Adler Museum of Medicine
Faculty of Health Sciences, University of the Witwatersrand, Johannesburg

The Adler Museum of Medicine was founded in 1962 and was situated in the grounds of the South African Institute for Medical Research, Johannesburg. It is now housed at the University of the Witwatersrand’s Medical School Campus in Parktown, Johannesburg.

In June 1974 the Museum’s co-founders, Drs Cyril and Esther Adler, presented the Museum to the University of the Witwatersrand which named it the Adler Museum as a token of the esteem in which the founders were held by the University. In addition, the University bestowed the degree of Doctor of Laws (honoris causa) upon Dr Adler and the degree of Doctor of Philosophy (honoris causa) upon Mrs Esther Adler. Until Esther Adler’s death in 1982 she was the Museum’s Honorary Curator while Cyril Adler acted as Honorary Director of the Museum. From 1982 Dr Cyril Adler was appointed by the University as Director/Curator of the Adler Museum, a post he held until his death in 1988.

1975 saw the inception of the Adler Museum Bulletin, the brainchild of Mrs Rose Meltzer. Mrs Meltzer produced the first edition single-handedly and she continued to edit it until her retirement in 1991 and was editorial consultant until her death in 1992.

The Museum contains interesting and invaluable collections depicting the history of medicine, dentistry, optometry and pharmacy through the ages. Items of medical historical interest on display include microscopes and other scientific instruments, early bleeding and cupping equipment with an exquisitely crafted incision knife, ceramic pharmacy jars dating back to the 17th century, a collection of bone china and ceramic feeding cups, some dating from the 18th and 19th centuries, an early 19th century wooden handled amputation set in a wooden case, diagnostic and surgical instruments, treatment apparatus such as one advertised as ‘Patent magnetic electrical machine for nervous diseases’ used by Queen Victoria to ease her rheumatism (19th century) and the first electrocardiograph machine (1917) used in the Johannesburg General Hospital, the original artificial kidney machine used in South Africa, early anaesthetic apparatus, ear trumpets and brass ear syringes (early 20th century), hospital and nursing equipment and medical ephemera.

There are reconstructions of an African herb shop, a patient consulting a sangoma (traditional healer), and a 20th century Johannesburg pharmacy, a doctor’s consulting room, a dental surgery, an operating theatre and an optometry display of the same period. A history of scientific medicine is augmented with displays of several alternative modalities. Other attractions range from a reconstruction of a patient being treated by the famous Persian physician Avicenna to an exhibition of early electro-medical equipment, and a collection of rare iron lungs.

A showcase containing new acquisitions to the collection is constantly changed as donations are received. The objects displayed provide an insight into the range and diversity of the collection.

In the foyer outside the Museum are panels relating to the history of the Cradle of Humankind (Sterkfontein and environs) and a display of replicas from the site give visitors a fascinating glimpse into this world heritage site.

The Museum has a rare book collection and a significant history of health sciences reference library. An archive arranged by subject matter is housed in the library. Biographical information relating to thousands of medical and allied health professionals is available for research purposes which includes photographs, notebooks, academic certificates, records, personal papers and memorabilia of prominent health professionals and academics.

The Museum arranges public lectures, tours, temporary exhibitions and provides excellent facilities for health sciences historical teaching and research.

Opinions expressed in this publication are those of the authors concerned and do not necessarily reflect the views of the Editors, the Editorial Board or the Board of Control of the Adler Museum of Medicine.

Application forms for membership of the Adler Museum of Medicine can be obtained from the Curator, Adler Museum of Medicine, 7 York Road, Parktown, 2193. Telephone and fax: (+11) 717 2081.


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ADLER MUSEUM BULLETIN

7 York Road, Parktown, 2193

Editors
JCA Davies MB BS (London)
Rochelle Keene BA(Hons)(Witwatersrand)

Email
Adler.museum@wits.ac.za
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In the light of the fact that South Africa continues to wrestle (unsuccessfully) with what may be one of the most serious epidemics of tuberculosis ever, is it eccentric to re-examine the relevance of sanatoria for the isolation of acute or chronic communicable diseases? Is it appropriate to use tuberculosis as an example and to select as a starting point a scholarly article by Professor Leonard G Wilson entitled The Historical Decline of Tuberculosis in Europe and America: Its Causes and Significance? Professor Wilson is Emeritus Professor of the History of Medicine at the University of Minnesota, and the article was published in 1990 in the Journal of the History of Medicine and Allied Sciences. Serendipitously, a copy of the article has just been sent to me by Professor Jock McCulloch, who recently delivered the AJ Orenstein Memorial Lecture at Wits, and has now started to explore the history of tuberculosis in southern Africa. Thus the provenance of what I am about to reiterate.

About the time Professor Wilson’s article was published I was listening with some apprehension to talks explaining the introduction of directly observed short course chemotherapy for pulmonary tuberculosis, accompanied by overt criticism of what we had been doing in Zimbabwe (successfully). Prior to the arrival of epidemic immunosuppression many of the individuals presenting with pulmonary tuberculosis were thin, sick, coughing and breathless, and many of their initial chest X-rays showed extensive bilateral cavitiated disease. About a third were slide positive (and inevitably culture positive), a third were slide negative but culture positive, and a third were closed cases, including all the non-pulmonary tuberculosis and all the children. When the changed regime of chemotherapy was introduced it signaled the end of cough mixture, vitamins, supervised feeding of the very ill, neglect of regular weighing, emphasis on slide positivity, restricted use of X-rays (particularly in case-finding), minimum stay in hospital, and the need for post-treatment surveillance, among others.

For a doctor who had worked in Zimbabwe this was an indigestible bolus of some size, implying that a great deal of money had been spent needlessly. In Zimbabwe all individuals thought to have tuberculosis were admitted to hospital and transferred to a sanatorium for careful investigation to establish the diagnosis, characterize the organism, exclude coincident disease, and in due course explain in detail the disease and its treatment. Recovery was carefully monitored. The original rules for prolonged hospital stay were modified in the early 1960s to allow discharge to be related to the extent of disease, the rate of recovery and access to adequate care and therapy at home or at work. No patient was allowed to cough incessantly for the lack of a dose of Linctus Codeine or Creosote Mixture, and all were encouraged to reject the belief that tuberculosis was caused by a serpent lodged in the chest. If the truth be known the 20th century chest hospitals in the major centres in Zimbabwe differed very little from those in Europe and North America fifty or more years previously, except in the important matter of effective chemotherapy.

The words repeated by many of my colleagues, “It is the drugs that do the work”, became the orthodoxy. Professor Wilson’s paper is not about cough mixture or vitamins or weight gain – it is about communicability. It is communicability which is the salient feature of tuberculosis. Prominent among the explanations for the steady decline of tuberculosis prior to the introduction of chemotherapy was the improved socio-economic and nutritional status of the population as a whole in most countries in Europe and North America, and this is no doubt correct and important. But the common inference that nothing else mattered is not true. It is in fact simplistic and damaging. My allegiance to the sanatorium idea is based on my training, which began in 1949, ended in 1955 formally, and continued in hospital appointments until 1958. The hospital appointments included, apart from house jobs, a junior post in a unit studying the merits of several modalities of treatment for bronchial carcinoma, followed by two years as a registrar in chest medicine and surgery at Preston Hall Hospital, part of the British Legion Village near Maidstone in Kent.

A place in the sun?
Professor JCA Davies
Reading Professor Wilson’s carefully argued exposition has been a revelation, and has taught me that my knowledge of history is inadequate in a field in which I ought to have been better informed. The origin of my (and many others’) explanation for the decline of tuberculosis in England and Wales was the work of McKeown and Record in their studies of the reasons for the growth of the population since the 17th century. McKeown and his colleagues concluded in a number of publications that the rapid increase in the British population was due to the decline in the death rate rather than an increase in the birth rate, and that fewer deaths from communicable disease were a significant factor in the decline.

The most important communicable cause of death was tuberculosis and they concluded that “specific preventive or curative therapy had no effect”. As is well known, they attributed the decline in tuberculosis to socio-economic change on a wide front. They, like me, had not read the history carefully enough. The first authority cited by McKeown and his co-workers, and whose work they appear to have ignored, was (Sir) Arthur Newsholme, the first Medical Officer of Health of Brighton (1885-1908) and later the Chief Medical Officer of the United Kingdom (1908-1919). Wilson deals exhaustively with Newsholme’s work while he was in Brighton, and my brief précis is lifted from Wilson’s paper:

“… Newsholme explained the decline of tuberculosis as the result of a reduction in exposure to infection brought about by the segregation of consumptives in England in Poor Law infirmaries”. In a chapter in his book entitled *The Elements of Vital Statistics* he concluded “… historically, before its rationale was understood, and when hospital treatment was being given solely for humanitarian reasons, this hospital treatment played an important part in reducing the death rate from tuberculosis”. It is clear that Newsholme extended the argument to make a number of important points, including the fact that modern doctors should not think in terms of the modern tuberculosis sanatoria, which they were busy denigrating, but in terms of the unintended epidemiological benefit of the provision of institutional care for the poor. The very poor frequently have tuberculosis and this may be not only the result, but part of the cause, of poverty. Death from tuberculosis is associated with the expectoration of very large numbers of mycobacteria, resulting in widespread and long-lasting contamination of domestic environments. In a series of publications from 1901-1923 Newsholme dealt with many aspects of the control of tuberculosis. Professor Wilson’s absorbing account of the discussion and research related to the reasons for the decline of tuberculosis can be downloaded from [http://jhm.as.oxfordjournals.org/](http://jhm.as.oxfordjournals.org/).

To cap it all Newsholme made a mathematical model demonstrating the concordance between the proportion of cases of tuberculosis segregated with the rate of decline of mortality due to tuberculosis. With due acknowledgement of my debt to Professor Leonard Wilson, and his debt to Sir Arthur Newsholme, here is the model in brief:

“After examining most carefully the influence of urbanization, the degree of overcrowding in housing, the general well-being of the people, and the influence of sanitary education and sanatoria in various countries, Newsholme demonstrated conclusively that the decisive factor in the decline of phthisis mortality in England and Wales had been the segregation of phthisis patients in institutions, principally workhouse infirmaries and insane asylums. At Brighton 20 percent of the total number of consumptives were segregated in the workhouse infirmary, and if their average period of stay, which was one-third of a year, represented one-ninth of their period of infectivity, estimated at three years, then their segregation prevented the spread of just over 2 percent of the total phthisis infection. The segregation of phthisis patients in workhouse infirmaries, therefore, ought to have produced an annual decline in phthisis mortality of about 2 percent and in fact from 1871 to 1905 the average decline in phthisis mortality had been less than 2 percent [because this was not uniform national policy]. Thus the influence of segregation was fully adequate to produce the recorded historical decline”.

Albert Debeila, with whom I worked for several years in Sekhukhuneland, during a discussion of the tuberculosis problem there said: “When you get to where the patients are [their homes], they have nothing”. Given the poverty and state of health of many of this country’s tuberculosis patients, and the very high death rate among them, a spell in a well-run sanatorium might qualify as a place in the sun and be welcomed – and a place in the sun where conceivably the chemotherapy might work more effectively if it was not viewed as the only horse in the recovery stakes. Brighton rules? OK?

REFERENCE

Dust, Disease and Politics on South Africa’s Gold Mines

AJ Orenstein Memorial Lecture, Wits Faculty of Health Sciences,
10 April 2013

Professor Jock McCulloch
International Development Program, School of Global, Urban and Social Studies, RMIT University,
Melbourne, Australia

ABSTRACT

The 20th century opened and closed with a crisis on South Africa’s gold mines. In between those two points the Rand mines were believed to lead the world in preventing silicosis, the oldest and most intractable of the occupational diseases. For most of the 20th century policy makers in Western Europe, North America and Australasia looked to Johannesburg, and in particular to its state sanctioned system of medical surveillance, as a model of how to make workplaces safe. Since majority rule a number of major studies have confirmed that rather than being safe South Africa mines are hazardous and it is probable that they have always been so. This paper explores how an illusion of safety was created and perpetuated.

The 20th century opened and closed with a crisis on South Africa gold mines. The first crisis, which lasted from 1902 to 1912, was finally resolved by a series of Mines and Miners’ Phthisis Acts and by the provision of the world’s first compensation scheme for hard rock miners. The second crisis, which has been running for over a decade, is currently being played out in courts in London and Johannesburg. That litigation may result in more than 200 000 claims for compensation from former miners and their families. In between those two points, South Africa’s gold mines were presumed to have conquered the problem of silicosis.

Miners’ phthisis or silicosis is one of the oldest and most common of the occupational diseases. Its emergence as a serious health threat at the end of the 19th century coincided with the introduction of industrial tools, and more especially the use of power drills and gelignite in hard rock mines. The inhalation of free silica dust can lead to scarring or fibrosis of the lungs. The classic symptoms are breathlessness or “air hunger”, a persistent cough and weight loss. Silicosis usually has a long latency period and, especially in its early stages, it may be difficult to diagnose. The disease is insidious and it will progress even after a worker has left a dusty occupation. In both the industrial and developing worlds silicosis kills thousands of workers each year. The World Health Organization’s (WHO) International Programme on the Global Elimination of Silicosis, launched in 1995, aims at the reduction and eventual elimination of what remains an incurable disease. South Africa is a signatory to that initiative.

Silicosis by itself can be fatal; it also greatly increases an individual’s chances of contracting pulmonary tuberculosis. That has been true especially in southern Africa where the rural communities from which migrant labour was drawn had little prior exposure to infection. The use of the term miners’ phthisis rather than silicosis in the South African legislation until 1946 was recognition of the synergy between the two diseases.

When mining began on the Witwatersrand in 1886, the high silica content of the host ore and the use of pneumatic drills and gelignite produced clouds of fine dust which could destroy a miner’s lungs within a few years. To overcome that hazard, the South African industry invested heavily in dust suppression technologies and medical surveillance.
In 1912 South Africa became the first state to compensate silicosis as an occupational disease. Four years later gold miners with pulmonary tuberculosis, a recognised sequela of silicosis, became eligible for compensation awards. From the 1920s successive governments in Pretoria were extolling the success of an important industry in preventing a life threatening disease which had plagued hard rock mines for centuries. South Africa’s system of medical review and compensation was much admired throughout the British Empire and a number of its features were adopted in Australasia. It also featured prominently in science and policy debates in France, Belgium, Germany, Italy, Japan, and the USA.

There were good reasons why South Africa’s mines had such an enviable reputation. The data published annually from 1916 by the Miners’ Phthisis Medical Bureau and its successor, the Silicosis Medical Bureau, was one of ceaseless improvement in which the Witwatersrand led the world in preventing lung disease. In the period from 1917-20, for example, the reported silicosis rate among white miners was 2.195 per cent. By 1935 it had fallen to 0.885 per cent. The rate for black miners was even lower. In 1926-27 it was 0.129 per cent and in 1934-35 it had fallen to 0.122 per cent. In the period from 1946 to 1947 the silicosis rate among black miners was 0.178 per cent and in 1948-49 it was 0.201 per cent.

The end of minority rule brought a dramatic change to South African science. It also brought a change to the perception of the industry. In 1994 the Leon Commission of Enquiry into health and safety found that dust levels on the gold mines were hazardous and that they had probably been so for more than fifty years. Subsequent research by Anna Trapido, David Rees, Tony Davies and Jill Murray at the National Institute of Occupational Health (NIOH) in Johannesburg and by Neil White, TW Steen, Rodney Erlich and Jonny Myers at the University of Cape Town, has identified a pandemic of hitherto undiagnosed and uncompensated disease. Those studies put the silicosis rate in living miners at between 22 and 30% or over one hundred times higher than the official rates in 1915, 1935 or 1955. Jill Murray’s post mortem research estimates that up to 60 per cent of miners will eventually develop silicosis. The previous under reporting raises the spectre of tens of thousands of compensation claims.

In addition to silicosis, the mines have played a major role in the spread of tuberculosis. According to current estimates the incidence of tuberculosis in South Africa is among the highest in the world, with the rate among miners being ten times higher than for the general population. David Stuckler has found that even allowing for HIV/AIDS, miners in sub-Saharan Africa, and especially those working on South Africa’s gold mines, have a higher rate of tuberculosis than does any other working population. The system of oscillating migration which has been a feature of the mines since they opened has led to the transmission of disease to rural communities and across national borders. There is evidence that the gold mines have much the same influence in spreading tuberculosis to the general population as have prisons in the former Soviet Union.

The current litigation against AngloGold Ashanti, Gold Fields and Harmony raises the question as to how the current disease rates can be reconciled with the data published between 1916 and 1994. There is no evidence, for example, that since majority rule work conditions have deteriorated dramatically, and while diagnostic methods continue to improve, at no point in the past twenty years have they been revolutionised. The mine workforce has become more stable since 1980, with migrant workers spending longer periods in continuous employment. But labour stabilisation has long been a feature of the gold mines and it alone cannot explain the discrepancy between the current and past data. Rather than any dramatic increase in silicosis and tuberculosis, it seems likely that the mines have always been hazardous and that the data published prior to 1994 is false. To understand how that was possible this paper will examine the system of mine medicine as it evolved on the Witwatersrand after 1912.

**THE MEDICAL SYSTEM**

South Africa’s gold mines were distinguished by their depth, their scale and the size of their labour force. Those features encouraged the dominance of a few mining houses which, because of their importance to employment, foreign exchange and state revenue, had great political influence. On the global stage the mines were just as important. In 1930 South Africa’s mines produced over 60% of the gold upon which the stability of the Western financial system rested. The mines employed a huge number of migrant workers drawn from rural communities inside and outside South Africa’s borders. The work force consisted of a unionised white labour aristocracy employed in supervisory
The labour system was racialised yet there is little reference to race in the silicosis legislation or in the mines acts. Instead, those laws were framed in terms of two categories: miners (whites) and native labourers (blacks). White miners were designated as skilled and native labourers as unskilled. They did different jobs for different rates of pay and for different contract periods. They also had very different levels of dust exposure. Whites and blacks worked under different compensation regimes, they were subject to different forms of medical surveillance, and they received different medical care: there were sanatoria for white silicotics and repatriation for blacks. The nomenclature of miners and native labourers pervaded both official discourse and South African science; it also influenced how the industry was viewed from the outside. The minutes from the International Labour Organisation’s (ILO’s) 1930 Silicosis Conference suggest that the overseas delegates were oblivious to segregation on the mines. So too were the editors of the ILO’s 1937 review of the South African compensation system.17

There were pre-employment medicals to select recruits who were free of pulmonary disease, and periodic examinations to diagnose early cases of silicosis and tuberculosis. Exit medicals were used to identify those entitled to compensation and prevent the spread of tuberculosis to labour-sending areas. White miners were examined by specialists at the Miners’ Phthisis Medical Bureau and they also had access to private physicians. From 1916, the screening of whites involved a clinical examination, a chest X-ray, and the taking of medical and work histories. Black miners were examined by mine medical officers or at the WNLA compound. In theory the system was comprehensive and Dr LG Irvine, long time Chairman of the MPMB, proudly told delegates to the ILO sponsored Silicosis Conference in Johannesburg in 1930: “The whole system draws around the mine natives a serviceably close net of opportunities for detection of cases of silicosis and tuberculosis”.18 South Africa’s success in preventing occupational diseases was discussed seven years later at the ILO’s Conference in Geneva. One delegate, Dr Langelez, represented Conference opinion when he commented that the South Africa examinations were so stringent that if they were adopted on Belgium’s mines it would lead immediately to a labour shortage.19 The reality was rather different.
The mine medical service consisted of a small number of senior officers supported by a group of young graduates who used the mines as a stepping stone to a better career. By the mid 1930s a full-time medical officer on a medium sized mine would be responsible for at least 6 000 men. The workload of the five full-time physicians at the WNLA hospital was also crushing. In addition to responsibility for more than 250 hospital patients each doctor examined between 300 to 1 200 black miners a day. During the 1930s there was a rapid increase in the number of black miners (from 214 155 in 1931 to 359 710 in 1940-41) but there was little increase in the size of the medical corps. The annual labour turnover which remained constant at around 90% added to the case loads. While the technologies used for diagnosis changed over time, the workloads of medical officers did not.

The industry was dependent upon migrant labour and the standards of fitness applied by medical officers tended to vary according to the mines production needs. One constant was the greater care taken with the entry medicals than was given to the periodic and exit examinations. In 1923 the Chamber of Mines issued a set of guidelines for the selection of recruits. Men with a weak chest, signs of tuberculosis or silicosis, pleurisy, chronic bronchitis or asthma were to be rejected. Under no circumstance were men who had been repatriated with tuberculosis or silicosis to be recruited. I have not found a parallel set of guidelines for the conduct of exit medicals.

In addition to the stethoscope, weight loss was used as a marker of early stage disease. Miners who had lost five pounds or more between weighings, or six pounds over three consecutive weighings, were given a more thorough examination. Irrespective of how the examinations were conducted, weighing was well recognised as a poor guide to diagnosis. Writing in 1927 Dr Watkins-Pitchford, the former Director of the MPMB, commented that weight loss was particularly unreliable with early stage silicosis and at best brought to light only half of the tuberculosis cases. Despite its acknowledged limitations weighing remained the system’s centrepiece until the introduction of mass miniaturization radiography.

The work environments on the Rand changed constantly. The technologies used by medical officers also changed. The most important innovation in regard to black miners was mass miniaturization radiography which was introduced in the early 1950s. The technique developed for the Chamber of Mines by Major KG Collender enabled a miniature X-ray to be taken cheaply, and quickly. Initially mass X-rays were used for selected groups, such as men with long service, and they were gradually extended to all migrant workers. The technology which promised to improve the quality of examinations was soon moulded to the imperatives of mine medicine. On average a medical officer at the WNLA would read between 800 and 1 000 films in an hour. Dr JA Louw proudly told a Transvaal Mine Medical Officers Association (MMOA) meeting in 1965 that in an hour he could review up to 2 000 plates.

The medical examinations soon created an orthodoxy about the pattern of disease. Tuberculosis was far more common in blacks than whites. The reverse was true of simple silicosis (where tuberculosis was absent) which by 1930 was fourteen times more common in white miners. The official explanation ignored the way the medical examinations were conducted and emphasised the six and twelve month contracts of black miners which protected them from continuous exposure to dust. The higher rate of tuberculosis among migrant workers was due supposedly to their racial susceptibility rather than to malnutrition in the rural areas and the unsanitary conditions in the mine compounds.

**THE CRITICS**

While outside South Africa the gold mines enjoyed a glowing reputation, the industry did have its domestic critics. The Miners’ Phthisis Commissions held between 1919 and 1952 routinely commented on the inadequacy of the medical examinations and the obstacles black miners faced in regard to compensation. The 1919 Commission was highly critical of WNLA’s medical officers for making it difficult if not impossible for the relatives of a deceased miner to receive an award. Death certificates often cited “general tuberculosis” as the cause of death when in fact a miner had died from pulmonary tuberculosis. The situation with living miners was no better. The Commission found medical examinations were poorly conducted and often there was no exit medical as required under the Act. That was so even in cases of seriously ill men who had spent months in hospital. Without a certificate a miner could not lodge a claim. The findings of the 1919 Commission were endorsed by the Young (1930), Stratford (1943), Allan (1950) and the Beyers Commissions (1952). They are also...
consistent with recent research by Jaine Roberts on former miners from the Eastern Cape. Among her cohort of 205 men, 85.3% had not received an exit examination as required by law.\textsuperscript{32}

The injustice of the compensation system is easy to quantify. In the period from May 1911 to September 1929, £1 208 015 was paid to white miners and their dependents who in addition benefited from grants in aid, free medical care, re-training and rural re-settlement schemes. There were also dowries to encourage widows to re-marry.\textsuperscript{33} From 1 May 1911 to 31 July 1929 black miners, who outnumbered whites by ten to one, received £702 036.\textsuperscript{34} In the period to 1946 a total of £24 487 000 was paid to white miners and their dependents. Black miners and their dependants received £2 million.\textsuperscript{35} That pattern was maintained.

MINE MEDICINE AND THE 1930 SILICO SIS CONFERENCE

The first silicosis crisis saw Johannesburg become the world centre for research. The leading researchers included IG Irving, A Sutherland Strachan, FW Simson, W Watkins-Pitchford, A Mavrogardato, Spencer Lister, AJ Orenstein, and Andrew Watt of the Rand Mutual Assurance Company. It was a close knit community. Scientists were employed either by the state or the mining houses with the same cast of experts appearing at the numerous commissions into miners’ phthisis. There were also regular transfers of personnel from one sector to the other. During a career which spanned the period from 1913 until the early 1960s, Dr Orenstein was variously Head of Sanitation for Rand Mines Ltd, Chair of the Chamber’s powerful Gold Producers Committee (GPC) and from 1956 the inaugural Director of the Pneumoconiosis Research Unit within the Department of Mines. He also served on a number of Commissions of Enquiry.

The 1930 Conference in Johannesburg which was sponsored jointly by the International Labour Organisation (ILO) and the Transvaal Chamber of Mines was a pivotal moment in the history of silicosis. Its sitting in Johannesburg was recognition of South Africa’s achievements in terms of research, data collection and state regulation. That data included the world’s largest collection of X-rays of a work force. The gold mines also assembled dust samples: by 1925 more than 100 000 readings were being taken annually.\textsuperscript{36} The Lancet remarked: ‘The statistics of South Africa are of the utmost importance to all countries which have to initiate legislation in connection with silicosis’.\textsuperscript{37} Travel was slow and expensive in the 1930s and it was probably the first time the leading scientists from South Africa, the USA, Germany, Belgium, Italy, the UK and Australia had met to discuss their research. The Conference resulted in a series of recommendations which influenced the direction of research and also established the Johannesburg mines as the model of workplace reform.

To the ILO, which had been founded in 1919, silicosis epitomised the new hazards of industrial production. The ILO carefully monitored the Johannesburg research and it took an active interest in the issues of prevention and compensation.\textsuperscript{38} The ILO also took a keen interest
in migrant labour and in particular migrancy in colonial Africa. Under the leadership of Dr Luigi Carozzi the ILO scrutinized recruitment regimes, hours of work, rates of pay, and the provision of medical care. Consequently there was a voluminous correspondence between the ILO, the Chamber, the Colonial Office and the administrations of Nyasaland (Malawi), Basutoland (Lesotho), Bechuanaland (Botswana) and Swaziland, all of which supplied miners to Johannesburg.

The ILO hoped the Johannesburg Conference would foster an international network of health specialists, thereby promoting workplace reform. The Chamber saw the Conference as a stage on which to publicise the safety of the mines and the achievements of South African science. In addition, there were other agendas at play. Perhaps the most important was the Chamber’s determination to secure access to migrant labour from surrounding colonial states and in particular from the tropical north. That market had been closed in 1913 because of the appalling mortality rates from infectious pneumonia.41

The initial impetus for the Conference came from Dr Luigi Carozzi of the ILO and William Gemmill, the General Manager of the Transvaal Chamber of Mines. William Gemmill regularly toured the recruiting stations and he always handled the major negotiations with Union officials and colonial administrations.42 Under Gemmill’s leadership the Chamber commissioned films and newspaper articles to counter criticism of the industry’s labour practices. In 1919 Gemmill served on the Low Grades Mines Commission which was chaired by the Government Mining Engineer, Sir Robert Kotze. In that same year South Africa sent three delegates to the foundation meeting of the ILO in Washington DC. South African employers were represented by William Gemmill. Gemmill also played a key role in the creation of South Africa’s new industrial relations system in 1923.43

At the end of the 1930 Silicosis Conference the delegates agreed on the need for a universal standard of dust measurement so that the risk in different industries could be compared. They also agreed that the best place to conduct such work was Johannesburg.44 There were no recommendations for global standards, and there was no support for external assessment of workplace safety. The delegates offered no criticism of work conditions in South Africa, nor of its migrant labour system. On the contrary, they adopted South Africa as a model of workplace reform. In his review of the Conference in The Lancet, Professor Arthur Hall referred to Johannesburg as “the mecca for silicosis researchers”. He praised South Africa’s gold mines and the South African state for leading the world in safety, medical care and compensation.45

The overseas delegates left Johannesburg believing that South Africa’s gold industry had brought silicosis under control. Disarmed by that illusion, the ILO pursued a modest reform agenda. In 1933 it revised the Convention on Occupational Diseases and in the following year silicosis was added to the Schedule of Diseases for which compensation should be paid. The ILO Conference in 1934 published a list of the trades and industries which posed a risk of silicosis and endorsed the importance of pre-employment and periodical examinations as a means of prevention.46 In the following decade there were no further Commissions in South Africa, the UK or Australia, and only a single Congressional enquiry in the USA. In each of those jurisdictions miners with silicosis were often denied compensation. The South African data continued to mislead the research community about the hazards of hard rock mining and it may well have contributed to the invisibility of silicosis as a political issue in the USA until the 1970s.47

**WHAT WAS HIDDEN**

The South African data presented to the 1930 Conference had a number of distinctive features, the most notable being the variations in the disease rates between whites and blacks. According to the official returns, the silicosis rate among whites was around fourteen times higher even though, because of their job specialisations, blacks had greater dust exposures. The obvious explanation lay in the cursory examinations conducted by mine medical officers, but that factor was not mentioned in any of the presentations, nor did it arise during discussion. Instead, the Johannesburg scientists focussed upon the short term contracts of black miners. Supposedly, after brief periods underground, migrant workers returned to their villages where they quickly recovered from the effects of dust exposure. White miners remained underground and developed silicosis. That explanation was cited routinely by the Chamber at Select Committee enquiries and Commissions. Dr Mavrogordato rehearsed the same orthodoxy at the 1930 Conference when he told his audience:
“Observation had shown that the smaller incidence of silicosis among natives, as compared with Europeans, was due to their intermittent employment.”48 Conversely, the tuberculosis rate among black miners was far higher than for whites. The official explanation focussed on poor hygiene in rural areas and the greater susceptibility of blacks to infection.49 No mention was made of the synergy between silicosis and tuberculosis. The Chamber and the Miners’ Phthisis Medical Bureau also ignored the low mine wages, rural poverty, and malnutrition which contributed to the spread of infectious disease.

Despite the voluminous data collected at Johannesburg, little was known about black miners. The Departments of Mines and Health, like the Chamber, took no interest in the post-employment health of migrant workers. Neither did they offer assistance to labour sending states keen to control the spread of tuberculosis. The Young Commission Report, which was released in 1930, found there was no information on the fate of mine beneficiaries once they had left the Transkei with tuberculosis in that year, seventy six nineteen were expected to “possibly die shortly”.51 Thirty were dead within six months and a further nineteen were expected to “possibly die shortly”.51 As late as the mid-1940s, the Chamber was not notifying the colonial administration in Malawi of black miners who had been compensated for tuberculosis we have from that period comes from 1926. Of the one hundred and ten repatriated to the Transkei with tuberculosis in that year, seventy six were dead within six months and a further nineteen were expected to “possibly die shortly”.51

In addition to the deficiencies in medical surveillance, there were more fundamental problems with the data. The science of epidemiology played no part in the compilation of the official rates of silicosis and tuberculosis. That rate was based on the number of successful claims made before the Miners’ Phthisis Medical Bureau: the disease rate was the compensation rate. There were numerous barriers which prevented black miners, who comprised 90% of the workforce, from receiving compensation and so they were grossly under-represented. The Chamber’s key committees were aware of the inadequacy of the data. The Gold Producers Committee (GPC) regularly faced protests from the administrations of Malawi, Botswana and Mozambique over the numbers of returning miners with tuberculosis who had not been compensated. Occasionally protests came from WNLAs’s own medical officers.53 William Gemmill and his colleagues participated in numerous Commissions of Enquiry into miners’ phthisis at which those issues were raised by the Department of Native Affairs.

Compensation was funded from a levy placed on the industry as a whole rather than on individual employers. Despite the falling disease rates from 1912, the compensation payments and hence the levy continued to rise, mainly as the result of lobbying by the white Mine Workers Union (MWU). By 1929 the financial burden on the industry was around £1,000,000 per annum.54 Conflict over compensation between the MWU, the Department of Mines and The Chamber resulted in most of the more than twenty Commissions and Select Committee enquiries held in the period from 1902 until 1974. The focus of those enquiries was the legislation’s retrospective reach in extending compensation to men with existing disabilities, the status of tuberculosis as an occupational disease, and the size of the levy.

THE SILENCE OF ANDREW WATT AND THE POST MORTEM DATA

While the official data showed that the mines were safe, at least one delegate to the 1930 Conference had a different view. Dr Andrew Watt is an important figure in the history of silicosis but he published little and his name is cited rarely in the scientific or historical literature. Watt was, however, much admired by his contemporaries and he played a major role in the development of ventilation technologies on the Rand.55 Dr Watt was educated at Edinburgh University and in 1902 he was appointed medical officer at the Simmer & Jack Mine in Johannesburg. The silicosis rate was very high and Watt became the first mine doctor to use X-rays for diagnosis.56 At Simmer & Jack, Watt initiated a research programme correlating miners X-rays with post mortem results. He also began a series of animal experiments, using rats and guinea pigs. Watt was a member of the 1912 Commission into Miners’ Phthisis and he conducted the X-rays which formed the basis of the Commission’s Report. Watt subsequently joined the Rand Mutual Assurance Company as chief medical adviser. His principle role was in assessing compensation claims. By 1930 he had worked on miners’ phthisis for almost thirty years.

The paper Watt presented to the 1930 Conference covers the period from the Weldon Commission in 1903 to the formation of the Miners’ Phthisis
Medical Bureau in 1916, but it contains little that is original and not surprisingly it is tucked away at the end of the published proceedings. Watt offers no criticism of the mining industry. On the contrary his tone is conciliatory: “The Witwatersrand has led the way in the study of this very ancient disease, because unfortunately the disease has been more prevalent here than in any part of the world, and the opportunity for research and study has been made possible by the generosity and business acumen of the leaders of the industry”.

In 1925 Watt had completed a never to be published manuscript of over one hundred pages on the history of miners’ phthisis. It is a distillation of a career’s research which contains path breaking data and observations on silicosis, and the synergy between silicosis and tuberculosis. In contrast to his conference paper, it presents a bleak assessment of the risks faced by miners.

In his manuscript Dr Watt remarks that free silica is hazardous even in the smallest quantities: therefore he doubted if water alone could make hard rock mines safe. Watt distinguishes between two forms of dust: income dust is the dust generated by each day’s work while capital dust is the dust always in circulation underground. Income dust had been greatly reduced by the introduction of water sprays but sprays had no effect on capital dust. He comments that if there was capital gas in a colliery, that colliery would not be worked. Capital dust also made it unsafe to use mine air for ventilation. Therefore to improve air quality would greatly increase production costs. Watt’s paper suggests it may be impossible to make the gold mines safe.

Like his colleagues, Watt was well aware of the relationship between silicosis and tuberculosis. That threat was even greater in South Africa than in hard rock mines in the USA or Australia where labour had greater immunity to infection. To assess the level of risk Watt conducted a series of animal experiments. His most important results came from rats inoculated with tuberculosis bacilli, and dusted with free silica. Silica was found to lower immunity to tuberculosis even in the absence of demonstrable lung damage. The results were particularly significant in animals with a high natural resistance to tuberculosis. Watt was sure that the small isolated silicotic areas in miners’ lungs may have been infective from the onset, making the synergy between silicosis and tuberculosis profound.

Reflecting on research from other parts of the continent, Watt concluded: “Experience with British and French native troops drawn from the northern tropical districts of Africa suggests that it may be suicide for a man with a negative tuberculin reaction (that is without prior exposure to tuberculosis) to enter a phthisis-producing industry”. Watt believed that it was impossible to reduce dust to a level at which fibrosis and hence tuberculosis would not occur. The mines were dangerous for white miners and lethal for migrant workers drawn from rural communities and especially for those from the tropical north.

As with the findings of the Miners’ Phthisis Commissions of 1919 and 1930, the research of Andrew Watt could well have alerted his colleagues at Rand Mutual and at the Department of Mines to a serious threat of silicosis and tuberculosis, especially for migrant labour. There was another source of information which was equally telling. On the initiative of then head of the MPMB, Watkins-Pitchford, from 1924 any miner who died suddenly was subject to a post mortem and the results published in the MPMB’s annual report. Under the Miners’ Phthisis Acts Consolidation Act of 1925 that procedure became law. Each year over five hundred miners perished in accidents and presumably most of the autopsies were performed on that random group. The MPMB data shows far higher rates of silicosis and tuberculosis than were identified in living miners. That in itself is not surprising as it had long been acknowledged that post mortems could uncover lung disease missed in a living subject. What is surprising is the dramatic difference between the two sets of data.

The sequence from 1924 until 1950 reveals a tide of disease. In 1924 the lungs of 122 white and 176 of black miners were examined. Silicotic changes were found in 97 or 79.5% of whites. Among that group 28 had been certified as free of silicosis, some within months of death. The lungs of the 176 blacks showed an even higher rate. Despite being subject to periodic examination, in 78 men the cause of death was tuberculosis without the deceased having been diagnosed during life. In another 60 tuberculosis with or without silicosis was present. In 1928 the lungs of 227 white miners and 429 blacks were examined. Of the whites 55% had compensatable disease while 81% of the blacks were affected. That trend continued, with the data consistently showing a disease rate one hundred times higher than the official figures published by the Bureau. The initial data must have shocked Watkins-Pitchford and it may explain his sudden
resignation from the SAIMR and the MPMB in March 1925.66

THE ENDMGAME

There was a great deal at stake in mine medicine. Labour stood to gain through safer workplaces and the right to compensation. No matter how much employers wanted to reduce morbidity and mortality rates, they also wanted to minimise the costs. If the disease rates were deemed by the state as too high, in addition to compensation there would have been the added burden of providing enhanced ventilation, more stringent blasting regulations and improving conditions in the mine compounds.

The mines were places of constant change. Over time they became deeper and their sources of labour shifted from South Africa to the colonial north then back again. The labour process, the technologies used to extract the ore and to diagnose miners’ phthisis became more sophisticated. The political environment was also unstable. Throughout all of that flux, in the period from the passing of the first Miners’ Phthisis Act in 1911 to the mid 1950s the conduct of medical examinations changed little. In the words of Dr GWH Schepers, who served as a specialist with the Bureau from 1944 until 1952, the examinations at the WNLMA and by the Bureau were “to meet a legislative requirement”, they were not to ensure that miners were compensated.67 Former miners with damaged lungs were excluded at the entry medicals while the periodic examinations were reduced to weighing, which was usually carried out by an orderly.68 Mass miniature x-ray plates were read in less than a second. In addition to those obvious flaws, perhaps the most telling evidence about the purpose of medical monitoring is in what was left undone.

Between 1924 and 1994 neither the Chamber nor the individual mining houses carried out surveys to see what happened to men after they left the mines. Employers also failed to warn miners about the risks they faced, and they made little effort to prevent the spread of tuberculosis to rural areas. Exit examinations were either performed poorly or not carried out. There is evidence that miners from the Eastern Cape are still not receiving an exit examination as required by law.69 Such stability over such a period is suggestive of a policy.

It is worth reflecting upon the consequences if Dr Andrew Watt had spoken candidly at the Johannesburg conference in 1930 or if the MPMB’s post mortem data had been taken up by the Department of Mines. Either would have demolished the myth of safe mines promoted by the Chamber, and at the very least encouraged the ILO to oppose the reintroduction of tropical labour. Watt’s manuscript could have shifted the research agenda to the sub-clinical effects of silica exposure and perhaps forced the SAIMR to conduct comprehensive studies of silicosis and tuberculosis in rural areas. Recognition of the complexity of the disease process could have put an end to diagnosis.
by X-ray alone and led to a surge in compensation awards.\textsuperscript{70} That in turn may have encouraged more adequate compensation regimes in the UK, the USA, Australia and elsewhere.

The mining companies presently facing litigation will no doubt cite various factors which contributed to the invisibility of disease. Silicosis is not easy to diagnose and the lack of biomedical capacity in the labour sending states of Lesotho, Malawi, Botswana, Swaziland and Mozambique obscured its incidence. While that explanation has some merit, on closer examination it tends to fall part. Since 1916 all gold miners have been subject to an elaborate state sanctioned system of medical surveillance. In addition, the industry invested heavily in research. During his annual address in June 1990, the Chamber of Mines’ President KW Maxwell commented: “Some 60 percent of the R65 million spent in 1989 by COMRO (The Chamber’s research organisation) was devoted to wide-ranging research projects directly related to safety”.\textsuperscript{71} That R65 million did not include the amount spent on pre- and post-employment medicals and on the continuous monitoring of long term employees. Such a level of investment makes it even more surprising how easily the pandemic of uncompensated disease was discovered after 1994. Anna Trapido was a PhD candidate with minimal resources when she uncovered a pandemic in the Eastern Cape, a pandemic apparently unknown to employers or the state.\textsuperscript{72}

Since the current South African litigation began the industry has made some admissions. In its 2006 Report to Society, AngloGold Ashanti acknowledged the failings of the compensation system. “Many of these former employees may not have been diagnosed as suffering from the disease (silicosis) at the time they left the industry or later, in retirement, and they may not have received due compensation from the Compensation Commissioner”.\textsuperscript{76} There is no evidence, however, that the mines are any less dangerous now than they were in 1930 or that their devastating impact on community health has been diminished. The annual incidence of tuberculosis amongst mineworkers is currently over 4 000 per 100 000. That is almost ten times the national incidence for South Africa of 550 per 100 000, a figure which is among the highest in the world.\textsuperscript{77}

The gold mines’ much admired system of medical surveillance, data collection and compensation underpinned the commercial success of South Africa’s most important industry by allowing the costs of production to be shifted back onto rural communities within and without South Africa’s borders. It is only now, more than a century after the first silicosis crisis, that those costs may at last be called in.

REFERENCES


21. On occasions it was as high as 12 000. See Dr Williams in discussion, *Proceedings of the Transvaal Mine Medical Officers’ Association*, May, 1935 Vol. XV, No.164, p. 81.


33. The dowries were designed to reduce the number of widows who, if they remained single, were entitled to life pensions.


36. Dr A.J. Orenstein, in *Silicosis 1930*, p. 32.


40. See for example ‘Employment of Natives on the Witwatersrand Gold Mines’, CO 525/173/2, the British National Archives, Kew.


43. See David Yudelman, *The Emergence of Modern South Africa*, pp. 156, 206.


53. In November 1924 Dr L. Bostock, District Manager of the WNLA at Lourenço Marques, wrote in protest to the Gold Producers’ Committee about the spread of tuberculosis amongst men returning from the mines. The issue was raised at a Mine Medical Officers’ Association meeting in Johannesburg in March the following year. See *Proceedings of the Transvaal Mine Medical Officers’ Association*, Vol. IV, No.11, March 1925.


57. Ibid, p. 596.


60. Ibid, p. 17.

61. Ibid, p. 53.

62. Ibid, p. 130.


The importance that most societies currently place on newborn babies has not always been the case. Infanticide has been practiced in most parts of the world throughout human history. Hunter gatherers and many Neolithic groups used infanticide to control their numbers so that their food supplies could support them. In Greek and Roman times little value was placed on the life of a newborn infant. Adults decided the fate of newborn infants and physically weak, sickly or abnormal infants were usually “disposed of” by exposure. Even in the case of normal infants the male head of the household could decide to accept or reject the infant depending on the gender, the number of other children, the family’s financial position etc. In Sparta in ancient Greece these decisions were made by the elders of the tribe at a public forum. Similar practices have been recorded in most parts of the world and even today infanticide is still carried out in a number of countries. Indeed, our own newspapers regularly carry stories of babies left to die or disposed of in various ways.

There is very little written in the historical records regarding special forms of care for premature babies in pre-industrialised societies. A medical historian quoted by Cone could find only two such examples. One group of Eskimos used the skin of a large sea bird turned inside out to wrap such infants and then keep them near a fire. In the other instance, the Tsonga people of southern Africa wrapped premature babies in leaves of the castor-oil plant and put them in a big pot which was exposed to the sun to keep them warm. As a result of the lack of such care in most parts of the world, the survival of very low birth weight (VLBW) infants, defined as those weighing <1500g at birth, was very uncommon until the latter stages of the 19th century.

Various types of incubators began to be developed during the 19th century, especially during the latter half, to keep premature babies warm. However, the first organised care of premature babies was initiated by a French obstetrician, Stephane Tarnier, around 1878 in Paris which led to the introduction of the first closed incubators and gavage (tube) feeding. This, together with the awareness of some infection control measures, resulted in a marked improvement in the survival of premature babies and these basic measures still form the basis of modern day neonatal care. Tarnier’s work was continued by another obstetrician, Pierre Budin, who further developed these methods of care and he has rightly become known as the pioneer of this new era of neonatal care that continues into the 21st century. While Tarnier and Budin were certainly motivated by humanitarian concerns, it seems that patriotism also played an important role. France had been defeated by Prussia in the Franco-Prussian War (1870-1871) and the war and the associated famine had resulted in a very high mortality. France had the lowest birth rate in Europe and the number of infants born in Germany at that time was twice the number of those born in France. Thus there was an added incentive to save more premature babies to ensure that France would have adequate numbers of soldiers to put into the field in future wars!

The success of Tarnier and Budin’s methods became widely known and similar units for premature babies developed rapidly in other countries of Europe and North America. There were significant advances in incubator technology and feeding techniques. However, premature babies also became a source of entertainment at fairs and exhibitions throughout Europe and the United States, beginning at the Victorian Era Exhibition, Earl’s Court, London in 1897 where the premature baby exhibit attracted up to 3 600 visitors a day. From 1903 until the 1940s, premature babies were part of the attractions at Coney Island, New York, and a relatively recent newspaper feature described how they could be seen alongside exhibits such as Violetta the Armless Legless Wonder, Princess Wee Wee and Ajax the Sword Swallower! People paid to see these babies and the man who put them on
Dr Martin Couney became rich on the proceeds. He was shunned by his colleagues as an unseemly showman, but was also credited with saving the lives of thousands of babies.\(^4\)

During the early decades of the 20th century, the main problems that were recognised in premature babies were sepsis, syphilis, anaemia and rickets.\(^2\)

Syphilis was a major problem as it was responsible for many premature deliveries and almost all premature infants who showed signs of congenital syphilis at birth died prior to the antibiotic era. A review published in 1920 and quoted by Cone reported that in St Louis, USA, 10-20\% of adult males and 10\% of married women were syphilitic and that syphilis was responsible for 3.5\% of all infant deaths in the city.\(^2\) While syphilis is no longer the killer that it was previously and various ways have been developed to reduce the prevalence of rickets and anaemia, sepsis remains a major problem in all neonatal nurseries.

The history of the use of oxygen for premature babies with respiratory distress has been elegantly described by Silverman.\(^5\) In the early 1940s it was noted that the breathing patterns of premature babies and their colour and blood oxygen levels improved if they were provided with supplemental oxygen. Further developments in incubator design made it possible to maintain high levels of oxygen in these incubators. The mortality of premature babies with respiratory disease at that time was very high and oxygen was seen as potentially lifesaving. By the late 1940s acceptance of the value of oxygen therapy was universal in the USA and many other countries. It thus became the norm to pipe as much oxygen as possible into the incubators of premature babies, even those without major respiratory disease.

The first description of retinopathy of prematurity (ROP, initially called retrolental fibroplasia) leading to blindness appeared in the literature in 1942 and, in some centres where oxygen was being used liberally, ROP was affecting significant numbers of premature babies by the mid-1940s. However, it was not until the early 1950s that the association between oxygen and ROP was suggested and this finally led to a multicentre trial in 1953-54 in the USA comparing liberal with restricted oxygen administration. The results showed that there was a strong association between liberal use of oxygen and ROP and the erroneous conclusion was drawn that ROP would not occur if the oxygen concentration administered did not exceed 40\%.

This then became standard policy and incubators were designed such that if more than 40\% oxygen was to be given, a red “flag” had to be raised as a warning. In spite of all the advances in the understanding of ROP (see below) many modern incubators still have this feature. Silverman later analysed this study and pointed out the methodological problems, the lack of statistical rigour and the failure to evaluate mortality and long term neurological sequelae properly.\(^5\) It was only five years later that reports began to emerge of increased mortality in premature infants with respiratory distress syndrome and it took even longer to discover that the rates of spastic diplegia had increased in surviving premature infants. One
later analysis suggested that for every one case of ROP prevented, 16 premature infants may have died as a result of hypoxia.\textsuperscript{6} It was only much later that it was established that it was not the inspired oxygen that needed to be controlled but the amount of circulating oxygen reaching the immature retinal vasculature i.e. a baby with severe lung disease in 100% inspired oxygen but with normal levels of circulating oxygen is at low risk for ROP while the converse is true. The story of oxygen given to premature babies is sad but provides extremely important lessons regarding the value of properly designed randomized controlled trials with long term follow up as an important component.\textsuperscript{5}

Respiratory disease thus remained a major cause of mortality in premature babies but the cause of this so-called respiratory distress syndrome or what was described microscopically as hyaline membrane disease was unknown. However, a series of studies starting in 1959 established that this was due to a lack of the surface tension lowering agent known as surfactant that normally lines the alveoli. Early attempts to produce an artificial surfactant failed and it would take another 20 years before an effective surfactant could be produced and a further 10 years for one to become widely available commercially. Advances in ventilator technology aroused interest in the possibility of keeping such babies alive with ventilatory support until endogenous surfactant was produced, typically after 48-72 hours. The first publication of artificial ventilation of newborn babies was by Smythe and Bull from Cape Town who described their experience of ventilating term babies with neonatal tetanus.\textsuperscript{7} Ventilation of premature infants with respiratory distress syndrome began in the early to mid-1960s and, although there was initial resistance to it by some, the reduction in mortality was soon evident and this led to the development of the modern day neonatal intensive care unit (NICU). From the early 1970s there was an incredible growth in the number of NICUs throughout the industrialized world and more recently in developing countries. The introduction of commercially available artificial surfactant around 1990 was probably the single most important advance over this period and the large randomized controlled trials that were performed prior to its widespread use serve as a model example of how the introduction of a new intervention should be managed. Survival rates of premature infants in modern day NICUs have improved dramatically and the concept of the limit of viability has been constantly pushed back

Handicap in surviving premature infants has been a major theme for over 50 years and remains so. One of the problems, however, is that it takes 5-10 years to collect adequate data on handicap rates and by that time, the rapid advances in neonatal care that are continually taking place may make those data inappropriate for similar premature babies born at the time the results are published! Nevertheless, as the limits of viability have been pushed back in industrialized countries with more extremely premature babies surviving, the rate of handicap in these survivors has increased over the past two decades. While these countries are able to provide the necessary medical and social services for these numbers of handicapped survivors, it would not be possible in developing countries.

**NEONATOLOGY IN JOHANNESBURG**

A neonatal service was started at the old Transvaal Memorial Hospital in the 1930s and an NICU was opened in the 1960s. Together with the rest of the paediatric service, this unit moved to the (then) new Johannesburg Hospital (now the Charlotte Maxeke Johannesburg Academic Hospital – CMJAH) in 1978 where it still is. Ventilation of premature infants in this unit was generally limited to those >1000g birth weight until the late 1970s when advances in the care of such premature infants had begun to result in better rates of survival. A neonatal unit at Coronation Hospital (now Rahima Moosa Mother and Child Hospital – RMMCH) was started after it opened in the 1940s, but a full NICU was not

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An example of a ventilated premature baby in a modern day Neonatal Intensive Care Unit
started until the late 1970s. During the 1990s the unit at the JG Strydom (now Helen Joseph) Hospital moved to and was combined with that at RMMCH.

The neonatal unit at Baragwanath Hospital (now Chris Hani Baragwanath Academic Hospital – CHBAH) was started in the early 1950s. In 1954 Kahn et al published an analysis of the first 1000 admissions to the unit and the survival of VLBW infants compared favourably with a similar study that had been done in Birmingham, England at the time. At that time all babies with birth weight <2500g were regarded as premature and it was only later that it was understood that many babies <2500g at birth were term babies who were growth restricted. Similarly VLBW infants may be premature but also small for their gestational age. It was not appreciated at the time that a large number of the CHBAH premature babies were also growth restricted and were thus more mature with less organ immaturity and, as a result, more likely to survive. In the late 1950s another important study from CHBAH showed that, although neonatal jaundice due to Rh incompatibility was uncommon, hyperbilirubinaemia and kernicterus were common, especially amongst low birth weight babies. This study also confirmed that low birth weight babies developed kernicterus at lower levels of bilirubin than those of normal birth weight and thus required exchange transfusion at lower levels to prevent brain damage from kernicterus, an important advance at the time.9

The unit at CHBAH grew rapidly, but it was not until 1979 that an NICU was opened and, due to the large numbers of VLBW infants, those <1000g at birth were not offered ventilation. Few data on survival of low birth weight babies at CHBAH were available during the 1960s and 1970s but accurate data were again collected from the beginning of the 1980s, two years after the opening of the NICU. The survival of babies <2000g at birth according to 500g categories between 1981-2 and 1995-6 can be seen in the table.10 While there had almost certainly been some impact on the mortality of those 1000-1499g by 1981-2 since the opening of the NICU in 1979, the survival improved over the next 15 years from 64% to 79%, while the survival of those 1500-1999g, already over 90% in 1981-2 improved less dramatically. What was surprising was that the survival of those <1000g for whom ventilation was not offered also improved dramatically from 14% to 32%. It would seem that the introduction of an NICU had a beneficial effect on all levels of care with positive spin-off effects for this group as well. In their analysis from the early 1950s, Kahn et al reported the survival of babies born weighing 907-1360g (2-3lbs) as 25% - by 1995-96 this survival figure had risen to over 70%, illustrating how much neonatal survival had improved at this hospital over a period of 45 years.

There were no local data available on the developmental outcome of VLBW infants and, with the dramatically improved survival of VLBW infants, neonatologists were frequently questioned about whether this was leading to an increase in

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<td>&lt;1000g</td>
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handicapped survivors, particularly relevant to communities that had limited resources to take care of such infants. For this reason a cohort of 113 surviving VLBW infants from CHBAH born in 1990 was enrolled and followed up for 18 months. There was a significant mortality during the first year of life of 13% after hospital discharge, but the rate of significant handicap (cerebral palsy, psychomotor retardation and/or blindness) was 9%, and occurred mainly in the group 1000-1499g who had required ventilatory support during the early neonatal period. The rate of handicap was similar to or lower than that being reported from industrialized countries at the time, partly as a result of the sicker and tinier babies having a higher mortality, but it was re-assuring that the vast majority of surviving VLBW infants were free of major handicap.

Artificial surfactant became available locally in the early 1990s and was used at all three hospitals. At CHBAH it was in very short supply due to its high cost in the first few years. However, as it became more freely available improvements in the mortality of infants with respiratory distress syndrome who needed ventilatory support were seen, falling from a mortality of around 50% in the early to mid-1990s to less than 30% by the end of the decade. The other major development in the 1990s was the removal of the racial classification of the hospitals with the result that the racial profile at all three hospitals became similar. A major advance in the last decade has been the administration of artificial surfactant by endotracheal tube to VLBW infants within a few hours after birth together with the provision of nasal continuous positive airway pressure (CPAP) via nasal prongs. The latter can be provided in a high care setting rather than in intensive care. This has resulted in fewer VLBW babies needing assisted ventilation and, several years ago, a decision was taken by our three neonatal units to lower the cut-off for ventilation to 900g. In addition, babies with birth weight as low as 750g are being treated with surfactant and nasal CPAP. A second major advance has been the introduction of kangaroo mother care. Once premature babies are stable and growing, this involves mothers (and sometimes fathers!) keeping their babies wrapped against the chest with skin to skin contact. This keeps the baby warm without the need for an incubator and can be done intermittently or, if the mother is able to stay in the hospital, on a 24 hour basis. This has resulted in less demand on nursing time, fewer nosocomial infections with resultant improvement in survival and an improvement in breast feeding rates of VLBW infants. A recent follow up study of VLBW babies at CMJAH confirmed the relatively low rate of major handicap even lower than that found in the earlier study showing that the further improvement in survival of VLBW infants in our units has not been accompanied by an increase in handicap.

NEONATOLOGY – THE NATIONAL PICTURE

Some dramatic successes have occurred with respect to neonates in South Africa over the past two decades. Seropositive rates for syphilis in mothers attending antenatal clinics have fallen from 15-20% to less than 2% with the result that, whereas symptomatic congenital syphilis was previously a major problem, the condition is now rarely seen. Neonatal tetanus which is still a major killer in many parts of Africa and the developing world is now also extremely uncommon even in rural areas, where it was previously a major problem, as a result of immunization of pregnant women against tetanus and clean management of the umbilical cord at the time of and after delivery.
However, significant disparities exist in neonatal care between the public and the private sectors. The most recent estimate of neonatal mortality for South Africa was 14 per 1000 live births, but the figure is less than half for the private sector which makes up around 10% of births in the country. In addition there are also significant disparities in neonatal care in the public sector between metropolitan areas and other parts of the country where NICUs are often not available due to a lack of adequately trained medical and nursing staff and equipment. One of the Millennium Development Goals set by the United Nations in 2000 was that under 5 mortality should be reduced by two-thirds from the baseline in 1990 by 2015. Amongst other factors, the HIV/AIDS epidemic has made this a difficult target to achieve for South Africa. However, neonatal mortality makes up around 30% of the under 5 mortality in South Africa and has been falling very slowly. A reduction in neonatal mortality is thus an essential component of the overall reduction in under 5 mortality.

The three most common causes of neonatal mortality in South Africa are prematurity, perinatal asphyxia and infections. Prevention of prematurity has been a long standing goal in many countries, but specific interventions to achieve this have had little or no effect on the whole. While it would be difficult to make NICU facilities available throughout the country in the near future for all premature babies who would need them, good basic neonatal care combined with the introduction of newer modes of treatment such as artificial surfactant, CPAP and kangaroo mother care for premature babies can be introduced to regional and district hospitals with appropriate training of the staff. Neonatal asphyxia rates could be dramatically reduced with a two pronged approach of improving the care of women in labour and ensuring that there are staff adequately trained in neonatal resuscitation in all facilities where babies are born. South Africa currently has extremely low rates of breast feeding and this predisposes many babies to infections not only in the neonatal period but beyond. Improvements in the rates of breast feeding and careful attention to infectious precautions as first outlined by Tarnier and Budin in Paris well over a century ago would have a major impact on mortality due to infections.

CONCLUSION

The dramatic technological advances that have been seen in neonatology over the past 50 years are likely to continue and the boundaries will continue to be stretched. However, the basic principles of care for newborn babies developed almost 150 years ago are still the foundation on which newer advances must be based and it is relatively “low tech” interventions with appropriate training of staff that will reduce neonatal mortality in those countries where it is still unacceptably high.

REFERENCES

I went to a primary school in Alexandra Township and to Tigerkloof High School in 1950. This was a boarding school in the district of Vryburg in the Northern Cape run by the London Missionary Society. From 1951 through 1953 I studied for a BSc degree at Fort Hare, which was an affiliate of Rhodes University. Its graduates received Rhodes University degrees. In 1954 I was accepted as a first year student in medicine at the University of the Witwatersrand. The competition for entry was intense and costly, beyond the capacity of most African students to pay without financial assistance. It was a moment of joy and ecstasy when I received a letter of acceptance to the Medical School and a Johannesburg City Council scholarship that paid my tuition. There were three African and five Indian medical students during my first year of Medical School, and all of us graduated. I received my medical degree in 1959.

Wits has a significant reputation in educational excellence and research. The University’s Institute of Human Evolution continues to keep Wits in the forefront of palaeoanthropological research where it has been since the discovery of the earliest hominin by Professor Raymond Dart in the North West Cape in 1924. When I entered Medical School in 1954 Professor Dart had retired, but was often seen in the anatomy laboratory assisting medical students. I remember him coming to my desk while I was looking at a histology slide of the testis. He enquired if I needed help. I said I was looking at a slide but was unable to find specialised elements referred to as interstitial cells. He looked through the microscope and shouted “testis.” I explained that I knew the slide was of a specimen of testis but I was looking for specialised cells within the testis. He responded: “You don’t have to know that.”

MEDICAL SCHOOL

Courses at Medical School were taught and supported by excellent professors with a superior ability to interact with students. These included Professor Phillip Tobias, Head of the Department of Anatomy, whose work with Mary and Louis Leakey on a jaw bone found in a gorge in Tanzania set him on a path of record breaking discoveries in Sterkfontein and recognition as a world authority in paleoanthropology. His anatomy teaching, both in the laboratory and lecture rooms, was a delight. Professor Joseph Gilman, Head of the Department of Physiology, was an energetic, vigorous personality. His course on malnutrition and vitamin deficiencies was illustrated by presenting patients from the ‘Non-European’ section of the General Hospital with signs of various nutritional and vitamin deficiencies. I remember cases of pellagra, a tryptophan deficiency. I also remember being told that scurvy was more common among sailors in the 15th and 16th centuries with mortality rates that were higher than those due to shipwreck and piracy. We learned that lives could be saved by investments in African school lunch programmes that included vitamin and iron fortified diets and pasteurised milk. Prenatal care by midwives familiar with maternal and child welfare services saves lives.

The final year of medical school was completed by passing both the written and oral examinations. One of my oral examiners was an orthopaedic surgeon from the University of Pretoria who specialised in hand injuries. He passed one of the wrist bones in my direction and said: “Name it, site it and tell me the structures related to it.” I identified the bone as the triquetrum, and named other bones related to it. He was obviously satisfied with the answer. I remember wondering why I was asked to identify a small wrist bone when I was not going to be an orthopaedic surgeon much less a hand surgeon.

A Medical Journey from Alexandra Township (Johannesburg) to Harlem (New York City, USA)

Stephen Matseoane

BSc (Rhodes), MBBCh (Witwatersrand 1959), FACOG, FACS
The results of the final examination were posted on a bulletin board just outside the conference room for all to see. I remember approaching the board and anxiously looking for my name. It was with joy that I identified my name on the pass section. There were no telephones in Alexandra to call my mother. I ran to the Alexandra bus stop in Noord Street, Joubert Park, where my father sold vetkoek [fat cakes] to passengers as they exited the bus. I told him I had passed the final medical examination and that I was now a medical doctor. We hugged and I shed tears of joy. We both got on the bus to Alexandra to share the news with my mother who was equally elated.

My mother and father joined me at the University Great Hall for the graduation ceremony led by the Vice-Chancellor of the University. I was given a certificate written in Latin. It was the happiest day of my life. I was glad to recognise the indispensable support of my father and mother who provided encouragement at every stage of my educational career. My mother and father did not complete primary education, but understood the value of education and encouraged their children to work hard on their school projects. We were proud to understand that despite apartheid and Bantu education of 1953, Wits affirmed the ideals of its principal Jan Hofmeyr that the University should know no distinction of class, race or creed in accepting students. These ideals were further confirmed recently by the then Vice-Chancellor and Principal, Professor Loyisa Nongxa, when he unveiled a memorial plaque in the Wits University Gardens in honour of its students, faculties and alumni.

When I graduated from Wits in 1959, the government passed a law called the Extension of University Education Act. The law prohibited Wits and other English-speaking Universities from accepting Africans as students except where a cabinet minister had given permission, contrary to the ideals of the university.

BARAGWANATH HOSPITAL

In February 1960 I joined the Baragwanath Hospital as an intern rotating at 3-monthly intervals through the departments of internal medicine, surgery, paediatrics, and obstetrics and gynaecology. On 21 March 1960, the hospital superintendent announced through a loud speaker that all physicians including those who were off duty needed to stay in the hospital until further notice. It soon became clear that there was a crisis when several ambulances arrived at the hospital. We were instructed to discharge home all patients who were not critically ill, and to assist emergency room staff in the triage of patients arriving from Sharpeville near Vereeniging. Many young people seen in the emergency room had gunshot wounds. They were shot in the back while running away from the police. Some of the patients were saved by the competent staff of physicians, surgeons and nurses working as teams. Regrettfully some were dead before arrival, others died in the operating rooms because of massive haemorrhage and/or shock. Sixty-nine people died and 180 were wounded. The violence spread to Cape Town and Durban. My rotation through obstetrics and gynaecology service sparked an interest in the discipline, and I expressed to the director my wish to pursue postgraduate training in obstetrics and gynaecology. I was denied the opportunity and advised to practice general medicine in Soweto or Alexandra township. I knew the outcome of the interview with the director. No African physician had previously been admitted for postgraduate training in Obstetrics and Gynecology in any of the Johannesburg complex of hospitals, including Baragwanath.

While in the library of Medical School, I saw a notice on the bulletin board that read: “Wanted medical school graduates of accredited medical schools to work as interns at Lebanon Hospital in New York City.” I saw this as an opportunity to work in New York and to pursue postgraduate training in obstetrics and gynaecology. My application to Lebanon Hospital was accepted. I prepared to write the ECFMG (Educational Council for Foreign Medical Graduates) examination in Johannesburg. I received a passport and a student visa to enter the United States in October 1960. I applied for a travelling grant on a cargo ship owned by Farrel Lines shipping company. I could not afford the cost of an airline ticket. My parents did not know my plans to go overseas. I knew they would discourage me from leaving South Africa and press me to practice in Alexandra. My passion was to pursue training in obstetrics and gynaecology overseas since the opportunity was not available in South Africa. I was reminded of the African-American spiritual called Higher Ground:

I am pressing on the upward way
New heights I am gaining every day

My heart has no desire to stay
Where doubts arise and fears dismay
Though some may dwell where these abound
My prayer, my aim is higher ground.
I received a travel grant from Farrel Lines in December 1960. I then told my parents of my plans to leave South Africa. They realised that I had been planning the trip for some time. They gave my trip a blessing. I thank them for hoisting me up as I climbed to reach higher ground.

I left Johannesburg for Durban and sailed aboard a Farrel Lines ship across the Atlantic Ocean to Charleston, South Carolina in February 1961. I took a Greyhound bus to New York City and to Lebanon Hospital. I was met by an administrator for education at the hospital. A room was assigned to me at the house staff building. I was advised to join the staff on hospital rounds the following day.

LEBANON HOSPITAL

The internship at Lebanon was a repetition of the work I did at Bara. In the second month of the medical rotation, I was on first call for the emergency room. I received a call from the emergency room nurse on the second week that I was on call. The call was about a 45-year-old white man with a sore throat and a fever. Following a history and examination I thought he had pharyngitis. I ordered routine blood tests and a throat swab for culture of the offending organism. I placed the patient on broad-spectrum antibiotics and an analgesic for pain and told the patient to return to the outpatient clinic in one week. The patient returned to emergency room two hours later with severe throat pain radiating to both shoulders and dizzy spells. I was called to re-evaluate the patient. I called a senior resident to see the patient with me. It was clear that he had suffered a myocardial infarction that was confirmed by an electrocardiogram. He was transferred to the intensive care unit for management. I was disappointed that I missed the diagnosis on the patient’s first visit. I had never seen a case of myocardial infarction in my year of training at Bara. I remembered white colleagues at Bara talking about cases of myocardial infarction among white patients they had seen in the Johannesburg General Hospital.

My rotation through the department of obstetrics and gynaecology at Lebanon Hospital confirmed my interest in the discipline. I applied and was accepted at Mt Sinai Hospital to train in obstetrics and gynaecology. Before leaving Lebanon Hospital I published two articles in peer reviewed journals:


While on residency training at Mt Sinai Hospital, I visited Dr Eric Kahn, who was the director of paediatrics at Harlem Hospital, which was affiliated with Columbia University. Dr Kahn was director of paediatrics during my internship at Baragwanath in 1960 where I rotated through paediatrics. During my visit I told him of my interest in working at Harlem Hospital on completion of my residency. He introduced me to Dr Donald Swartz, a director of obstetrics and gynaecology at Harlem Hospital. I accepted a fellowship position in the department of obstetrics and gynaecology following completion of the residency in July 1965. After passing the New York State Licensure examination, I was able to participate in patient care services. I was an Instructor, Assistant, Associate and finally Clinical Professor of Obstetrics and Gynaecology at Columbia University in 1998. I was promoted from junior to senior attending physician for obstetrics and gynaecology at Harlem Hospital in 1980.

HARLEM HOSPITAL

Historically Harlem Hospital serves the poor and the uninsured patients of Harlem, a major African-American residential and cultural neighborhood. Harlem has an arts centre that has inspired generations of poets, musicians, and writers. Harlem Hospital and Harlem evoke memories of Baragwanath Hospital, Alexandra and Soweto.

I was appointed president of the New York Gynecological Society in 1998. This society is a forum of New York City gynaecologists and provides monthly conferences on the state of the art in gynaecology. I was appointed a member of the New York Obstetrical Society in 2000. The society holds monthly meetings on maternal and foetal medicine.

I was appointed director of the Obstetrical and Gynecological Department and Clinical Professor of Obstetrics at Columbia University in 1998.

During my tenure as director, the following goals and objectives were vigorously pursued:

• Providing women of Harlem medical services of the highest quality consistent with the
mission of the hospital irrespective of race, colour or ability to pay.

- Increasing patient satisfaction.
- Assisting women in planning pregnancies that result in healthy babies and healthy mothers and encouraging responsible fatherhood.
- Providing family planning services for those in need of safe, effective contraceptives with reduction of unintended pregnancies.
- Making the Department of Obstetrics and Gynecology the provider of choice in its primary service area which includes the area between Central Park North at 110th Street to 155th Street in the North and the Hudson River in the West and the Harlem River in the East.
- Offering surgical and non-surgical care for disorders of women of all ages from adolescence to menopausal and postmenopausal.
- Offering HIV/AIDS testing and management.

The primary HIV/AIDS rate in Harlem has been on the increase. All patients who enter the hospital are encouraged to test. Those who test positive and are pregnant are placed on antiretroviral drugs to reduce maternal-foetal transmission of the virus to the foetus.

As director of the department my responsibilities included the care of patients with complex gynaecological problems and female cancer patients. High risk obstetrical patients were under the supervision of the maternal medicine specialist.

On 18 June 1966, while screening patients in the gynaecology clinic, I received a telephone message from the office. The message said two white men with foreign accents were anxiously looking for me. I recalled at the time that my student visa which allowed me entry into the United States had expired. My suspicion was that the white men were South African security officers who wished to interrogate me. I left the hospital and called the director, Dr Donald Swartz. He told me there was nothing he could do to help but to get in touch with the Reverend Adam Clayton Powell, pastor at the Abyssinian Church a block away from the hospital. Reverend Powell was also congressman for Harlem. I met with Reverend Powell and told him that I had overstayed my student visa requirement and that the South African security men were at the Harlem Hospital to interrogate me regarding my continued stay in the hospital and the United States. Approximately 10 days later I received a letter from Reverend Powell on the congressman’s stationery. He had contacted the Commissioner of Immigration and requested my immigration status be changed to permanent status and allowed me to continue my medical work at Harlem Hospital without interruption by government officials.

During my tenure at Harlem Hospital I was able to introduce the following procedures:

(I) LAPAROSCOPY

This procedure was introduced to Harlem Hospital after completing a two-week course with Dr Neuwirth who had established a reputation as an expert endoscopist. The procedure provides excellent visualisation of the pelvic organs and permits the diagnosis of various gynaecologic disorders and surgery without formal laparotomy. The procedure has a low morbidity rate and short convalescence period. It is a cost-effective outpatient procedure. Since that initial introduction of the procedure in the department of gynaecology in the 1970s, the procedure is now extensively used by general and paediatric surgeons.

Laparoscopy lends expediency to the diagnosis and treatment of ectopic pregnancy, endometriosis, chronic pelvic pain. It is widely used in the investigation of patients with a history of infertility and total sterilization performed as an outpatient procedure.

(II) COLPOSCOPY

A colposcope is a binocular microscope used for magnified visualization of the uterine cervix. The most common use of a colposcope is in patients with abnormal pap smears without a visible lesion of the cervix. Harlem Hospital did not have trained personnel in the 1960s to evaluate patients with abnormal pap smears using the colposcopic technique. I attended a course in colposcopy given by Dr Ralph Richardt who is credited with having described precursor lesions of cancer of the cervix that he characterized as cervical intraepithelial neoplasias. These lesions are not visible to the naked eye, but are seen on colposcopy as abnormal epithelial changes which on biopsy may show histological changes consistent with intraepithelial neoplasia.

I acquired a Zeiss colposcope in 1971 and started a colposcopy clinic in Harlem Hospital. The clinic
Fortunately many gynaecologic cancers have higher survival rates because of improved diagnostic techniques that detect early cases and therapeutic interventions. There are, however, many challenges and unfortunately many cancers are fatal.

(III) GYNAECOLOGIC CANCER

Harlem Hospital is certified by the American Cancer Society to diagnose and manage cancer patients. For a number of years I have been responsible for the management of gynaecologic cancers both in an outpatient and in the inpatient areas of the hospital. With the assistance of a team consisting of members of the attendant staff and residents I have managed complex surgical malignancy cases. The volume of new information in gynaecologic cancers continues to increase beyond the capacity of a single individual to muster. Successful cancer programmes rely on a multidisciplinary team approach consisting of pathologists, radio therapists, chemotherapists, trained gynecologists and social workers.

I have authored several medical articles published in peer-review medical journals during my tenure at Harlem Hospital – Columbia University affiliate, including a multicenter randomised control trial: “Periodontal Treatment and Risk of Preterm Birth,” The American Academy of Periodontology (October 27, 2007) described the article as a “scientific manuscript with direct clinical relevance to the practice of periodontics.”

Columbia University trustees unanimously approved my appointment to Emeritus Professor of Obstetrics and Gynecology as from 1 October 2009. President Bollinger states: “This affirmation of your importance to our scholarly community recognises your contributions to the university.”

Since leaving Alexandra in 1961, I have had a long and varied medical career which owes a great deal to my marriage to my lovely wife Carol, with whom I have three daughters, Dara, Joyce and Karen. Dara and Karen are medical doctors practicing in New York. Joyce is in Iowa. She is a speech therapist. My wife passed away on 6 January 2006. She was a delightful, pleasant, bright African American woman born and raised in Brooklyn, NYC.

I have received many awards from peers, community health organisations and Harlem Hospital administrators for long, dedicated community service. What a pleasure to bask in the approval of honoured colleagues. It is a joy to be recognised so generously. I share these compliments with members of my family who have given me the support to complete this exciting medical journey that lasted over half a century.
from them and to dust. Collections are most often affected by moulds through water damage, although severe mould cases also occur when materials have been stored in humid conditions for a long time. Therefore, the environment must be controlled to inhibit their growth.

Temperature and humidity are interrelated; fluctuations and extremes thereof can affect the state of collections and archival materials. According to the NPS Handbook for Museums, fluctuations in temperature cause destructive stress to artifacts. The ideal combination is a low temperature and moderately low relative humidity in monitored conditions. The presence of airborne mould species may indicate mould growth on surfaces within buildings. The damage caused by mould is preventable by storing collections in clean, dry and stable conditions and ensuring good air flow.

This survey was conducted in response to a request from the Museum and its Board members who were concerned about mould growth on objects that were stored. As moulds were suspected, an indoor air quality survey was conducted.

METHODS OF SAMPLING AND ANALYSIS

Walkthrough inspection
A walkthrough inspection was conducted prior to the sampling by two occupational hygiene assistants from the NIOH, accompanied by Professor Tony Davies, Board member and Mr Sepeke Sekgwele, staff member of the Adler Museum.

Environmental parameters
IAQ parameters which included temperature (T) and relative humidity (RH) were measured simultaneously on the day of sampling, using a Kanomax IAQ monitor (SKC PTY).
A comprehensive time series measurements of T and RH were taken over a period of 7h and 5 min, using a TSI Q-Trak™ Indoor Air Quality meter. The interval and the mean values were used to plot a time series graph (Figure 1). The guidelines used by various groups for these parameters are outlined in Table 1.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Recommended levels</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature (°C)</td>
<td>Unbound records 18-21°C</td>
<td>Purafil Inc. Technical Bulletin-600A Environmental control of museums, libraries and archival storage areas, Georgia, USA (2004)⁶</td>
</tr>
<tr>
<td></td>
<td>Bound records 18-21°C</td>
<td></td>
</tr>
<tr>
<td></td>
<td>16-25°C</td>
<td>The International Group of Organizers of Large-Scale Exhibitions (The Bizot Group) (2009)⁷</td>
</tr>
<tr>
<td></td>
<td>Unbound records 25-35%</td>
<td>Purafil Inc. Technical Bulletin-600A Environmental control of museums, libraries and archival storage areas, Georgia, USA (2004)⁶</td>
</tr>
<tr>
<td></td>
<td>Bound records 40-55%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>45%-60% for wood, leather, textile and ivory</td>
<td>NPS Museum Handbook (1999)³</td>
</tr>
<tr>
<td></td>
<td>40%-60%</td>
<td>The International Group of Organizers of Large-Scale Exhibitions (The Bizot Group) (2009)⁷</td>
</tr>
</tbody>
</table>

Air and swab sampling for moulds
The MAS 100 air sampler (Merck Pty; Germany) was used for collection of airborne fungi by impaction method on Malt Extract agar (MEA) at a flow rate of 100 L per minute. Swabs were also taken from the surfaces with visible moulds and were cultured on the same medium and all plates were incubated at 25°C for 3-7 days. Fungal identification was done using microscopy and the phenotyping system (Biolog Inc, USA). Samples collected from outside were used as reference samples as there is currently no occupational exposure limit (OEL) for airborne biological agents. The results are summarized in Table 2.

RESULTS AND DISCUSSION

Walkthrough observations
The museum storeroom is located in the basement of the Wits Education Library and it contains various collections which are part of the history of medicine and allied health sciences. During the walk-through the area was cluttered, without enough space to walk around as many items were stored on the floor. The artifacts were made mainly of wood, leather, metal, paper and glass, and white substances, suspected to be moulds, were observed on all the types of materials.

<table>
<thead>
<tr>
<th>Lab no.*</th>
<th>Sample ID</th>
<th>cfu/m3</th>
<th>T (°C)</th>
<th>RH (%)</th>
<th>Mould Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>IM 2067/12</td>
<td>Base right</td>
<td>280</td>
<td>21.6</td>
<td>56.8</td>
<td>Cladosporium sp</td>
</tr>
<tr>
<td>IM 2068/12</td>
<td>Base left</td>
<td>310</td>
<td>21.4</td>
<td>58.1</td>
<td>Cladosporium sp Penicillium sp</td>
</tr>
<tr>
<td>IM 2069/12</td>
<td>Tunnel inlet</td>
<td>385</td>
<td>20.8</td>
<td>61.2</td>
<td>Aspergillus sp Penicillium sp A. flavus</td>
</tr>
<tr>
<td>IM 2070/12</td>
<td>Wood swab</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Cladosporium sp Penicillium sp</td>
</tr>
<tr>
<td>IM 2071/12</td>
<td>Leather swab</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Aspergillus sp</td>
</tr>
<tr>
<td>IM 2072/12</td>
<td>Copper swab</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Rhizopus sp</td>
</tr>
<tr>
<td>IM 2065/12</td>
<td>Outdoor</td>
<td>620</td>
<td>26.2</td>
<td>44.4</td>
<td>Cladosporium sp Penicillium sp</td>
</tr>
</tbody>
</table>

*For laboratory reference only; N/A - not applicable
The room is kept locked for security purposes and there was concern expressed about the lack of air in the storage area which was evident. The windows are sealed and there is no air-conditioning system with resultant inadequate ventilation. The roof had evidence of water leakage at the inter-leading door (Figure 2) near a tunnel leading to the outside. There was also a small hole made for a pipe inlet near the tunnel which was not sealed. Almost all the types of materials that were stored had some form of white mould-like growth on the surface, including metal and glass materials (Figure 3).

Environmental parameters
Relative humidity and temperature were measured at each of the points of the mould sampling; the results are shown in Table 2. The temperature in the room was slightly higher (20.8-21.6°C) than the acceptable limits of 21°C, whereas humidity was above the recommended limit of 25-35% for general unbound records and 40-55% for bound records (Table 1).

The changes in the temperature and humidity over 7 hours and 5 minutes were recorded.

The relative humidity ranged from 55.4% to as high as 65.6% (Figure 1) which suggests that the environmental conditions in the storage area are not constant throughout the day. It is emphasised in the NPS Museum Handbook that the changes in RH must not exceed +/-5% from the set point in one month. It was also highlighted that 50% RH might be reasonable for storage areas, but if the temperature drops at night, relative humidity may rise, which is what was observed from this survey. Relative humidity of more than 65% may promote mould growth.3

The temperature recorded during the time series measurements was an average of 19.5°C, which is within all the guideline ranges. The maximum measurement of 21.1°C was however slightly higher than the recommended standards, excluding the range by the Bizot group 7 (Table 1).

Microbial counts and identification of moulds8
Total fungal counts of all areas in the Museum storage area were below the outdoor sample (620 cfu/m³), which is the reference sample due to the lack of local standards for microorganisms. Cladosporium and Penicillium sp were the most common genera isolated from both outdoors and indoors. The seasonal distribution of fungi indoors could be related to the outside fungal distribution. Other genera that were identified from the storage area were Aspergillus and Rhizopus sp. These species have also been isolated in libraries and archive storage facilities and were found to be the most frequent sensitizers in museum workers.1,2,9

Aspergillus spp and Penicillium spp are widespread in the environment and commonly found as
contaminants in soil, decaying vegetation, dust and other organic debris; however these organisms are opportunistic invaders. *A. flavus* which was isolated near the tunnel inlet is found globally as a saprophyte in soils and is an important fungal pathogen in crops which causes pre-harvest and post-harvest infections. *A. flavus* grows and thrives in hot and humid climates. *R. oryzae* has a world-wide distribution with a high prevalence in tropical and subtropical regions. It has been isolated from many substrates, including a wide variety of soils, decaying vegetation, foodstuffs, and animal faeces and bird droppings. 8

*Health risks for museum workers and visitors* 
*Aspergillus* could cause aspergillosis which could be in the form of allergy, invasive infection and toxicoses; whereas *Penicillium* is known to cause corneal, cutaneous, external ear, respiratory and urinary tract infections. *Cladosporium sp* is widespread and known to cause type 1 allergies (hay fever and asthma). In mammals, *A. flavus* can cause liver cancer through consumption of contaminated feed or aspergillosis through invasive growth. 8

**LIMITATIONS OF THE SURVEY**

The results of this survey reflect the conditions during the day of the assessment. Daily and seasonal fluctuations in IAQ parameters can be expected depending mainly on the variability in environmental and weather conditions. There is currently no occupational exposure limit for microorganisms globally. In addition, the South African regulations for hazardous biological agents do not classify moulds species as it does with bacteria, viruses and parasites.

**RECOMMENDATIONS**

The following corrective actions were recommended:
1. Install a ventilation system and ensure an adequate supply of fresh air into the storeroom.
2. Train museum staff in the importance of air circulation, indoor air quality and regular monitoring of the environmental conditions.
3. Repair roof leakages and promote hygienic conditions including regular dusting of the stored materials and vacuuming the storage area to prevent microbial growth and protect materials from being damaged.
4. Install an indoor air quality monitor which will constantly monitor and record the temperature and relative humidity and should include overnight assessments.
5. Workers should wear disposable dust masks when carrying out housekeeping tasks in the storage area to minimize inhalation of hazardous biological agents.
6. Install shelves for storage of all objects and clear the aisles from clutter.
7. Monitor the environment post intervention of the recommendations made. Continuous monitoring should be done in accordance with section 7 of the Hazardous Biological Agents (HBA) Regulations.

**CONCLUSION**

The indoor air conditions in the storeroom of Adler Museum of Medicine are clearly favourable for mould growth. There was a significant fluctuation of relative humidity (between 49.9% to more than 65%) in the storage area, with temperature that was slightly higher than the recommended limit. Water damage, lack of ventilation and a poorly maintained storage area aggravate the problem. By controlling temperature, humidity and other environmental contaminants, long term sustainability can be achieved by maximizing the life of the objects and minimizing the risk of exposure of workers to moulds.

**ACKNOWLEDGEMENTS**

Thanks to Professor Tony Davies and Mr Sepeke Sekgwele for their assistance to the team during the assessment.

**REFERENCES**

7. The International Group of Organizers of Large-Scale Exhibitions (The Bizot Group) (2009).
Tributes to Professor Ronald Dorfman (MBBCh 1948)

For the better part of four decades, Dr Ronald Dorfman, Wits Medical School graduating class of 1948 and Professor of Pathology at Stanford University School of Medicine, was one of the leading experts on the pathology of Hodgkin and non-Hodgkin lymphomas. He was one of a small international group of pathologists who developed the modern clinically relevant pathologic classification of lymphomas that linked pathology characteristics to prognosis.

I had two personal encounters with Ron Dorfman. The first in 1960 when he taught a class in lymphoma pathology as part of the year-long third-year pathology curriculum at Wits Medical School, and again 49 years later, when I had the pleasure of meeting him and his wife, Zelma, at a Wits Medical School alumni reunion in Laguna Woods, California in 2010. As a radiation oncologist with special interest in lymphomas, I was very aware of Ron Dorfman’s important contributions over many years to the medical scientific literature on the subject of lymphoma pathology, and had consulted him directly many times by mail, telephone and email regarding difficult cases. Throughout that time, each time I saw a publication by him, or a reference by someone else to his work, it filled me with great personal pride and satisfaction to know that he had been one of my teachers at Wits, and that I could claim a little reflected glory in that personal direct connection to him.

In about 1964, and again in about 1969, the then internationally renowned pathologist, Lauren Ackerman, Professor of Pathology at Washington University in St Louis, Missouri, spent time as Visiting Professor of Pathology at Wits, and had a huge impact on many students, registrars, and faculty with whom he had contact. That was a time when many South African physicians, rebelling against the oppressive impact of apartheid, sought careers abroad, Ron Dorfman among them. Recruited by Lauren Ackerman to the prestigious Washington University, Ron served on the faculty there for five years, before moving to the equally prestigious Stanford University in 1968, where he remained until his retirement in 1993. At the time he joined the Stanford faculty, it was becoming the focal centre for basic and clinical research and treatment of lymphomas, and Ron took his seat at the multidisciplinary table of lymphoma giants, with radiation oncologist, Henry Kaplan, and medical oncologist, Saul Rosenberg. A generation of Stanford medical students and residents learned about lymphomas from those three great men, and the outstanding colleagues whom they attracted to Stanford over the next 40 years.

Even today, twenty years after Ron Dorfman retired, his name features prominently in text books and journal articles about the lymphomas, and one of them has the eponym Rosai-Dorfman disease, carrying the name of Ron Dorfman into medical history.

It was only at the 2010 alumni reunion, that I really had the opportunity to get to know Ron personally, and it gave me a thrill to be able to discuss his career and achievements with him, to reminisce over some of the cases I had sent him over the years, and to tell him of my admiration for him. I found him to be a warm, friendly, unassuming, and modest person, and had looked forward to seeing him again at the 2012 Wits reunion in Santa Fe, New Mexico, which he and Zelma planned to attend. We received the sad news a few months before we gathered in Santa Fe that Ron would not be joining us.

Martin Colman, MD, FRCR, FACR, FACRO, John Sealy Distinguished Centennial Chair, Professor and Chairman, Department of Radiation Oncology, University of Texas Medical Branch, Galveston
I didn’t know Ron Dorfman well. When I was a medical student, he lectured to me in pathology. He must have somehow imprinted something in me – ignited a spark, but it took many years before I became a pathologist, moreover one with an interest in haematopathology and the lymphomas. Wherever I go my entry card is to say I am a South African trained haematopathologist; and when people ask: do I know Ron Dorfman I say yes, he lectured to me, he taught me what I know. After that they think I know everything. He has a disease named after him. There aren’t too many people who have diseases named after them. The disease is "Rosai-Dorfman" disease. Juan Rosai and Ron Dorfman described it, collaborating with each other. The nice thing about it is that it is one of those rare diseases of the lymphoid system that is actually a benign disease, not malignant like most of the diseases of lymph glands. I think this goes along nicely with the fact that Ron was a very nice, a very benign person himself. Although Ron was at Stanford and I was at UC Davis, we used to consult his expertise on difficult cases. I met him two years ago at the Wits reunion held at Laguna Beach. I’m sorry that he didn’t make it to this one.

Ralph Green MD, PhD, FRCPath
Distinguished Professor, Department of Pathology and Laboratory Medicine and Internal Medicine, University of California, Davis

I first met Ron Dorfman at the home of Dr TonyPerlman in Baltimore in 1968. He had just completed five years in the surgical pathology department at Washington University in St Louis, and was due to enter Stanford University as an associate professor in surgical pathology. He and Tony had been classmates at King Edward School, where he had been an outstanding sportsman in rugby and athletics; they had both taken time from their medical training to serve in Italy during World War II.

Ron graduated in December 1948, served as a houseman and registrar to ‘Mosie’ Suzman and to Jack Douglas, and then went to England to further his training in internal medicine. At the Royal Postgraduate Medical School at Hammersmith, he renewed a friendship with another King Edward classmate, the late (and great) Kenny Weinbren, who inspired him to become a pathologist. He returned to Johannesburg to study at the South African Institute for Medical Research (SAIMR) where he developed an interest in diseases of the lymphoreticular system. During his years at the SAIMR, he encountered two cases of young African boys who presented with marked lymphadenopathy with rather unique and unusual pathological changes. In both instances, the clinical course was benign.

Ron had the good sense to take the slides of these two patients with him when he accepted a position at Washington University in 1963. During this period, an Argentinian Fellow in surgical pathology named Juan Rosai, showed him the slides of two infants with lymphadenopathy and a very similar histologic pattern to the two South African boys. Rosai and Dorfman subsequently published 34 cases of “Sinus Histiocytosis with Massive Lymphadenopathy (SHML)” now commonly known as “Rosai-Dorfman Disease”. Ron continued to have an outstanding career at Stanford and at his retirement in 1993, the “Ronald Dorfman Chair of Hematopathology” was established in his honour.

In 2006, when Julien Hoffman offered to host a reunion in NAPA Valley, California, I persuaded Ron and his wife Zelma to join us, since we were relatively close to Palo Alto. Ron was not able to attend the 2008 Houston/Galveston reunion due to a recurrence of Meniere’s disease; however, he and Zelma were present at Laguna Woods in 2010.

It was a sad moment to hear that he had died in June 2012 at the age of 89, apparently of heart failure. Our deepest sympathies to his wife, Zelma, his daughters Erica, Carol and Anne, his brother Stanley, two nephews and two grandsons.

Bernard Tabatznik BSc, MBBCh, FACP, FRCP
Formerly Chief of Division of Cardiology and Associate Chief Department of Medicine, Sinai Hospital of Baltimore; Chief, Department of Cardiology, North Charles General Hospital Baltimore, MD; Emeritus Assistant Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore

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It saddened me to learn only last week that Martin Spencer Israel died in 2007 [born 1927]. You may or may not remember him from our days at Wits. He was a good friend and we had been in close contact over several years.

Always a shy person, he was an only child. His parents were Lithuanian Jews. His father was an ophthalmologist. Martin excelled as a pianist. He had a difficult decision between pursuing a career as a pianist and going to Medical School. That he had a brilliant intellect goes without saying.

In his clinical years, he used to sit in the front rows of the Harveian Lecture Theatre. By chosen custom, we non-Whites used to sit in the front rows if only to assert our presence. Amongst the most assertive was one Vusimusi Gumede. This was in contrast to the Hospital Lecture Theatre, where we were obliged to sit in the back rows just in case a white patient was brought along for presentation when it was obligatory for non-White students to leave the Lecture Theatre. Gumede, who practised in Verulam near Durban has passed on; he authored a book on the evils of alcohol.

In preparation for the final examinations Martin and I used to travel to Coronation Hospital to see patients and quiz each other on clinical findings. He used to pick me up in his old 2-seater on Hospital Hill and drive to Coronation Hospital. Like so many of us at the time he left South Africa and sought further training in the UK. He was appointed House Physician to Professor John (afterwards Sir John) McMichael at Hammersmith Hospital. As irony would have it he passed MRCP examination at first attempt at a time when his Registrar, Tim Counihan, failed at the same sitting. Martin wished to pursue a career in paediatrics and was short-listed for trainee appointment at the Hospital for Sick Children, Great Ormond Street. He went through the ritual as I did myself, making appointments to see the various consultants at their convenience before the final interview but he never was appointed at Great Ormond Street, presumably because of his gentle and non-assertive attitude.

Martin changed track from clinician to pathologist and to stabilise his base in Britain he volunteered for National Service and was sent with a British team to Kaduna in Northern Nigeria. Continuing his career in morbid anatomy and histopathology, he was appointed lecturer at the Royal College of Surgeons at Lincolns Inn Fields. He ran the course in pathology for part 1 of the FRCS examinations. He became a co-author of a successful text, Walters and Israel: General Pathology, a text that ran into 6 editions.

I met Martin when I arrived in London via a year spent in India in 1952. We went to shows and had cake and tea, usually on a Sunday evening. We continued to exchange letters and I always met up with him on leave visits from Makerere University in Uganda every two years. I learnt that Martin had been doing counselling in his spare time, mostly in the care of older persons. When the Royal College of Surgeons ceased funding the course for part 1 examination, Martin wrote to say that he had become a Christian and that he was to be ordained as cleric. He wrote profusely and sent me copies of some of his work. His best known work, Living Alone, ran into several impressions and I believe is still in print. He was appointed to a church behind the Albert Hall in Kensington where the congregation was itinerant rather than permanent. On one of my visits to London I attended incognito in the back pews and heard him preach an interfaith sermon. It was a time when I had just arrived in the UK as a refugee from Uganda. He was most kind and supportive.

Martin always had a flail leg, presumably resulting from polio. He used to live in Earl’s Court. He had a fall and felt he could not go on living on his own. He moved to a terrace house in Battersea which was where I last visited him in the late 1990s. He had live-in carers, a young New Zealander, and his wife.

Professor Krishna Somers
MBBCh (Witwatersrand) FRCP (Lond & Edin)
FRACP, FAC, FCSANZ, DCH
By the time you have read the preface it will be clear that this is a complex and important story. It is a vital source of information for scientists concerned about occupational lung disease in South African hard rock mines; essential reading for citizens of the country worried about the failure of development in rural labour-sending areas; a challenge to the captains of South Africa’s biggest and richest industrial undertakings. The book offers grist for the mill of human rights activists and an account of some of the adverse consequences of the migrant labour system.

Professor McCulloch, whose AJ Orenstein Memorial Lecture in April 2013 is published in this issue, has gone to great lengths to analyse the history of South Africa’s gold mines and the impact of the industry on the country, on its internal labour reservoirs, and, more widely, on the adjoining states from which migrant labour was drawn from 1867 onwards. That impact is now being felt acutely in the persistent labour unrest precipitated by the events on the platinum mines around Rustenburg, and specifically at Marikana. The story is set against the background of the unprecedented conjoined epidemics of silicosis, pulmonary tuberculosis and HIV infection affecting miners, not only in gold mines but in the platinum mines which have burgeoned as gold mining has waned. In media reports, the contribution of work-related disease and injury in promoting industrial unrest is ignored as a rule.

This book is a timely wake-up call to the industry, setting out as it does the neglects of the 20th century, and implying plainly that unless deliberate action is taken the current damaging situation will persist.

The title reflects the fact that the political principles operating in South Africa almost throughout the 20th century were a dominant influence, not only in society but disastrously in the workplace, in particular as a result of the development and exploitation of the migrant labour system. The mining industry played a central role in the formation of the society in which South Africans now live. The preface begins with a lengthy paraphrase from the 100th Presidential Address to the Chamber of Mines of South Africa’s Annual General Meeting on the 19 June 1990, in which the then President, Mr KW Maxwell, listed some of the adverse features of the country about to embark on the transition to democratic rule. “Per capita disposable income was falling and more than five million South Africans were unemployed. Half of the adult population was illiterate and half of the country’s children were not attending school. South Africa had just 60,000 students in technical and higher education. With half the population Australia had over 800,000 [such students]”. The author adds: “Maxwell was no doubt aware that the mines which had made the country the dominant industrial power on the continent had helped to create one of the world’s most unequal societies”. Against this background the (Leon) Commission of Inquiry into Safety and Health in the Mining Industry reported in 1995 that the evidence submitted to it had satisfied the commissioners that the dust levels in South African mines had not changed for about fifty years. The Commission expressed its concern that no results of recent dust control studies were available and that much of the evidence relied heavily on the work of Beadle carried out more than twenty-five years previously.

Chapter 1 (Gold Mining and Life-Threatening Disease) begins, appropriately, with the story of the late Mr Thembekile Mankayi’s legal case against AngloGold Ashanti, and the reversal of the lasting assumption that current statute precluded litigation by a miner or ex-miner who had contracted occupational lung disease against his employer. After his attempts to establish his right to sue had failed in the High Court, and in the Supreme Court of Appeal, the Constitutional Court gave Mr Mankayi leave to have his case heard. This has been followed by the preparation of a number of
new cases (class actions) on behalf of the many miners with undiagnosed and uncompensated occupational lung disease who have returned to the labour-sending areas from which they were originally recruited. The chapter ends with the following summary: “For almost a hundred years South Africa’s gold mines claimed to be leading the world in safety, medical surveillance and compensation, but a careful reading of the history suggests that this was an illusion. The industry’s failure to create safe workplaces and to compensate migrant workers for occupational disease underpinned its commercial success and allowed the costs of production to be shifted to rural communities”, a subject set out in great detail in Dr Anna Trapido’s thesis submitted to the University of the Witwatersrand in 2000 and frequently referred to in this book.

This book is strong medicine, based on extensive research and comprehensively referenced. It includes vital new information from archival collections not yet widely known or studied, in particular the archival material donated to the University of Johannesburg by The Employment Bureau of Africa. Chapter 2 (Creating a Medical System) tackles the sensitive and controversial matter of what the mine doctors have been doing in the mines all these years. In the beginning the industry was faced with a health crisis which led to the appointment, by Lord Milner, of the Weldon Commission within months of the end of the South African War (1899 – 1902). About the same time an open letter to the British Medical Journal from an occupational health doctor in England advised miners not to work in South Africa’s mines because it would be bad for their health, and the Chamber of Mines offered prizes for innovative methods of dust control. “Mine medicine was a difficult career made no easier by the tension between the industry’s desire for labour and its determination to control costs” the author concludes at the end of a graphic description of the impossible logistics of what AJ Orenstein described as “medical mass production”. The outcome was captured by Dr Anna Trapido as follows: “One of the most effective disincentives to hazardous working conditions is employer cost. Failure to compensate occupational disease means that the full costs are not borne by the employers. The economic link between hazard and disease is lost”.

A number of carefully cherished mining industry orthodoxies are challenged and found wanting. The notion that tuberculosis spread from the labour-sending areas to the mines is one of these, and possibly the most often repeated. There is plenty of historical evidence that the reverse is the case. Chapter 9 entitled The sick shall work ends like this:

“In addition to spreading disease the migrant labour system is one of the major obstacles to medical care. Treatment at the mines, in the public health systems of South Africa and in the neighbouring states such as Lesotho is uncoordinated. Some mines provide on-site care or refer miners to public health centres while some return them to Lesotho for treatment. Each system captures a different set of patients and generates a different set of data. The restructuring of the labour force has created a further problem. Most men are employed directly by the mines, but those who are employed by private contractors are not entitled to the same health benefits. South African immigration law forbidding the employment of non-citizen novices on the mines has led to a growing pool of undocumented migrant workers. There is an urgent need for the coordination of health care between employers and the Departments of Home Affairs, Labour and Health in South Africa and Lesotho [and the other neighbouring states from which labour is drawn]. There is also need for comprehensive education about HIV/AIDS, tuberculosis and silicosis and miners’ rights to compensation”.

There are ten chapters in the book, all ten of which include passages which are worth quoting in full. This would make this review unduly long. Apart from that, the present reviewer is far too close to the current problems of miners and their lung disease, and should exercise restraint for fear of boring the reader. Suffice it to say that writing this book has been a major undertaking successfully completed. Read a chapter at a time and leave space to ponder each one. The final paragraph of the book ought to impel many to do this. “The failures of the South African system of medical surveillance, data collection and compensation underpinned the commercial success of the nation’s most important industry. The costs of production were shifted to rural communities within and outside South Africa’s borders, and it is only now, more than a century after the mines began production, that these costs may at last be called in”. Professor McCulloch has knowingly, I am sure, repeated the passage in Chapter 1 quoted earlier because it is arguable that what we see as “neglect” was an integral part of a business plan. In consequence I conclude with two questions: Is reparation required? If reparation is required how is it to be made?
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Reference examples

Dr Frack had been a member of the 1919 Class, the Tin Templers.¹

It did not, however, include anything about osteology, for bones would have doubled the size of The Pocket Gray.²

Direct quotes should be in italics or in inverted commas

Military medicine, surgery, and nursing were matters too important to be left to private charity, however well intended…³

“The tenth edition of Aids to Anatomy appeared in 1940…. It had been edited by Professor Stibbe, who, sadly, in 1923 left the University of the Witwatersrand.”⁴

References


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The Editors, Adler Museum Bulletin, 7 York Road, Parktown, 2193, South Africa
Email: adler.museum@wits.ac.za
Enquiries to the Curator: Telephone: (011) 717 2081;
Fax: 0865532483