patients with chronic beryllium disease have peripheral blood and lung lymphocytes which undergo blast transformation in response to beryllium salts. We have evaluated the beryllium-specific lymphocyte transformation test (LTT) as a workplace screening tool for early beryllium disease. Fifty-one workers (88 percent of those with current beryllium exposure) completed questionnaires and gave blood specimens for LTT. Their chest radiographs were reviewed by a NIOSH-certified B reader. Twenty workers gave repeat blood specimens within one month for assessment of test reproducibility ($r=0.98$, coefficient of variation 21%). No persons changed LTT classification as either normal (2.6-fold stimulation index) or abnormal. Six of 51 workers (11.8%) had elevated LTT results, ranging from 5.7 to 16.7-fold stimulation. Four of five abnormal cases undergoing clinical evaluation had beryllium disease, demonstrated by granulomata on transbronchial lung biopsy and elevated LTT by bronchoalveolar lavage fluid cells (ranging from 18.6 to 44.3-fold stimulation). These data show that 1) minimally symptomatic cases of beryllium disease can be identified by this peripheral blood test; 2) not all beryllium-sensitized persons have beryllium disease at the time of their positive peripheral blood test; and 3) LTT reproducibility is good, justifying further evaluation of this test for screening. We conclude that the peripheral blood LTT may prove useful in preventing clinical chronic beryllium disease by early diagnosis in a subclinical phase.

No Paper provided.

MODIFIED NUCLEOSIDES IN ASBESTOS INSULATION WORKERS AT HIGH NEOPLASTIC RISK

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ABSTRACT

Occupational exposure to airborne asbestos is associated with increased neoplastic risk. A latency period of 20–40 years between first onset of exposure and clinical manifestation of malignant disease is usually present.

Patients with certain cancers, including those with malignant mesothelioma, excrete high levels of modified nucleosides (transfer RNA breakdown products) in their urine.

In an attempt to investigate the usefulness of measuring such cancer markers in a population at high neoplastic risk, we have examined 1000 asbestos insulation workers with long-term exposure to asbestos but with no clinical evidence of malignancy at the time of examination.

A high prevalence of abnormal excretion patterns of several nucleosides, such as pseudouridine, 1-methyladenosine and 1-methylguanosine was found among the high-risk population as compared to a control group.

The results suggest that measuring levels of these biochemical markers among asbestos exposed workers, and perhaps others exposed to carcinogenic agents, has the potential for identifying, through multivariate statistical techniques, individuals who are at high neoplastic risk.

No Paper provided.

CHEST RADIOGRAPHIC AND CLINICAL FINDINGS AMONG IRONWORKERS AND MILLWRIGHT AND MACHINERY ERECTORS

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ABSTRACT

Asbestos associated diseases are prevalent in the construction trades primarily as a result of the use of asbestos containing insulation materials. Workers in metal related trades, employed at construction sites and power facilities, but not routinely using such materials, may also be at risk for asbestos hazards.

In order to assess such risk, a cross-sectional investigation was conducted of 867 ironworkers and 111 millwright and machinery erectors from the New York metropolitan area.

A high similar prevalence of roentgenologic abnormalities was found in both groups.

Forty three percent of the millwright and machinery erectors had pleural abnormalities. Duration from onset of employment was significantly longer for those with pleural abnormalities (mean 33 years) as compared to those with normal findings (mean 18 years). Ten percent had signs of interstitial lung disease. Correlations between roentgenologic and clinical findings will be presented. These findings demonstrate that individuals employed in trades with indirect exposure to airborne asbestos may be at high risk of adverse effects from such exposure.

No Paper provided.