OCCUPATIONAL HEALTH

The following papers were presented at the 1970 annual meeting of the M.A.P.A.O.'s Committee on Occupational Health held in Toronto on May 20, 1970. The papers deal with problems of improving mine working conditions in the areas of dust control and noise abatement, with special emphasis this year on methods of dust collection.

Health hazards of dust inhalation (pneumoconiosis)

By J. E. COWLE*

There are many definitions of the word pneumoconiosis but for practical purposes it may be considered to mean the deposition and retention of dust in the lungs. The term does not necessarily imply that significant structural change has occurred in the lungs or that disability has been produced even though extensive and abnormal shadowing may be observed on the chest x-ray film.

How the lung handles inhaled dust

When the dust-laden air is inspired it passes through the nose and/or mouth, down the trachea (windpipe) and through the various bronchial tubes to the alveoli (small air sacs) across whose walls the exchange of oxygen and carbon dioxide takes place.

Dust particles of greater than five microns in diameter are almost invariably trapped in the layer of sticky mucus which coats the nose, sinuses, trachea and bronchi. Because many of the cells of this membrane are ciliated and because the waving action of these little cilia moves the mucus layer upward toward the throat, the large dust particles are either expectorated or swallowed. Particles of less than 0.1 micron diameter behave differently and, although some may be retained in the lung tissue, a great many remain within the alveolar air and flow out again on expiration.

Particles whose diameter is between 5 and 0.1 microns are, therefore, the important ones insofar as the various pneumoconioses are concerned, (the exception being asbestos fibres). Dust particles in this size range settle on the walls of the alveoli and many of them gain entrance to the lung tissue by passing through the alveolar walls in some manner which is not fully understood. They are then engulfed by phagocytes (dust cells) whose task is to remove foreign material from the lungs. It is believed that the dust laden phagocytes then enter the lymphatic system and flow to the lymph glands in the hilae (lung roots) where they are usually arrested. If the inhalation of dust continues over a period of years, the hilar lymph glands appear to become saturated with dust and the phagocytes then begin to be arrested in the much smaller aggregations of lymphoid tissue scattered throughout the lungs, predominantly at the points where the bronchial tubes bifurcate. It is in these locations that the dust if opaque (as in the case of iron) can be seen on chest x-ray and it is here that the beginning of fibrosis of the lung (if the dust is fibrogenic) occurs.

Classification of mineral dusts

It is doubtful that any dust can be regarded as harmless under conditions of extreme exposure. In general, however, dusts may be divided into two broad groups depending on their ability to evoke a fibrous tissue reaction in the lungs.

For practical purposes they may be classified as follows. (See Table I).

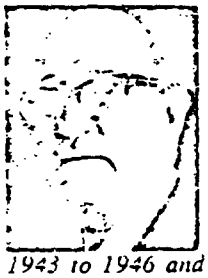
Inert dusts

The dusts in this group do not as a rule produce a fibrotic reaction within the lungs. They may as in the case of iron, barium and tin which are radio-opaque produce extensive shadowing on chest x-ray. They are chiefly important because the x-ray changes which result from their retention in the lungs may be misinterpreted and considered to represent disabling pneumoconiosis.

There is some difference of opinion as to whether pure carbon or graphite can produce significant pulmonary fibrosis but there is little evidence in the medical literature to support the occurrence of disabling pneumoconiosis due to this exposure in the absence of fibrogenic dust.

Fibrogenic dusts

Although the inhalation of fibrogenic dust is essential for the production of disabling pneumoconiosis, *individual*



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susceptibility seems to play an important part in determining the degree of fibrosis produced.

Other factors also are of prime importance in the production of pneumoconiosis. Of these the chief ones are:

a) Intensity of exposure (dust counts)
b) Particle size
c) Content of the fibrogenic substance in the dust (e.g., SiO_2)
d) Length of exposure — usually in years
e) Age at first exposure
f) Presence of other pulmonary condition, e.g., tuberculosis

Since this meeting is mainly concerned with prevention of occupational illness and injury as it affects Ontario miners the following remarks will be confined to a consideration of the more important aspects of silicosis. It should be remembered that while these remarks refer in general to silicosis regardless of industry and geography they are specifically based on experience in the province of Ontario.

Silicosis

Silicosis, because of the numbers of men involved and the degree of disability produced, has been and probably still is the most important of the pneumoconioses. It results from the prolonged inhalation of dust containing a significant concentration of free crystalline SiO_2. Dusts containing not more than 10% silica rarely produce problems. Dusts with higher concentrations of silica are, of course, more important and broadly speaking the risk of producing silicosis increases with the silica content. As previously noted the significant portion of the inhaled dust embraces the particles whose diameter falls between 0.1 and 5.0 microns.

The previously described manner in which the lung handles dust applies to silica. The reaction of the lung to the silica-laden dust cells is to lay down fibrous tissue in a whorled arrangement so as to produce a nodular lesion. As previously noted these tend to follow the distribution of the lymphatics which are intimately related to the tiny blood vessels and the bronchial airways. The earliest silicotic nodulation is found in the glands at the lung root and immediately beneath the pleural surface of the lung. These early changes are not specific on chest x-ray and produce no disability because they do not involve the respiratory portion of the lung. When further progression has taken place, silicotic nodules occur within the lung tissue and it is at this stage that the nodules cast their shadows on x-ray and begin to diminish the breathing capacity of the lung by causing structural change.

TABLE I Classification of dusts according to their ability to evoke a fibrous tissue reaction in the lungs

Inert dusts	Fibrogenic dusts
Iron dust	Free crystalline silica (SiO_2)
Limestone	Amorphous silica if calcined
Marble	Coal
Most silicates	Asbestos (fibrous silicates)
Tin	Talc
Barium	Nepheline syenite (low grade fibrogenicity)

While under poor environmental conditions silicosis can develop quickly (eg., 2-5 years in an unprotected sandblaster) with effective dust control this time can be greatly lengthened and the numbers of men affected kept to a minimum. Of all men who have entered Ontario mines since 1940 only seventy-six have developed radiological evidence of silicosis as of October 1969 and of these 78% had significant previous exposure elsewhere. This means that since 1940 of all men who have worked only in Ontario mines, only seventeen are, at this time considered to have silicosis.

While all figures concerning men who began mining since 1940 are not yet available it appears that the mean elapsed time from first exposure to the earliest evidence of silicosis in chest film is in the order of 25-30 years.

Simple silicosis

In the absence of complications, the most important of which is tuberculosis, silicosis causes little disability. Retained silica in the lungs produces fibrous nodules along the course of the lymphatics, the bronchioles and the blood vessels which bear an intimate relationship to each other. The production of fibrous nodules may progress for several years after *cessation of exposure* and then gradually arrive at a state where the reparative processes of the lungs successfully contain the fibrogenic silica particles. In most such cases the tremendous functional reserve of the lungs is such that the amount of lung tissue replaced by the fibrotic nodules is usually of minor importance and causes little disability. If the amount of nodulation is sufficient to interfere with the elasticity of the lung then this pliable organ becomes less elastic and the amount of air flowing in and out on respiration is decreased. At this stage of silicosis the chest x-rays exhibit nodular shadowing and the workman may complain of some degree of shortness of breath on exertion.

Complicated silicosis

Complicated silicosis is considered to be present when the nodulation seen on chest x-ray begins to coalesce with the development of larger conglomerate shadows. This usually, although not invariably, indicates that a tuberculous infection has become superimposed upon the silicotic process even though the tubercle bacilli may not be found in the sputum.

The most serious feature of silicosis lies in the fact that its presence predisposes to tuberculous infection. In some silicotics the tuberculosis runs an acute course; more commonly, however, the combination of tubercle bacilli and inhaled quartz produces chronic fibrosis of the lungs which slowly spreads over a period of many years. Contraction of the masses of fibrosis grossly distorts the lung tissue and often results in the formation of areas of emphysema in the less affected portions of the lung. Such a condition causes considerable shortness of breath and if severe may produce total respiratory disability. The usual symptoms of tuberculosis such as fever, night sweats, loss of weight and productive cough frequently are not present. The dyspnoea, (shortness of breath) and

Fig. 1 This section demonstrates nodular silicosis. The silicotic nodules appear as rounded black densities since they contain many carbon particles which produce the black colorization. Silicosis of this degree usually does not produce significant disability

Fig 2 This section demonstrates the presence of silicotic nodules in the lung. The main feature is that many of the nodules in the upper lobe of the lung have coalesced producing a massive lesion. This was due to superimposed tuberculous infection. Lesions of this type distort the lung architecture and produce disability

Fig. 3 Nodular silicosis with a massive conglomerate lesion in the upper lobe due to superimposed tuberculosis. The disease in this case was so severe as to cause actual breakdown of the massive lesion producing a tuberculous cavity. Serious disability accompanied this advanced type of lesion

Fig. 4 Massive silicosis is beautifully demonstrated in this section. At least two-thirds of the lung is the site of a mass of silicotic nodules which have run together to form a solid lesion. These solid lesions, of course, contain no air and the patient whose other lung was equally affected was breathing with no more than one-third of his total lung substance. Such far advanced cases were more common previously when tuberculosis among silicotics was more common and before we had drugs for treating this condition. Fortunately, we see relatively few cases with this far advanced and crippling condition nowadays

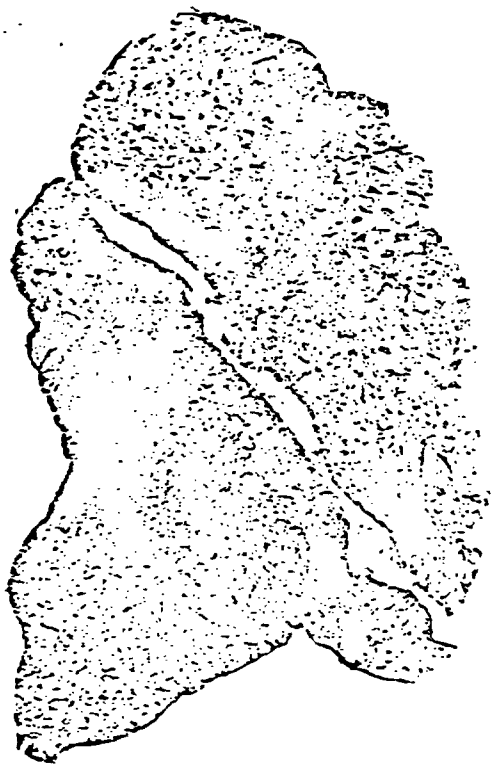

Fig. 1

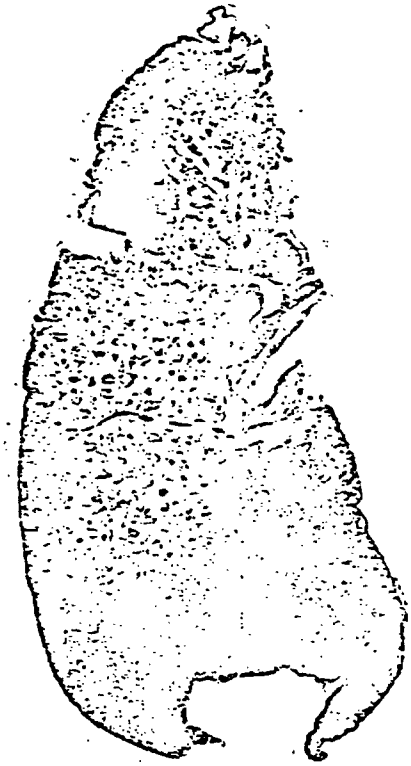

Fig. 2

Fig. 3

Fig. 4

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disability which were formerly believed to be characteristic of silicosis were in most instances due to the massive fibrosis and secondary emphysema which follow the combined action of tubercle bacilli and silica. Simple silicosis alone does not appear to cause emphysema with its resultant disability.

In the 1920's and 1930's the cause of death in the overwhelming majority of silicotics was pulmonary tuberculosis and the mean age at death was below fifty. By the mid 1940's silicotics were living longer and the number of tuberculosis deaths declined. Within the last few years silicotic miners are living at least as long as the average adult male in the general population and the present death rate from tuberculosis is down to about 3%. Most silicotics are now dying of conditions unrelated to their occupation.

From time to time the question of whether there is an increased incidence of deaths from lung cancer among silicotics has been raised. Our records as well as those from Great Britain and South Africa demonstrate that this is not the case. Lung cancer has in recent years very greatly increased in the male population but there appears to be no statistically significant excess among silicotic miners.

Prevention

While a knowledge of the manner in which silicosis develops, the disability which it produces and the complications which may accompany it is of importance, the prime objective of all efforts in this field must be prevention. This should hold the uppermost priority in our approach to the problem and we must never become complacent in this respect even in the light of vast improvement in the incidence of new cases. *Silicosis is preventable* and the ultimate aim of all concerned should be its total extinction. The measures aimed at the prevention of silicosis are:

a) The engineering control of dust in the mining environment.
b) The medical measures offered by pre-employment selection and periodic screening.
c) The inhalation of McIntyre aluminum powder.
d) The hope that P.V.N.O. may be useful in prevention when inhaled and in treatment when given by injection.

Engineering methods

It would be presumptive for the writer to comment seriously on the engineering aspects of dust control. It is, however, felt in order to mention that surface workers such as those sharpening drills and those working in apparently clean mills and assay offices have on occasion developed silicosis. Such occupations should never be overlooked in any assessment of dust conditions and where indicated should be considered in every respect on the same basis as underground workers. It would be folly to place in such work areas, "poor risk" men who are considered medically unfit to work underground.

In addition, in mines and mills where the free silica content of the dust is in the order of 70-90%, extraordinary measures are indicated and constant care is essential to maintain dust control at a very high level. As long as men are required to work in an atmosphere containing a significant amount of free silica, the engineer must never relax his vigilance.

Medical methods

The injurious effects of mining on the lungs and the lives of miners have been known for over four hundred years. As early as 1556 Agricola wrote concerning the work of mining "when the dust is corrosive it ulcerates the lungs and produces consumption". Even three hundred years before the discovery of the tubercule bacillus, it was known that miners ran an extremely high risk of dying of this disease which was referred to as consumption. With the discovery of the tubercle bacillus in 1881 it was soon recognized that it was infection by this organism which produced virtually all of the serious chest complications in miners.

It became apparent shortly after the turn of the century that persons with silicosis are extremely susceptible to tuberculosis. In addition, it was also observed that persons with tuberculosis of the lungs whether active or "healed" are poor risks for work in silica exposure since they are likely to develop active tuberculosis or silicosis or a combination of the two. The establishment of the miners' Phthisis Bureau in South Africa in 1916 began an era of attack on tuberculosis and silicosis which has continued to the present.

As a direct result of the earlier efforts in South Africa, steps were taken to set up a similar program of examining applicants and miners in Ontario. In 1928 the Mining Act was amended to require the compulsory pre-employment and annual examination including chest x-ray of all persons in Ontario mines exposed to silica dust for at least fifty hours per month. The legislation came into effect in January 1929 and miners' chest examining stations were opened by the Workmen's Compensation Board in all mining camps.

Since that time applicants found to have x-ray evidence of present or past tuberculosis of the lungs are rejected for silica exposure work. In addition compulsory annual examinations are conducted on all miners in order to remove from the exposure area any men who develop tuberculosis. The significance of this action is that 'poor risk' men are prevented from entering exposure in the mines and those miners developing tuberculosis are removed from exposure as early as possible and treated. This is of benefit not only to the men with the infection but also to their fellow workers who might contract tuberculosis by working and associating with them.

Since compulsory examinations began there has been a very significant drop in the numbers of new cases of silicosis which have developed. Of course, it would be folly to attribute this entirely to the medical examinations because over the same period of time much has been done to decrease dust exposure by engineering methods. In addition, tuberculosis in the general population has gradually declined due to a vigorous public health program. The improvement is due to the combined effects of these and probably to other factors.

In addition to the above, drugs for the effective treatment of tuberculosis became available about 1950 and shortly thereafter the prophylactic use of these same drugs was made available to men with silicosis. Since 1957 most silicotics have been given a course of the same drugs which are used to treat tuberculosis in an attempt to prevent this serious and often very disabling complication. A study of silicotics given these drugs as a preventive measure up to December 1967 revealed a dramatic drop in the development of active tuberculosis among those treated and an accompanying decline in the numbers of silicotics dying from tuberculosis.

McIntyre aluminum powder

The use of inhaled aluminum powder (McIntyre) as a prophylactic agent against the development of silicosis was introduced into the Ontario gold mines in 1944. It has now been dispersed in change rooms for 25 years and therefore is deserving of mention.

To begin with it should be noted that its use is based on earlier experimental work in animals where it was shown to prevent or retard the development of silicosis under carefully controlled conditions. It was, therefore, in the hope that similar benefits would follow its use in humans, that its dispersal in change houses was initiated. It should further be noted that aluminum prophylaxis has never been claimed to be a substitute for good dust control and careful medical selection and regular examination of miners. It must be looked upon as a supplementary measure which may benefit miners by re-

tarding or preventing the development of silicotic lesions in the lungs. The ultimate proof of its effect in humans is still not certain but there are some definite observations which can be made at this time.

To begin with there is no evidence that the inhalation of aluminum powder in the concentrations used has produced ill effects. This statement is based on a great many physical examinations of silicotic and non-silicotic miners and on a significant number of post mortem examinations, performed on the lungs of silicotic and non-silicotic miners. There is likewise no evidence to suggest that aluminum powder inhalation enhances the development of tuberculosis.

When one considers that the average gold miner who develops silicosis (and this is a very small percentage of those at risk) requires about 25 years of elapsed time from first exposure to diagnosis, it is at once obvious that the prophylactic use of aluminum has still not significantly exceeded that time. If aluminum powder does in fact prevent or even retard the development of silicosis a further period of observation is still required before such effects can be documented. Unfortunately, there is no specific group of gold miners in Ontario which might be looked upon as "controls" against which to compare the experience of those who have inhaled aluminum powder as a prophylactic. In addition, the number of newly developed silicotics in the gold mines is now so small that it would require a very dramatic change before a firm conclusion could be drawn. It should be kept in mind, however, that if aluminum powder has had a beneficial effect this has occurred during a period when the engineering-medical efforts to control silicosis have also been carried out. It may well be impossible to identify which has been the most effective.

Polyvinyl-pyridine-N-oxide (PVNO or P204)

The problems of disabling pneumoconiosis which motivated the research with aluminum powder in Ontario in the 1930's have also been vigorously approached by a group of research workers in Western Germany since 1953. The magnitude of the West German problem can be realized from the fact that there are presently about 700,000 men employed underground in coal mines and some 85,000 receiving workmen's compensation payments for disabling pneumoconiosis.

This research work has been carried out at the state sponsored Medical Research Institute of Silicosis and Air Hygiene at the University of Dusseldorf. In 1960 the present director of this institute published a paper on the beneficial effects in animals of polyvinyl-pyridine-N-oxide, a highly polymeric powder which is readily soluble in water. This substance is more simply referred to as PVNO or by its test number P204.

Professor Schlipköter's observations are summarized by him as follows:

"1) PVNO suppresses the experimental development of silicosis in several kinds of animals and protects the cell from quartz necrosis, ie., it annuls the cytotoxicity of siliceous dust.

2) The highly polymeric N-oxide has not only an inhibitory effect on the development of the fibroblastic reaction to quartz, but also a genuinely therapeutic one on an existing quartz silicosis. Parenteral application, particularly the intravenous injection of 100 to 200 mg. of PVNO per kilogram of body weight, achieves the retrogression of an advanced silicosis in the animal experiment.

3) PVNO markedly improves the bronchial clearing of the lung and also reduces the SiO_2 contents of the lymph nodes, i.e., it reduces the penetration of the dust through the relatively dust proof barrier between the alveoli and the interstitium. An increased elimination of the quartz dust and a delay in the transfer of dust into the parabronchial lymph nodes can be achieved by weekly inhalation of a highly diluted PVNO aerosol.

These three effects of this substance could be proved by numerous animal experiments. Since no side effects of the PNVO have been observed to date and the toxicological investigations have been concluded, the clinical testing on patients suffering from silicosis have been initiated."

Professor Schlipköter visited Canada in April 1969 and presented a preliminary review of this work. He warned that the results in animals should be interpreted with caution insofar as they might be projected to humans. He did, however, report that in January 1969 a selected group of about sixty miners with pneumoconiosis were started on treatment with PVNO in six hospitals in West Germany and Austria. In a recent personal communication to Dr. John F. Paterson he indicated that this clinical trial is continuing and is under the direction of Dr. Grundmann. As yet no reports concerning the results have been published.

In addition to the trial to ascertain the value of PVNO in treating established pneumoconiosis, Professor Schlipköter advised that permission was granted by the West German Ministry of Economics early in 1969 to institute a prophylactic trial of PVNO among the underground workers at one coal mine. The trial is to take the form of the inhalation of the powder for one hour once a week. To date no reports have been forthcoming regarding this trial.

Much interest has been stimulated by this work and it is hoped that within the next year or two more will be known concerning the effectiveness of PVNO.

Results of preventive measures

It is only by a careful study of the experience among miners that the effectiveness of preventive measures can be assessed. The need for a statistical study designed to prepare and analyse data concerning the incidence, prevalence and progression of silicosis in the mining industry in Ontario was recognized over twenty years ago by a committee appointed by the Honorable Prime Minister in 1949. As a direct result of recommendations made at that time a Medical Statistical Unit was established under the Workmen's Compensation Board. Since that time this unit has not only reconstructed, from available records, the silicosis picture in Ontario from the earliest examinations in 1928-1929, but has maintained complete records on the examinations of all miners and former miners and has at appropriate intervals

TABLE II Deaths-silicotics (after S. W. McIntosh, March 1970)

Period died	No.	Ave. age	Due to tuberculosis		
			No.	% Total	Ave. age
1926 — 1929	42	43.3	33	78.6	43.4
1930 — 1934	79	47.9	61	77.2	46.8
1935 — 1939	101	50.7	70	69.3	49.0
1940 — 1944	125	52.8	87	69.6	51.9
1945 — 1949	163	58.8	92	56.4	57.7
1950 — 1954	160	62.4	59	36.9	60.0
1955 — 1959	193	65.1	43	22.3	65.1
1960 — 1964	171	67.9	19	11.1	68.5
1965 — 1969	178	69.8	6	3.4	72.2
1970 — 1974					

TABLE III New 5 ratings from 1927 to 1969[1] by period of first Ontario exposure (excluding original examinations) (after W. C. Wheeler, B.A.)

Period of First Ontario exposure	Ontario Exposure only	Exposure Outside Ontario	Total
Before 1910	7	51	128
1910 - 14	132	71	203
1915 - 19	124	59	183
1920 - 24	236	107	343
1925 - 29	189	147	336
1930 - 34	49	57	106
1935 - 39	27	28	55
1940 - 44	6	12	18
1945 - 49	3(1)	4 (1)	7(2)
1950 - 54	3(1)	16 (5)	19(6)
1955 - 59	5(5)	25 (21)	30(26)
1960 - 64	—	2 (1)	2(1)
1965 - 69	—	—	—
Other (2)	—	11	11
Total	851	590	1,441

(1) To September 30, 1969.
(2) Men with no exposure in Ontario.

published reports. The figures referred to today are as up to date as possible and are taken from a brief report prepared in October 1969. This has been kindly made available by W. C. Wheeler, B.A., Chief Statistician of the Workmen's Compensation Board of Ontario.

Figure 3, of this report (Fig. 5 here) shows the numbers of newly diagnosed cases of silicosis based on x-ray evidence, per thousand miners in dust exposure. It is apparent at a glance that an overall steady decline in the incidence of new cases has occurred. The upper solid line represents all miners and the lower broken line those who have mined only in Ontario. Both lines indicate considerable improvement since 1930 but the difference between the two indicates the added risk involved for men who have mined elsewhere where conditions probably were less well controlled. There can be no doubt that the situation has improved tremendously and in my opinion this is the direct result of the preventive measures which have been taken.

Table III from Mr. Wheeler's report shows the same data in a different form. The declining number of new silicotics is very clearly demonstrated when they are considered by period of first exposure. I draw your attention particularly to the small number of cases which have developed in men who have begun mining since 1940. The improvement in the situation is obvious and it is hoped that it will continue but this will depend in large part on the care with which the preventive measures are maintained and possibly improved.

Table II is a most informative tabulation drawn up by S. W. McIntosh. It supplies further evidence of the effects of the preventive measures even in men who have developed silicosis. It shows dramatically how miners with silicosis are living as long as their non-silicotic counterparts in the general population of the province. It also shows that the numbers of silicotics dying from tuberculosis of the lungs has dropped from a devastating 78% to 3.4% in the years 1965-1969. While this latter figure is extremely good it should be interpreted with no complacency when we note that the death rate for the general male population of Ontario in the 60-69 age group during 1968 was only 10.3 per 100,000 and that it was considerably lower in men under 60. The continuing medical supervision and the prophylactic use of anti-tuberculous drugs still appear to be very strongly indicated in men with silicosis.

I wish to draw particular attention to Figure 5 with respect to the adverse trend in incidence since 1962. This in my opinion can be accounted for by a new and important factor. In the mid 1950's uranium mines came into operation in two areas of the province — Bancroft and Elliot Lake. The native rock in the Bancroft area contains in the order of 10% free SiO_2 whereas in the Elliot Lake area is in the order of 70%. As one would expect the Bancroft area mines have not produced any cases of silicosis.

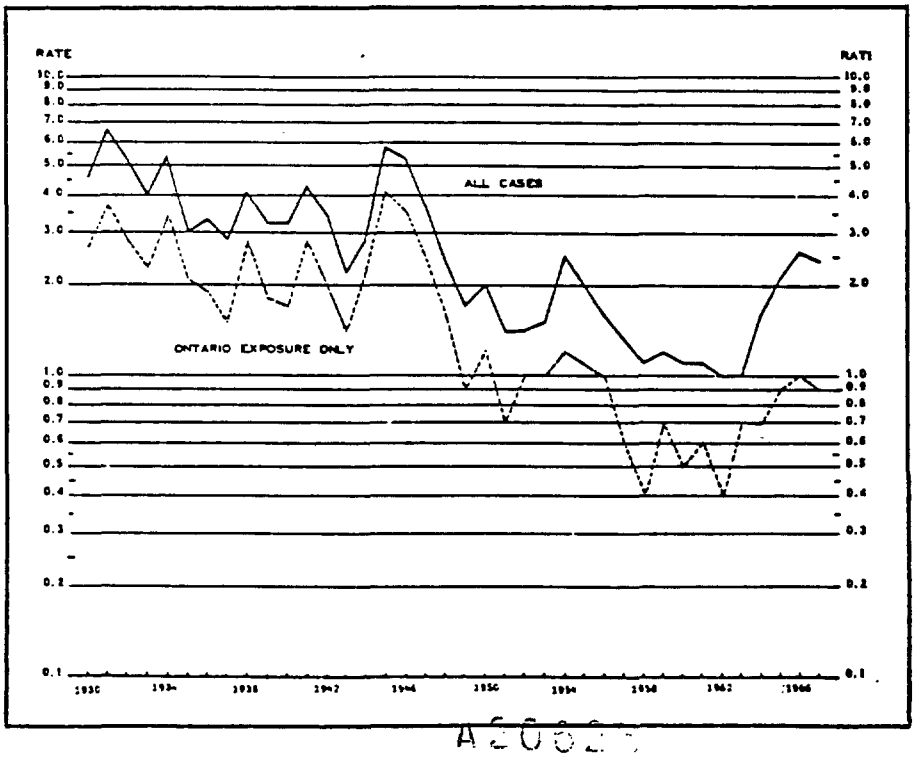
On the other hand 35 of the last 58 new cases of silicosis in the province have had some and a few have had all of their exposure at Elliot Lake. These men are indicated by the numbers in brackets in Table III which I have taken the liberty of superimposing on Mr. Wheeler's table.

The pathological findings based on examination of small portions of the lungs of seven of these men have shown a type of pathology which while possessing some of the classical changes of silicosis also show other changes which are of a different nature. This pathological picture is something not previously encountered in Ontario silicotics.

It is not considered that radioactivity in the inhaled dust is modifying the picture as similar changes have not been observed in the lungs of silicotic miners from the radioactive dusts inhaled in the mines in Joachimsthal, Czechoslovakia, St. Lawrence, Newfoundland and the Colorado plateau of the United States.

We do know that exposure in the Elliot Lake area can produce silicosis in 5-8 years and that, therefore, relatively young men can be affected. We also know that in many of the cases the lesions seem to become stationary within a few years after exposure ceases. It would appear, therefore, that a modified type of silicosis results from this exposure, which requires further investigation. It also is obvious that vigorous dust control measures

Fig. 5 New 5 ratings per 1,000 mine employees in dust. All cases, and Ontario exposure only 1930-1967 (after W. C. Wheeler)



are, required to meet the problems created by this exposure.

Conclusion

The facts mentioned in this paper are strong evidence that a great deal has been achieved in Ontario in the prevention of disabling silicosis and in the improved expectancy of life and the freedom from tuberculous complications among our miners. Only through the continued efforts using all available means will this situation continue. In fact renewed efforts should be put forth by all concerned in an attempt to further protect the well being of the miners. As long as new cases of silicosis are being produced there is no reason to feel satisfied.

In this respect the engineering control of respirable dust is of paramount importance and the engineer is obviously a person who must face this problem with every means at his disposal. □

Industrial noise control

By C. H. WOOD*

■Noise has been with us for a long time but the question of noise control in industry has received a great deal of publicity only recently. Noise is now being classified as a type of pollution which is to be cleared up just like other forms of pollution. Most of the information available on how noise problems are to be corrected is presented in the form of charts or graphs generally showing noise spectra giving decibel levels on a base of frequency. Unfortunately, this information is of little use to operating personnel who are more familiar with physical things that can be seen, touched and measured. Noise cannot be seen or touched so it is an intangible condition. However, it certainly can be heard.

The purpose of this paper is to put the characteristics of noise and the means of solving noise problems into readily understandable terms. First, to define and demonstrate noise, and then to show how to get rid if it.

Noise, for the purpose of this discussion, consists of minute fluctuations in the ambient air pressure. These pressure pulses travel through the air and register on our ear drums. We hear this sensation as noise. The frequency at which these pressure pulses occur varies very widely, but the frequencies that the human ear can sense vary from about 50 cycles per second to about 15,000 cycles per second, or Hertz (Hz). The ear is most sensitive in the range from about 250 to 10,000 Hz. The amount of pressure fluctuation is very small and is impractical to measure with normal pressure measuring equipment. Noise is measured on a sound level meter and the unit of measurement is the decibel. Now most readers have all heard this type of information before, but would you know a decibel if you heard one? And just what is frequency?

Well, for those who are musical, middle 'C' on the piano is 261 Hz and 'A' is 440 Hz. The hum from small transformers and from fluorescent lighting fixtures is 120 Hz. Some common sounds might be worth mentioning. When you just crack open the vent on your car at 60 mph, the hiss is primarily 500 Hz. The whine of a jet engine inlet is primarily high frequency in the 5,000 cycle range, while the rumble of a freight train is mostly in the medium to low range of 200 Hz. So much for frequency. The loudness of a sound is measured in decibels, and to give you some idea of general overall Db levels . . . That in most rooms is about 42 Db, and that hiss from your car vent is about 80 Db. A teenage daughter threw a party recently and the noise level in the living room was generally from 110 to 115 Db.

A demonstration of sounds

Various frequencies to which the ear is sensitive are readily demonstrated by means of proper sound producing and measuring equipment. Pure tones are given to acquaint the ear with sensations produced by this or that frequency. However, in practice pure tones rarely exist. A demonstration readily shows the difference between a pure tone of 1000 Hz and a typical power saw having its sound mostly in the 1000 Hz band. While these are similar on the frequency meter, the sound to the ear is quite different. Another useful demonstration is to reproduce sounds of various decibel levels. The reason for this demonstration is that on occasion when discussing a proposed enclosure with a client we comment that it would reduce the overall level from 90 to 80 Db. This has no real meaning unless he knows what 90 Db and 80 Db sound like.

In addition to showing frequency and noise levels, another demonstration is designed to give consideration to getting rid of industrial noise problems. (The basic problem is that noise bothers people). The logical conclusion then is that in any new installation try to group the noisy equipment in areas that are removed from people. Put the noisy MG set, compressor or other equipment off in a little concrete house by itself and put the people somewhere else. In those installations where this is not possible the usual approach is to build a house around the noise source or a house around the people it is bothering. In the case of air moving equipment, silencers can be designed to handle most ducting noise problems and enclosures can control radiated noise.

Let us first consider enclosures. To do this we must first think about the nature of sound, then select the type of enclosure construction best suited for the particular application. I already mentioned that noise consists of minute pressure fluctuations.

It can be seen from demonstrations that a very stiff or a very heavy material will act as a good barrier. Soft or very thin materials are not good. It is possible by laminating certain materials to obtain a sound barrier much superior to that which would be obtained by the individual materials used separately. For instance, 3 inches of glass fiber has a sound transmission loss of about 3 Db at 1000 Hz, 18 gauge steel sheet has a transmission loss of about 100 Db for a total of 13 Db. If, however, these are laminated so the glass wool is under some degree of compression, the transmission loss can be as high as 30 Db. To demonstrate this we have a noise source and a variety of enclosures such as those of plastic and regular panel construction.

Design of enclosures

An enclosure, by its very nature restricts access to the equipment it encloses. Where access is required only for maintenance or inspection, it is usually possible to design the enclosure to provide the necessary noise reduction and at the same time have enough doors or other openings to provide the required access. Enclosures around fans, pumps, com-

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