EVALUATION OF RESPIRATORY HAZARDS IN THE WORKING ENVIRONMENT THROUGH ENVIRONMENTAL, EPIDEMIOLOGIC AND MEDICAL SURVEYS

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HISTORICAL AND CURRENT CONTEXT

Respiratory disease consequent on work in dusty trades has been recognized since ancient times when man first turned to tools to help him to exploit the riches at the earth's surface. In the past, distinctions have been blurred between various disease processes involved (fibrotic, infectious, malignant), all of which may follow occupational exposures.[1] The term pneumoconiosis was introduced in the 19th century to describe the rather specific nature of the lung's fibrotic reaction to inorganic dusts, such as silica, coal and iron. In keeping with its Greek roots, the term is currently defined by the World Health Organization as the "accumulation of dust in the lung and tissue reactions to its presence."[2] Over the past century, industrialization, the growth of populations, and the increased demands for the raw materials of the earth's crust have led to an increase in the number of workers whose jobs expose them to mineral dusts.

In consequence, the early years of this century saw an increase in the burden of dust diseases in industrialized countries, and post World War II in the newly industrializing countries. There are no global estimates of the number of workers currently at risk; Table I refers to the 1970's[3-5] and is mainly based on information furnished to the International Labour Office by those countries which report on their mining, tunnelling and quarrying operations.[3] The considerable between country differences in rates are no doubt largely due to differences in methods of reporting. Nor is the coverage comprehensive.

Not only the distribution but also the nature of some of the pneumoconioses may be changing. For instance, since the first International Pneumoconiosis Conference held in Johannesburg, in 1930,[6] the profile of diseases such as silicosis appears to have changed, at least in the large controlled industries.[7,8] Whereas in the early decades of this century, these were diseases which disabled young and killed prematurely, they are now increasingly diseases of primarily radiologic manifestation with little morbidity or impact on longevity. Reasons no doubt include improved living standards, better medical care and tuberculosis control in addition to improved environmental controls at the workplace.[8]

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However, outbreaks of acute disease continue to appear, usually in new processes or small uncontrolled industries, even in the technologically advanced countries.[1,8-12] The mid-century epidemic of asbestos-related disease is another example of the failure to apply known control technologies to commercial exploitation, in this instance due perhaps in part to the exigencies of World War II.[13]

The perspective envisaged for the VIIth International Pneumoconiosis Conference as reflected in the themes selected for discussion is considerably broader than that of the First Conference, held in Johannesburg in 1930. The players are also different. Clinical, engineering and industrial hygiene scientists were the major contributors at the First Conference, with the major contributing laboratory sciences being pathology and microbiology. Today all branches of the clinical laboratory sciences are represented, in particular, epidemiology. This is a late comer on this scene and has become increasingly important as it adapted the techniques developed for the study of epidemic infectious disease to the study of chronic noninfectious disease of multifactorial etiology. It is well accepted that environmental and medical surveys can be used to evaluate hazards in the working environment. However, today I wish to indicate how they may be combined using the approaches and methods of epidemiology, and statistics which together offers 3 powerful tools: i) a basis for sampling when numbers to be studied exceed resources; ii) a means of estimating power when sample size is limited (workforces are after all finite) and iii) the methods of analysis which enable the simultaneous consideration of more than one factor in these diseases of multifactorial etiology. The examples chosen to illustrate this presentation are from my own field of endeavour.

ROLE OF EPIDEMIOLOGY

Epidemiology is defined as the study of the "distribution and determinants of health related states or events in specified populations and the application of this study to the control of health problems."[14] I agree with those who argue that it is a discipline rather than a science, i.e., a field of learning or practice applicable to the study of natural phenomena (biological, sociologic or other), rather than a science, i.e., a systematized theoretical body of knowledge about a particular category of natural phenomena.[15] As such, it is a

Table I

Selected Information on Dusty Occupations in Various Countries: Number of Current Workers at Risk, Reported Prevalences of Pneumoconiosis (total cases) and Incidences (new cases each year) per 1000 Exposed Workers

Continent /country	Sources of exposure	Years	Number at risk	Total cases/ 1000	New cases/yr 1000
Europe					
France	mines, pits, quarries	1977	89,391	51.1	5.1
Germany	coal mines,	1977	111,992	228.9	7.5
	other	1977	c.7,000	116.9	2.4
Poland	mines, other	1978	c.90,000	34.4	3.7
UK	coal mines,	1977	252,600	119.1	2.1
	other	1977	5,800	103.4	9.4
America					
US	coal	1973-78	118,579	'20.0	na
Ontario	mining	1970	17,355	na	1.4
Quebec	mining	1967-77	12,556	na	1.8
Mexico*	mining	1973	4,815	8.9	8.9
Peru	not stated	1976	56,819	c.36.0	na
Australia					
NSW	coal	1973-76	15,970	28.8	na
	other	1976-78	4,484	10.6	0.5
Queensland	coal	1968-72	1,387	na	6.4
	other mines	1968-72	3,903	na	8.1
W Australia	mines	1978	6,923	27.0	2.6
India	mines,other	1973-77	c.600,000	c.25.0	
Africa					
Kenya	small mines	1977	3,359	0.0	na
S Africa	hard	1977	19,504	na	11.1
	rock	1977	300,357	na	1.6

Table shows information derived mainly 3 countries reporting to ILO on the number of subjects exposed in dusty occupations as well as pneumoconiosis rates: (ref 3): figures for Ontario, Quebec, and for S Africa were derived from ref 4.

* refers to 1 company only; each company keeps its own statistics

discipline which must be of interest and of use to all participants here today, whatever our branch of science.

Epidemiology can be used to address in populations the same issues which a clinician addresses in the management of a single case: namely, the description and recording of its features (the history, examination and laboratory tests); the explanation contained in the diagnosis and the formulation of prognosis, and in light of the above the planning of the management and the evaluation of its success. Thus population based (epidemiologic) studies may have as their objective, description (prevalence or incidence of disease) and/or explanation (who in a population is affected and why: who is not and why not). These findings can then be used to formulate corrective measures, and once in place, their effectiveness can be evaluated by further studies. The key in the clinical as well as the public health management of occupational disease is how to establish the link between the biologic outcome of interest (abnormality, dysfunction or disease) and the pertinent exposure.

ESSENTIAL ELEMENTS OF AN EPIDEMIOLOGIC SURVEY

These can be summarized in four interrogative adverbs: why (the objectives of the survey), how (its design), who (the target population or workforce(s)) and what is to be studied (referring to the measurements made of dependent and independent study variables).[13] Most important is the first, what McDonald calls the "fundamental ingredient of any scientific endeavour," namely, "an obtainable objective or answerable question ... clearly and unambiguously defined."[16] He also recommends that a subsidiary question be asked: "and what will I do with the answer?" Thus an epidemiologic study is neither "a data gathering exercise with a nebulously defined purpose and no hypothesis to test;" nor is it a study "which misses a truth because it is buried in a mass of data." These are both popular misconceptions which relate to the false belief that the key characteristic in epidemiological study is that it is based on large numbers of subjects.[17] Indeed some of the most effective epidemiologic studies are very economical in this regard.

Design

At the heart of the scientific method is the experimental design. In its complete form it requires that the researcher have control of all aspects of the study including the option of testing the entire target population (or sampling at random from it); control of the assignment of test units to intervention (exposure) or not, as well as the opportunity to examine all test units before and after the intervention with no loss to follow-up.[16] When study units are cells, or plants or animals, this is possible; when the subjects are human, and exposure the result of natural experiment, this is rarely so. Indeed, the definition of a survey (the word used in the title of this presentation) is "an investigation in which information is systematically collected but in which the experimental method is not used."[14]

Other than randomized control trial, for instance, of tuberculosis drug therapy, most occupational health surveys must of necessity use a less-than-complete experimental design. While the strongest designs include measurements before and after exposure (i.e., are longitudinal or cohort in concept), prevalence (i.e., cross-sectional) designs are often all that is feasible, and are most frequently used for chronic non-malignant diseases such as pneumoconiosis whose onset is difficult to pinpoint. Indeed, the prevalence study has been not inappropriately dubbed the "workhorse" of chronic disease epidemiology.[17]

By contrast, the case control design is an elaboration of the traditional clinical case series, in which clinical case experience is described without reference to the population from which they were derived. The case control study also starts with identification cases of the disease under study; persons without the disease (controls) are then selected from as far, as can be determined, the same population as generated the cases, and the past of cases and controls are compared for evidence of exposure. Hybrid designs, using the case-control approach within a cohort, have been creatively exploited in establishing relationships between occupational exposure and malignant diseases,[18] and they are now increasingly being used in the study of non-malignant diseases such as the pneumoconioses.[17] Nor does the case series study necessarily merit the scorn often accorded it by editors and reviewers: it was after all such a clinical case series reported by a missionary doctor, the surgeon to whom his cases were referred, and the pathologist on the surgical pathology service which first drew the attention of the medical community to the link between mesothelioma and asbestos exposure.[19] Indeed, it has been pointed out that shrewd clinical observation remains the most powerful tool in detecting new disease patterns linked to workplace exposure,[20] also in identifying recognized disease patterns in workplaces or associated with exposures not previously thought to be at risk.[17]

Dose Response Relationships

Dose response relationships form the scientific basis of pharmacology (which deals with desired responses) and toxicology (which deals with undesired responses). In both, dose refers to the amount of the agent delivered to the target organ and retained for a period of time sufficient to evoke a response. In occupational surveys of chronic diseases like pneumoconiosis and chronic obstructive pulmonary disease, dose-response relationships are important in establishing causality.[16,17,21] However, estimates of exposure have until recently, been the only available indicator of dose; obviously a very poor substitute given the low deposition rates and highly efficient clearance of so much of what we breathe in. What is surprising, given the impossible task of represent ing exposure over a working lifetime accurately, is that exposure-response relationships are usually demonstrable in workplace surveys even using quite simple indicators of exposure.

The development of new methods, such as the quantitative measurement of lung dust residue represent a quantum advance in the study of the dose variable and these have already contributed to our understanding of why exposure response relationships differ between workforces. For instance, there is now evidence in support of mass rather than fiber number being the determinant of fibrosis scores for asbestosis.[22] This topic is rightly one of the key themes of this Conference.

New technologies of this sort to obtain the most precise estimates of dose possible may not however always be available, and the value of what is surely the simplest estimate of exposure, the worker's personal assessment, should not be overlooked. Thus several recent community-based studies have shown clear evidence of association between indicators of chronic obstructive pulmonary disease (COPD) such as FEV_1 and occupational exposure to dusts at work, evaluated subjectively by study participants.[23,24] Subjective estimates of personal exposure have also proved as useful as objective dust exposure measurements in demonstrating exposure response relationships in workforce based studies, an observation of relevance in situations where resources for objective environmental control measurements do not exist, for instance in certain industrializing countries.

Modelling Exposure Profiles

Whether or not lung responses are influenced by exposure profiles (such as the occurrence of peaks or gaps in exposure versus steady level exposure) remains a matter of concern, with implications for setting control levels. However it is not an easy matter to investigate. One approach is to use mathematical modelling based on biologically plausible models.[26,27] For instance, in Quebec asbestos miners, temporal patterns of exposure appeared to influence the different respiratory responses;[27] thus for asbestosis, the strongest predictor was cumulative exposure; for pleural change exposure, peaks and residence time of dust in the lung; for airway reactivity, both with early and recent exposure, and for airflow limitation and bronchitis, dust level and dust load over time as well as smoking.

MEASUREMENT TOOLS OLD AND NEW: APPLICATIONS AND EXAMPLES

The effect of measurement error, whether of exposure or response, is attenuation of exposure response relationships. This has led to concerted efforts to improve standardization and reduce measurement error. In the case of the chest radiograph, the traditional health measurement tool in pneumoconiosis surveys, the ILO has taken the lead in standardizing techniques of film reading.[28] Subsequently, respiratory questionnaires and lung function tests have been included in most workplace surveys,[1,17] originally in support of the diagnosis of pneumoconiosis, but subsequently as outcome measurements in their own right to characterize among other things airway function and standardization procedures for their use in surveys has been developed by various professional bodies.[29,30] Despite its modest status (it is cheap and despised by clinicians as inaccurate), the respiratory questionnaire has proved a surprising but powerful measurement tool. For instance, exposure-response relationships for the complaint of shortness of breath when hurrying on the flat are readily demonstrated in asbestos exposed workers, consistent with the clinical conviction that shortness of breath is an early, characteristic and essential feature of asbestosis.[8,13] Recently there has been a resurgence of interest in this and other symptoms such as wheezing as response variables coinciding with the increasing appreciation of the fact that acute and chronic airway responses occur following a wide range of occupational exposures.[31]

Pulmonary Function Tests

Obsession with the importance of reproducibility of lung function tests for epidemiology studies often led researchers to exclude subjects whose results failed to meet specified criteria for acceptability.[29] A careful analysis by one research group of subsequent health experience in subjects with and without test failure brought to light a very interesting source of bias, namely, that test failure in itself carries a greater chance of a less unfavourable outcome.[32] This observation has now been confirmed in several cohorts and the underlying mechanism(s) are under investigation.

The "healthy worker effect" is a term originally coined to describe the lower mortality experience of employed workers compared to the general population,[14] presumably due to their better than average health status. There may be a similar explanation for the better than average lung function often seen in workers engaged in physically demanding jobs. For instance, in a survey of Paris workers employed in a number of plants, younger workers with pollutant exposure had consistently better (not worse) values for FEV than those whose jobs did not involve exposure; in older workers the situation was reversed.[33] Nor is this experience unique.[34] It is also biologically plausible: dusty jobs are traditionally heavy jobs likely to attract those of above average performance. This potential source of bias has implications for analysis as well as for interpretation, and suggests that cross-sectional studies of older workers are likely to underestimate exposure effects on lung function even when external reference values are used to take account of confounders.[35]

Complex Health Measurements as Tools in Epidemiologic Studies

The laboratory measurements now available to characterize pulmonary abnormality, dysfunction and disease are remarkable for their variety and precision, but also for their complexity and cost and their optimal integration into research into pneumoconiosis and other diseases of occupation can be challenging. This is often possible through the use of hybrid study designs, such as case-control within a cohort or within a prevalence study. This allows the target population to be described by low-technology measurements (e.g., questionnaire, job, and if necessary lung function or x-ray), and within this framework, stratification by exposure, or response, or both can be done prior to sampling. In this way it is possible to address well formulated objectives by comparison of selected but small groups of subjects using high technology tools. Note the population description should respect basic epidemiologic principles including a complete definition of the target population with an assessment of selection bias into and out of the workforce (respectively the "healthy worker" effect and the "survivor" effect). For example, it was possible to use questionnaire, x-ray and lung function data gathered in a cross-sectional study of the Quebec asbestos miners and millers[36] to select smaller subsets of subjects in whom further measurements were carried out to address additional questions on the early effects of exposure,[37] and whether lung geometry was a risk factor for the development of asbestosis.[38]

UNRESOLVED ISSUES, FUTURE RESEARCH AND DIALOGUE

A conference like this brings to light many unresolved issues, and perceptions vary as to their importance. One which deserves careful scrutiny is how best to evaluate the effectiveness of current pneunoconiosis control measures including health surveillance and environmental control levels. Most current survey research is descriptive (for instance, health hazard identification or evaluation) or etiologic (examining exposure response relationships), little is evaluative (determining the effectiveness of controls). Despite the probably billions of chest radiographs, and the probably millions of spirometric test records carried out in health surveillance programs, it is still not clear whether medical surveillance and/or current environmental control levels for silica[12] and asbestos[35] if respected, do indeed protect human health.

A second and related question concerns the links between pneumoconiosis and tuberculosis, an issue of great importance in those countries of Africa and Asia with both high tuberculosis infection rates, and extensive mining operations.[4] Under such circumstances, mine medical services may be responsible for extensive surveillance and treatment programs which could provide the framework for important research. For instance, a recently completed study in goldminers in the Orange Free State evaluated several short tuberculosis treatment regimens, and in a subset of the data showed that continued mining exposure while on treatment did not affect the outcome unfavourably.[39] This important finding went contrary to the current practice which precluded miners on treatment for tuberculosis from further underground service, in the belief that continued silica dust exposure diminished the chance of treatment success. Nor was outcome unfavourably influenced by the presence of silicosis. As a result of these findings, regulations now permit miners to continue in underground service, while under treatment, without loss of income, an important consideration in a largely migrant and rurally based workforce.

A third issue is how better to exploit the many existing data banks (including case registries and health surveillance data) for research and health control purposes. For instance, the Swedish silicosis case registry,[40] set up in 1933, has been used to study i) progression (shown to be greater if cases continued in a job with exposure after the earliest radiologic manifestation); ii) the relationship to lung cancer (silicosis cases have a greater risk than non-cases); iii) tuberculosis rates (still a frequent complication in cases of silicosis, even after the introduction of drug therapy in 1951). The PATHAUT data file, another registry containing machine readable autopsy reports on some 33,000 South African miners,[41] has also been used as a data base for a case control study which showed hard rock mining to be a risk factor for emphysema.[41] Other uses of case registries will be reported at this meeting.

Finally there is the issue of dialogue, within and between disciplines, within and between researchers, and within and between professionals. Each of us tends to believe the other is ignorant of what we have to offer. Dialogue is less difficult in the context of a conference such as this, when participants are free of daily tasks; dialogue is also less difficult perhaps in institutes dedicated to a common theme "Dialogue" should also include user-responsiveness: those who are in the workplace on a daily basis are often the first to perceive the unexplained or the unexpected and yet their comments are often not sought or heard. Finally, research into the diseases of occupations (whether it be basic laboratory research, cellular biology, environmental or clinical research) should always and only be driven by hypotheses which have biologic credibility as well as user plausibility, in the context of good study design. In addition, if there is a sound answer to Dr. McDonald's question: "and what will I do with the information" before starting a survey, then the survey is likely to be one which will furnish a useful evaluation of respiratory hazards in the working environment.

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PROGRESS IN ETIOPATHOGENESIS OF RESPIRATORY DISORDERS DUE TO OCCUPATIONAL EXPOSURES TO MINERAL AND ORGANIC DUSTS

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INTRODUCTION

Thank you for giving me the honour of introducing the theme on progress of Etiopathogenesis of Respiratory Disorders due to Occupational Exposures to Mineral and Organic Dusts.

In the first place, I would like to say how pleased I am to follow Margaret Becklake in the setting of these themes. We both did our Graduate and Postgrauate training in Johannesburg which was the scene of the first of these conferences in 1930. Of course, we were both too young to attend.

Secondly, I have been given a vast field to cover in a very short time. I will paint with a very thick brush on a large canvas. I will concentrate on my personal experiences and views to offer a provocative base to the further sessions of this conference. I trust this will stir up sufficient controversy to satisfy our sponsors.

I note that my remit covers both mineral and organic dusts. On organic dusts my experience is brief—I do not believe that there is such a disease as byssinosis. The biological effects of cotton dust are part of the vast new field of study covered by the term "Industrial Asthmas," which is now a separate field from pneumoconiosis.

SILICOSIS

There are two facets of silicosis research in which we have developed new ideas since the 1930 conference. One is positive and requires explanation, the other I feel is a false lead which requires most serious scrutiny.

Although historical evidence goes back to neolithic times, it was in the industrial revolution that it was realized that exposure to mineral dust could have fatal consequences. By 1912 South African workers had shown that quartz was responsible for these lesions and by the Johannesburg conference it was proved that tuberculosis was the main killer of silicotics. The problem is how does silica do the damage and why is there this promotion of tuberculosis? My views are now considered simplistic and I hope that at a later stage of this conference someone will produce a more scientific hypothesis. I believe that silicosis is a disease of the monocyte macrophage system and the destruction of numerous macrophages by the inhaled quartz crystals produces a local milieu promoting infection from the mycobacteria when they are present. As far as I will go in explaining this are the studies of Tony Allison and Jack Harington. Briefly the quartz crystal is taken up by the macrophage forming phagosomes with the relevant lyzosomes which are released, but fail to digest either the quartz crystal or the wax coat surrounding the tubercle bacillus. The quartz crystal then by some means disrupts the membrane of the phagosome releasing the "enzyme soup" which destroys the macrophage: the unscathed quartz crystal is freed to destroy further macrophages and the bacillus to reproduce.

The other facet which disturbs me is the suggestion that quartz is an important carcinogen. We have as pathologists studied numerous cases of silicosis and exposed a vast number of animals to quartz dust. In all the human cases I know of where carcinoma does occur in silicotics it is either associated with cigarette smoking or much more rarely with radon release. In the experimental evidence only two series of experiments are quoted in which malignancy occurs. I am responsible for one of these studies and our results have been incorrectly interpreted. In 1960 I inoculated quartz into the pleural cavity of Wistar rats. Some of these rats subsequently died of tumour which were not mesotheliomas. In 1962 the experiment was repeated with two further strains of Wistar rats in which a much higher incidence of these tumours occurred. These tumours were subsequently studied by my wife who showed that these tumours were in fact histiocytic lymphomas of macrophage origin. She was unable to produce a significant number of these tumours by using different routes of exposure. It is unlikely in human exposure that silica would reach the pleural cavity.

The other study was carried out by Dave Smith at Los Alamos where Fischer rats were exposed to very heavy clouds of quartz and developed severe pulmonary fibrosis. Some of these animals subsequently died of peripheral carcinomata. These peripheral tumours occur in the animals with severe pulmonary fibrosis and these lesions are not specific for silica exposure.

COAL WORKERS PNEUMOCONIOSIS

When I first became involved in the study of coal workers pneumoconiosis I was informed that the disease could be divided into simple and complicated forms. The simple form did not cause disability; the complicated form did because

of the production of massive pulmonary lesions consisting of vast chunks of fibrous tissue. All these facts have been disproved. The majority of coalworkers do not develop any pathological change apart from having excessive coal dust blackening their lungs. About 10% of these then develop pulmonary nodulation, so at this stage the disease becomes "complicated," and with further exposure, these nodules tend to form vast coalescent masses if exposure is sufficient. The main disease in these men is not the nodulation *per se*, but the associated emphysema and interstitial fibrosis in some cases. In the massive lesions, the lumps are not fibrous tissue. In fact, the amount of collagen and pre-collagen amino-acids present in them is the same as in the non-involved lung tissues. Working with Dr. F. Wusterman of the Biochemistry Department of the University College of Wales in Cardiff and Professor P. McGee at Oxford, we were able to show that the main constituent of these lesions is fibronectin, a glycoprotein which occurs as 3% of the normal serum proteins.

ASBESTOS AND ASSOCIATED DISEASES

There are as we all know, a group of fibrous minerals that can be split longitudinally and have commercial uses. These are chrysotile, crocidolite, amosite, tremolite / actinolite and anthophyllite. The term "asbestos" was originally used for chrysotile. If this had been maintained and the other materials referred to as the amphibole fibres, the present confusion in assessing the risk hazard would not have occurred. In the amphiboles the risk hazard depends on the ultimate length diameter ratio of the fibre and this has been clarified with the studies of the biological effects of tremolite, an amphibole with a widespread occurrence in the earth's crust, usually as a contaminant of chrysotile, talc, anthophyllite, and other minerals. It also occurs in small deposits and is frequently used all over the world as a soil conditioner in agriculture.

The physical features of tremolite vary in all forms from thick flakes to very fine fibres. The electron microscopic appearance of some fibres is shown. Under the transmission electron microscope it can be seen that the finest and straightest of the fibres is crocidolite followed by amosite and the coarse anthophyllite. Now tremolite covers this whole spectrum. By far the finest of all fibres are chrysotile fibres particularly when they break up into fibrils, one chrysotile fibre having the equivalent diameter of at least 100 chrysotile fibrils. However, due to the coiled wave-like configuration the aerodynamic efficiency of chrysotile depends upon that of the full coil. Before venturing into an account of the biological effects of these different fibres, it is necessary to state the hypothesis of selective retention of fibres in the lungs. This contends that it is the fibres retained in the lung parenchyma which are significant in the causation of the disease.

Now I will briefly state our belief in the correlation of disease with fibre type. I am sure this will be contended and defended during this conference.

Asbestos Bodies

Asbestos bodies develop around amphibole and other straight mineral fibres and are seldom on chrysotile fibres.

Pleural Plaques

All types of asbestos are associated with development of pleural plaques particularly tremolite, amosite and anthophyllite. The incidence of environmental plaques is extremely high in agricultural situations and these are usually associated with tremolite.

Asbestosis

All forms of asbestos dust if inhaled in excessive quantities will cause asbestosis.

Carcinoma of the Lung

Initially carcinoma of the lung occurred in people with severe asbestosis with long term survival. Since the 1950's the incidence of carcinoma of the lung has greatly increased due to the association with cigarette smoking. We still contend, and will present supporting evidence, that the association is between cases of definite asbestosis and carcinoma.

Diffuse Pleural Mesotheliomas

Diffuse pleural mesotheliomas are associated with exposure to crocidolite, very fine tremolite, very fine amosite; and if associated with pure chrysotile this must be an extremely rare occurrence. These associations have been occupational, para-occupational or familial.

In 30% of cases of diffuse mesotheliomas in adults, there is no evidence of an association with actual asbestos exposure as defined above. The amount of asbestos in the lungs of these cases is similar to that seen in the the general population living in the same environment.

Diffuse Peritoneal Mesotheliomas

These tumours are not as common as those originating in the pleural cavity.

Experimental Mesotheliomas

We have produced these tumours by the intrapleural inoculation of various types of asbestos dust, including chrysotile. In the majority of the chrysotiles used there was tremolite contamination. The exception to this was the chrysotile that gave the highest rate of experimental tumours. This was a specially prepared preparation containing numerous long straight fibrils and the actual dosage was at least one thousand million times greater than occurs in human exposures. When we used this dust in an inhalation study the tumour rate was similar to that seen in the controls.

Significance of Fibre Body Burden

Chrysotile

Chrysotile fibres are difficult to count as they tend to form clumps, and fibres break up into a myriad of fibrils, so that amphibole fibre is equivalent to about 100 chrysotile fibrils. The present opinion is that exposure to chrysotile has a much milder effect than the amphiboles, and that the association with mesotheliomas is minimal.

Amphiboles

The total amphibole count, a mixture of fibre types, with different length and diameter, can be used in the assessment

of effect, taking 5×10^6 fibres per gram dried weight of lung as the absolute upper limit of non-occupational exposure.

In significant asbestosis there are 100×10^6 fibres and in severe asbestosis 1000×10^6 fibres.

Significant Fibre Size

Mesotheliomas Diameter <0.25 μm, length >8.0 μm
Pulmonary Fibrosis Diameter <3.0 μm, length >8.0 μm

Diffuse Mesotheliomas

Crocidolite—1 million fibres probably minimal but there have been familial cases with counts of 500,000.

Other Amphibole Fibres

Again, only fibres in the size range of less than 0.25 μm and greater than 8.0 μm in length are regarded as significant. The tremolite and amosite are probably equivalent to crocidolite.

It must be borne in mind that these studies are in a developmental stage and the criteria recorded above are those of our present state of knowledge. Further modifications will be reported as the studies continue.

The number of fibres recorded as millions per gram weight of dried tissue depend on the technique developed by Fred Pooley in Wales in collaboration with Patrick Sebastien in France. There have been modifications of their methods which I understand can be made comparable in some circumstances.

MAN-MADE MINERAL FIBRES (VITREOUS)

I have used the above title deliberately as we have only undertaken extensive studies on samples of rockwool, slag wool, glass wool and sub-micronic glass fibre. I am not in the position to report on detailed studies of the other synthetic fibres such as the ceramic fibres; but hope that later in the conference others will give reports.

In our extensive studies with fibres given to us by both European and American industries, we were only able to produce significant tumour incidents following the intrapleural inoculation of the sub-micron glass fibre. No increased incidence of tumours or significant fibrosis was seen following inhalation experiments.

It should be recorded that in the numerous specimens of lung extracts from tumours that Professor Pooley has studied, only a handful have contained commercially prepared man-made mineral fibre. If the material does not get retained in the lung it is unlikely to cause disease.

ABSORBENT CLAYS

These clays are part of the palygorskite group and are used for cat litter and containing spills on factory floors. Another use is in the preparation of drilling mud for the oil industry. Our detailed studies have been confined to the attapulgite and sepiolite produced in Spain. In our experimental studies only fibres from a small deposit in western Spain were shown to be of a length/diameter ratio to be regarded with suspicion. These fibres produce mesothelioma following intrapleural inoculation into rats. On our advice, the production of this fibre has been discontinued. Other attapulgite fibres and sepiolite fibres did not produce tumours following both intrapleural and inhalation studies. Later in this conference Dr. Kathryn McConnochie will report on a clinical and radiological study of the workers who produce the sepiolite.

ERIONITE

The most fascinating new development in the fibre studies are those on erionite. We all know of Professor Baris' fascinating studies in which erionite was shown to produce a higher incidence of mesotheliomas than any other fibre.

From our experimental studies we obtained fibres from one of the houses in Karain and also from other sources in Oregon State and following intrapleural inoculation it was shown that the sample from Oregon produced 100% tumours and only a slightly lower rate was found in the dust from Karain although it had a lower fibre content.

In inhalation studies the Oregon fibre produced mesotheliomas in 27 out of the 28 animals exposed, 1 animal dying of leukaemia. In repeated experiments tumours rose to 100%.

In comparison to this in our much larger experiments in which animals inhaled asbestos dust, we were only able to produce a very low incidence of mesotheliomas.

Therefore, as I retire from this field, I leave you with a fibre which is a very potent carcinogen and must be of value in unraveling the mineral fibre mesothelioma mystery.

.....oOo.....

ADIEU!

PROGRESS IN PREVENTION: EARLY DIAGNOSIS AND MEDICAL CONTROL OF OCCUPATIONAL LUNG DISEASE

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I would like to touch the old—nevertheless very interesting—history of dust-related lung diseases very briefly. I will concentrate more on later results and on data available for further research and strategies for dust-exposed persons, especially miners.

PARACELSUS already mentioned the miners' disease, and he called it consumption of miners ("Bergsucht"). In this term, the relationship to tuberculosis is obvious. At this time and till the early fifties of this century, silico-tuberculosis was one of the main problems of complications of miners' dust-related lung disease. This is more or less history but not in all parts of the world.

RAMAZZINI of Padua (1780) described bakers' asthma for the first time which was caused by the organic flour dust as an asthma-like disease.

The term "pneumoconiosis" was introduced by ZENKER (1867) for the first time, and at this time pathologists showed us all the changes of the structures in the lungs caused by dust, mainly by quartz and coal mine dust. In Germany, mostly the term "silicosis" was used for "coal workers' pneumoconiosis" as it is called in English speaking countries. At present, coal workers' pneumoconiosis is still the most important dust-related disease from the sociomedical point of view. Many of us may remember the tremendous basic contribution given by pathologists and some may remember that for physicians' better understanding of this disease radiology was the key to a new era. These different pictures led to different X-ray classifications. The first internationally used classification was that of Johannesburg (1928) followed by the classification of the International Labour Office (ILO 1980/81). With one set of standard films edited by ILO we have an instrument world-wide available to control the development of pneumoconiosis by the X-rays and for comparative studies.

We leave history now and we move on to the present time.

The development during the last 30 years has shown tremendous progress not only on behalf of our knowledge. Our improved understanding of this disease "coal workers' pneumoconiosis" and very similarly of pneumoconiosis caused by organic dusts like bakers' asthma have had important progress for the expectation of life as well as for the quality of life of dust-exposed and disabled persons due to exposure to harmful dust.

The development of new methods in basic research work was followed by much better insights in the etiology and pathogenesis of these diseases. At the same time, new drugs, very efficient drugs, were developed which could not prevent these diseases till now, but could control the complications responsible for disablement and early death. Both early disablement and early death were terrible facts connected with most of the pneumoconiotic disorders and with the complications related to the different forms of pneumoconiosis.

To remember some of these steps, it may be useful to understand our plans and projects for the future.

There is no doubt that the improvement of dust control at all levels is a very important step for the control of dust-related diseases, but besides the improved dust control tremendous medical progress took place. All the coal workers who really develop problems in relation to coal workers' pneumoconiosis have obstructive airway diseases. This kind of airway obstruction starts on the basis of chronic bronchitis and is followed by obstructive bronchitis. In case of less strong X-ray changes, the obstructive airway disease is not more frequent than in non-dust-exposed men (REICHEL et al., 1969).

In categories B and C of the ILO classification this means large massive fibrotic lesions the incidence of which is twice as high as in non-dust-exposed men (the smoking habits of miners agree with the control group of non-dust-exposed men) (Figure 1).

Like patients with idiopathic obstructive bronchitis, the obstructive bronchitis of coal miners dictates the clinical situation of these patients. Fortunately, the obstructive bronchitis of coal workers with coal workers' pneumoconiosis can be treated in the same way with the same success as the idiopathic form of chronic obstructive bronchitis (Figure 2).

We control our coal workers with coal workers' pneumoconiosis very carefully. This means, coal workers with coal workers' related obstructive bronchitis stay under a controlled regime of treatment. Under the long-term treatment the expectation of like of our miners with large opacities and fibrotic lesions on the X-ray is now at least as long as that of the general population (Figure 3).

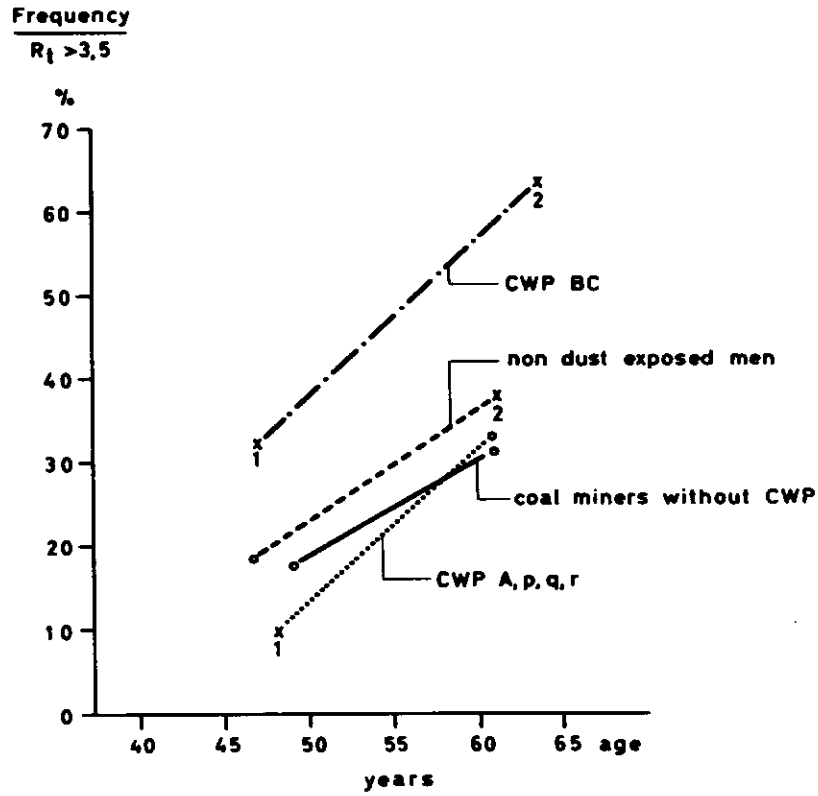

Figure 1. Age dependency of patients with obstructive airway diseases at different stages of coal workers' pneumoconiosis and of non-dust-exposed men (obstructive airway disease = R_{aw} >3.5).

At first glance, the situation may give satisfaction, but we have to realize that the age at which the coal workers develop obstructive bronchitis is nearly the same as 20 years ago. The mean age of the manifestation of obstructive bronchitis is 57 years. In the fifties the expectation of life after airway obstruction was 3.5 years on the average, and now the expectation of life is 16 years on the average. But during this time, coal workers with coal workers' pneumoconiosis and obstructive bronchitis are disabled and have more or less dyspnea, and some even develop cor pulmonale.

Therefore, for the future we have to avoid the development of coal workers' pneumoconiosis and we have to learn to avoid manifestations of chronic obstructive airway diseases.

First the development of coal workers' pneumoconiosis on the X-ray: The correlation between the ILO 12 step classification (from −/0 to 3/+) is relatively linear. Figure 4 shows the results of one mine in W.-Germany as mean value and also the progression of the worst and the best case (Figure 4).

From these curves we can calculate the ILO classification step time: it is the time in years necessary to get from one classification step to the next one (e.g., 0/1–1/0). In the example in Figure 4, the ILO classification step time is 8 years on the average. These curves allow an extrapolation at a relatively early time. From such curves we may learn more about the causes of the different ILO classification step times for different individuals as well as for different mines.

There are clear differences between different mines as Figure 5 shows (Figure 5).

We proposed that:

a) the X-ray development of coal workers' pneumoconiosis should be documented for each coal worker on ILO classification step times/exposure times curves;
b) an interval of 4 years for X-ray examination of coal miners is adequate and without any risk for coal miners at present exposure levels.

In order to prevent the pneumoconiosis due to obstructive airway disease the prevention of airway obstruction is the most important factor as already mentioned above. Today, we dispose of sensitive methods to detect early signs of lung function changes related to airway obstruction. In W.-Germany, we examined in 4 mines the miners by careful lung

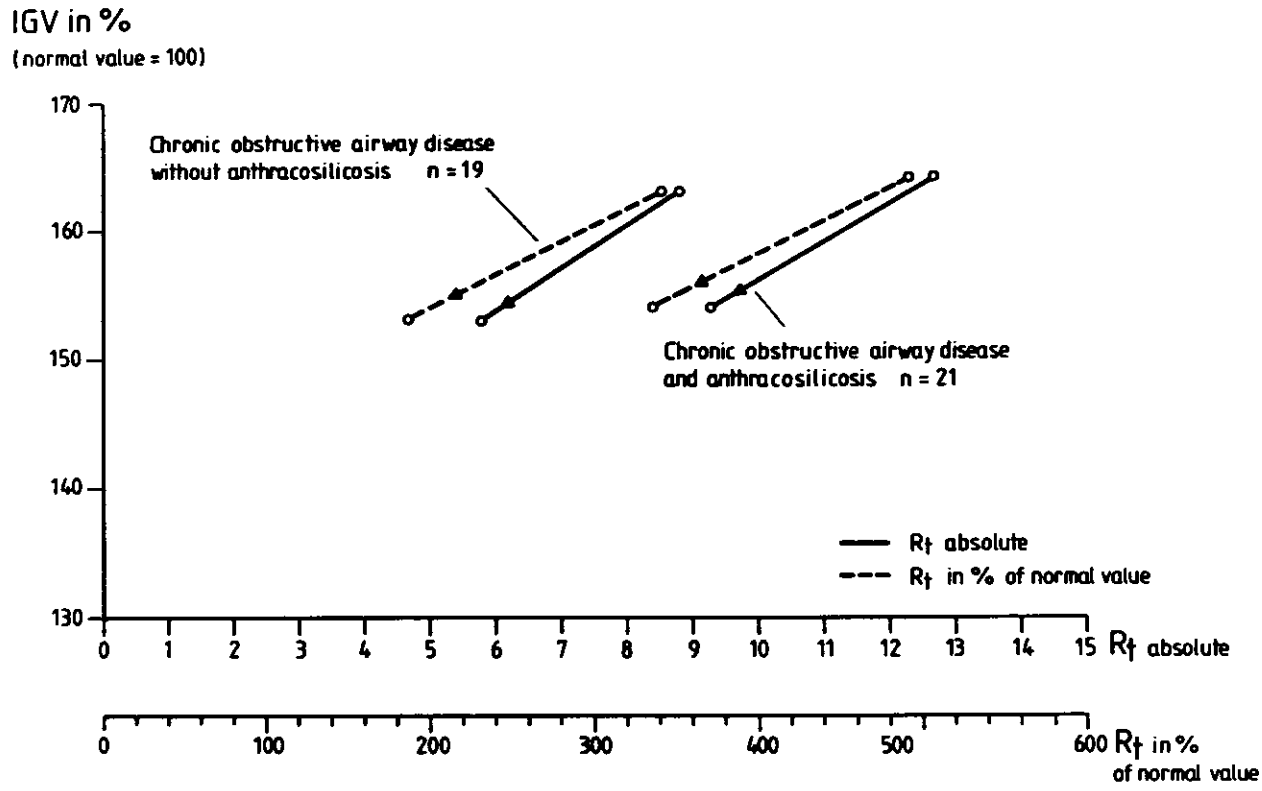

Figure 2. Decrease of airway resistance (R_t) and of intrathoracid gas volume (IGV) under typical treatment with bronchodilators and glucocorticoids in patients with pneumoconiosis-related obstructive bronchitis (n = 21) and of patients with idiopathic obstructive bronchitis (n = 19).

function tests. Among the miners is a relatively high percentage still at work who has obstructive airway disease, and there is quite a number of miners with oversensitivity (hyperreagibility) of the airways.

It is very important to detect persons with signs of obstructive airway disease as early as possible to control the development of the lung function of these persons:

1. to start with an adequate treatment at adequate time and
2. to avoid progression of this disease. It is very likely that an early treatment can control this disease and can avoid progression.

We have to control the lung function of dust-exposed persons with adequate methods. Time intervals for re-examination could be 4 years but not longer.

Dust concentration decreases in the inhaled air are most important for prevention. The very effective dust masks are normally worn for short times only. The acceptance of normal light masks depends on the isolation, and therefore on the development of sweat under the mask. The loss of the possibility of communication is also important and problems of increased airflow resistance may be a factor, too.

The light masks (Figure 6) comparable to those masks worn in hospitals, have in this respect many advantages, although they decrease the dust concentration only for about 70%.

1. They allow communication with other persons without effort;
2. They soak up the sweat around the mask;
3. The decrease of dust concentration in the inhaled air is about 70%, and therefore the ILO classification step time increases so that during the life time coal workers' pneumoconiosis responsible for dust-related obstructive airway disease will not develop (Figure 7).

CONCLUSION

In addition to the best available dust suppression we should emphasize that the light (one-way) masks will be used continuously. With this strategy, coal workers' pneumoconiosis could be controlled so that dust related obstructive airway diseases never will occur.

These results shown mostly for coal miners and coal workers' pneumoconiosis can be transferred more or less to other types of pneumoconiosis.

Our knowledge about the development of pneumoconiosis increased tremendously. During the last decades we could dispose of strategies which are able to slow down the development of coal workers' pneumoconiosis suddenly and which can avoid the coal workers' pneumoconiosis-related obstructive airway diseases. Furthermore, we can improve the health situation of the miners we are responsible for.

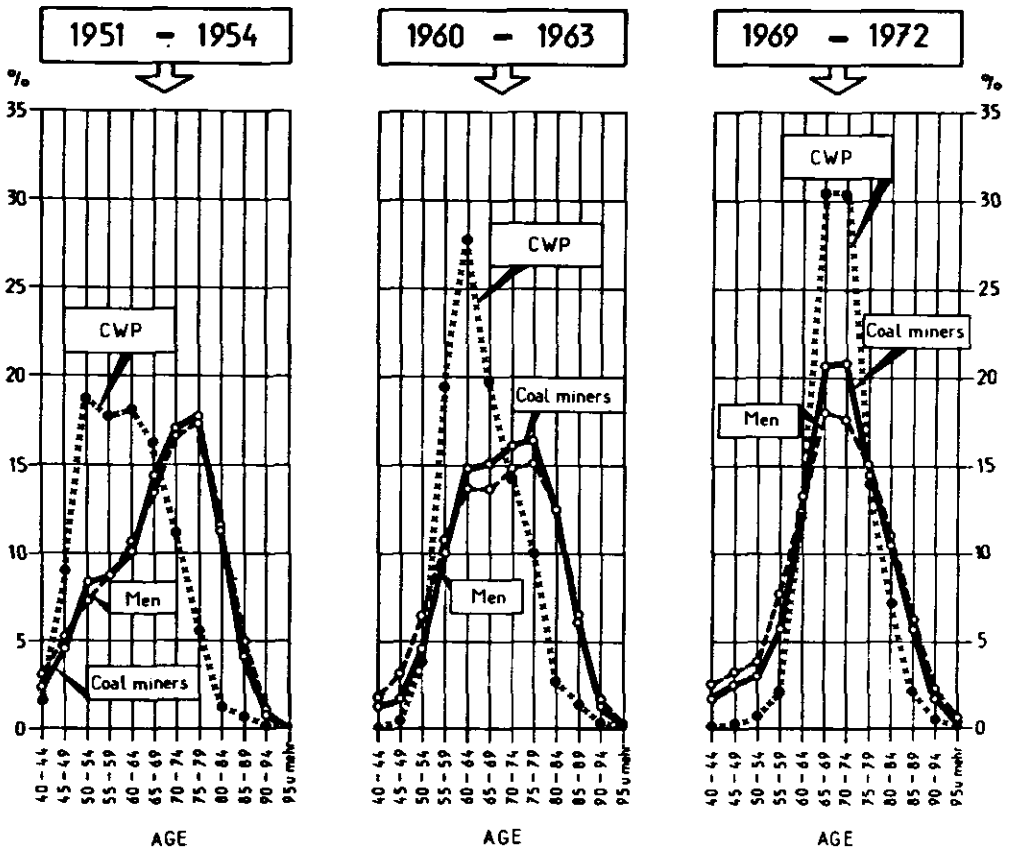

Figure 3. Expectation of life of coal workers with obstructive bronchitis and of miners without coal workers' pneumoconiosis and of non-dust-exposed men in the years 1951–1954, 1960–1965, 1969–1972.

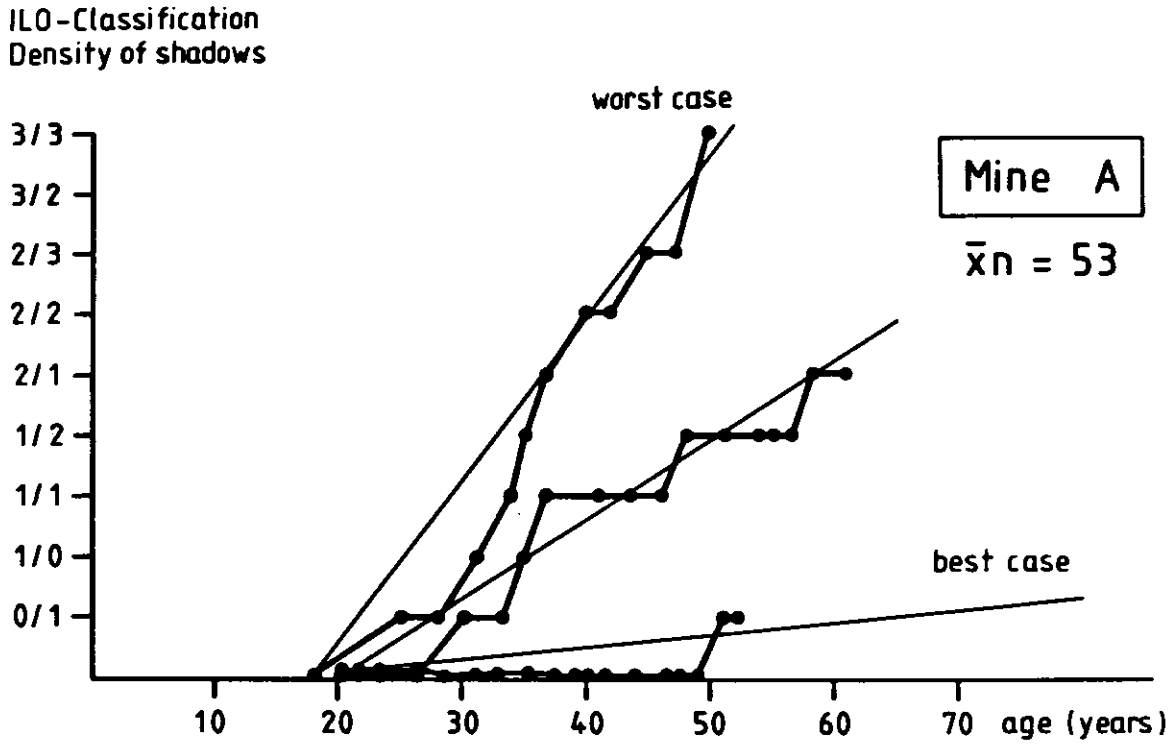

Figure 4. Correlation between ILO classification and exposure time (mean values of 53 miners and the best as well as the worst individual case).

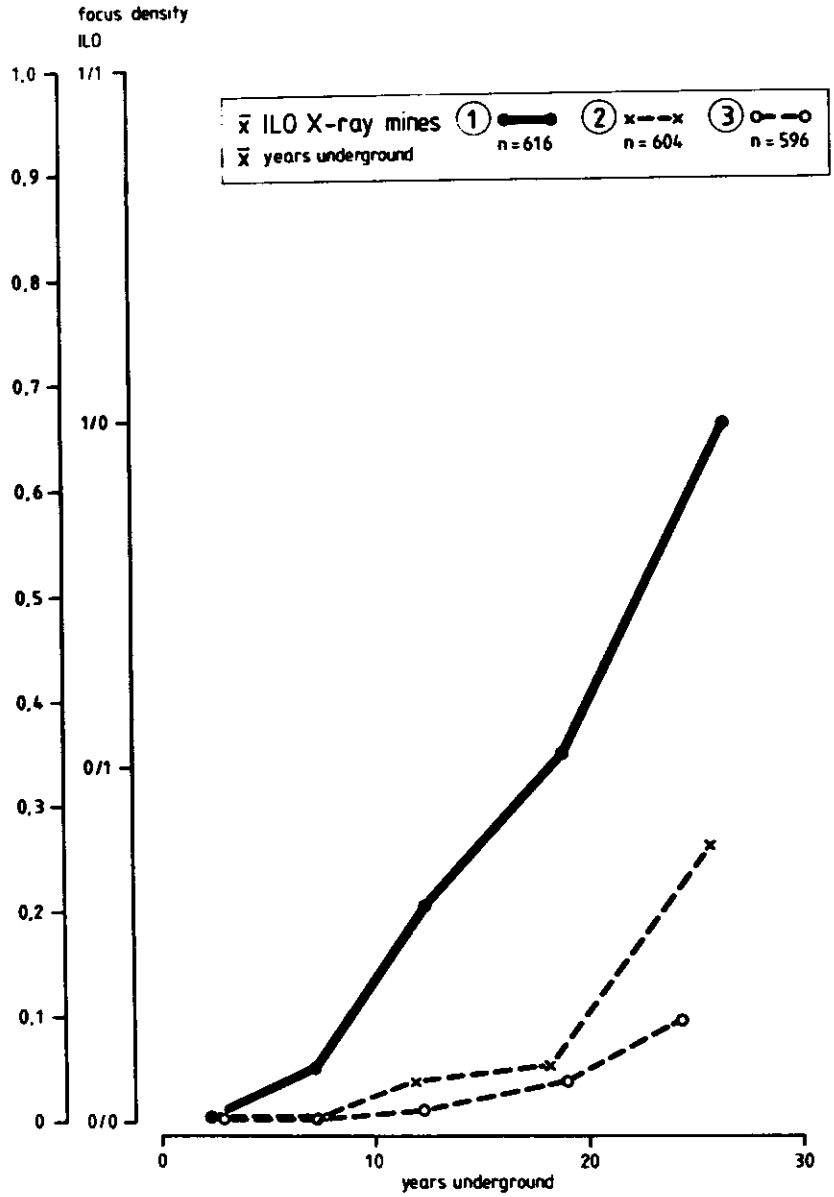

Figure 5. Correlation between ILO classification steps and exposure time on 3 different mines in W.-Germany (mine 1 n = 616, mine 2 n = 604, mine 3 n = 596).

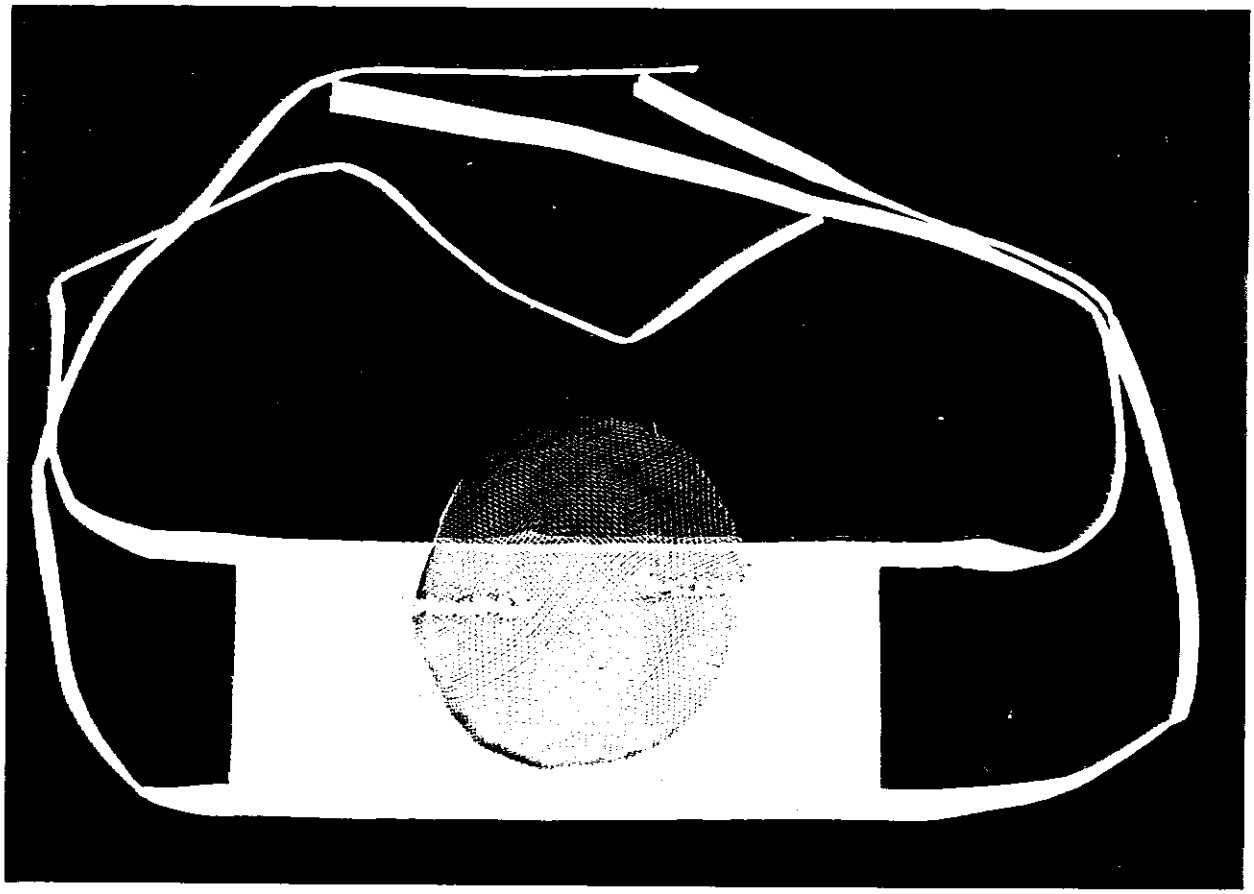

Figure 6. Light mask with very good acceptance and many advantages protecting against the development of coal workers' pneumoconiosis (decrease of dust concentration in the inhaled air ~ 70%).

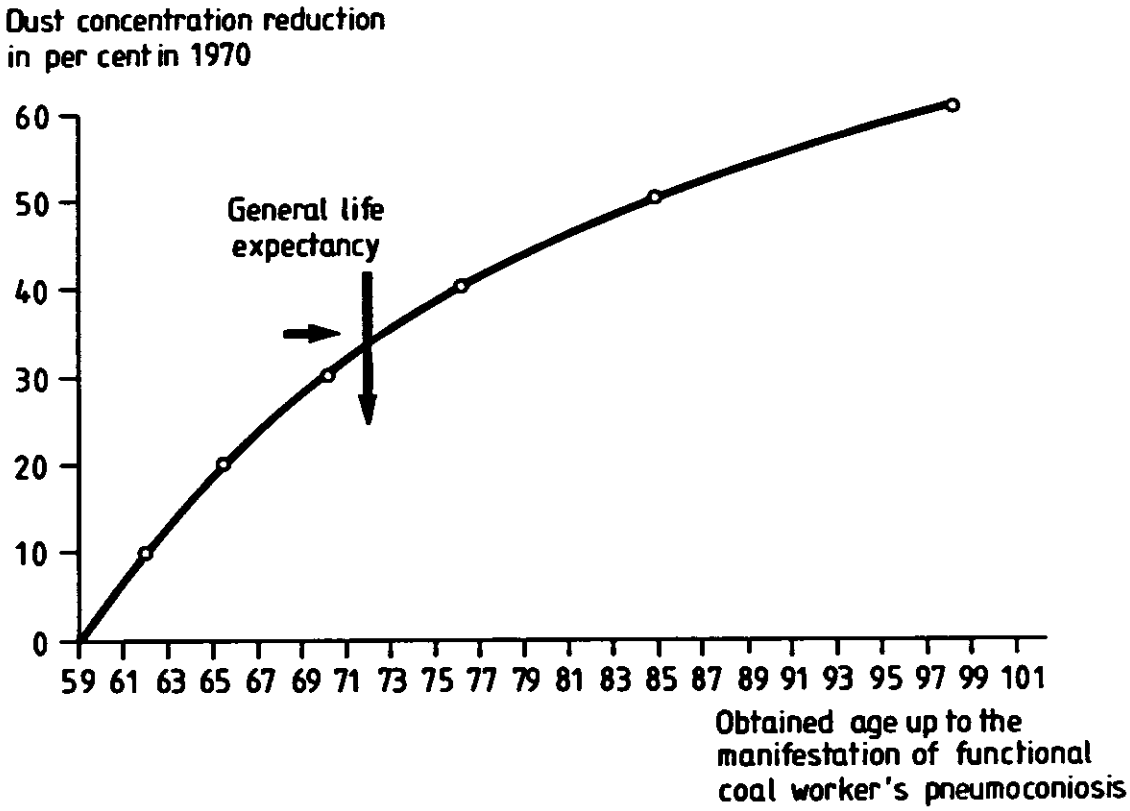

Figure 7. Relationship between decrease of dust concentration in percent of the values from 1970 and the age at which X-ray changes take place which could be responsible for coal workers-related obstructive airway diseases (a decrease of about 35% would be enough to prolong the manifestation time to the values of the normal expectation of life).

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REFLECTIONS ON PROGRESS WITH MINE DUST CONTROL AND DUST CONTROL TECHNOLOGY

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INTRODUCTION

It is obviously impossible to provide a detailed historical or even present-day account of the control of dust in mines and dust control technology in the brief time allotted to me. Therefore, as a compromise this theme paper highlights major selected subject areas of scientific and technical knowledge that have culminated in the current degree of dust control and control technology in U.S. mines. What were the historical understandings and emphases; what types of knowledge were gained through laboratory and applied research that permitted us to effectively implement dust control strategies through either voluntary or regulatory societal mechanisms? In different nations there has been a shared concern with this occupational problem, and the contributions to understanding have been multinational. Bear with me if I tend to oversimplify; it is my belief that at times we must sit back and take a long look at what has been called the "drum roll of history." This enables us to discern "the big picture" from the many necessary and essential details that punctuate progress in any field of human endeavor. It also enables us to better consider where we are at present and to define further, needed progress.

CONTROL OF MINE DUST

Recognition of Coal Workers Pneumoconiosis as a Disease State

The report by Bedford and Warner[1] in Great Britain in 1943 must be regarded as a major turning point in our understanding of the impact of inhaled coal dust and of dust control in mines in Great Britain. This report stimulated the adoption of airborne dust standards for "approved dust conditions" in conjunction with employment underground. No specific dust concentration limits or standards were set by law, but the adopted standards in the attainment of dust suppression continued for almost 30 years. The standards were the result of extensive studies of pulmonary disease in South Wales coal miners conducted by the Medical Research Council. These studies were conducted in five mines; they associated dust with x-ray abnormalities. The British later extended these studies to a larger number of mines, i.e., the so-called 25 Pit Studies.

The proposal for a standard was that not more than 10 milligrams per cubic meter of anthracite dust or 1 milligram per cubic meter of minerals other than coal for particles 5 microns or less in size, should be achieved. Note that the particle number standards for approved mine dust conditions introduced in Britain in 1949, remained basically the same until 1970, when gravimetric standards were introduced, again resulting from epidemiologic studies relating dust and pneumoconiosis. Thus, in Great Britain there was recognition of the disease state and adoption of standards for approved dust conditions.

In the United States, a major 1936 report by the Public Health Service[3] indicated that the term anthracosilicosis, as used, was a descriptive title for the form of pneumoconiosis commonly called miners' asthma. It was diagnosed by occupational histories, clinical examination and x-ray exams. The report indicated that the correlations "between exposure to dust and the evidence of constitutional changes left little doubt as to the etiological significance of the dust in the air breathed." Similar correlations were found between the silica exposure and the extent of pulmonary changes. Investigators concluded that employment in an atmosphere containing less than 50 million dust particles per cubic foot would produce a negligible number of cases of anthracosilicosis when the quartz content of the dust was less than 5%. This report was also an extraordinarily important one in that it lead to adoption of the standards for free silica in the United States, which are in effect to this day under our Occupational Safety and Health Act, if the user chooses to utilize midget impinger sampling and dust counting methods to evaluate dustiness.

My point in citing these two reports is to indicate that acknowledgement of the correlation between the disease state and the etiological agent is essential before control efforts can take place. In the 1950s the Commonwealth of Pennsylvania pioneered in studies of coal miners that lead to the recognition of the disease state of coal workers pneumoconiosis in bituminous coal miners, as contrasted to anthracite miners. Thus, the 1930's U.S. investigations resulted in differentiation between free silica in the dust causing silicosis, and miners' asthma occurring in hard coal mines. We spent another 25 years in the United States

debating legitimacy of the disease state of coal workers pneumoconiosis, which seriously hampered efforts at dust control. In this regard, the British reached consensus on this point before we did.

The Physics of Dust

In order to control the dust associated with a disease, one must know a great deal about dust physical and chemical properties. Although there were scientific treatises on dust properties as early as 1934,[2,37] these volumes were very limited in the technical information provided that could be translated to the measurement and control of mine dust. The dust measurement techniques in effect during the 1930's were the midget impinger[16] developed in the United States, and the Kotze' Konimeter,[26] developed in South Africa. Gravimetric techniques were not extensively utilized to assess airborne dust for disease prevention until the late 1940's and 1950's. In fact, calibration of the impinger for coal dust particles (the only particles for which, to my knowledge, the instrument was ever calibrated) was reported by C.N. Davies in 1951.[9] The concept of aerodynamic particle size remained to be elucidated. However, even without these understandings enormous progress in mine dust reduction could be, and was made, as evidenced in South Africa in the 1930's through the 1950's.

The ability to advance beyond the qualitative understanding that inhalation of dust is dangerous to your health and that dust concentrations measured as described correlate with disease prevalence in exposed workers, also depends upon insights into the hygienically significant sizes of inhaled particles. The development of this area of understanding is my third selected critical area of knowledge for effective dust control.

Dust Deposition in the Human Respiratory Tract

Drinker and Hatch[11] traced the examination of particles in exhaled air to studies by Tyndall in 1882, and also cite a hygienic study by Saito in K.B. Lehmann's laboratory in 1912. Studies in the 1930's on nasal filtration by normal men were performed by Lehmann and by Torangeau and Drinker. The studies by Brown in 1931 were the first major studies on dust retention in man.[5] Experimental investigations by Davies,[8] Landahl and Hermann,[23] Van Wijk and Patterson[36] and Wilson and LaMer,[38] supplemented by theoretical calculations by Findeisen,[13] all advanced the state-of-the-art of particle size deposition in the human respiratory tract. The particle inhalation study that dictated U.S. views on deposition for two decades was that by Brown, et al., in 1950.[6] It was the major source of definitions for respirable dust in the U.S. and Europe, and for the very important report on dust deposition and retention in the human respiratory tract by the International Congress on Radiological Protection in 1966.[35]

These were the first of a long series of experiments, still continuing, to define the particle sizes of significance for chronic disease developing in the pulmonary compartment of the respiratory tract. The studies have been refined and there has been international agreement on the deposition curves in healthy men after inhalation at standard volume. The subject was reviewed by Lippman[24] and the new definitions of compartmental deposition in the human respiratory tract have been published.[27]

The significance of this work was that it provided a target in terms of the spectrum of particle sizes of hygienic importance in dusty environments, and enabled those interested in control to take aim at that target.

It should be noted that during all of this work the characteristic "size" of a particle was the projected area diameter, because the predominant instrument for viewing collected dust particles was the light microscope. In the light microscope one observes a silhouette, a two dimensional representation of a three dimensional object, the dust particle. Work performed at a later date would differentiate between the aerodynamic size of a particle and the silhouette size of the particle that one observes in the microscope. While this may appear to be a minor physical differentiation, it is of the utmost significance. It explains why we observe fibers 200 microns in length in the human pulmonary compartment; their aerodynamic size is equivalent to a less than 10 μm diameter sphere of unit density which could penetrate to that region of the respiratory tract. It took many years for the significance of the aerodynamic size to be recognized.

The understanding of dust aerodynamic behavior and deposition in the lung required decades to crystallize. The control of dust could progress, but there was great uncertainty if one was capturing the sizes appropriate to the disease state. An analogy is use of a shotgun versus a target rifle. The standardization of so called "respirable dust" and the development of an instrument to simulate that dust size waited until 1954 when B.M. Wright in England introduced the horizontal elutriator.[39] Dust control efforts were proceeding in all industrialized nations, but it is fair to say that permissible dustiness was far above the concentrations we have today in mines of industrialized nations. There was also great uncertainty in the long term benefits associated with the control efforts, because the largest particles have the greatest weight associated with them. There could be extensive reductions in dust measured in terms of weight per unit volume; there could, however, still be only conjectural impact on the disease state, because the smallest particles reaching the pulmonary compartment are associated with the least weight per particle.

West Germany began a series of epidemiologic studies in their mines in the search for the dust parameter that would best correlate with disease. They concluded that the surface area of the dust was an appropriate parameter and developed their dust measurement techniques accordingly. At a later date, they too would recognize the respirable dust concept with cyclone precollector sampling to determine respirable dust weight. During all the years of the 1950s and 1960s the British counted particles using a thermal precipitator instrument and reported their dust concentrations in numbers of appropriately sized particles per cubic centimeter of air.

Instruments to Measure Respirable Dust

It is interesting to peruse the first volume of air sampling instruments entitled "The Encyclopedia of Instrumentation for Industrial Hygiene."[40] The volume is concerned with many different types of air sampling instruments. The cas-

cade impactor, the midget impinger, and the electrostatic precipitator were the major instruments available for particulate sampling in 1956. Indeed, at a Governor's Conference in the Commonwealth of Pennsylvania in 1964 a leading U.S. industrial hygienist who was director of industrial hygiene for our major steel firm, and a previous President of the American Industrial Hygiene Association, stated publicly that the respirable dust concept then in vogue in England and Germany was not applicable to United States mines. Thus, one reason that efforts to develop instruments that would apppropriately sample respirable dust in coal mines did not rapidly progress in the U.S. was because the respirable dust concept was not readily accepted.

Subsequently, appropriate instruments were developed in the 1960s, utilizing a United States Atomic Energy Commission cyclone preseparator and the definition of the United States Atomic Energy Commission for respirable dust. It approximates the BMRC respirable dust acceptance curve defined by the horizontal elutriator.[20] These instruments approximated the accepted pulmonary deposition curve at that time, mainly based on the data of Brown et al.[5]

The dust standards in mines enforced in Great Britain during the period 1949 to 1970 were summarized by Chamberlain et al.[7] The British abandoned the particle counting standards in 1970 and adopted gravimetric standards. The introduction of gravimetric standards in the United States accompanied the Coal Mine Health and Safety Act of 1969.

The Mine Health and Safety Act of 1969 required that beginning June 30, 1970 the operator of each coal mine was required to maintain the average concentration of respirable dust in the active working at or below 3.0 milligrams per cubic meter. The standard was reduced to 2 milligrams per cubic meter after December 30, 1972 and has remained at this level.[32] Because of the difficulty of adapting to this standard an Interim Compliance Panel was authorized to issue a permit for non-compliance for a dust concentration as high as 4.5 milligrams per cubic meter while the standard was 3 milligrams per cubic meter, and for 3 milligrams per cubic meter when the standard was 2 milligrams per cubic meter. However, by December 30, 1975 the 2 milligrams per cubic meter was to be met. In the U.S. we are still not meeting that standard in all mines. The Mine Safety and Health Administration has developed an elaborate sampling procedure to insure compliance with this standard. The procedure involves sampling key occupations or key locations in the mine. The progress in dust control in mines has been achieved with a regulatory inspectorate for approximately 275,000 miners that equals the inspectorate of the U.S. Occupational Safety and Health Administration, which has responsibility for over 75 million workers at all types of worksites. Mine Safety and Health Administration inspectors in the United States visit every mine many times in a given year; the probability for a visit by an OSHA inspector to a workplace are, on the average, less than one in 50 for most businesses.

RISK LEVEL OF PRESENT STANDARD

Since 1982, there has been major emphasis on risk assessment in the regulatory process in the U.S.[30] The 1969 U.S. standard for permissible dustiness in mines was keyed to the British standard. It is interesting to review the risk level estimated to be associated with that standard. The interpretation of risk level can be derived from the British and the German epidemiological studies. In England the 25 pit study provided the data base.[22] In the British studies the quartz in the coal dust varied from 0.8% to 7.8% (respirable dust), with an average of 4.1%. The progression of the disease seemed to be associated with the quartz content of the respirable dust. Nonetheless, the probability of occurrence of 0/1 ILO classification x-ray for mean dust concentration of 2 milligrams per cubic meter for 35 years of exposure, is approximately 4%. The probability that a man starting with no pneumoconiosis (category 0/0) will be classified into 2/1 or higher after 35 years exposure to 2 milligrams per cubic meter is about 1 1/2% for low rank coal; 3% for high rank coal.[31] The U.S. estimate of CWP category 1 at 2 mg/m^3 is 9%; category 2 is 1–2%. In terms of current risk levels being discussed in the United States for other airborne contaminants, this is a somewhat high risk level. For example, the current estimate of risk at 0.2 fibers per cc for 35 years asbestos exposure is 0.7% for lung cancer and mesothelioma, with virtually zero risk of asbestosis. The 35 year time base for estimate of the risk is the same as that for respirable coal mine dust.

The German epidemiological studies occurred in 10 coal mines over a 10 year period.[29] A cumulative dust index was utilized based on light scattering measurement of the dust. Thus, the dust measurement was dependent on some function of the dust surface area. The German investigators related the Tyndallometric fine dust concentrations to the gravimetric fine dust concentrations measured with a cyclone/filter collecting device. They concluded that the ratio varied with coal rank; therefore, there was considerable uncertainty in a general correlation, but they did correlate by high, medium and low ranks of coal. Using the index developed, a cumulative dust value of 50,000 was associated with definite pulmonary change. The parameters influencing the conversion from light scattering to gravimetric measurements were a dirt concentration factor and the fineness factor of the dust, which influences the degree of forward scattering of light in the instrument. The concentration range of 0.9 to 1.5 milligrams per cubic meter as measured by the cyclone, was estimated to correspond to a cumulative fine dust concentration measured by light scattering of about 125,000. If I correctly interpret the publication describing these results, there would be a risk of about 5% of light to medium pulmonary changes with a 6,000 shift exposure to approximately 1.5 milligrams per cubic meter, indicating a risk level about that encountered in Great Britain and the U.S. These estimates are very intimately associated with the rank of coal and my conversions are therefore a rough estimate.

Chemical Composition of Dust and Coal Miners' Pneumoconiosis

The pathophysiology of coal miners pneumoconiosis is still not well understood. The presence of quartz in the dust is a confounding factor. The present tools for disease diagnoses, namely x-ray and pulmonary function testing, cannot dif-

ferentiate in the living miner between silicosis and coal workers pneumoconiosis. There is considerable disagreement at the lowest ILO classifications re: the disease state. Promising efforts to understand the disease state, as reflected in the present research emphasis, appears to be correlation of residual dust components for dust retained in the lungs and analyzed post mortem, with components of the exposure dust.[34] In particular, there seems to be increasing emphasis in the United States on free silica content of mine dust. MSHA is increasingly stressing the silica content of the dust. The classification in 1986 by the International Agency for Research on Cancer of crystalline free silica as Class 2A gives further impetus to the emphasis on free silica.[33] The inability to estimate the free silica of the airborne dust on the basis of settled dust has long been known.[12] It is now possible to measure free silica in respirable dust samples with a sensitivity of 1–10 micrograms, depending on technique, and it is anticipated that with the IARC classification there will be a change of the current U.S. silica standard, which is presently stated as a sliding scale for permissible dustiness based on free silica content.

In summary, the control of dust in mining has witnessed enormous progress during the past 20 years, stimulated by increased regulation in many countries, including the U.S., and innovative development of standards in South Africa, Germany and England, standards preceded by extensive epidemiological investigations that provided an estimate of the risk levels associated with adopted numbers. Areas of knowledge that required development to efficiently implement dust reduction in mines were the deposition of dust in the respiratory tract and the physics of dust, the latter required for instrument development and sensitive analytical techniques to measure the dust collected. Different nations took different approaches to the evaluation of dustiness but almost all now utilize gravimetric methods, for both feasibility and for scientific reasons. When compared to risk levels associated with standards now being adopted for other airborne contaminants in the United States, there is need to further consider the airborne respirable mine dust standard. Recent classification by IARC of crystalline free silica as a Class 2A carcinogen strongly suggests the need to better understand the exposure to and impact of free silica in coal mine dust, in particular. Having discussed selected topics in the control of mine dust, I will now briefly look at the progression of dust control techniques in mines. What are the technologies that have brought about this progress and how much further can we exploit these technologies, or are other "understandings" needed to make further progress?

DUST CONTROL TECHNOLOGY

Ventilation

The Office of Technology Assessment in its 1984 report Controlling Hazards in the Workplace[27] introduced the terminology of the "hierarchy of controls," with engineering controls at the top of the hierarchy and administrative procedures and work practices following; personal protective equipment is the last intervention to control exposure. Among the engineering controls are control at the source and control by substitution. The seven engineering controls listed by OTA are shown in Table I. In 1950 in a review of literature on dusts,[14] the authors quote Harrington, a 1934 reference[18] with regard to the control of dust in mines. Harrington indicates that ventilation, fire protection and prevention, health, safety and efficiency are very closely interlocked in mines. He indicates that ventilation is perhaps the major route for control of hazards in mining, both in metal and nonmetal mines, permitting the worker to exert himself in comfort at maximum physical capacity without endangering his health. He focuses very heavily on "the best remedy for the dust menace in mines, other than preventing its formation, is the universal coursing of currents of air to remove the dust, as it has been proved that the very fine, most dangerous dust in metal mines remains suspended..." Harrington indicates that spraying devices available to reduce dust while drilling may be effective if used intelligently. However, they may even intensify the air dustiness if used without intelligence and, unfortunately, the latter is generally the case. He points to the availability of efficient water drills. Harrington also indicates that while finely divided dust "in mines is probably the chief cause of miners consumption, it is now recognized that there may be other factors of almost equal influence, such as high temperatures and humidities, harmful gases, and lack of air movement; all of these defects are readily remedied by ventilation." Table II is a summary of approaches or "lines of attack" for dust control in mines, as presented by Hamilton in 1972.[17] It differs little from Harrington's approach.

Table I

OTA Hierarchy of Controls: Engineering Controls

Elimination
Substitution
Isolation
Enclosure
Ventilation
Process Change
Product Change

Table II

"Lines of Attack" for Dust Control in Mines[17]

1. Removal and dilution of dust by ventilation.
2. Contol of the formation and dispersion of dust by attention to the method of mining and the way in which machines are operated.
3. Application of water, either to limit the dispersion of dust into the air, or to suppress airborne particles.
4. Use of exhaust ventilation to contain dust sources, followed either by ducting the dusting air to unoccupied parts of the mine, or by filtration before returning it to the main ventilation current.

The use of water in drilling and in mining has a long history. The British Coal Mines Act of 1911 required that a drill worked by mechanical power "shall not be used for drilling in ganister, hard sandstone, or other highly siliceous rock, the dust from which is liable to give rise to fibroid phthisis, unless a water jet or spray or other means equally efficient is used to prevent the escape of dust into the air."[21]

Water

Water has also been used, particularly in Western Europe to infuse the coal seam prior to drilling. Coal piles have been wetted after blasting and after cutting. Permanent use of water is not possible because moisture can be detrimental to certain processes in minerals and in some mines limited quantities of liquid must be used if the product is to be marketed. Wetting agents have been added to water and in recent years droplets have been electrified during spraying to increase contact with the dust. Foams has also been utilized, the theory being that dust particles will be trapped in the individual cells of the foam and subsequently wetted by the liquid as the cells collapsed.

The development of our understanding of aerosols and particles owes much to the concerns of mining. In particular, the work of the Safety in Mines Research Establishment in Sheffield, England and the Bergbaustaubsverein and the Silicosisforscungs Institut in the Ruhr were major contributors to the pool of knowledge of the physical properties of dust as reflected in compendium volumes such as that by Green and Lane.[15] Other summaries of particulate knowledge also reflect the contributions of investigators at these institutes who, although they were pursuing applied research, recognized the necessity for basic contributions on the physics and chemistry of dust. The names of Cartwright, Hodkinson, Davies, Robock, Hamilton and Timbrell immediately come to mind. It is not my purpose here to dwell on the specific research investigations that lead to progress in the control of mine dust. Rather, I believe it is possible to discern the trends in this area, as reflected in comparing a 1980's review published in the United States with the earlier literature on dust control in mines.

The review by Breslin and Niewiadomski[4] of the United States Bureau of Mines was published in 1984 and reviews progress in dust control technologies for U.S. mines from 1969 to 1982. In this report the authors stressed the control of dust formation, primarily. The relationship of coal cutting to the generation of airborne dust is highest on the priority list of the Bureau's dust control "understandings" for control technology. This is an extension of the innovative work by Hamilton in England.[17] The type of cutting bit, the depth of cut, the possibility for injecting water through the bit, the number of bits used, are all aspects of this research program. Because the mining methodology in the U.S. is shifting very rapidly to longwall production the applications of these techniques to the longwall operation both at the cutting site and upstream are focused upon. As indicated in the earlier statements by Harrington, the dust movement caused by the application of water is of great concern in these investigations. Ventilation is still our major workhorse in the dilution and removal of dust through both blowing and exhaust, but a substantial gain is achieved through the use of water injection and control of cutting.

The Bureau also focuses upon the use of dust collectors for trapping the dust; these operate both on scrubbing and filtration principles. The trapping of the dust after generation permits the use of the air without its burden of respirable mine dust. The largest fraction of work performed by the Bureau in these years was for in situ testing of these techniques after laboratory evaluation. A great deal has been learned about the equivalent volume of air cleaned versus water pressure as a function of different types of spray nozzles in the wet type scrubbers. Wetting agents have also been tested and there is some reported incremental gain due to their use. In the course of advancement of this technology the Bureau lists the following basic "understandings" which have come from this work.

- Laboratory studies showing the relationship between dust generation and the specific energy used to cut coal.
- Studies of deposition of aerosol on electrostatically charged surfaces.
- Experimental research on the dynamics of water drops impacted on surfaces.
- Development of laboratory apparatus for generating water drops of uniform size and for measuring drop size.
- Measurement of the adhesion force between dust particles and surfaces.
- Characterization of the physical and chemical properties in mine dust.
- Development of technology for automatic measurement of particle size, shape and composition using a scanning electron microscope.
- Studies of the efficiency of dust sampling inlets.
- Development of apparatus for generation of laboratory aerosols.
- Studies on the effect of water sprays on air movement and dust suppression.

The Bureau indicates that fundamental research was done with the ultimate long term goal of improving technology for control of dust in mines and that meant many of these areas of knowledge have applications in areas other than mining. The National Academy of Sciences in 1980 issued a report[25] in which the Academy directed the Bureau towards research which "should be directed more toward obtaining fundamental understanding of the origin, transport and characteristics of respirable coal mine dust." One could say this is expected from an Academy report, but I prefer to think that there is finally broad recognition of the need to understand fundamentals in order to develop technology.

The future goals of the dust control technology in the Bureau are also of interest. They are stated as:

- Optimization and in mine application of the new water spray system ("Shearer/Clearer") for longwall dust control.
- In-mine evaluation of new and emerging longwall dust control technology.
- Determination of the applicability and effectiveness of water powered scrubbers at longwall operations and on continuous miners.
- Completion of field evaluation and application of a mine worthy twin scrubber system for continuous miners.

- Development and testing of optimal ventilation systems for dust control during continuous miner operations.
- Redesign, testing, and application of an improved canopy-air curtain system in underground as well as surface operations.
- Development of a basic understanding of the formation of transport of dust during the cutting cycle for development of more effective controls.
- Development and testing of improved bagging machine dust controls, bag ceiling, cleaning and disposal techniques for the mineral processing industry.
- Development of dust suppression systems for cutter machines and other equipment used in conventional mining operations.
- Development of improved dust controls for conveyors, transfer points and stage loaders.
- Determination of cutting force in coal seam for use in development of deep cutting machines.
- Development and testing of improved personal dust exposure.

It would be interesting to compare these research goals for control of dust in mines with those of other nations committing significant expenditures to development of dust control technology at the national level. As one reviews progress in this field over the past 50 years, it is striking that the impetus for sharing of information has come from the professionals and the professional associations and not through their governments. Thus, the first International Pneumoconiosis Conference was held in South Africa, very much due to the efforts of Dr. Beadle, who was preeminent in development of a dust control and medical surveillance program in South Africa. The inhaled particles and vapors series of conferences, a major stage for sharing of information by investigators and practitioners, was sponsored by the British Occupational Hygiene Society. The overlapping of many research program areas is apparent in the past. One wonders if we can become more efficient in our approaches to development of these new technologies. The scientific community will always share results, but the planning of research could greatly benefit by such an international effort.

It is impossible to not be struck by our utilization of the same workhorses for making progress with dust control in mines. We have reached the limit for bringing air to the face and diluting the generated dust. We are probably on the asymptotic portion of the curve for extracting greater efficiency from the application of water, either through the cutting tool or after the cut. We are in need of some new, innovative approaches. In view of the enormous progress over the last 2–3 decades with our understanding of disperse systems and aerosols, it would appear there is opportunity for introducing new and innovative ideas into dust control technology. There are analogies in other fields to this need. The treatment of hazardous waste is receiving major impetus because the major workhorse heretofore has been burial and storage in the ground, which has run its course and is associated with great risks for the future. New technologies are appearing and will undoubtedly have a major impact on the quantities of materials disposed of to ground by 1995. The Superfund Act and its recent renewal have stimulated this work. While the impression in England and perhaps also in the United States is that we have "solved" the problem of coal workers pneumoconiosis and dust in mines, in general, it is incumbent upon us to make clear that this is by no means true. We have made enormous progress, but it remains to bring our risk levels in concordance with those accepted for other work environments. Doing this efficiently requires knowledge and knowledge requires investment of funds.

On this note I would like to end. The story of dust control in mines has some logical development. It is troubling that the current perception is that the job is done, and that diversification of scientific effort and funds from this subject area is occurring in many countries. We must correct this erroneous perception in order to maintain and continue the hard won gains to date.

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