

Should we have paid more attention to occupational lung disease in South Africa?

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South Africa is faced with a public health catastrophe due, in part at least, to the mining activity, which laid the foundation of our economy. Since the discovery of diamonds around Kimberley in 1870 and gold on the Witwatersrand less than twenty years later, mining has been the mainspring of our economy. As a direct result of the need for large numbers of unskilled workers, and the recruitment methods developed to ensure that this need was met, the migrant labour system became deeply entrenched in southern Africa. In the mines millions of men – many women and some children – have worked in dangerous and dusty conditions.

Dust levels the same over 50 year period

There have been three very high risk exposures in South African workplaces: silica, asbestos and tuberculosis. The technology change in the 1890s, from hand drilling to drills powered by compressed air, and the sharp rise in levels of dust (containing silica) that followed, led to lethal lung disease among the skilled miners from Europe working in the Transvaal gold mines. The South African Miners' Phthisis Commission heard evidence in 1902 from Mr. Thomas Pryce Rosser, a shift-boss at the Wolhuter Deep (1). He was asked 'In your opinion is it quite possible to allay all dust in a mine?' to which he replied 'I think so, simply by meeting it on the spot where it is created.'

Surgeon-General William Crawford Gorgas, at the time one of the two foremost public health practitioners in the world, repeated Tom Pryce Rosser's opinion a decade or so later (2): 'The hygiene of this disease is obvious, that of laying the dust.' Work done in South Africa by Beadle between 1956 and 1970 led him to conclude that there was little evidence of a decline in dust levels between 1938 and 1969 – a period of 31 years during which the industry was highly profitable and major expansion into new and even more profitable areas took place.

Evidence submitted in 1994 to the (Leon) Commission of Inquiry into Safety and Health in the Mining Industry satisfied the commissioners that occupational disease had not been adequately controlled by the industry, that there had been a failure to collect and use data effectively, and that dust levels

had remained roughly the same over a period of about fifty years (3). It is probable that some simple analysis of the trends in occupational lung disease (prevalence or incidence) might have stimulated intensive preventive activity decades ago.

Contemporary evidence from field surveys in a Botswana village (Thamaga) (4) and an Eastern Cape magisterial district (Libode) (5) show that about one in five former miners have pneumoconiosis. A similar prevalence was found among currently employed gold miners over the age of 40 with more than 20 years service (6). PATHAUT data (data from the pathology database at the National Institute for Occupational Health in Johannesburg) show that about a third of black gold miners dying in service in 2005 had silicosis (7). The prevalence in mostly retired white miners (25.4%) was not markedly different from that among working black miners (31.6%).

The most recent study of a group of retrenched miners returning to Lesotho confirms the existence of a very serious burden of progressive and inter-related occupational lung diseases, and supports the opinion that the social damage due to uncontrolled exposure of a very large workforce to silica-containing dust is manifest as an epidemic of tuberculosis (8).

Ultimate of asbestosis exposure

In 1931 the medical officer at an asbestos mine in the De Kaap valley near Barberton completed his study of the health effects of chrysotile on the workforce, in particular on workers in the mill (9). In the mill 'the concentration of dust in the atmosphere ... is at all times excessive, and frequently sufficiently so as to render indistinguishable objects at a distance of a few yards. The mill works continuously day and night with the exception of Sundays. Thus the dust is never allowed to settle'.

He concluded that 'exposure to high concentrations of asbestos dust produces definite early symptoms and physical signs. These symptoms and physical signs present a characteristic clinical picture.' About twenty years later Sir Richard Doll showed that asbestos dust exposure and lung cancer were associated (10). At the end of the 1950s South African workers in Johannesburg and Kimberley showed beyond reasonable doubt that malignant pleural and peritoneal

mesotheliomas followed exposure to blue asbestos dust in the workplace and in the environment surrounding the mines and mills of the Northern Cape (11).

The mining and milling of asbestos continued and proved to be a profitable enterprise. Dust levels remained high. In 1949 Dr. Schepers, an official of the Silicosis Medical Bureau in Johannesburg described the children jumping up and down in the asbestos sacks at Penge Mine (12). He believed 'these children to have had the ultimate of asbestos exposure.' In 1970 Mr. R.E.G. Rendall collected 286 personal dust samples from underground and surface workers at Penge Mine (13). The highest dust exposure was found to be among the workers 'hand tamping' – that is compressing the final product into sacks by hand. The average exposure of the six workers sampled was 326.7 fibres per cubic centimeter of air in their breathing zone. The 'ultimate of asbestos exposure' in the same job, in the same place, twenty years later!

South Africa mined and milled almost the entire world production of amphibole (blue and brown) asbestos and almost all of it was exported. The present position in respect of malignant mesothelioma in South Africa is unclear. The position in England and Wales is representative of the situation in the industrialized countries of the northern hemisphere. In the years 1968-78 (11 years – both genders) there were 1860 deaths due to malignant mesothelioma in England and Wales. Current figures indicate that this number of men (only) die annually from the tumour.

Tuberculosis, a “model” disease

Annik Rouillon wrote a very important essay in 1979 – at the time she was the Executive Director of the International Union against Tuberculosis (14). This is the opening paragraph: 'Tuberculosis, a “model” disease. This is how I would summarise tuberculosis over the last century and particularly over more recent decades. It is trite to say that rarely, if ever, has a disease played such a role in the history of humanity and of clinical, experimental and social medicine.'

In 1920 the incidence of new cases of tuberculosis in South Africa was 40 per 100 000 of the population per year. There were 3000 new cases in that year. In 1960 the rate was 359 and there were 60 000 new cases. The rate had increased nearly tenfold and the number of cases roughly twenty-fold – a neat demonstration of the effect of population growth on incidence rates. During the next two decades the population continued to increase, but the number of new cases of tuberculosis stayed roughly the same – in 1970 64 000 and in 1980 55 000 cases. As a result there was a gratifying reduction in the incidence of new cases which in retrospect seems to have been accepted at face value. This was followed by a sharp

rise in the number of cases to about 70 000 in 1990 – an increase of more than 25%.

The geographical breakdown of the numbers of new cases of all forms of tuberculosis notified between January and December 1990, and the calculated annual incidence per 100 000 population, show three startling anomalies (15):

1. The calculated rate for the Transkei was 10.4 – 345 notified cases in a population of 3.3 million. This rate is less than the rate for white, American born, citizens of the USA, and is less than 4% of the rate for the Ciskei.
2. The rate for Bophutatswana was 9.4 – 158 cases in a population of 1.7 million. This rate is less than that for whites in the USA and less than a fifth of that for Kwanabele.
3. The rate for Lebowa was 41.4 compared with 93.4 for Gazankulu and 81.5 for Venda.

If the rate for the Ciskei is used to estimate the likely number of cases in the Transkei, the rate for Kwanabele applied to Bophutatswana, and the mean of the rates for Gazankulu and Venda applied to Lebowa, an extra 10 000 cases seems possible, raising the total to nearly 85 000. This is an increase of more than 50% in a decade. In passing it is worth noting that in the (roughly) 40 years (1952-91) since treatment for tuberculosis began to be developed the number of cases per annum had increased more than threefold.

Recently published figures suggest that South Africa now has more than 300 000 new cases of tuberculosis per year, and that the rate is now above 500, or 100 times the current rate in the USA. Gorgas, himself an American, told us in 1914 that 'I am inclined to think that for the future, present conditions continuing, tuberculosis will cause you more trouble among natives than does pneumonia at present' (2). South Africa has, in addition, more than five million HIV positive individuals. By even the most optimistic foot-rule the health service is faced with a formidable task. Given unlimited resources, sustained political will and intense staff commitment the control of these diseases will take many decades.

What is a possible way forward? I have been intrigued by the response of the public health service in the USA to the rise in the number of new cases of tuberculosis which followed the spread of HIV infection. My personal experience in the Midlands Province of Zimbabwe between 1963 and 1974 persuaded me (but not many others) that tuberculosis was eradicable, and that even with limited resources rapid results could be obtained. The situation in New York City described in the opening paragraphs of an article by Frieden et alia in the New England Journal of Medicine is one which most South African health professionals will recognize (16). The fact that a rapid result can be obtained may come as a surprise to many.

The fact that the national response to the modest rise in the incidence of tuberculosis in the latter half of the 1980s led to a sustained decline in tuberculosis is further evidence that the methodology developed for tuberculosis control, and the tuberculosis service developed as a result, are enduring assets.

My proposal is to upgrade the tuberculosis service by introducing regional tuberculosis officers, stabilising the staff of existing tuberculosis units and reverting to a vertical service, to which the HIV control and ARV distribution functions can be added.

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