SILICOSIS AMONG CAPE GEMSTONE WORKERS : TIGERS' EYE PNEUMOCONIOSIS.

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CONTENTS

ABSTRACT.

Silicosis continues to be an important occupational disease in South Africa, particularly in small, poorly regulated industries. A case series is described of six workers who developed silicosis whilst involved in the processing of semi-precious gem stones. They had been employed as stone sculptors in lapidaries where they processed tigers' eye, rose quartz, amethyst, quartz crystal and a variety of other locally occurring semi-precious stones. In five of the cases exposure was in small and poorly regulated lapidaries without specific dust control measures. The sixth was detected during the course of a health and hygiene survey (including dust sampling) that I conducted in one of two lapidaries still operating in the Western Cape. These workers developed serious disease. Progressive massive fibrosis (PMF) was noted in 4 of the 6 cases, three of whom had progression of their disease after cessation of exposure. With the development of PMF the initial restrictive pulmonary function abnormalities were followed by steadily worsening airflow obstruction. Lung biopsies confirmed silicosis in one case and were suggestive in a further two. Tuberculosis was confirmed in two cases and suspected and treated in a third. Workmens' Compensation was awarded in five cases. The survey confirmed that in semi-precious gem stone processing, the risk of silicosis appears to be confined to stone sculptors. Tried and proven

techniques of general and local exhaust ventilation combined with water or oil to control dust at source were capable of effectively reducing dust emission to acceptable levels.

ACKNOWLEDGEMENTS.

I was a third year medical student in the year that one of the patients that I describe first presented himself to Groote Schuur Hospital. All, except one of the cases that I describe are patients seen, diagnosed and cared for by physicians other than myself, including Professor S.R. Benatar, Professor E.D. Bateman, Professor P. Potgieter, Dr S.J. Louw, Dr S. Morrison and Dr P. Willcox.

Professor Bateman readily agreed when I proposed that our collective experience with tiger's eye pneumoconiosis should be written up and the current situation in the industry evaluated. We evaluated the case records together, including a consensus description of the radiographs according to the ILO Guidelines. Together we agreed on the protocol for the health and hygiene survey and he gave me valuable editorial advice in preparation of the text. Dr R. Chetty, Department of Anatomical Pathology, U.C.T. recalled and reviewed with us the pathology samples available on this condition. Prof Bateman and Dr Chetty were my co-authors when a shorter version of this report was published in the American Journal of Industrial Medicine.

Dr J.T. Mets, Department of Community Health, U.C.T., loaned me equipment and instructed me in its use for dust sampling in the lapidary. The National Centre for Occupational

Health, Johannesburg made laboratory services available to us as detailed in the text.

In the health and hygiene survey I was assisted by Mrs J. Etheridge, who carried out spirometry and Sr M.E. McIntyre, who completed the questionnaires. Radiographs were taken in the Department of Radiology, Groote Schuur Hospital/U.C.T., according to ILO specifications, as facilitated by Professor R. Kottler. Necessary funding was made available through the Department of Medicine, U.C.T. and Groote Schuur Hospital.

INTRODUCTION.

Silicosis is a very old problem. Available evidence suggests that it is also very much a current problem in South Africa and in other regions of the world. Research is on-going. Epidemiology and laboratory science both continue to provide us with new insights into the natural history and pathogenesis of silicosis. This dissertation contributes to our knowledge about silicosis by providing the first detailed description of pneumoconiosis occurring in the industrial processing of semi-precious gemstones of southern African origin. The features of this pneumoconiosis are described in the light of a review of recent and relevant literature on the effects of human exposure to silica.

In the first instance this dissertation is a case series. Case series may be criticised as a form of 'armchair epidemiology', since the data is relatively easy to collect and it is usually not possible to subject it to meaningful statistical analysis. In fact much of the present core knowledge of clinical medicine is based on the careful documentation and description of case histories. The importance of the case series reflects our reliance on 'natural experiments' in documenting a wide variety of exposure-response relationships in human beings. Where meaningful control groups can be assembled the case series

can be developed into a case-control study, but in this instance this was not appropriate.

My literature review located only two reports of silicosis amongst jewelry or gemstone workers. Radiological and pulmonary function abnormalities have been described amongst Hong Kong workers who process gemstones such as jade or lapis lazuli and use silica flour as an abrasive (1). Similar findings have also been reported in abstract form in relation to working the harder quartz gemstones occurring in Southern Africa (2), but there is no detailed description of this pneumoconiosis in the currently available texts on occupational health (3,4,5,6,7), nor is there any mention of it in any of the recent review papers on silicosis (8,9).

A series of six sculptors of semi-precious gem stones is described. They have been seen, evaluated and followed up at the Respiratory Clinic, Groote Schuur Hospital, since 1976. They presented with a variety of manifestations of silicosis. These were skilled workmen who hand-sculpted gem stones. The stone is worked by hand holding carefully pre-selected pieces against a mechanical abrasive belt or disc (usually carborundum) and thereby gradually shaping it. Included in the case reports is one spanning a fourteen year follow-up of a man who developed silicosis with progressive massive fibrosis. In my review of the literature on silicosis I have not yet found a similar report that

provides such longitudinal detail. This contributes to our understanding of the relationship between radiology, pulmonary function changes and the patho-physiological processes observed in this condition.

Processing of semi-precious gemstones occurs in small enterprises in South Africa, Namibia, Lesotho, Botswana and elsewhere. Significant quantities of mineral stone are exported, chiefly to East Asia. The lapidary industry in the Western Cape is small with enterprises employing a total or fewer than 100 persons. In the last ten years economic pressures have resulted in the closure of three out of five of these lapidaries. Five of the six workers had been employed in factories that have ceased operation. The skilled workmen in these lapidaries knew one another and requested referral to our clinic once the risk of pneumoconiosis became known. From their descriptions and our inspections it is apparent that the general ventilation in these lapidaries was poor (9). Minimal and ineffective steps were taken to control the dust at source and respirators were not provided. Only case 6 had the benefit of protection against dust in the form of continuous watering of the abrasive belt, combined with reasonable general ventilation.

An evaluation of the respiratory health of the workforce at one lapidary was combined with analysis of air quality. The results of this survey are presented.

This dissertation is divided into two parts. The first is a comprehensive review of recent articles and texts on the subject of silicosis. This review covers recent information on silicosis internationally and in South Africa, interesting and relevant aspects of mineralogy, exposure assessment, pathogenesis, pathology and clinical features. The second part of this dissertation contains description and discussion on the cases seen in our clinic and the health and hygiene survey carried out at a local lapidary.

OCCURRENCE AND PREVALENCE OF SILICOSIS

It appears that silicosis is a truly ancient occupational disease. Silicosis has been reported in flint-knappers, a widespread industry in Neolithic times (5). Similarly, 'hut lung', or Transkei silicosis, which arises from the use of hand-held stones to grind maize, is a 'stone age' variant of this disease (10). Evidence of silicosis (possibly acquired environmentally, rather than occupationally) has also been found in Egyptian mummies (11).

Many occupations have been reported as having an association with silicosis. These occupations are so many and varied that is difficult to compile a complete list. Table I is a summary of the sources of silicosis compiled by Cotes and Steel (7) and lists major categories of risk occupations.

The prevalence and severity of silicosis is thought to be chiefly related to the intensity and duration of exposure to free silica dust. Brief exposures to dusts with high silica content may give rise to progressive disease even after relatively short exposures. Endemic and epidemic occurrence of silicosis has been described by authors as illustrious and varied as Hippocrates, Pliny, Agricola and Ramazzini (9). Notable outbreaks of silicosis since 1900 have

Table I : Sources of silicosis.

Substance	Process	Uses and related occupations
A. Potentially high risk from crystalline SiO_2		
Granite	Q, cutting, dressing, tunnelling	Construction, monuments grinding, road building laying sewers, cables etc.
Sandstone	Q, cutting, dressing, crushing,etc. tunneling.	Construction, monuments sandblasting, refractories foundries, abrasives, scouring powders, polishes, boiler scaling, manufacture of fibre glass, carborundum, optics.
Flint	Separation from chalk, calcining,	China ware, filler, abrasive, as 'flints' for facings, etc.
Slate	M/Q, sawing, splitting, trim and polish	Construction, table tops, electric panels, pencils.
Shale	M/O-C, grinding etc.	Ceramic, aggregate, extraction of oil.
Silicon	Vaporisation of quartz	Electronics, lasers etc.
B. Risk from variable contamination with SiO_2.		
Granite ores.	Mining gold, silver, copper, mica, iron, platinum, tungsten, fluospar, barium.	
China and fire clay	M,Q, crushing, milling, mixing.	paper, paint, china and stoneware, refractories.
Feldspar	M, crushing, milling etc.	Ceramics, filler, enamel.
Bentonite	Q, crushing, milling.	Crayons, filler, lubricant.
Diatomite	O-C, crushing, screening, calcining.	Refractories, abrasives, filler, filter, insulation.

M = mining, Q = quarrying, O-C = open-cast mining.

included rock drillers in Witwatersrand gold mines, British sandblasters (12) and U.S. tunnelers (13).

Until the Second World War, silicosis was the most important and widespread form of pneumoconiosis. Since then it appears that substitution by other materials and hygiene measures have resulted in a declining incidence of new cases in the majority of industrialised countries (5,9). Despite this, silicosis still appears to be the most prevalent chronic occupational disease in the world (14). In developing countries reliable statistics are lacking, but from what is available it appears that the incidence and prevalence of pneumoconioses are high and appear to be rising (15).

There is a paucity of recent and detailed information on the occurrence of silicosis in secondary industry in South Africa. The most recent detailed publication is a review of silicosis cases seen and diagnosed at the National Centre for Occupational Health (NCOH) clinic (Johannesburg) between 1972 and 1986 (16). This paper reviews previous research endeavors in this area. The only prior major survey was one conducted by the Department of Labour in 1963 (17). At that time it was estimated that 106 000 industrial workers were exposed to silica dust. Workers in a number of industries, who had at least two years exposure to silica and who had never worked on the mines, were radiographically examined.

A total of 5 531 radiographs, taken from a sample of 161 factories, was surveyed by 3 readers. Of these, 356 were considered to have pneumoconiosis, yielding a crude prevalence of 64/1 000. Comparison of the Department of Labour study with more recent surveys in ferrous foundries conducted by the NCOH (18) suggested that there had been no substantial gains in silicosis control in the intervening two decades. This conclusion is supported by dust measurements conducted in foundries by the NCOH during 1986. Eighty percent of samples in 6 foundries exceeded the threshold limit value for respirable silica (16).

In their review of cases seen at the NCOH, Ehrlich et al. found that four industries accounted for 83% of the 217 cases - foundries, ceramics factories, refractories, and ore and stone crushing. A very similar distribution had been found in the 1963 Department of Labour survey. Some of the striking findings in the review included the relatively young age of the black workers with silicosis (21% were $\leq$ 40 years at diagnosis), the relative frequency with which accelerated silicosis (18% had $\leq$ 10 years of reported exposure) and progressive massive fibrosis (PMF) (21%) were encountered. These findings were taken as indicating high dust exposures and risk of silicosis in some sectors of South African industry. At the present time there is no recent reliable information on the numbers of workers exposed to silica outside of mining, nor are there any

recent reliable estimates of the prevalence or incidence of silicosis among these exposed workers. It is reasonable to assume that many cases go undetected (16).

Additional sources of information on silicosis in South Africa are statistics concerning compensation of silicosis (19,20). These are summarised in Table II. The figures given for mines and works are for silicosis only, regardless of source, although the overwhelming majority of cases are from gold mines. The abbreviations used (1^{o}, Pn+TB, etc.) are not explained in the report consulted and the relevant legislation must be consulted for detailed definitions. Amounts awarded were not stated in the available report. Up to the present this has been based on racial categories. At the time that these statistics were current the award for Pn+TB (white miner) was R34 300 and for CD+TB (black miner) R1 790. The 1983 MBOD report was the most recent that I was able to locate. Comparison of the figures given in this report with previous MBOD reports indicates a slow but progressive increase in the total number of cases of CD among black miners over the preceding decade (21).

The figures given in Table II for secondary industry are for compensable silicosis and asbestosis combined. There is no regular form of surveillance in secondary industry such as exists in mines and works. The low overall figures suggest that there is under-reporting of pneumoconiosis in secondary

industry. The NCOH clinic alone is probably responsible for about one third of all cases referred to the compensation authorities. Awards in secondary industry are based on wages, not on racial category. In the permanent disability category the "cost of accidents" in 1988 for 43 blacks was R573 632, compared to R562 371 for 21 non-blacks.

The 1963 Department of Labour report (17) spelt out the measures by which silicosis in industry could be controlled. These included extension of legislation, medical examination of workers in prescribed industries, a properly equipped factory inspectorate and a medical inspectorate. A quarter of a century later, some of these recommendations are only starting to be applied.

Table II : Certification and Compensation of Silicosis in South Africa.

a) Mines and Works (1982/83):

i) New certifications (Living Whites and Coloureds)

1^{o}	2^{o}	2^{o}+TB	Pn50-75%+^{o}TB	Pn>75%+^{o}TB	Pn+TB	Total
125	18	23	4	7	4	181

ii) New certifications (Deceased Whites and Coloureds)

1^{o}	2^{o}	2^{o}+TB	Total
178	8	17	203

iii) New certifications (Living Blacks)

CD	CD + TB	Total
444	725	1169

iv) New certifications (Deceased Blacks)

CD	CD + TB	Total
242	55	297
	SUB - TOTAL	1950

b) Secondary industry (1988):

i) Permanent disability

Whites, Coloureds and Asians	Blacks	Total
21	43	64

ii) Fatal

Whites, Coloureds and Asians	Blacks	Total
4	5	9
	GRAND TOTAL	2203

MINERALOGY

Silica or silicon dioxide (SiO_2) exists in nature in amorphous and crystalline forms. Amorphous silica (natural glass, diatomaceous earth) is much less toxic than the crystalline form. Crystalline silica forms under conditions of extreme heat and pressure in the earth's crust. The crystals consist of silicon-oxygen tetrahedrons in a number of polymorphic forms such as alpha quartz, cristobalite and tridymite. Alpha quartz is the most common and commercially important of these minerals, being the major constituent of igneous (granite), metamorphic (sandstone) and sedimentary deposits (slate and shale) (9).

Silicates are closely related minerals where various cations (Na, Mg, Al, Ca, Fe) and anions (F, OH) are substituted into a crystalline silica matrix. Kaolin (aluminium silicate), talc (magnesium silicate), vermiculite, micas, bentonite, feldspar and Fuller's earth are all commercially important silicates. Many rock minerals are composed of mixtures of silica and silicates. In general terms, while fibrogenicity of dust is proportionate to its silica content and is evident when this component comprises > 2% by weight, the presence of certain other silicates is believed to modulate the pathogenicity of quartz (9).

The semi-precious stones processed by sculpturing in the lapidary industry of the Western Cape Province are either crystalline or crypto-crystalline quartz ($SiO_2.nH_2O$) containing quantities of trace or other elements. The particular conditions under which they crystalize give them their special qualities. The various stones include rose quartz, jasper, agate, amethyst, aventurine and quartz crystal. The most important and prized of the semi-precious gem stones of southern Africa is tigers' eye (See plates 1 - 4). This unique form of salicified crocidolite occurs almost exclusively in an area of the northern Cape Province of South Africa where it is associated with deposits of crocidolite asbestos. Significant quantities of mineral tigers' eye and other gemstones are exported for processing, particular in East Asia.

In view of the close association of tigers' eye and crocidolite asbestos, the possibility needed to be entertained that the spectrum of pathology in lapidary workers might include some of the features of asbestos exposure. To study this we had to assess both the nature of the dust and the lesions produced.

PLATE 1

Mineral and processed tiger's eye.

PLATE 2

Mineral and processed rose quartz.

PLATE 3

Mineral and processed amethyst.

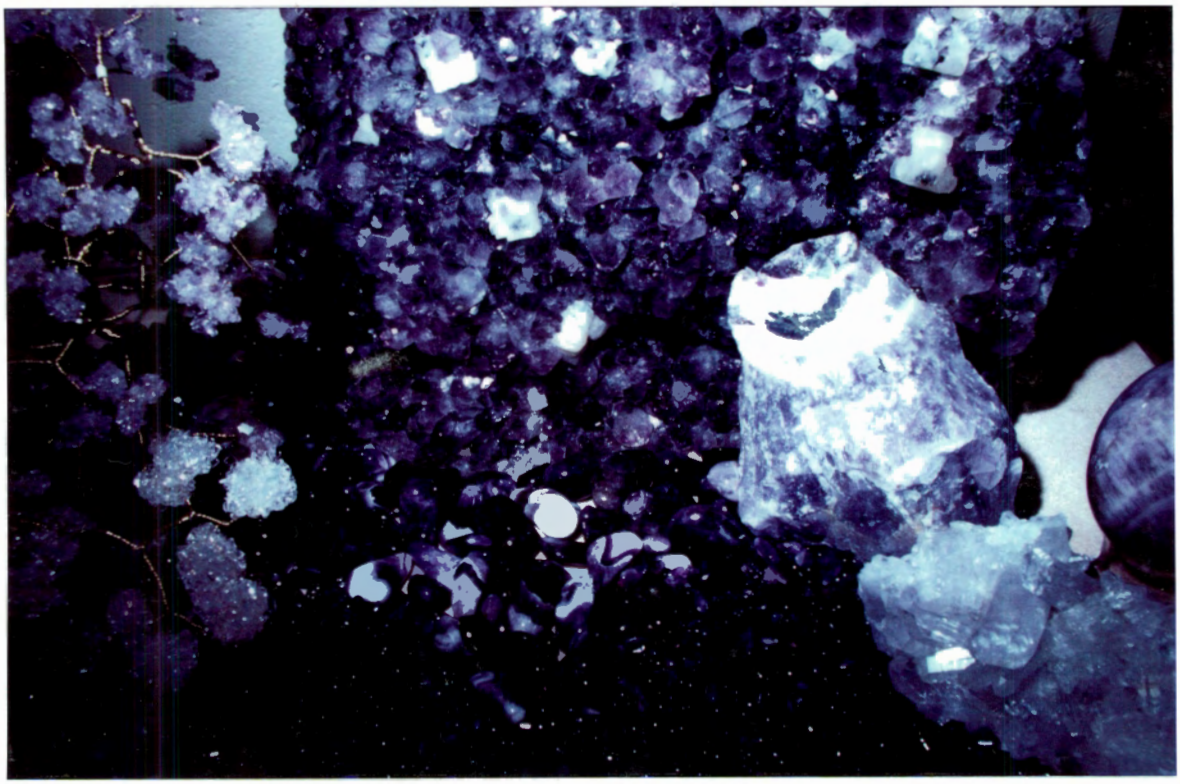


PLATE 4

Mineral and processed aventurine.

EXPOSURE ASSESSMENT.

In the first instance assessment of exposure requires an accurate understanding of the work done by an individual. In the clinical setting this requires an adequate history, including specific tasks over the years, preventive measures and the types of rock or mineral handled (22). In the workplace this involves the use of dust sampling apparatus and both the quantitative and chemical characterisation of dust.

In the mid-1960's there were approximately 60 routine sampling instruments in use for assessment of silica exposure. Since that time the number of types of instrument has been very much reduced in favour of those based on filtration with or without sedimentation (using cyclones), and optical instruments which measure the amount of light diffused by the dust particles.

At the present time the instrument in general use is the personal gravimetric dust sampler (8). This is the only method which will be described here. The personal sampler is composed of a cyclone assembly and filter holder containing a pre-weighed filter that is attached to a battery-powered pump (see figure 1). The cyclone assembly

Figure 1 : Schematic view of the CPM3 sampling instrument (ILO 1983).

Key : 1. Air entry. 2. Cyclone. 3. Rotating filter. 4. Air exit. 5. Switch. 6. Motor. 7. Voltage regulator. 8. Batteries.

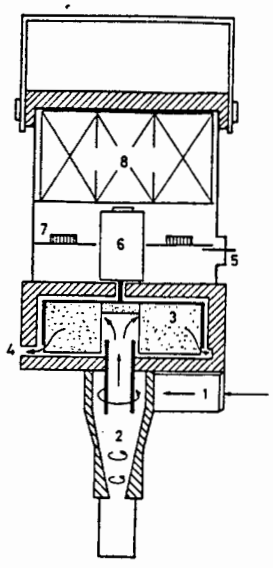
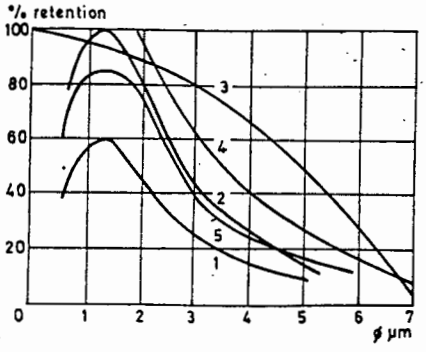


Figure 2 : Curves showing dust retention and typical characteristics for pre-separators (ILO 1983).

Key : 1. Typical alveolar retention. 2. Curve reduced to unity (i.e. maximum 1 = 100%). 3. Separation curve for MRE instrument. 4. Separation curve for AEC instrument. 5. Separation curve for CPM3 instrument.



uses centrifugal forces and collection is effected by the inertia of particles in suspension in a current of air with a pre-determined flow (usually 2.0 litres per minute). The instrument retains only those particles with an impact velocity within certain limits, depending on the air flow, the air velocity, the dimensions of the system and particle size distribution. Figure 2 shows the pattern of alveolar dust retention and the acceptance curve of a number of models of cyclone (6).

The sample weight collected is estimated by subtracting the pre- from the post-sampling weight of the filter used. Sampling strategies for duration, frequency and site will vary according to sampling objectives and relevant protocols. In general, sampling should be of more than two hours duration. Filters will require further analysis of the sample collected to determine the free silica content. If a sample contains more than 200 micrograms of free silica then X-Ray diffraction can be used. However, particularly with small samples, this method has about a 30% margin of error. If silicates or amorphous silica are not present then chemical analysis or infra-red spectrophotometry may be used. Results are usually expressed as milligrams per cubic meter of free silica.

In Table III the major factors that are thought to determine the extent or severity of silicosis are summarised (23).

Exposure assessment addresses the questions of silica dose, nature and composition of the dust. Availability of serial data concerning dose over a period of years will give the best estimate of total exposure dose when combined with duration of exposure. These factors are relatively easily quantified when compared with host factors such as between subject variation and complicating disease. Between individual variation is important since not all workers in the same environment will develop silicosis to the same degree.

Table III : Factors that determine the extent of silicosis

Category	Example
Silica dose	ambient dust level, job type, hygiene
Duration of exposure	years of employment
Nature of dust	silica polymorph, quartz
Composition of the dust	free silica percent, other silicates
Subject variation	genetics, respiration, work habits
Complicating disease	tuberculosis, rheumatoid arthritis

PATHOGENESIS

Evidence concerning the pathogenesis of silicosis has been collected over at least three decades by hundreds of investigators (biological and physical scientists) studying human cases, animal models and in vitro systems involving cells and cell lines. The published literature on this subject is vast. Given the size of the available literature and the multiplicity of factors involved it is necessary to focus this review. Three crucial questions will be addressed.

(1) How does silica arrive at sites where it causes disease;

(2) How does silica exert its toxicity; and

(3) What are the essential elements of the host response to silica?

(1) How does silica arrive at the sites where it causes disease?

The size, shape, mass and aerodynamic characteristics of inhaled dust particles determine their deposition within the respiratory tract. The sites at which they land determine, in part, whether these particulates are rapidly cleared from the lung, or remain to interact with pulmonary tissues and host defence mechanisms (24).

Particles with aerodynamic diameters > 10 μm are deposited against the turbinates of the nose and the posterior oropharynx; particles 1 - 10 μm deposit in large and medium sized airways; particles 0,5 - 5 μm reach and deposit in small airways and alveoli, while very small particles < 0.5 μm may be deposited or carried away suspended in the exhaled airstream. An example of an alveolar dust retention curve was shown in figure 2.

The inertia of larger particles leads to their deposition at airway bifurcations whilst smaller particles contact the respiratory epithelium either by sedimentation or by Brownian motion as the airstream slows. Again deposition occurs preferentially at alveolar duct bifurcations (25).

Particles that land on ciliated epithelium may be cleared by the mucociliary escalator within several hours. Particles

deposited between islands of bronchial mucus, or which land on the non-ciliated membrane of the lower respiratory tract may either be phagocytosed by alveolar macrophages or may penetrate the respiratory epithelium directly where interstitial macrophages will ingest them. Thereafter, resident and recruited pulmonary macrophages will demonstrate intimate contact with the silica particles.

Some alveolar macrophages carrying particles may gain access to the mucociliary escalator where clearance of dust deposited at alveolar level may take place over a period of days. Dust laden alveolar macrophages are also thought to be capable of penetrating the airspace epithelium. Dust that gains access to the interstitial compartment may remain at that site or is slowly (over months) transported along lymphatic channels to regional lymphoid tissue, hilar lymph nodes and sub pleural lymphoid aggregates.

(2) The toxicity of silica.

The toxicity of crystalline silica appears to be related to the chemical characteristics on the surface of particles produced by the breakage of SiO^2 tetrahedrons. New surfaces are created at the time of dust liberation from the mother lode. The toxicity of this silica can be modified by geological origin, interaction with accompanying minerals, grinding mode and consequent distribution of particle sizes generated, time after grinding, thermal treatment, acid etching, chemical purity of surface and absorbed organic molecules (26).

In free silica, silicon and oxygen are covalently bonded: ≡ Si - O - Si ≡. Fracturing in a heterolytic way leads to the formation of ≡ Si+ and ≡ S - O-, or in a homolytic way to produce ≡ Si and ≡ SiO types of radical. These radicals may interact with ambient air molecules and their concentration markedly decreases with time (27).

In contact with water and physiological environments a number of reactions may take place on the surface of quartz. Silanol groups ≡ SiOH may form. Silicon-based radicals may react to produce hydrogen peroxide (H^2O^2) or free oxygenated radicals (OH or O^{2-}) (28). The presence of Fe^{++} can increase this OH production (26).

Hydroxyl radicals (OH) are highly reactive. Peroxidation of lipids is one of their well-identified actions. The formation and accumulation of lipidic hydro-peroxides on a cellular membrane will alter its functions and can provoke its collapse. There are some indications that this surface activity could be catalytic in nature with a low but continuous production of free oxygenated radical - an important finding given the long latency period of silicosis.

The ≡ SiOH silanol groups may also be membranolytic. This may occur through hydrogen bonding with membranes that may bring the membrane into contact with a local concentration of free oxygenated radicals beyond the capacities of the cell defense system (29). Surface ≡ SiO- anionic sites may also play a membranolytic role by creating strong electrostatic interactions between quaternary ammonium groups of some lipids.

These interactions between the surface of quartz and membranes are especially important following macrophagic phagocytosis of particles.

Early in vitro observations about the interactions of silica and alveolar macrophage emphasised the toxicity of silica that followed membranolysis of phagolysosomes (30). In vivo observations now suggest that lipids such as surfactant may

reduce the cytoxicity of silica macrophages, resulting instead in altered function as will be detailed below.

(3) The host response to inhaled silica.

Current hypotheses on aspects of the host response to silica at the cellular and cytokine level have been well summarised by Davis (23,31). Figure 3, which has been adapted from his publications, provides a useful summary of what are thought to be the mechanisms of this interaction.

Deposition of silica particles in the lower respiratory tract may activate complement C5a present in alveolar lining fluid or may result in chemotaxin release by the first macrophages to ingest dust particles. Further macrophages are then rapidly recruited to this site of deposition. These macrophages may become activated without necessarily ingesting silica particles (32). Animal (33) and lavage studies in healthy human granite workers (34) suggest that macrophages activated in this manner have a normal term of viability. The toxicity of silica particles ingested by macrophages is exerted through secretion of pro-inflammatory mediators. Having ingested silica particles, four outcomes for the macrophage can be envisioned : i) the macrophage might transport the particles to lymphoid sites via lymphatics or up and out on the mucociliary escalator; ii) the silica-containing phagolysosome of the macrophage might be disrupted, causing macrophage death and release of both particles and constitutive macrophage enzymes; iii) the macrophage, although damaged, might be stimulated to release

or secrete elastase, collagenase and toxic O_2 species; or iv) the macrophage is stimulated to produce pro-inflammatory mediators, particularly interleukin-1 (IL-1) (35).

Silica does not have recognisable antigenicity. It appears to induce inflammation and fibrogenicity non-specifically. However, the ample evidence of cell-mediated immune mechanisms in silicosis suggest that silica is capable of participating, probably in an adjuvant role, in immunological events. These events are played out over a long period of time, usually at a low level.

As depicted in Figure 3, IL-1 calls into action the extensive bi-directional communications between macrophages and lymphocytes. Proliferation of an expanded and activated population of helper T-cells is induced via interleukin-2 (IL-2). This population can in turn secrete a variety of mediators such as macrophage activating factor (MAF), interferon gamma (IF-g), macrophage migration inhibiting factor (MIF), and macrophage fusion factor.

Stimulation of immunoglobulin production accompanies silicosis and anti-nuclear antibodies, rheumatoid factor and circulating immune complexes may be present (36). Non-smoking healthy silica exposed workers also have increased immunoglobulin concentrations in their bronchoalveolar lavage fluid, despite normal serum immunoglobulin levels

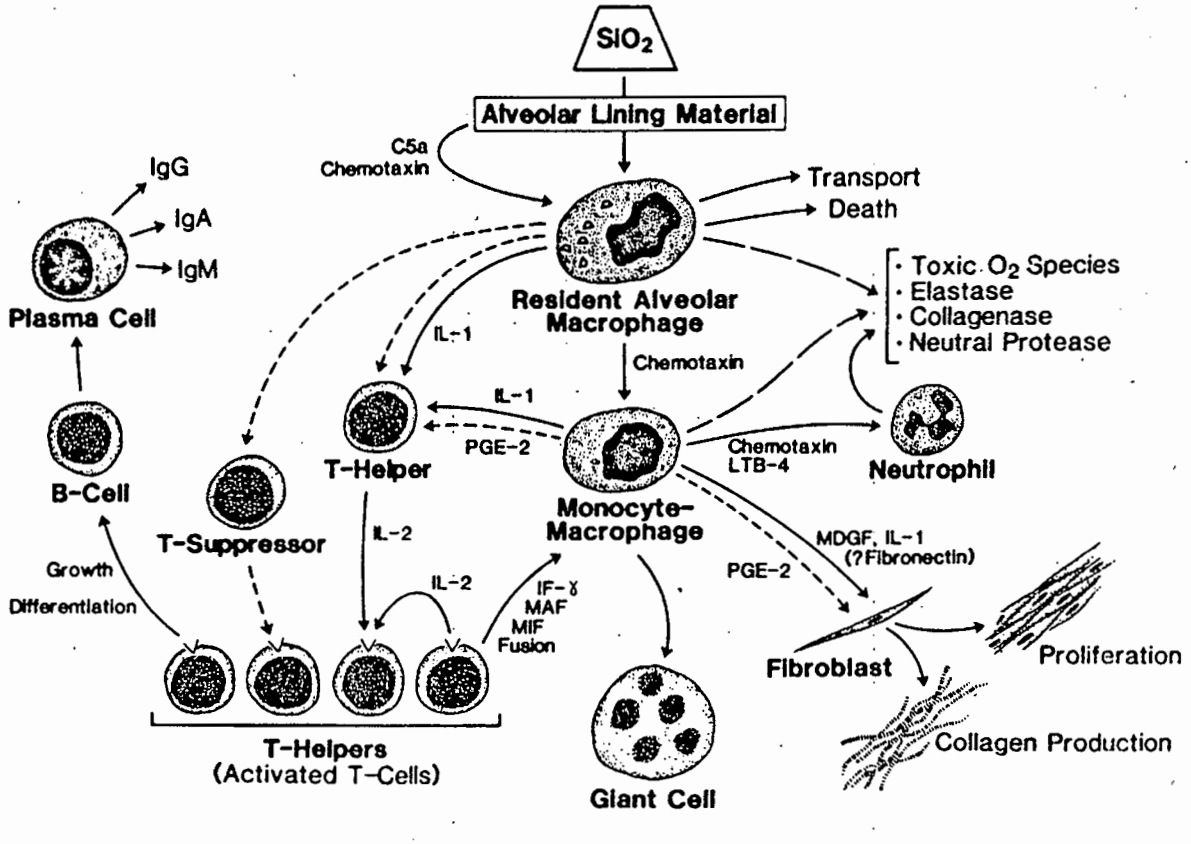
(37). These findings are taken to suggest non-specific polyclonal stimulation of local production of immunoglobulins in the lung. This could be the consequence of maturation of uncommitted B cells to plasma cells under the influence of activated T cells and possibly IL-1. Notably plasma cells are prominent in silicotic nodules.

Macrophages activated by silica secrete chemotactic cytokines for neutrophils that include leukotriene B-4 (38). The role that neutrophils play in the pathogenesis of typical human chronic silicosis is not clear. Proteolytic enzymes and free oxygen radicals produced by neutrophils are capable of causing lung injury but neutrophils are not prominent in bronchoalveolar lavage fluid, nor histology in human silicosis.

Advanced silicosis is characterised by pulmonary fibrosis. Again the interaction of silica and macrophages appears to be implicated (39). It appears that activated T cells producing IF-g stimulate activated monocytes/macrophages to secrete macrophage-derived growth factor - a much more potent fibroblast growth promoter than IL-1. Fibroblast proliferation is followed by collagen production. Fibronectin also accumulates at these sites and is an important constituent of silicotic nodules.

Figure 3 : The mediators and cell-cell interactions postulated to govern the development of silicosis (After Davis, 1986).

Key : Details are discussed in the text. Stimulators are shown as solid lines, suppressive influences as dashed lines. SiO_2 : crystalline silica; C5a, 5th complement component active fragment; CHEMOTX, chemotactins; IL-1, interleukin-1; IL-2, interleukin-2; PGE-2, prostaglandin E-2; IF-g, interferon-gamma; MAF, macrophage-activating factor; MIF, migration-inhibition factor; LTB-4, leukotriene B-4; MDGF, macrophage-derived growth factor.



PATHOLOGY

Microscopic appearances.

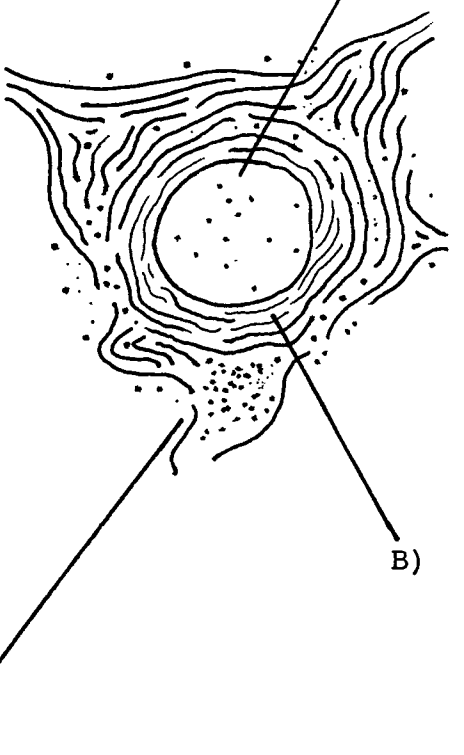
A series of histopathological lesions evolve in human and animal models of silicosis. A sequence of immune-inflammatory events is evident within these lesions. Once exposure has occurred the changes that occur correlate roughly with time.

The earliest 'minimal lesion' comprises an interstitial collection of macrophages with dust particles concentrated within it, mixed with a loose collection of reticulin fibres. These changes are often adjacent to respiratory bronchioles, pulmonary vessels and in the sub-pleural regions (9).

With time hyalinised collagen fibres accumulate in the centre surrounded by dust containing macrophages, lymphocytes and reticulin. Occasional neutrophils and multinucleated macrophages are also present. As the central zone gradually expands the collagen fibres assume a concentric configuration. A classical silicotic nodule appears to represent a specific tissue response to crystalline silica. The structure of the classical silicotic nodule is diagrammatically represented in Figure 4 and examples are shown in plates 8 and 9.

As these nodules enlarge they coalesce and collapse into one another with consequent destruction or distortion of the lung parenchyma, airways, blood vessels and the pleural membrane.

Figure 4 : Diagrammatic representation of a classical nodule in silicosis



C) Peripheral zone: contains more randomly organised collagen fibres mixed with dust-laden macrophages and lymphoid cells.

Macroscopic appearances.

The cut surface of the silicotic lung exhibits a variable number of well-demarcated, round, firm palpable nodules of variable colour that (for unknown reasons) are usually more marked in the upper zones.

In simple silicosis individual nodules 2-6 mm in diameter are scattered irregularly in the parenchyma. Nodules may become calcified, only rarely do they undergo necrosis. Pleural and sub-pleural focal fibrosis associated with silicotic nodules may also occur. Irregularly enlarged air spaces may develop in the lung parenchyma associated with silicotic nodules and scars.

In more advanced disease individual lesions may become linked together to form large, complex, confluent, nodular masses. Conglomeration of nodules may reach the point where progressive massive fibrosis (PMF) may be said to be present. PMF is usually found in the upper zones of the lung where considerable volume loss may occur. Ischaemic necrosis is very unusual in silicotic PMF and if cavitation occurs it is usually due to tuberculosis.

The presence of PMF is defined by the Committee of the College of American Pathologists (9) as a histological

lesion at least 2 cm in diameter, whereas in the ILO Classification of Radiographs of the Pneumoconioses (1980), (6) PMF is considered to be present if an opacification in the lung on roentgenogram exceeds 1 cm in diameter (Category A large opacities are 1-5 cm in diameter).

Pulmonary hypertension and cor pulmonale are features of advanced silicosis. The mechanism of this may be hypoxic pulmonary vasoconstriction related to either severe parenchymal disease or complicating COAD. In addition structural alterations in pulmonary vessels may result from the accumulation of dust in the adventitia of large vessels and by destruction, distortion and stenosis of smaller vessels by silicotic nodules.

Additional pulmonary pathologies associated with silica exposure but not detailed here are i) silicotic alveolar proteinosis; and ii) Caplan's syndrome (rheumatoid pneumoconiosis); iii) tuberculosis; iv) There is growing epidemiological evidence that carcinoma of the bronchus may be related to silica dust exposure (40,41,42).

SILICOSIS FOR THE CLINICIAN

Diagnosis

A recent review (14) re-affirms long established strategies for the diagnosis of silicosis. This involves the triad of i) careful assessment of past exposure, ii) exclusion of masqueraders (eg. sarcoidosis), and iii) pathological evaluation of tissue. In most cases pathological evaluation is unnecessary if exposure is unequivocal and other processes seem unlikely. Less invasive procedures, including bronchoalveolar lavage, computerized tomography of the chest and determinations of serum angiotensin converting enzyme activity, have not demonstrated any strongly positive or negative predictive value.

Clinical features and natural history of silicosis

It is not my intention to produce a detailed review of the clinical features of silicosis. The basic descriptions of the clinical syndromes are widely available in the major texts on occupational lung diseases and in reviews consulted. Instead, this review will focus on issues in need of clarification, issues of interest to the clinician and recent data on the natural history of silicosis.

There are four distinct non-malignant pulmonary syndromes associated with silica exposure. On first becoming acquainted with them there could be some confusion because of a certain looseness in definition and a plethora of names. The four are:

i) Acute silicosis/silicotic alveolar proteinosis/silicotic alveolar lipoproteinosis;

ii) accelerated silicosis;

iii) chronic/simple/nodular silicosis; and

iv) progressive/conglomerate/complicated silicosis or progressive massive fibrosis (PMF)

Acute and accelerated silicosis are relatively rare conditions related to (often short-term) exposure to high concentrations of relatively pure silica particles. The highest exposures tend to produce acute silicosis which is characterised by desquamation of type II pneumocytes and leakage of lipoproteinaceous material into the alveoli. Silicotic nodules are small and poorly defined. Acute silicosis usually progresses, causing death by respiratory failure within about a year. Repeated therapeutic bronchial lavage may be useful in this condition (43).

In accelerated silicosis the major features are very similar to the chronic disease, but the time from exposure to radiological appearance of disease and death is shorter. The time periods quoted vary somewhat. Ziskind, Jones and

Weill (8) state that radiological changes appear within 4-8 years, whilst the NIOSH (9) committee states 5-10 years. Ziskind, Jones and Weill state that the condition is usually fatal after 10 years of exposure, whilst NIOSH states that it is fatal ten years after the onset of symptoms. Neither Parkes (5) nor Cotes (7) offer a special definition of accelerated silicosis. Accelerated silicosis appears to be distinguished from chronic silicosis both by its progressive course and in that the latter appears only after at least 10 and often after 20 years of exposure to free silica (8;9).

Using time periods to distinguish the clinical syndromes is artificial and unsatisfactory (44). An additional pathological feature of accelerated silicosis may be predominance of early silicotic lesions including 'minimal lesions' and lesions that have not developed concentric lamination of collagen fibres (45). Early lesions may co-exist with more mature nodules depending on the time-course of the disease.

Chronic silicosis is the most common manifestation of silica dust exposure. Interstitial fibrosis is not a prominent feature where nodules are isolated and less than 1 cm in diameter. Chronic silicosis causes relatively minor physiological impairment. In conglomerate silicosis nodules become confluent and enlarged thereby encroaching on, and replacing the lung parenchyma. Conglomerate silicosis or

progressive massive fibrosis may produce significant physiological impairment. One longitudinal study of lung function and radiological progression in a 'survivor population' of Hong Kong granite quarry workers (46) showed that baseline FEV1 and FVC in simple silicosis was near-normal, being 94.0% (± 17.5) and 96.5% (± 15.7) predicted value respectively. In complicated silicosis these values were 83.6% (± 23.7) and 86.7% (± 18.4) predicted value respectively.

Another study of a survivor population receiving disability benefit for silicosis has shown a difference in estimated decline in FEV1 between simple and complicated silicosis in non-smokers (18 ml/year and 38 ml/year respectively) (47). In this study FEV1 was shown to decline with increasing X-Ray category of silicosis irrespective of smoking habit. This effect was most marked in subjects with symptomatic chronic bronchitis. A similar, but not statistically significant trend was noted for vital capacity.

Radiological progression from simple silicosis to PMF may occur in periods of less than 16 months (48,49). From the various descriptions of progression of simple silicosis it appears that the known determinants of this are i) cumulative dust exposure; ii) category of simple pneumoconiosis; iii) young age of onset; iv) continued

dust exposure in the presence of simple pneumoconiosis (8,46,48,49,50).

It is generally held that simple silicosis does not affect life expectancy, although complicated silicosis may do so (9). This is supported by multivariate analysis of prognostic factors influencing survival of workers compensated for silicosis in Quebec from 1938 - 1985 (51). On the basis of their model, the authors found that patients with only small opacities on their chest radiograph and without symptoms or abnormal breath sounds had a survival similar to the average Quebec man. Other patients had a poorer survival. Using their prognostic index a 55 year old male, compensated, silicotic, smoker with regular expectoration, dyspnoeic with minimal effort, abnormal breath sounds, VC < 80% predicted and large opacities on chest radiograph would have a probability of 0.49 of surviving 5 years, of 0.21 of surviving 10 years and 0.06 of surviving 15 years. By comparison a hypothetical 55 year old Quebec male from the 1980-82 lifetable had a 0.93 probability of surviving 5 years, 0.83 for 10 years and 0.70 for 15 years.

Complications of silicosis

Silicosis may be complicated by a variety of other diseases or processes. Tuberculosis is the most important of these and will be dealt with in some detail. Other complications include:

i) Pulmonary heart disease: The presence of pulmonary hypertension appears to be a serious adverse prognostic sign in silicosis (52) although it seems that right ventricular failure only supervenes in a small proportion of cases with advanced conglomerate silicosis (5)

ii) Chronic bronchitis frequently co-exists with silicosis, as may irreversible airflow obstruction. There is substantial epidemiological evidence linking these entities, which satisfies epidemiological criteria for causality (53,54). However, in individuals we still lack methods of accurate quantification for the interaction of smoking and occupational dust exposure in the causation of chronic bronchitis (55). There is some evidence that silica exposure may have different effects in smokers and non-smokers (56) as seen in one cross-sectional study in U.S. hard-rock miners. A restrictive defect was most common in never-smokers, whilst in smokers the accent was on irreversible airflow obstruction.

iii) Spontaneous pneumothorax: appears to be associated with silicosis as with other types of interstitial lung disease (5).

iv) Segmental and middle lobe collapse: May occasionally occur due to bronchial compression by silicotic lymph nodes (5).

v) Scleroderma (progressive systemic sclerosis): Appears to be more common among men exposed to silica-containing dust than in the general population (5). The better-known presenting features of systemic sclerosis, including Raynaud's phenomenon and dysphagia are unusual. Initial presentation is often with non-specific features. Pleural, interstitial lung and pericardial involvement is relatively common whilst renal disease is rare (57).

vi) Nephropathy: Glomerular damage has been ascribed to silica exposure.

vii) Rheumatoid syndrome: Progression of silicosis associated with rheumatoid factor with or without active arthritis has already been alluded to.

viii) Carcinoma of the bronchus has already been mentioned.

ix) Tuberculosis: Silicotic patients have heightened susceptibility to a range of opportunistic infections, including cryptococcus and sporotrichium (9). Anaerobic bacteria have also been implicated as a cause of infection in patients with advanced silicosis (58). MOTTs such as M. kansasii and M. avium - intracellulare have also been documented in silicotics. The most prevalent and therefore serious superinfection by far, is that of M. tuberculosis (59). Silicotuberculosis may be either a) clinically active, acute disease with positive sputum; b) chronic lesions with or without cavitations; or c) quiescent and fibrotic. Recognition of tuberculosis accompanying silicosis may be extremely difficult. Symptoms, signs and non-specific laboratory findings may be misleading. 'False negative' sputum cultures are not uncommon (59). It has been suggested that the following radiographic changes are strongly suggestive of tuberculosis superimposed on a silicotic process: a) the rapid appearance of new infiltrates, especially in the upper third of the lung fields; b) coalescence of nodules, especially in the upper third of the lung fields; c) the development of bronchial stenosis or occlusion; d) development of a pleural or pericardial effusion; e) the appearance of any massive unilateral, non-retractile opacity limited by a fissure; and f) the appearance of a cavity, especially one with an irregularly shaped inner wall, within a conglomeration of nodules (59).

It appears that silicotics have greatly increased susceptibility to tuberculous infections. This is the only pneumoconiosis in which this is the case and appears to be related to the altered functioning of pulmonary macrophages laden with silica particles. Preventive strategies that have been tried in silicotics have included use of BCG vaccine. This is now considered contra-indicated in the light of evidence that BCG may accelerate and worsen silicosis whilst giving no protection against tuberculosis (60). The evidence that INH is effective in preventing tuberculosis in silicotics appears to be equivocal (59).

The treatment of silicotuberculosis has been greatly improved by the sterilizing powers of rifampicin and pyrazinamide. One trial of short-course chemotherapy for silicotuberculosis (61) suggests that either of two regimens (A: rifampicin 600 mg, isoniazid 300 mg, pyrazinamide 2 gm, streptomycin 1 gm for 5 days a week for 100 doses; or B: the above given 7 days a week for two months followed by daily thiacetazone 150 mg for seven months) are as effective in black miners with silicotuberculosis as they are in matched controls with tuberculosis only. Using these regimens 36-month life-table relapse rates of 6 - 11% were encountered. It is not possible to judge the severity of the silicotuberculosis encountered by the authors of this report and whether short-course chemotherapy is also likely to be

effective in cases with PMF, or if treatment should be more prolonged in such cases.

Causes of death in silicotics.

None of the various studies of mortality patterns in silicotic subjects are directly generalizable to South Africa. One interesting study will be quoted to give some insight into this matter (62). This study included 595 workers who had been compensated for silicosis in the Latinum region, Italy and had died before December 1984. Mortality rates and smoking rates were compared with the general population. Respiratory diseases (ICD 460 - 519), tuberculosis, lung cancer, bone cancer and cirrhosis of the liver all showed significantly increased risk ratios (4.1, 3.7, 1.5, 4.1, and 1.9 respectively). Excesses of brain cancer and leukaemia did not reach statistical significance. Smoking habits did not appear to differ meaningfully between cases and the general population. The excess risk for lung cancer (64 cases) was only statistically significant in mining and pottery workers.

Treatment.

There is currently no specific form of therapy for silicosis. Interventions therefore revolve around primary prevention of the disease and secondary prevention of complications. Primary prevention revolves around measures such as i) wetting down work areas to prevent dust generation; ii) requiring exposed workers to wear effective masks or hoods; iii) institution of effective local and general ventilation where silica-containing dusts are generated; iv) substitution of silica containing substances where possible; v) limiting exposure times of workers. Routine radiographs for early detection of disease also have value.

Secondary prevention in established cases of silicosis includes regular monitoring and high clinical suspicion of tuberculosis and other opportunistic infections. Once silicosis has become radiologically apparent, removal from exposure reduces the risk of progression to complicated disease (14,50).

PART II : SILICOSIS IN THE LAPIDARY INDUSTRY OF THE WESTERN CAPE PROVINCE.

In the late 1970's there were four small industrial establishments in metropolitan Cape Town where semi-precious gemstones were processed from mineral to their finished form. By the mid-1980's there was only one operational company left in Cape Town with a second, newly-established plant in Hermanus. The precise reasons for this contraction of the industry are not known to me, but they must be related in part to the superior competitiveness of larger and better-run enterprises, the effects of imports from East Asia and the establishment of new enterprises in areas where wages are traditionally lower than the Western Cape, such as Bophutatswana and Lesotho.

The effects of the economic decline of the lapidary industry are seen in the retrenchment of most of the workers described below, and are probably also reflected in the very poor occupational hygiene prevalent in most of the industry. The labour process in all of these enterprises appears to have been very similar with the only apparently important variations being in the local and general measures taken to control dust. The entire labour process is described in detail below in my account of a health and hygiene survey of the only operational lapidary in Cape Town. However, all of the cases described have in common that they worked in one

part of this process - the hand sculpting of carefully selected pieces of virgin stone by holding them against a grinding wheel (see Plates 5 and 6). Production of a single piece (see Plate 7) may take several hours, or even days.

As skilled workmen many of these workers knew each other personally. This informal network was important as a means of facilitating the referral of some of these men to our clinic, although traditional referral routes, e.g. from tuberculosis clinics were also important.

PLATE 5

The usual method of sculpting semi-precious gem stones using a wet carborundum wheel.

PLATE 6

A close-up of the same process.

PLATE 7

Examples of sculpted semi-precious gem stones.

CASE REPORTS.

Case 1.

A 35 year old man who worked for 19 years as a gemstone sculptor and presented to our clinic in 1976 with symptoms of dyspnoea, intermittent wheezing, weight loss and a non-productive cough, including a single small haemoptysis. He smoked 20 cigarettes a day. Pulmonary function testing showed a modest non-obstructive reduction in lung volumes (as compared to predicted values) (63) and reduced carbon monoxide transfer factor (64)(Table IV). The chest radiograph was compatible with simple nodular silicosis (Table V)(Plate 10). Because of uncertainty about the diagnosis he under-went open lung biopsy. The biopsy showed the features of silicosis as described below.

Radiographs taken in 1978 showed changes compatible with progressive massive fibrosis (Plate 11). Nodules were present throughout both lungs with large confluent areas (type C large opacities) in both upper lobes and in the apical segment of the right lower lobe. Serial chest radiographs over the next few years revealed the evolution of upper lobe bullae and areas of compensatory emphysema in the lower lobes. Pulmonary function tests were carried out regularly between 1976 and 1990 (see Figure 5). These revealed a gradually worsening restrictive disorder until

1981, followed by a progressive decline in FEV1 of about 100 ml per year as severe airflow obstruction developed. Accompanying the worsening airflow obstruction was air trapping as the residual volume and total lung capacity increased in inverse proportion to the FEV1.

He continued in his occupation until 1981 when Workmens' Compensation was applied for and awarded. He was subsequently retrenched. He also stopped smoking in 1981. He has continued to be followed up regularly in our clinic. When last seen and re-evaluated by myself at the end of 1989 he was severely symptomatic and limited by dyspnoea. He was being treated with bronchodilators which gave some symptomatic relief, although there was no evidence of reversibility in his lung function tests.

Comment : This patient's case record is of special interest and value because of the long follow-up period and the periodic detailed evaluation of his lung function. The changes observed can be interpreted as showing a close correlation of lung function changes with radiological evidence of the underlying pathological process. Once PMF developed the progressive fibrosis resulted in traction on adjacent structures with consequent compensatory emphysema, bullous formation and increasing residual volume. Distortion of airways from the second generation onwards would be measured as airflow obstruction. Obstruction would

worsen air trapping and would progress with the fibrosis. The patient stopped smoking in 1981 and therefore smoking is unlikely to have played a major role in his progressively worsening airflow obstruction.

It is unfortunate that he continued with silica exposure for a considerable period of time after simple silicosis in high profusion had been diagnosed. This placed him at increased risk for the development of PMF.

Case 2.

A 32 year old man presented to our hospital in early 1981 with right sided pleuritic pain and was found to have a spontaneous pneumothorax and diffuse nodularity on chest radiograph. He smoked 5 cigarettes a day and had been working for 15 years in lapidaries, mostly as a sculptor. He was particularly experienced in the working of tiger's eye. He initially refused further evaluation but was seen again in November 1981 on the advice of his colleagues. At that time he was asymptomatic and his pulmonary functions were within normal limits. The chest radiograph showed the presence of simple silicosis with some minor pleural thickening. He continued to work despite advice to the contrary. He returned to our clinic again in 1985. He had recently been retrenched because the company he had worked

for had closed down. On this visit he was symptomatic with dyspnoea and a radiograph (Plate 14) showed established progressive massive fibrosis (Table V). Workmens' Compensation with 100% disability was successfully obtained. With subsequent follow-up a progressive decline in his pulmonary function with worsening fixed airflow obstruction was documented (Table IV). He began to have repeated admissions to hospital for the treatment of respiratory tract infections. In 1985 his lungs were found to be colonised by Pseudomonas sp.. By 1989 he had deteriorated markedly with signs and symptoms of cor pulmonale. He received domiciliary oxygen but died in respiratory failure in March 1990.

Comment : Spontaneous pneumothorax is a described complication of silicosis and other interstitial lung diseases. The occurrence in 1981 represented an opportunity lost for this man to withdraw from silica exposure. At this time he had simple silicosis with large nodules in high profusion. In returning to work he was at risk from PMF. This occurred with devastating consequences. The radiograph showed bullae formation and possible cavitation. At one period his respiratory tract became colonised by Pseudomonas sp., another described complication of silicosis and a possible cause of cavitation.

Case 3.

This 41 year old man was first seen in our clinic in 1979. He had first been diagnosed as having silicosis following evaluation and open lung biopsy at another hospital in 1977. He had worked for 15 years as a gem-stone cutter, primarily with tiger's eye and rose quartz, in a lapidary in Oudtshorn. He had an estimated 40 pack year smoking history. At presentation he was severely dyspnoeic and unable to work. Observed (and predicted) pulmonary function test results were : TLC 4370 ml (5460), RV 3150 ml (1690), FEV1 520 ml (3110), FVC 1120 ml (3760), T'LCO 5.5 ml/mmHg/min (27), and KCO 3.0 ml/mmHg/min/l (5.0). The chest radiograph showed the features of PMF and marked bullous change with compensatory emphysema. A small right apical pneumothorax was present. In view of the possibility of co-existent tuberculosis he was subjected to fibre-optic bronchoscopy and transbronchial biopsy. In spite of negative findings he was given a full course of anti-tuberculous therapy, together with bronchodilators and corticosteroids. There was little subjective or objective response and the patient died from respiratory failure in early 1981.

Comment : This patient had very severe airflow obstruction. The residual volume is greatly increased, to a degree not

seen in any of the other cases with PMF. Gas transfer is similarly more depressed than in case 1. This suggests a role for smoking in the severity of his lung function abnormality.

Case 4.

This 56 year old man worked in lapidaries as a gemstone sculptor and foreman from 1947 until he was retrenched in 1981. He described his working conditions as having been very dusty. Pulmonary tuberculosis was diagnosed at a local tuberculosis clinic in 1981 on the basis of direct examination of a sputum specimen. The chest radiograph failed to improve with treatment and he was therefore referred to the Respiratory Clinic for an evaluation. He felt improved from the treatment and was only mildly dyspnoeic. He had smoked 20 cigarettes per day for many years. The chest X-ray showed PMF with marked bilateral upper lobe destruction and contracture, worse on the right than on the left. No cavities were evident. There was a diffuse nodular infiltrate in the lower zones. Both lower zones appeared hyperlucent with compensatory emphysema. His pulmonary function showed a mixed pattern of moderate obstruction and restriction with reduced carbon monoxide transfer factor : TLC 4460 ml (5870), RV 1190 ml (1980), FEV1 1313 ml (3010), FVC 3215 ml (3890), FEV1/FVC = 40.8%, T'LCO 12.6 ml/mmHg/min (24.4), and KCO 3.18 ml/mmHg/min/l

(4.4). He was awarded Workmens' Compensation. Subsequently we have been unable to recall him but he is known to have been alive in 1989.

Comment : This patient's lung functions show the same mixed pattern of abnormality seen with PMF in the previous cases. He had acute active tuberculosis without cavitation seen on the radiograph.

Case 5.

This 29 year old man had worked in the same enterprise as Cases 2 and 4 for 13 years but had spent only the last three years as a stone sculptor. He presented to us in 1985 on the advice of his colleagues, subsequent to his retrenchment. He was asymptomatic and had pulmonary functions within normal limits : TLC 5600 ml (5960), RV 940 ml (1450), FEV1 3940 ml (3640), FVC 4780 ml (4510), T'LCO 30.2 ml/mmHg/min (32.1), and KCO 5.6 ml/mmHg/min/l (5.4). His chest X-ray showed diffuse nodular opacities with bilateral hilar adenopathy. A transbronchial biopsy was obtained by fibre-optic bronchoscopy. He remained well until 1986 when he presented with a cold abscess of the sternum. The chest radiograph showed some coalescence of nodules and upper lobe bullae (Plate 15). He received a full course of anti-tuberculous therapy. When recalled in 1989 he was asymptomatic, the chest radiograph was unchanged

and the pulmonary function tests were still within normal limits.

Comment : This man acquired simple nodular silicosis with a high profusion in a relatively short period of time. On the basis of this exposure he could be termed as having accelerated silicosis, although his subsequent course is clearly not that of accelerated silicosis. Large opacities developed after he had ended his exposure but these had not become confluent and appeared to have no effect on his lung function. His early withdrawal from exposure probably prevented progression of his disease.

Case 6.

This 46 year old man was detected in 1989 during the health and hygiene survey of a lapidary described below. He had an 18 year history of lapidary work, 8 years of which had been spent as a sculptor with his current employer, the balance of time having been spent in a number of similar establishments in the Eastern Cape. At the first consultation he had symptoms of chronic bronchitis and dyspnoea on moderate exertion. He was a smoker of 10 cigarettes a day. Pulmonary function testing showed : TLC 7645 ml (4690), RV 4060 ml (1460), FEV1 1360 ml before bronchodilator and FEV1 1560 ml (2750) after, FVC 3400 ml (3230), T'LCO 8.9 ml/mmHg/min (28.7) and KCO 1.9

ml/mmHg/min/l (4.8). The chest radiograph showed thickening of the oblique fissure on the right. There was a diffuse parenchymal abnormality with nodularity (type p) of low profusion (1/0).

Comment : This patient illustrates a not uncommon problem in evaluation of workers with suspected occupational lung disease. The airflow obstruction is out of proportion to what might be expected in simple silicosis. The smoking history and his symptoms suggest that smoking is largely responsible for his COAD and that he has co-existent COAD and simple silicosis of low profusion.

PATHOLOGICAL FINDINGS.

Histological assessment of all three available biopsies only showed specific features in case 5. The open lung biopsy in case 1 showed some fibrosis accompanied by accumulations of carbon laden macrophages. Cuboidal metaplasia of cells lining air spaces had occurred and no focal granulomatous lesions were seen. The changes observed could have been the result of silicosis though they were in no way diagnostic.

Similarly, in case 3, the transbronchial biopsy showed non-specific fibrosis. In case 5 the transbronchial biopsy on first attempt revealed normal cartilage and bronchial mucosa. The second attempt was successful and sections showed nodular but non-granulomatous collections of histiocytes associated with fibrosis and scattered lymphocytes as are typically found in silicotic nodules. The histiocytes had abundant eosinophilic cytoplasm. There was no evidence of anthracosis but polarization revealed speckles of refractile material. Iron stains were positive, but no asbestos bodies were present. The fibrosis was variable in extent, some nodules lacking collagen while others showed transformation to a concentric pattern. There were also small foci of necrosis but acid-fast bacilli were not detected. The intervening alveolar walls were unremarkable as were the fragments of bronchial tissue present. There were no significant vascular changes.

In case 3 almost the entire spectrum in the evolution of silicotic nodules was seen. Some of the nodules were 'early' in that they lacked the concentric lamination or 'onion skin' configuration of the mature silicotic nodule. Others were mature and had this configuration. In the prior review of the clinical features and natural history of silicosis it was stated that the finding of a spectrum of early to more mature nodules was possibly a feature of accelerated silicosis. The clinical course of case 5 is certainly not that of accelerated silicosis, indicating that the finding of a spectrum of changes is by no means specific for an accelerated natural history.

PLATE 8

A representative example of the pathological findings showing typical early angiocentric silicotic lesions within the lung parenchyma.

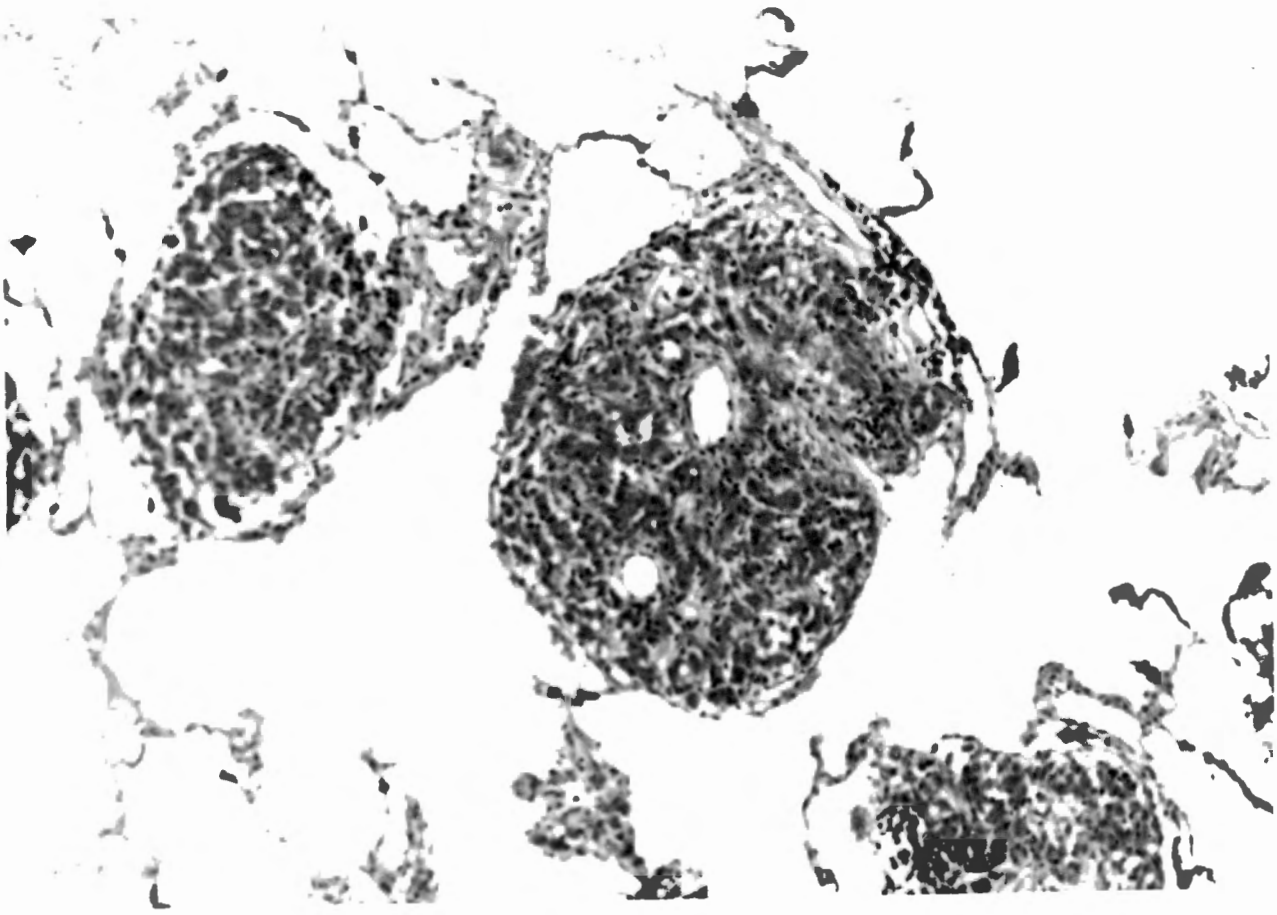


PLATE 9

An example of a more mature silicotic nodule.

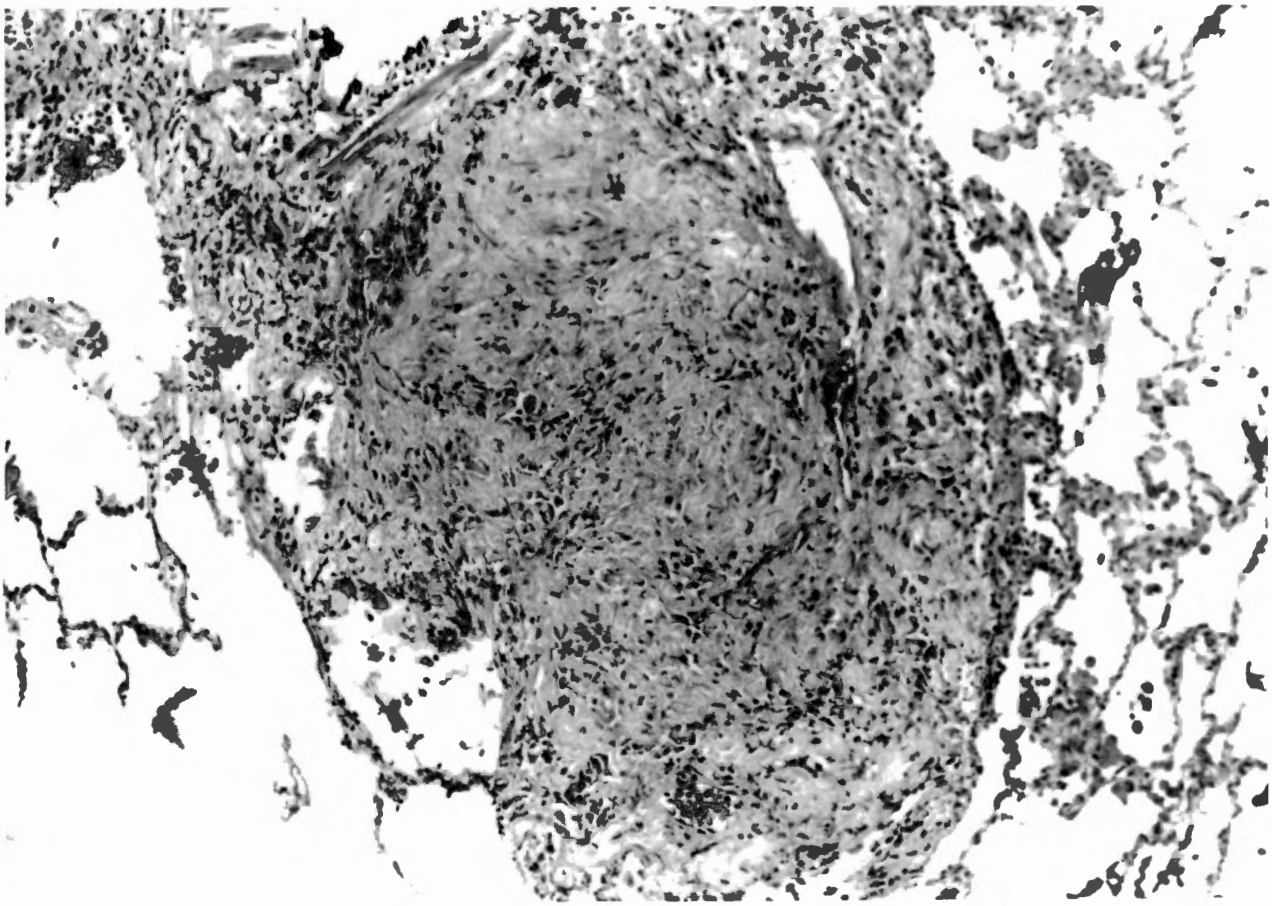


PLATE 10

Radiograph of case 1 : 02/05/76
(see Table 2 for description).

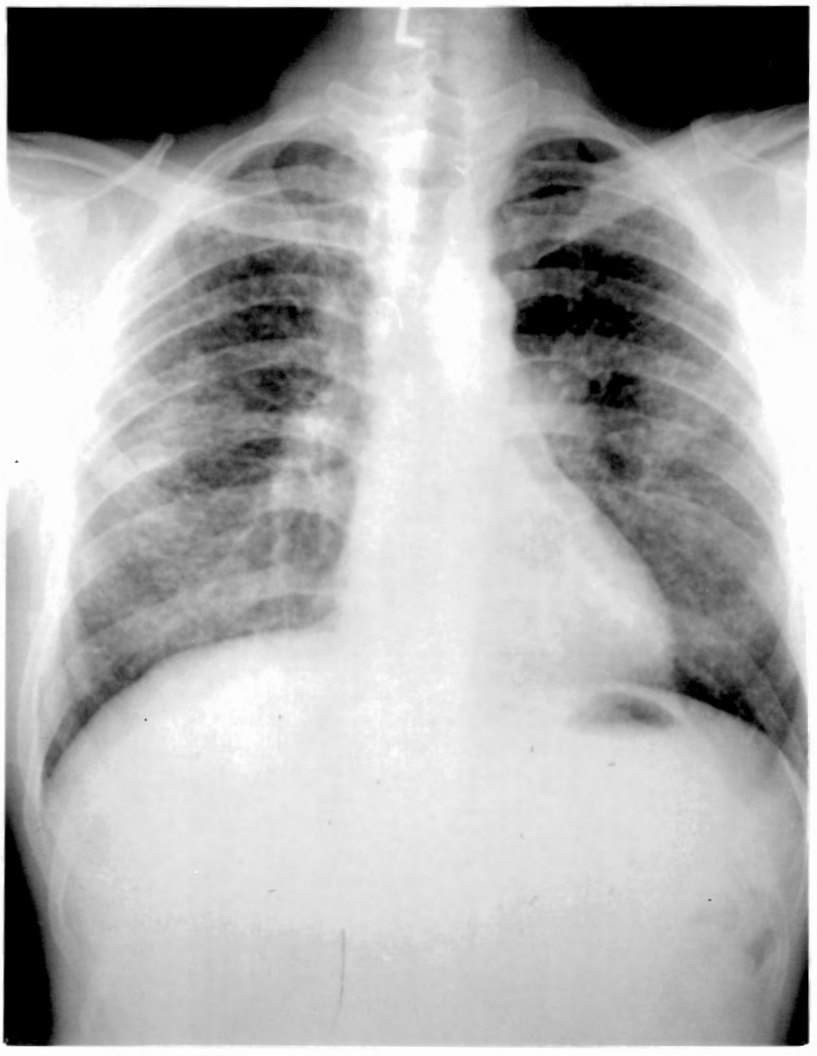


PLATE 11

Radiograph of case 1 : 21/09/78
(see Table 2 for description).

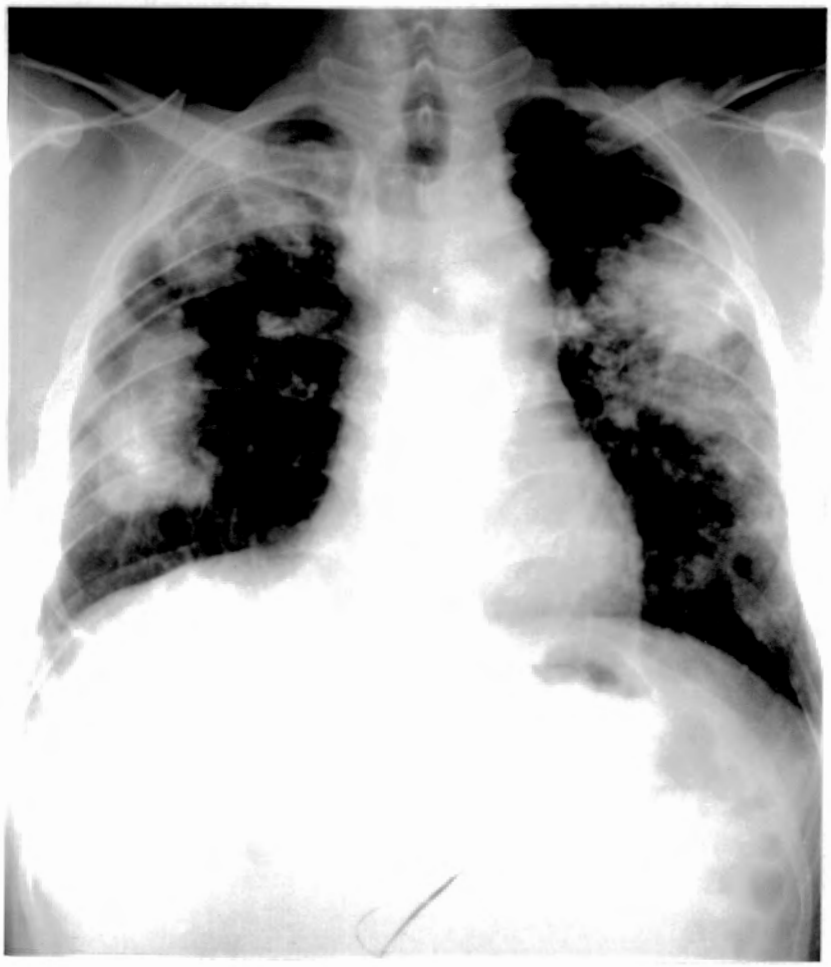


PLATE 12

Radiograph of case 1 : 07/04/83
(see Table 2 for description).

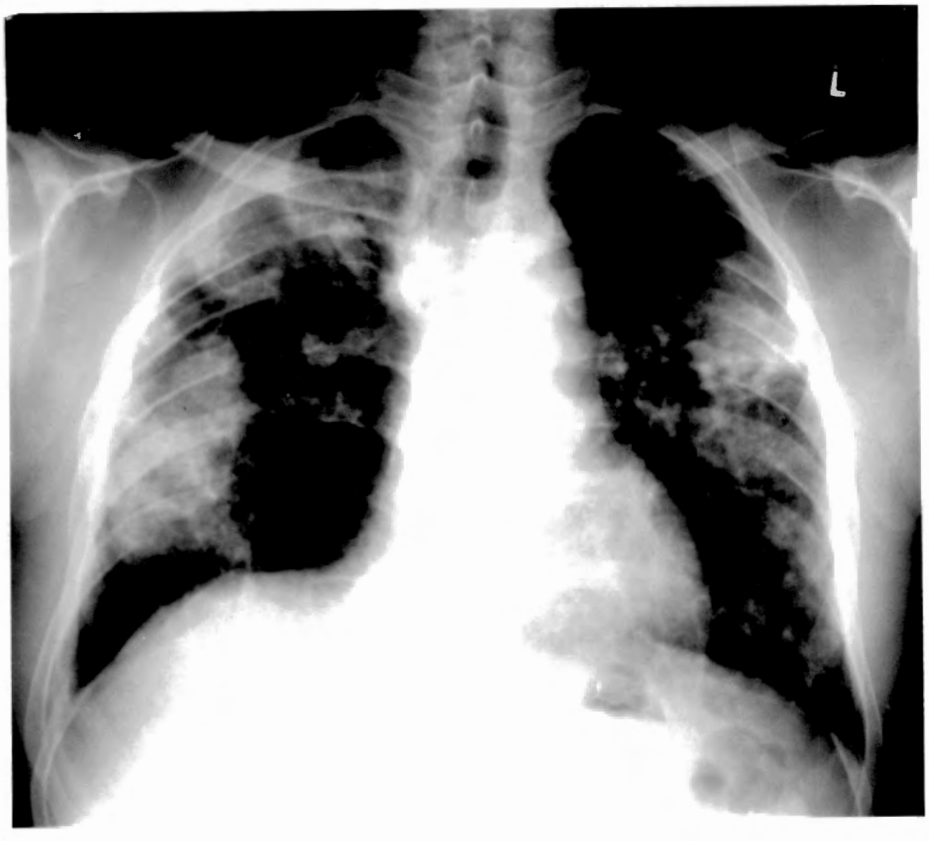


PLATE 13

Radiograph of case 1 : 20/11/89
(see Table 2 for description).

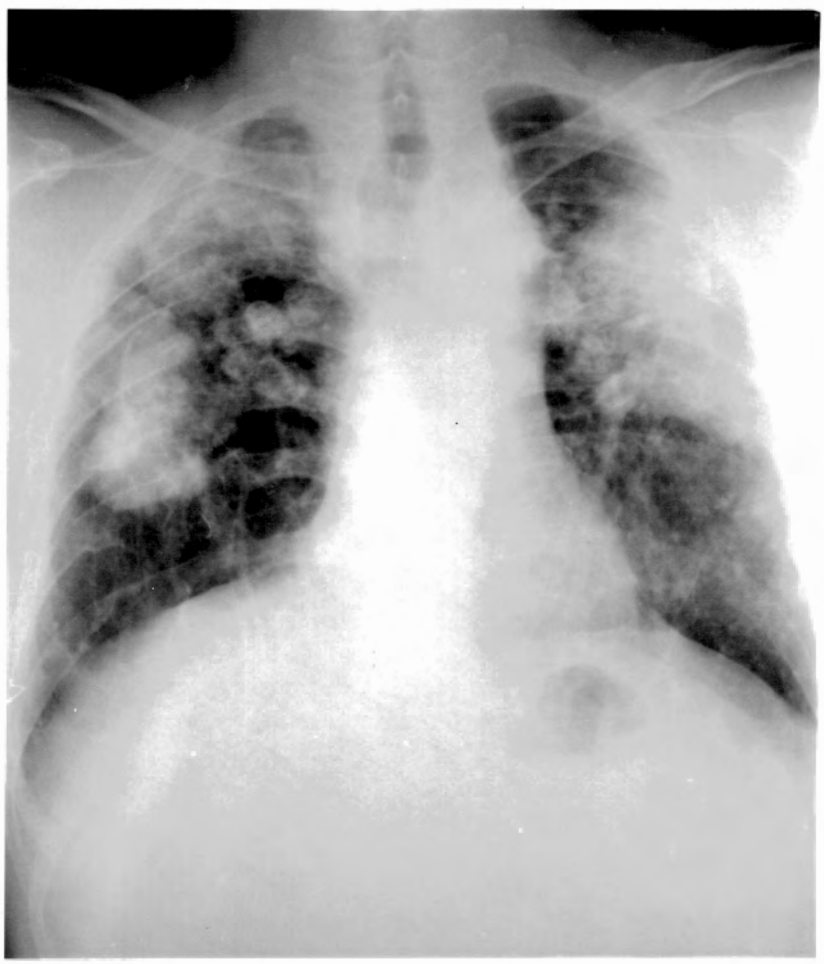


PLATE 14

Radiograph of case 2 : 05/12/85
(see Table 2 for description).

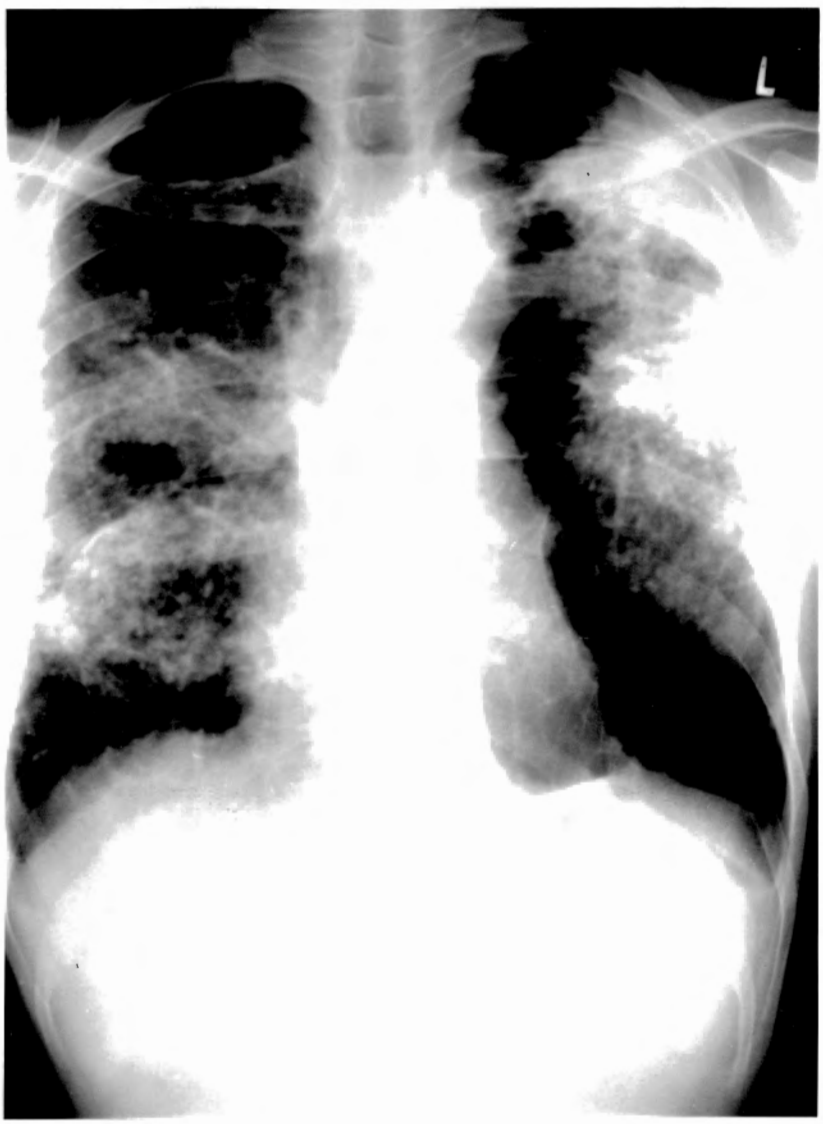


PLATE 15

Radiograph of case 5 : 30/05/86
(see Table 2 for description).

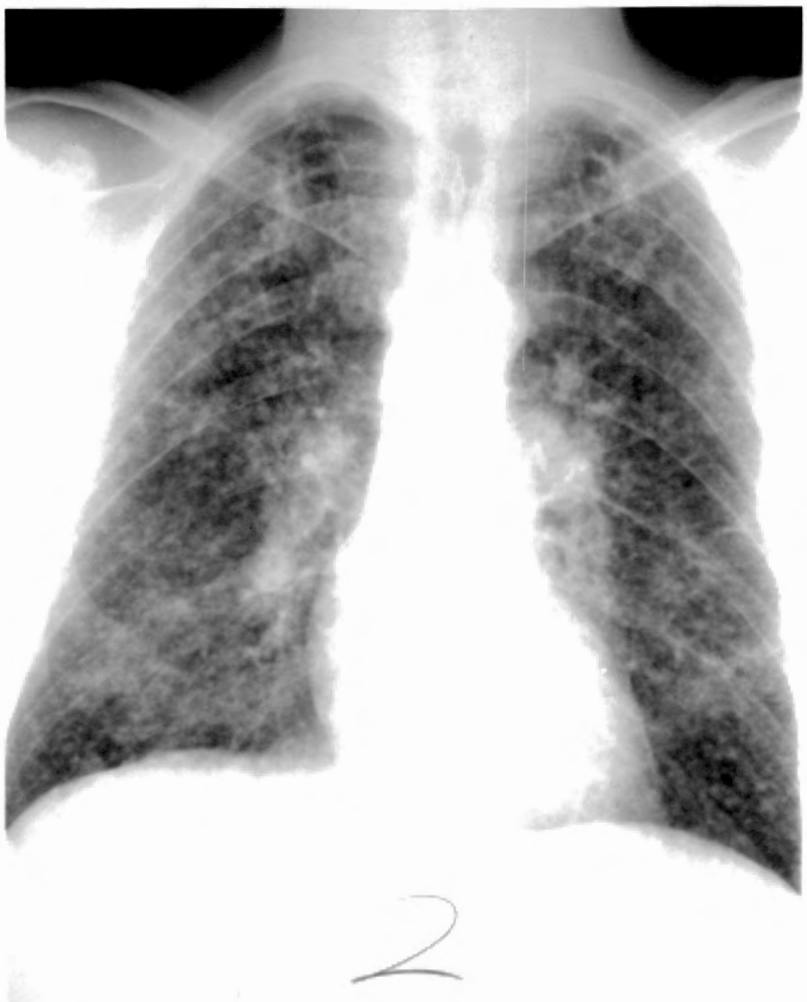


Figure 5 : Serial pulmonary function tests results in case 1.

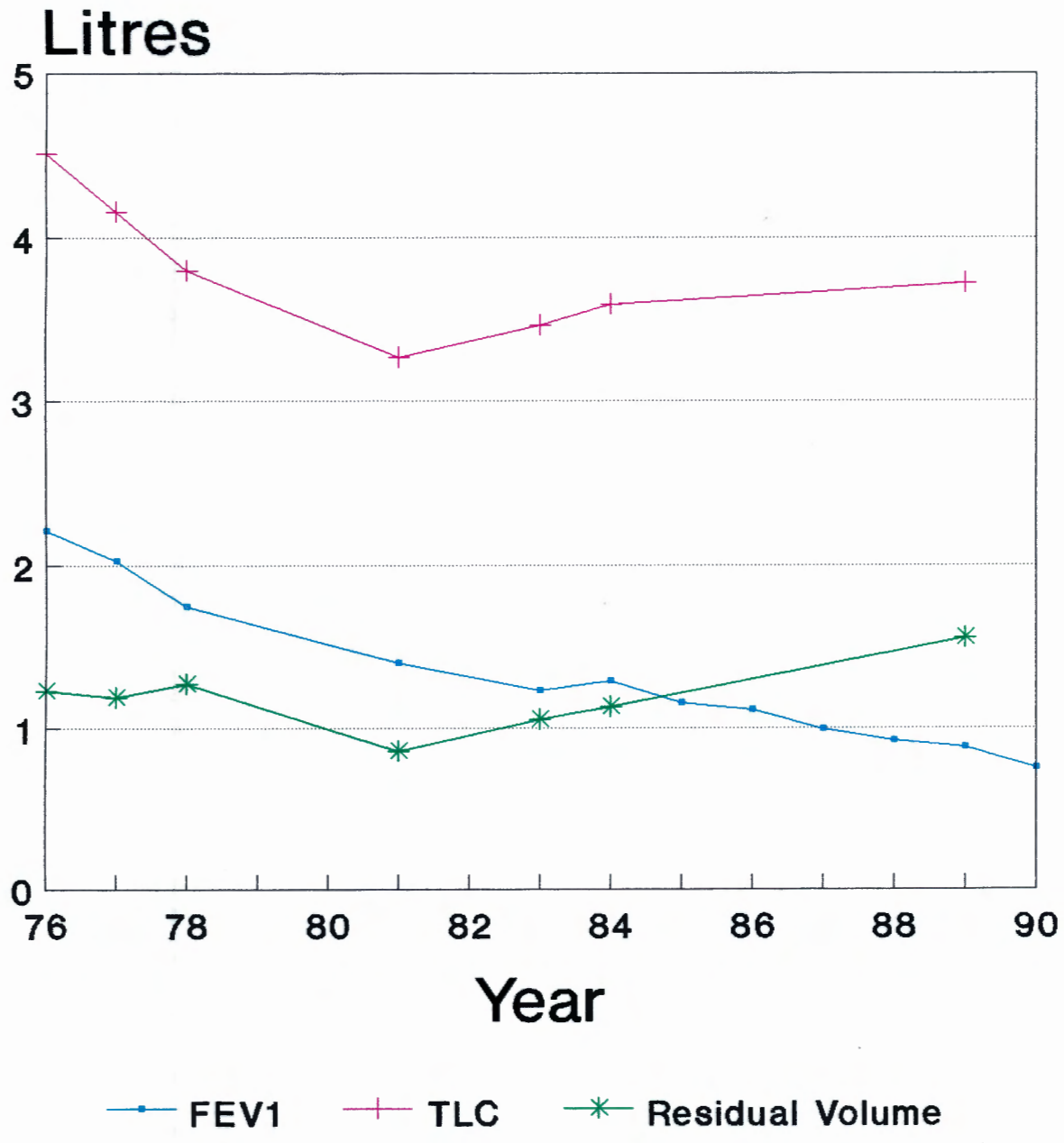


Table IV : Pulmonary function test results : cases 1 and 2.

A : Case 1

Date	FEV1/FVC	FEV1	FVC	TLC	RV	T'LCO
1976	67.3%	2210	3280	4510	1230	10.6
(Predicted)		(3470)	(4380)	(5960)	(1570)	(30.0)
1977	68.3%	2030	2970	4160	1190	
1978	70.0%	1750	2500	3800	1270	
1981	58.1%	1400	2410	3270	860	
1983	50.2%	1230	2450	3460	1050	
1984	52.4%	1290	2460	3590	1130	
1985		1150				
1986		1100				
1987		990				
1988		920				
1989	40.5%	880	2170	3720	1550	14.1
1990		850				
(Predicted)		(3200)	(4220)	(5860)	(1640)	(27.7)

B. Case 2.

Date	FEV1/FVC	FEV1	FVC	TLC	RV	T'LCO
1981	75.8%	2200	2900			
1985	50.2%	1280	2125	3475	1280	14.8
(Predicted)		(3200)	(3900)	(5460)	(1570)	(28.6)
1986	52.3%	1100	2100			
1988	54.9%	790	1440			
1990	49.1%	390	795			
(Predicted)		(3030)	(3770)			

Table V : Description of Radiographs according to ILO Guidelines (1980)

Case No. Date	Quality	Small Opacities			
		Profusion	Shape Size	Extent R	Extent L
1.					
02/05/76	2	2/2	r/r	*	*
21/09/78	2	2/2	u/r	*	*
07/04/83	2	3/3	u/u	*	*
20/11/89	2	2/2	u/u	*	*
.....	.....	.....			
2.					
16/11/81	3	2/2	r/r	*	*
05/12/85	2	2/2	r/r	*	*
.....	.....	.....			
3.					
06/02/79	1	2/2	r/r	o	*
.....	.....	.....			
4.					
07/07/82	2	2/3	u/r	*	*
.....	.....	.....			
5.					
09/02/82	3	3/3	r/r	*	*
30/05/86	2	3/3	r/r	*	*

Table V continued

Large Opacities	Pleural Thickening				
	Chest Wall			Diaphragm	Costophrenic Angle
	Plaque	Diffuse R	Diffuse L		
No	No	No	No	No	No
C	No	b1	No	No	No
C	No	b2	No	No	No
C	No	b2	a1	No	No
.....	.....	.....	.....	.....	.....
No	No	No	a1	No	No
C	No	No	a1	No	No
.....	.....	.....	.....	.....	.....
C	No	a1	No	No	No
.....	.....	.....	.....	.....	.....
C	No	a2	a1	No	No
.....	.....	.....	.....	.....	.....
No	No	No	No	No	No
A	No	No	No	No	No

Table V continued

Pleural Calcific.	Symbols	Comments
No	hi;	
No	ax; di;	
No	ax; bu; es;	Decreased lung size
No	ax; bu; cn; em; es; hi;	
.....	.....	.....
No	ax	
No	bu; em; di; ax;	
.....	.....	.....
No	id; bu; em; ax; px; di;	
.....	.....	.....
No	ax; bu; em;	
.....	.....	.....
No	hi;	
No	bu; hi; ax;	

LAPIDARY HEALTH AND HYGIENE SURVEY.

Background : The protocol for this survey was developed with the objective of documenting current occupational exposures in the lapidary industry and to screen exposed persons for occupational lung diseases. The management of the only lapidary still in production in Cape Town in 1989 was approached directly and agreed to co-operate with our protocol for a health and hygiene survey. When approached, the management of a second lapidary in Hermanus refused to co-operate.

Subjects : Twenty-one persons were employed in the processing of semi-precious gem stones from the natural state to the point where the stones are ready to be mounted in jewelry.

Labour process : The process proceeds from crushing to tumbling and is followed by cutting or ultrasonic drilling or sculpting. In this plant crushing of mineral is carried out by a stamping machine in the open air. Smaller fragments are directed to tumbling and polishing. Tumbling is a wet, enclosed process carried out by abrasive material mixed in with the stones in large rotating drums. After tumbling suitable polished stones are selected for cutting or drilling to prepare them for mounting. Both cutting and drilling were carried out under an enclosure hood with

diesel oil being constantly dripped onto the diamond cutting or drilling edge, serving the purpose of both lubrication and dust suppression. Only two employees in this establishment were engaged in stone sculpting. They would carefully select virgin stones, or fragments produced by crushing. These were then hand shaped against a carborundum disc or belt which had water continuously running over it (Plates 5 - 7). Polished and shaped stones were then forwarded to a make-up department in a separate part of the plant. There was no apparent dust hazard in make-up and it was not evaluated further.

Methods : All 21 employees were evaluated as follows : an ATS Respiratory Symptoms questionnaire (64) was administered by a trained nursing sister; a Vitallograph dry wedge spirometer was used to measure pulmonary function with ATS criteria (65) for reproducibility being applied; and chest radiographs were taken in the Department of Radiology at Groote Schuur Hospital according to ILO specifications for the radiological detection of pneumoconiosis (66). These radiographs were subsequently read by two observers (myself and Prof. Bateman). Any abnormalities on questionnaires, pulmonary function testing or chest radiograph were subsequently evaluated by myself.

Dust exposure was estimated using a personal sampler with an MSA cyclone head. The sampler was calibrated to a flow rate

of 2 l/min. both before and after use and was found to remain accurate. The cyclone was loaded with a matched weight Millipore filter (MAWP 037/pm). Only one sample was taken because of the logistic problems involved (the plant is 35 km from Groote Schuur Hospital). Samples of settled dust from the areas of crushing and sculpting operations were sent to the National Centre for Occupational Health laboratory in Johannesburg. They used an X-ray diffraction method to estimate the free silica content of samples.

Results : The mean age of the 21 employees surveyed was 35.0 years (S.D. 11.33) and their mean length of service was 7.7 years (S.D. 6.53). Two employees, both cigarette smokers, were found to be symptomatic and had significant chronic obstructive airways disease on pulmonary function testing. A further two had chest radiographs suggestive of pulmonary tuberculosis. One of then was direct sputum positive for acid and alcohol fast bacilli (AAFB), the other was direct sputum negative but had a known contact. Both were started on anti-tuberculous therapy. A third had an X-ray appearance suggestive of previous pulmonary tuberculosis but direct sputum was negative for AAFB. He declined further investigation since he felt well.

There were two stone sculptors employed in the plant. One was asymptomatic with normal pulmonary functions and chest radiograph. The other was symptomatic with cough and sputum

production. He had moderately severe airflow obstruction with slight reversibility. The chest radiograph showed hyper-inflated lung fields and small nodular opacities in a profusion of 1/0. This employee is described as case 6 above. The remaining 14 employees had no significant abnormalities on the screening tests used. Therefore there was only one employee with an X-ray appearance suggestive of silicosis.

Using the personal sampler an estimated 830 l of air was sampled over a working shift of the gem stone sculptor described as case 6 above. I was informed that he performed a normal day's work during this time. However, when I arrived the sampler had been turned off and this was contrary to my instructions. The estimation of 830 l is based on the time that I was informed that the sampler was turned off by the foreman. Since I was not able to perform spot checks on the type of work that this man was actually doing during the sampling time, and because of the unauthorised stopping of the sampler I do not have full confidence in the result obtained. Based on this sample the respirable dust level was estimated at less than 0.0001 mg per cubic meter.

The X-Ray diffraction analysis of settled dust by the NCOH indicated that the free silica content was very high - being

in the region of 80 - 90% in all of the samples submitted. No fibres were present in the dust.

Conclusions : It appeared that the local and general measures taken in this establishment to reduce silica exposure were adequate. Although one of the stone sculptors appeared to have developed simple silicosis in low profusion, this was not necessarily related to his current employment. I do not have full confidence in the measurement of dust exposure carried out using the personal sampler for the reasons outlined above. Despite this I concluded that this particular lapidary had taken sufficient measures to reduce the risk of silicosis (67).

DISCUSSION.

The results of this study indicate that lapidary workers are at risk of developing silicosis and, in some instances, PMF. The natural history of those workers who developed PMF was unusual in its severity, being the cause of death in two patients. Although abrasives such as silica flour (1), sand paper (containing flint and quartz) (8) or carborundum (68) have been implicated in pneumoconiosis, the labour process and the analysis of settled dust in this lapidary effectively excludes these as possible causes of these workers' pneumoconiosis. The prevalence of tuberculosis in this workplace (2/21 or 95/1000) was high but the nature of this study did not permit a full evaluation of this. Finally, it appears that there are technical solutions to dust suppression that are effective in reducing silica exposure in high risk occupations in this industry.

Radiological features of progressive massive fibrosis are well known and were amply illustrated in cases 1 to 4. Cases 1 and 2 progressed radiologically from simple silicosis to PMF within two and four years respectively. Cases 2 and 3 progressed to respiratory failure, cor pulmonale and death. Newly-fractured crystalline silica of high purity has long been held responsible for serious forms of pneumoconiosis and predisposition to tuberculosis. The danger of progressive silicosis resulting from intense

exposure to high concentrations of free silica is well known from activities such as sandblasting and tunneling. Cutting and smoothing tools in the monumental masonry industry are known to increase the risk of pneumoconiosis (8). Stone dressing and polishing in the monumental masonry industry is similar in many respects to lapidary work.

Silica dust exposure is known to be associated with restrictive, obstructive and mixed patterns of ventilatory impairment. In gold mines dust exposure may produce airflow obstruction independently of silicosis and cigarette smoking (69). In cases 1 and 2, where we have longitudinal documentation of lung function, there was a pattern of progressive airflow obstruction accompanying PMF. Our knowledge of the longitudinal changes in pulmonary function changes in silicosis is inadequate because it is based on the study of survivor populations and is cross-sectional in nature (46)(47).

In case 1, where we have 14 years of follow-up, we initially observed a mild mixed obstructive/restrictive disorder accompanying the simple silicosis seen radiologically. As progressive massive fibrosis developed there was a worsening of the restrictive disorder to moderate severity with FEV1, FVC, TLC and RV decreasing in a proportional manner over a five year period between 1976 and 1981. Between 1981 and 1989 there was a continued decline in FEV1 ($\pm$ 100 ml/year)

with a proportional increase in RV and the development of severe airflow obstruction. Gas transfer remained in the region of 50% predicted throughout this period.

Our longitudinal measurements of lung function apparently reflect the pathological changes taking place in PMF. The effects of progressive contractural or bullous emphysema, peri-bronchial fibrosis and airway distortion were reflected in the development of severe airflow obstruction and this appeared to be the major factor responsible for worsening impairment. The pulmonary function abnormalities encountered in progressive silicosis must therefore be dynamic and the precise pattern encountered will differ depending on the stage of the disease that measurements are made.

Our findings on the severity of silicosis in lapidary workers contrast with those of gemstone workers in Hong Kong (1). In both situations work was carried out in small, poorly ventilated work-rooms. In the Hong Kong study 27% of 157 workers were found to have radiological pneumoconiosis, defined as opacities with median profusion 1/0 and above. The highest profusion observed was 1/2 and this occurred among gemstone polishers who were using silica flour as an abrasive. Silica flour could contribute to a quartz content of respirable dust exceeding 90% whilst jade and lapis lazuli, the most commonly processed gemstones, are complex

silicates or carbonates and seldom contributed quartz in excess of 5% of respirable dust. As noted above silicates may modulate and ameliorate silicosis. The spectrum of disease documented among workers in Hong Kong was therefore less severe than we have encountered.

In a short report (2) on a lapidary in Lesotho where tiger's eye is processed there were 24 stone cutters with more than one year of exposure. 22 had radiological evidence of pneumoconiosis. This high prevalence again emphasises the dangers involved in processing such pure forms of quartz.

The frequency with which active pulmonary tuberculosis was diagnosed both in our case series and in our lapidary survey is noteworthy. Notwithstanding the moderately high background incidence of tuberculosis in our area the frequency with which the disease occurred in these patients underlines the importance of a very high index of suspicion about this complication.

Our experience with pneumoconiosis among lapidary workers is illustrative of silicosis and a number of enduring basic principles in occupational health. Case 6 had a long history of exposure but relatively mild disease. We attribute this to the fact that he had been protected by the application of basic principles of dust control, namely liberal local use

of water or oil to dampen dust at source, combined with adequate ventilation.

The lapidary industry is illustrative in a general way of the problems attendant on guaranteeing a safe and healthy workplace and providing occupational health services in small industrial enterprises. In this industry only a small minority of a small workforce are exposed to the health hazard. Given an awareness that there is a problem, the employer in a small enterprise would probably seek to 'contract out' the provision of a health service. Such services are usually provided by general practitioners, although there are now some private corporations that will contract to provide primary health care at a factory level. However the majority of small enterprises do not provide any form of health service. One survey on the Witwatersrand (70) indicated that only 18% of industrial enterprises (usually the larger ones) have some form of occupational health service.

There are a number of services that general practitioners can utilise for specialist opinions in occupational health, such as private specialists, the N.C.O.H. clinic in Johannesburg, and the Occupational Health clinic at Groote Schuur Hospital. Support and expert opinion in the field of industrial hygiene is much more difficult to obtain because of the relative scarcity of industrial hygienists.

Problems arise when there is low level of awareness of the possible health effects of industrial hazards and/or an unwillingness or lack of motivation on the part of the employer to do anything about it. Unfortunately as a consequence of ignorance, apathy or frank neglect hazards are overlooked until an unfortunate 'index case' has suffered injury. Prevention of these consequences requires an effective inspectorate of factories. Although the Machinery and Occupational Safety Act (Act 6 of 1983) specifically provides for factory inspections and vests considerable powers in such inspectors, at the present time the Department of Manpower employs insufficient properly trained inspectors for the system to operate effectively. Safety committees established under the Machinery and Occupational Safety Act could conceivably perform the role of an early warning system, but do not appear to do so at present.

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APPENDIX : A summary of the ILO Radiological Classification of the Pneumoconioses.

		Codes	Definitions
Small opacities	Rounded Profusion		Profusion is based on assessment of the concentration of opacities in the affected zones. Standard films define the mid categories.
		0/- 0/0 0/1	Category 0–small rounded opacities absent or less profuse than in category 1.
		1/0 1/1 1/2	Category 1–small rounded opacities definitely present but relatively few in number.
		2/1 2/2 2/3	Category 2–small rounded opacities numerous. The normal lung markings are usually still visible.
		3/2 3/3 3/4	Category 3–small rounded opacities very numerous. The normal lung markings are partly or totally obscured.
	Type	p q r	The nodules are classified according to the approximate diameter of the predominant opacities.
			p–rounded opacities up to about 1.5 mm diameter.
			q–rounded opacities exceeding about 1.4 mm and up to about 3 mm diameter.
			r–rounded opacities exceeding about 3 mm and up to about 10 mm diameter.
	Extent	Lung zones	The zones in which the opacities are seen are recorded. Each lung is divided into thirds–upper, middle, lower zones.
	Irregular Profusion		Profusion is based on assessment of the concentration of opacities in the affected zones. Standard films define the mid categories.
		0/- 0/0 0/1	Category 0–small irregular opacities absent or less profuse than in category 1.
		1/0 1/1 1/2	Category 1–small irregular opacities definitely present but relatively sparse. The normal lung markings are usually visible.
		2/1 2/2 2/3	Category 2–small irregular opacities numerous. The normal lung markings are usually partly obscured.
		3/2 3/3 3/4	Category 3–small irregular opacities very numerous. The normal lung markings are usually totally obscured.
	Type	s t u	The dimensions used for rounded opacities cannot be used, but the irregular opacities can be roughly divided into three types.
			s–fine irregular or linear opacities.
			t–medium irregular opacities.
			u–coarse (blotchy) irregular opacities.
	Extent	Lung zones	The zones in which the opacities are seen are recorded. Each lung is divided into thirds–upper, middle, lower zones.
Large opacities	Size	A B C	Category A–an opacity with greatest diameter between 1 cm and 5 cm, or several such opacities the sum of whose greatest diameters does not exceed 5 cm.
			Category B–one or more opacities larger or more numerous than those in category A, whose combined area does not exceed one-third of the area of the right lung.
			Category C–one or more large opacities whose combined area exceeds one-third of the area of the right lung.
	Type	wd id	As well as 'A', 'B' or 'C', the abbreviation 'wd' or 'id' should be used to indicate whether the opacities are well or ill defined.
Other features	Pleural thickening Costophrenic angle	Right Left	Obliteration of the costophrenic angle is recorded separately from thickening over other sites. Lower limit standard provided.
	Other sites	1 2 3	Grade 0–not present or less than grade 1.
			Grade 1–up to 5 mm thick and not exceeding one half of the projection of one lateral chest wall. Lower limit standard provided.

	Codes	Definitions
		Grade 2–more than 5 mm thick and up to one-half of the projection of one lateral chest wall *or* up to 5 mm thick and exceeding one-half of the projection of one lateral chest wall.
		Grade 3–more than 5 mm thick and extending more than one-half of the projection of one lateral chest wall.
Diaphragm Ill defined	Right Left	The lower limit is one-third of the affected hemidiaphragm. Lower limit standard film provided.
Cardiac outline Ill defined (shagginess)	1 2 3	Grade 0–up to one-third of the length of the left cardiac border or equivalent.
		Grade 1–above one-third and up to two-thirds of the length of the left cardiac border or equivalent.
		Grade 2–above two-thirds and up to the whole length of the left cardiac border or equivalent.
		Grade 3–more than the whole length of the left cardiac border or equivalent.
Pleural calcification Diaphragm Walls Other sites	1 2 3	Grade 0–no pleural calcification seen.
		Grade 1–one or more areas of pleural calcification, the sum of whose greatest diameters does not exceed 2 cm.
		Grade 2–one or more areas of pleural calcification, the sum of whose greatest diameters exceeds 2 cm but not 10 cm.
		Grade 3–one or more areas of pleural calcification, the sum of whose greatest diameters exceeds 10 cm.

Other symbols

Obligatory	Optional	
ca–suspect cancer of lung or pleura.	ax–suspect coalescence of small rounded pneumoconiotic opacities.	hi–marked enlargement of hilar shadows.
co–abnormality of cardiac size or shape.	bu–bullae.	ho–honeycomb lung.
cp–suspect cor pulmonale.	cn–calcification in small parenchymal opacities.	K–Kerley (septal) lines.
es–eggshell calcification of lymph nodes.	cv–cavity.	px–pneumothorax.
tba–opacities suggestive of active tuberculosis.	di–marked distortion of the intra-thoracic organs.	rl–pneumoconiosis modified by rheumatoid process.
od–other significant disease not covered by one of the other obligatory or optional symbols.	em–marked emphysema.	tb–inactive tuberculosis.