OCCUPATIONAL HEALTH EFFECTS OF AIRBORNE
COAL DUST AND MINE GASES

by
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ABSTRACT
The last 40 years has seen a dramatic reduction in the prevalence of pneumoconiosis through improved mining engineering associated with continuing medical surveillance of those at risk. Presently the dust particle count is being correlated with gravimetric dust standards. Chronic Bronchitis and Emphysema are also coal mining related diseases.

Methane is the commonest of a number of coal mine gases. Oxygen deficiency symptoms may result from high gas content of mine air. The explosive nature of methane-air mixtures is well known.

Mine gases will always present a potential threat to safety.

COAL DUST: COAL MINERS PNEUMOCONIOSIS

It has been long recognised that the occupation of coal mining has been associated with the development of a chronic disabling chest disease. "Miners Phthisis", "Black Lung", "Anthrocosis" and "Colliers Asthma" were some of the names given to this disease by the early medical investigators who knew little about the precise nature of the condition.

The disease is now universally known as "Coal Miners Pneumoconiosis".

It was not until the 1920's with the advent

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of the widespread use of chest x-rays, and some preliminary post mortem studies, that some basic knowledge was gained as to the nature of the disease.

The British Medical Research Council began more systematic research in 1936, and research still continues today in many coal producing countries, including Australia.

Coal Miners Pneumoconiosis results from the retention of coal particles in the tissue of the lungs where gas exchange takes place; the alveoli. Not all the dust that is inhaled reaches the alveoli; larger particles by virtue of their inertia do not negotiate the tortuous, branched, conducting airways and are deposited in the upper airways from where they are subsequently eliminated in the next 24-48 hours. Only particles with aerodynamic diameter of 0.5 to 7.0 microns are likely to reach the alveoli, and even then, many of these particles are likely to be exhaled rather than deposited. Of those particles which are deposited in the alveoli, most are removed over a period of weeks and months by a special clearance mechanism. It is those particles that are still retained that cause pneumoconiosis. Special cells called macrophages move this retained dust to special localised areas to form aggregates of coal dust. If sufficient particles are retained over many years they may form focal nodules.

These nodules are stellate shaped and can be seen at post mortem and on chest x-ray.

Since 1948 the Joint Coal Board has been

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The Aus. I.M.M. Illawarra Branch Symposium,
conducted periodic medical examinations on all
coe miners at 2-3 year intervals. The examina-
tion includes a chest x-ray which is assessed
for any evidence of pneumoconiosis.

An International Classification of Pneu-
moconiosis, devised by the International Labour
Organisation is used to quantify the chest x-rays.
This code not only helps with the clinical
assessment of pneumoconiosis in individual
mines, it also assisted with the surveillance of
the effects of occupational exposure in
specific mines and areas and also forms a code
for epidemiological research.

The grading is based on four profusion
grades (0, 1, 2, 3) with three finer sub-
divisions in each, 12 grades in all. (0/-, 0/0,
0/1, 1/0, 1/1, 1/2, 2/1, 2/2, 2/3, 3/2,
3/3, 3/-)

There are three size grades:-

"P" Nodule less than 1.5 mm
"Q" Nodule 1.5 mm - 3 mm
"R" Nodule 3 mm - 10 mm

There needs to be considerable dust
accumulation (x-ray category 2 or worse) before
pneumoconiosis causes any disability, and
generally at least 20-30 years exposure is
necessary to reach such a state. This disability
is manifested as shortness of breath on exertion
and limitation of work capacity.

There is no medical or surgical treatment
for pneumoconiosis, so the approach is based
on preventive medicine, with compensation for
those who have already been affected. Fortunately
there has been a dramatic decline in the
prevalence of pneumoconiosis in the New South
Wales Coal Mining Industry (Table 1).

Since 1948, the overall prevalence
(category 1 or worse) has declined from 16 per
cent to 1.6 per cent. The prevalence of
category 2 or worse (i.e., the stage where
symptoms can occur) has declined from 4.5

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* Artifact due to changes to ILO classification

per cent to 0.06 per cent. These figures reflect
the advances made in mining engineering with
better dust control being achieved by improved
mine ventilation and water sprays on mining
machinery.

Miners who had been dust affected were
compensated out of the industry. The Joint Coal
Board pre-employment medical examinations have
sought to screen out new entrants with pre-existing
pneumoconiosis that has developed as a result of
working in other countries.

MONITORING OF DUST HAZARD

Since 1943 a programme to monitor airborne
dust concentrations in New South Wales Coal Mines
has been in operation. This has been based on
a particle – counting technique, using the Owens
dust pump, to count the numbers of particles in
the 1 - 5 micron (respirable) range. The current
particle count standard is 175 particles per cc.
Although this statutory limit has worked well,
as evidenced by the declining prevalence figures
the Owens instrument is now almost obsolete. It
is proposed to change to a system of gravimetric
sampling. This will follow British and United
States practice.

Extensive research in the United Kingdom
has shown that the mass of respirable dust is a
better index of the hazard to health than the
dust particle count. It was also shown that a time
average concentration rather than a short period

The Aus. I.M.M. Illawarra Branch Symposium,
maximum level is more significant in terms of effects on the lungs.

The Standing Committee of Dust Research and Control of the Joint Coal Board has been evaluating a number of gravimetric dust samplers to determine the precise type of gravimetric sampling to introduce and the initial standard to set.

As pneumoconiosis has been successfully controlled with the current particle count standard of 175 particles per cc (1 - 5 micron range) it would seem logical to attempt to maintain the gravimetric equivalent.

The gravimetric equivalent has been determined by a series of side-by-side measurements in 20 collieries throughout New South Wales. The instruments used were the Owen Dust Counters, the SIMPDES personal sampler, the MSA model G personal sampler, the MRE 113A gravimetric sampler and the SIMSLIN. Regression analysis over all mines enabled the calculation of a figure of \(3.14 \pm 0.11 \text{mg/m}^3\) (95% confidence limits) gravimetric SIMSLIN optical reading corresponding to a particle count of 175 particles/cc. This is the closest equivalent; other samplers are long term and 175 particles/cc over a shift was equivalent to:

- \(3.98 \text{mg/m}^3\) MRE 113A
- \(2.69 \text{mg/m}^3\) MSA
- \(3.97 \text{mg/m}^3\) SIMPDES

Personal sampling is recommended for all mining, bord and pillar and longwall, as this is the best estimate of exposure and the most reliable index from which to calculate risk of pneumoconiosis.

**OTHER RESPIRATORY DISEASES**

It is now generally recognised that coal mining is also related to the development of two other respiratory diseases: Chronic Bronchitis and Emphysema. The relationship is strongest for Emphysema, with a weaker, but definite association for Chronic Bronchitis.

The association of coal mining and these two diseases has not always been accepted by various research workers around the world. Confusion and dissension have occurred because various non-occupational factors - notably smoking - are known to be associated with these two diseases.

Chronic Bronchitis is a condition which is associated with the proliferation of the mucus producing cells in the air conduction vessels. This leads to increased resistance to airflow on exhalation. Miners with this condition have a productive cough and they become short of breath on exertion: "Rhonchi" (whistling sounds) can be heard in the chest on physical examination. Spirometry testing shows a characteristic "obstructive" pattern. It cannot be seen on a chest x-ray, but can be measured at post mortem by measuring the ratio between the width of the mucus gland layer and the total width of the bronchial vessel wall - this is known as the Gland/Wall Ratio.

Emphysema is a disease that results in the enlargement and destruction of groups of alveoli. This causes a reduction in the elastic recoil of the lung on exhalation which produces premature closure or collapse of airways. The main symptom is shortness of breath on exertion. It can be detected on chest x-ray when greater than 20% of the lung is involved, and can be readily seen at post mortem. Spirometry tests show an obstructive pattern.

The Joint Coal Board's Medical division has conducted extensive research into the occupational related respiratory diseases of coal miners. This research has been based on a post mortem study of the lungs of several hundred deceased miners. Post mortem pneumoconiosis, emphysema, and chronic bronchitic findings have been correlated with the antemortem clinical and x-ray findings that were obtained during the periodical medical examinations. This series is unique, and the work of The Joint Coal Board...
has demonstrated the occupational association of coal mining with Chronic Bronchitis and Emphysema (Leigh et al., 1982, Leigh et al., 1983).

**MINE GASES**

**OXYGEN**

Oxygen is essential for the biochemical reactions that the body requires to function. Oxygen deficiency can occur in poorly ventilated areas in mines when oxygen is consumed by the oxidation of coal, the combustion of coal (fire and heatings) and by humans trapped in a confined space. Oxygen is normally present in air in 20.93% concentration. The clinical picture depends on the extent of the oxygen deficiency:

- 16-14% O$_2$: Breathing and pulse rate increased. Exertion becomes more difficult. The ability to think clearly and maintain attention is diminished.
- 14-10% O$_2$: Judgement becomes more faulty. Emotions are aroused with abnormal readiness. Muscular efforts lead to rapid fatigue.
- 10-6% O$_2$: Nausea and vomiting appear. Inability to perform muscular movement. Pulse increases rapidly. Loss of consciousness may occur.
- Less than 6% O$_2$: Respiration consists of gasps and then fails. Convulsions are followed by death.

**METHANE**

Methane is a colourless, odourless gas that occurs in all coal mines. It is not poisonous in itself, but it can cause asphyxia when its concentration is sufficient to displace oxygen and cause an oxygen deficiency.

This can occur dramatically in sudden methane "outbursts". Its main danger however occurs when it forms an explosive mixture in air. Any ignition can propagate to a much more violent coal dust explosion, and cause injury or death by blast trauma, burns or carbon monoxide poisoning.

**CARBON MONOXIDE**

Carbon monoxide forms in mines after fires, explosions, heatings and in small amounts after shotfiring. Of all deaths during colliery explosions 80-90 per cent are caused by carbon monoxide poisoning.

Carbon monoxide toxicity is related to its ability to disrupt oxygen transported by blood. Oxygen is transported in the blood as a combination with the haemoglobin molecule. However carbon monoxide preferentially binds to the site on the haemoglobin molecule where oxygen usually binds and forms a complex known as carboxyhaemoglobin. The affinity or "combining power" of carbon monoxide for haemoglobin is about 250 times that of oxygen, therefore relatively small concentrations of carbon monoxide can cause high concentrations of carboxyhaemoglobin.

The clinical effect depends on the percentage of blood saturation. Symptoms of throbbing headaches, nausea, weakness and incoordination occur with greater than 20% blood saturation. Death occurs after 50% saturation. Treatment involves moving the affected individual from exposure and administering oxygen. Ideally oxygen should be administered at an elevated atmospheric pressure in a hyperbaric oxygen chamber if available.

**CARBON DIOXIDE**

Carbon dioxide is produced in mines as the product of oxidation and combustion of coal, and as expired air from miners. In some mines it is given off from the strata in blowers and outbursts in a nearly pure condition.

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Carbon dioxide exerts toxic effects on the human body. Unconsciousness and death can occur when concentrations exceed 10%. At lesser concentrations, it causes headache and marked increase in the depth and rate of respiration. Concentrations above 5% in air are also usually accompanied by appreciable lowering of oxygen content, which increases the serious effects.

OXIDES OF NITROGEN
These occur in the exhausts from diesel engines and also in the fumes from shotfiring and other explosions.
The most dangerous of these is nitrogen dioxide, which has a reddish brown colour and an acrid smell. It is very poisonous and a concentration of 200 parts per million can lead to pulmonary oedema. Pulmonary oedema is a flooding of lung tissue by body fluid in response to chemical irritation of that tissue. The onset of pulmonary oedema can be delayed up to 24 hours after exposure, so that a person may feel well when he/she finishes work and later becomes ill.

HYDROGEN SULPHIDE
Hydrogen sulphide is produced in the vicinity of heaters involving high sulphur coal. It is at least as poisonous as cyanide, and like cyanide acts by blocking the cellular utilisation of oxygen by the inhibition of the enzyme cytochrome oxidase.

Exposures to low concentrations (20-50 p.p.m.) cause irritation of the eyes, lungs and respiratory tract. In higher concentrations (greater than 500 p.p.m.) it is rapidly fatal.

CONCLUSIONS
The last 40 years has seen a dramatic improvement in the prevalence of pneumoconiosis through improved mining engineering associated with continuing medical surveillance of those at risk.

Mines gases will always present a potential source of danger, but this can be minimised by good mining practice, well designed self rescuing equipment, and by a preventive educational approach at the individual level.

REFERENCES

DISCUSSION

J.M. ANSELL (Dept. Industrial Relations): Following up, once again, this oxides of nitrogen, 5 parts per million from one diesel are accepted as being a normal standard. Are there any figures for any growth of the pulmonary oedema, in such concentrations of say 20 parts/million or 35 or even 50 for any extended periods? In local modern mines there could be 10 diesels travelling inbye and its' quite possible that men are being exposed to such quantities as 35 or even 50 parts per million for extended periods.

R.K.S. GRIFFITHS (Cessnock District Medical Officer, Joint Coal Board): The figure I had for the part which produces pulmonary oedema is 200 parts per million which gives you considerable latitude above the statutory limit at present. But is is an extremely toxic gas. Did that answer the question? I'm not quite sure about lower levels.

A.J. HARGRAVES (Hargraves Mining Engineering Pty. Ltd.): In speaking about the carbon monoxide and the oxides of nitrogen for instance, they were toxic and carbon monoxide has a chemical effect on the blood and oxides of nitrogen attack the tissues of the lungs and so on. You say that carbon dioxide is toxic and the effect that has is over ventilation at 5% or more. Does it have any other effects? Is it sufficient to call it toxic because it has this physiological effect?

R.K.S. GRIFFITHS: Taking the matter a little further, not only does the chemical composition of the gas change with the passage of time but the isotopic composition of the carbon in the respective gases changes with time. There are a lot of unknowns about seam gas emission and it is useful that Barnes, deeply involved in seam gas analysis in the Illawarra is geared to measure Helium whenever he wishes. It would be useful to pursue these matters in collaboration, but in the mean time the figures of "partitioning" of mixed gases in seam gas emission, apparently with the coal somewhat as a chromatographic column holding some gases back will be forwarded to Dr. Jeger.

R.K.S. GRIFFITHS: It was stated that a concentration of 200 p.p.m. was necessary to induce pulmonary oedema, a figure well in excess of the statutory limit. This figure is, nevertheless, not clear cut. Whether any effects may occur with lower concentrations is uncertain.

A.J. HARGRAVES: About carbon monoxide and oxides of nitrogen for instance it was stated that they are toxic as carbon monoxide has a chemical effect on the blood and oxides of nitrogen attack the tissues of the lungs and so on. It was stated that carbon dioxide is toxic and the effect that this has is on ventilation with concentrations of 5% or more. Does it have any other effects? Is it sufficient to call it toxic because it has this physiological effect?

R.K.S. GRIFFITHS: The toxicity of carbon dioxide is complex. Carbon dioxide combines with water in the body to form carbonic acid, which in turn interferes with the body's pH. The body's pH is controlled within pretty strict limits. It is normally around 7.4 and the lowest it can drop to, without death, is about 7.2. The highest it may go is about 7.6. If the acid-base balance of the body is disturbed, it destroys the effectiveness of many of the thousands of enzymes in the body, which are essential for a host of chemical
reactions necessary to sustain life. The symptom of a throbbing headache is related to the direct effect of carbon dioxide on the blood vessels supplying the brain. It causes them to increase in size and that causes headaches. Carbon dioxide has a physiological effect on the respiratory centre. About 1% carbon dioxide causes a doubling in the respiratory rate and 5%, triple the respiratory rate. There are specific receptors in the body which very carefully analyse the concentration of carbon dioxide in the body and respond rapidly by increasing the respiratory rate. This is done in an attempt to blow off the excess carbon dioxide. The faster the rate and the depth of respiration the more carbon dioxide that is lost and the more oxygen that is taken in. Well that's alright for normal air, but in an environment of excess carbon dioxide more carbon dioxide is inhaled, which further increases the respiratory rate. The body is not designed to exist in a carbon dioxide environment.

M. CARR (Dept. Industrial Relations): Does smoking lead to a predisposition for either pneumoconiosis or emphysema or chronic bronchitis? Secondly the paper says that a blood saturation of carboxyhaemoglobin in excess of 20% causes severe symptoms. Would it be true that people who do smoke are less likely to be affected in the initial stages of contamination by carbon monoxide in a mine atmosphere than those who are virtually free of carboxyhaemoglobin because they're non-smokers and may have a lower tolerance?

R.K.S. GRIFFITHS: Concerning the relationship of smoking to pneumoconiosis, emphysema, or chronic bronchitis in coalminers, quite a lot of research has been done around the world on this point and there have been a few differing opinions. The studies by the Joint Coal Board which have involved the correlation of the clinical findings during life with post mortem pathology have shown that the people who smoke have a slightly lower incidence of pneumoconiosis. No-one is quite sure why this is so; perhaps smokers tend to cough more and thus tend to clear the coal dust they inhale. As far as chronic bronchitis and emphysema are concerned, it has long been recognised that they are related to smoking. It has only recently been accepted that the coal mining environment per se can cause emphysema and chronic bronchitis - previously it was attributed solely to smoking. Smoking would probably be a much bigger factor in producing chronic bronchitis and emphysema in coal miners than coal dust exposure would be, but nevertheless coal dust is also important in the aetiology. Concerning carbon monoxide poisoning in smokers it is felt that smokers would be more readily affected because they've already converted a percentage of their haemoglobin to carboxyhaemoglobin and further exposure would tend to push the level up even higher. They should be affected more quickly than non smokers.

B. HAM (M.I.M. Holdings): Could a comparison be made of dust levels in both bord and pillar and longwall operations? In any cases of pneumoconiosis reported over the last few years, what sort of split in relation to those types of mining methods is there?

R.K.S. GRIFFITHS: There hasn't been much utilisation of longwall mines in Australia in the past. They will be more common in the future, and, that's one of the reasons for the proposal to introduce gravimetric sampling. As a generalisation, the dust exposure to miners appears to be higher in longwall mining systems than in the bord and pillar type mining. This perhaps, does partly explain the difference in our prevalence rate of pneumoconiosis compared

to that of the U.K., ours is quite considerably lower. The gravimetric sampler should produce more meaningful results than the currently used Owens pump. The experimental use of gravimetric sampling has only just begun. Alf Hewitt would probably have an idea of the dust figures obtained from the use of personal gravimetric samplers in the longwall and continuous miner units.

A. HEWITT (Joint Coal Board): The Joint Coal Board recently carried out trial gravimetric sampling in three mines on the South Coast, Westcliff, Coalcliff and Corrimal. Westcliff and Corrimal were selected because of the longwalls. A six weeks trial has been completed at these mines and some results from the continuous miner units were higher than from longwalls, both the longwalls sampled during the trial have come out with good results. At one longwall not one sample exceeded three milligrams per cubic metre which is the proposed standard for New South Wales. On the second longwall during the six week period only one sample exceeded three milligrams per cubic metre. The stage 2 of the trial will be completed by 17 or 18 May, then a report will go to the Joint Coal Board and probably will be released to the industry. It will be June or July before all the results are tabulated. Following the trials it is expected that the Standing Dust Committee will recommend to the Joint Coal Board that New South Wales go on to gravimetric sampling. Queensland has been on gravimetric sampling for about 4 or 5 years now. The recommendation in New South Wales is that every continuous miner unit be sampled every production shift once every 12 months, and each longwall unit every six months, on every production shift. That's the recommendation now being worked on but there has been no legislation yet and it hasn't been fully decided if New South Wales will be going on to gravimetric sampling.

C.H. MARTIN (C.H. Martin and Associates): The Joint Coal Board commenced all its medical operations about 1948. The graph shown exhibited a dramatic decrease in pneumoconiosis from then to 1958. That decrease seemed to be out of proportion with the technological changes in ventilation and such things. Why was the drop so sudden - was there an extraneous cause also?

R.K.S. GRIFFITHS: When the Board's medical scheme came into being in 1948 a system of compensation was introduced for miners who had been shown to be affected with pneumoconiosis, the large drop in the percentage is the effect of the compensation out of the industry of these people on a pension. Now the industry has dropped down to a low level of around 1.6% total pneumoconiosis. Further elaboration on that figure of 1.6% that includes all grades of pneumoconiosis, and the majority of that percentage is made up of people with very minimal disease. Most would be asymptomatic. The fact that the percentage is remaining low is the indication of the effectiveness at modern dust control procedures.