THE WHITE DEATH:
SILICOSIS (MINERS' PHTHISIS) ON THE WITWATERSRAND GOLD MINES 1886 - 1910

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To Sessel and Victor

ABSTRACT

In its chronic form silicosis had always been been taken for granted as one of the occupational hazards of mining. But both during and shortly after the Anglo-Boer War it manifested itself in a new accelerated form amongst former Witwatersrand rock drillers. Despite the appointment in the Transvaal of a commission of enquiry in 1902 and the promulgation of dust precaution measures, by 1912 the prevalence of and mortality from the disease amongst the Witwatersrand miners had not diminished. This finding suggests two of the purposes of the study: first, the reasons for the continued prevalence of the disease; and second, the extent of the mortality from silicosis amongst the miners. Because of the apparently low prevalence of and mortality from the disease amongst African mineworkers, the disease was ironically nick-named the "white death". Therefore another aim of the study is to examine the validity of the medical claim that the short contracts of African migrant workers safeguarded them from contracting accelerated silicosis.

As the subject is complex, the study uses a thematic approach. Chapters two to nine deal with significant themes: first, the growing medical knowledge concerning silicosis, the mining and medical precautions against the disease and the age-old disregard for the occupational illness in its chronic

form; second, the industrialists' need to reduce working costs, the development of mass-production technologies and the resort by management to "speeding up"; and third, the miners' needs for job and wage security, the encroachment of African competitors in semi-skilled and skilled spheres of mining and the introduction and the extension of the colour bar. Chapter nine deals with underground health conditions. Chapter ten explores the awareness of the new form of the disease, accelerated silicosis, and the establishment in 1902 of the first Transvaal commission on silicosis. Chapter eleven discusses the failure to implement remedies. Finally, chapter twelve explores the prevalence of and mortality from silicosis and the impact of the disease on the workforce of the mines. In chapters ten, eleven and twelve a synthesis is offered of the themes and findings of the previous chapters. The project is based almost entirely on contemporary primary and published sources. Apart from silicosis, a unifying theme throughout the discrete sections is the perceptions of miners of their vocation in general, and of this occupational disease in particular.

The study requires periodisation. As silicosis is a slow-developing disease the starting point of the investigation is 1886, when gold was discovered on the Rand. The study ends in 1910 because the establishment of Union in 1910 and the legal award of compensation in 1911 heralded a new era in the history of silicosis on the South African gold mines.

The following are the conclusions. First, almost an entire generation of overseas miners, most of whom remained migrants and whose skills pioneered the South African gold mining industry, died from silicosis. Second, for reasons of self-interest, some of which they shared with one another, both the Transvaal state and the Randlords did virtually nothing to remedy the occurrence of the disease. Both parties were culpable for the neglect of the health of the industry's workforce: using only perfunctory dust safeguards, management intensified production through the deployment of both modern technology and labour intensive practices, peculiar to the Witwatersrand; and most of the state's interventionist initiatives were the result of pressure from the British House of Commons. Third, the fear and anger of miners at being the victims of a preventable occupational disease, provided the catalyst for their militancy during the period. Fourth, the industry's power was partly responsible for causing both the press and medical profession to be silent about the problem until 1910. Finally, the mineowners both seized and promoted the unsubstantiated medical orthodoxy, namely that the short contracts of African mineworkers protected them from accelerated silicosis, as an important rationale for perpetuating the migrant labour system.

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CITATION

In citing government publications I have used the series number. For instance, *Reports of the Transvaal Labour Commission. Minutes of Proceedings and Evidence* is referred to as Cd. 1897, 1904. The full titles of all government publications are listed in the primary sources under section 3. I have cited unnumbered government publications according to their full titles.

In citing secondary source material I use only the surname of the author or editor to indicate the work cited. Precise authorship, title and publication details are fully listed in the bibliography. For instance, Ashworth, p. 5, refers to Ashworth, William, *An Economic History of England 1870-1939*, London: 1960, p. 5. Where more than two editors or authors have been responsible for the compilation of a study, I cite only the first surname listed on the title page and indicate the others by the phrase et al.

In cases where different authors or editors have the same surname, to avoid ambiguity I give both the forenames and the surname in the footnote. For instance, J. Pratt Johnson, p. 334, (author of "The Miners' Phthisis Problem", *South African Medical Record*, 11 November 1916, pp. 331-337), distinguishes

this writer from Paul Johnson, whose study is entitled *Consolidated Gold Fields: A Centenary Portrait*, Johannesburg: 1987.

Where a single author has been responsible for several works, the citation indicates both the surname of the author and the title of the particular study referred to in the text.

ABBREVIATIONS

For simplicity and brevity I have used the following abbreviations. They are used mainly in the footnotes.

AEJ	*Amalganated Engineers' Journal* (Great Britain)
AHFA	Air Hygiene Foundation of America
AMM	Association of Mine Managers
ASEMJ	*Amalgamated Society of Engineers Monthly Journal* (South Africa)
BMJ	*British Medicial Journal*
BRA	Barlow Rand Archives
CAD	Central Archives Depot
CAT	Cornish Association of the Transvaal
Cd.	Great Britain. Parliamentary Papers
CHA	Corner House Archives
CO	Colonial Office

col./s.	column/s
GMEAR	*Government Mining Engineer. Annual Report*
GOV	Governor of the Transvaal Colony
HE	Hermann Eckstein
JCMMS	*Journal of the Chemical, Metallurgical and Mining Society of South Africa*
JSAIE	*Journal of the South African Institution of Engineers*
JXM	John X. Merriman
LTG	Lieutenant Governor of the Transvaal Colony
MJSA	*Medical Journal of South Africa*
MM	Secretary of Mines. Transvaal
MNW	Secretary of Department of Mines and Industries. Transvaal and Union of South Africa.
n.	note
par./s.	paragraph/s
p./pp.	page/pages
PRO	Public Record Office
q./qq.	question/s

CONTENTS

CHAPTER 1

INTRODUCTION

> "Such a death rate [from miners' phthisis] is a disgrace to the richest mining field in the world"---an anonymous miner, 1910.[1]

> "The primary fact is forgotten that we are face to face with the great danger of metalliferous mining - a disease [miners' phthisis] which is far more fatal than accidents, which is the one complaint which besets an otherwise health occupation which is readily avoided by...simple precautions."---*Mining Journal*, 1912.[2]

In his year-end report, in December 1901, the Transvaal Government Mining Engineer stated that during the period between the beginning of the Anglo-Boer War, in October 1899, and the resumption of mining on the Reef, in May 1901, 227 pre-war Witwatersrand rock drillers, or 16,5 per cent of the rock drillers, had died from the occupational disease, silicosis.[3] Fifteen months later the Weldon Commission, which investigated the causes and the occurrence of the silicosis, reported that the disease was prevalent amongst 22,6 per cent of the white underground workforce on the Witwatersrand mines: of

the 4 403 white underground mineworkers 15,4 per cent were definite silicotics, while 7,2 percent had symptoms which probably indicated the disease.[4]

In 1912 the Van Niekerk Commission, also known as the Medical Commission, concluded that neither the death rate from silicosis nor the prevalence of the disease amongst white underground mineworkers on the Witwatersrand gold fields had diminished.[5] As important, in 1912 the average age of death of rock drillers was thirty-three years and the commonest age was twenty-nine.[6] Miners on the Witwatersrand who specialised in rock drilling had an average working life of only seven to nine years.[7]

In other metal mining centres the position was different. In Bendigo, for instance, the average age of death for miners was fifty years.[8] In Cornwall the rapidly progressive form of silicosis amongst rock drillers had been reduced. By following the recommendations of the Haldane Commission, which reported at almost the same time as the Weldon Commission in the Transvaal, the Cornish mining industry improved miners' health. It was asserted that by 1910 the working lives of Cornish tin miners, particularly rock drillers, had been extended by ten years.[9]

At the turn of the 20th century silicosis was not a new disease amongst metal miners, as it had a history which spanned centuries. In its more usual

chronic form it caused premature death or severely crippled miners at an average age of fifty and after they had worked for approximately thirty years.[10] Employers and governments had taken it for granted as one of the occupational hazards of mining. The miners, too, were inured to the disease and anticipated short working lives. But the disease manifested itself in a new way amongst rock drillers on the Witwatersrand and in Cornwall. Its form was so rapidly progressive that it caused miners to die when they were young men.[11]

Constant and continuous exposure to excessive amounts of respirable silica dust caused "dense fibrous nodules" to replace the spongy lung tissue. When removed from the body and placed in water, such a silica-damaged lung sank "like a stone", its weight being three time as great as that of an average lung.[12] Also, such a fibrotic lung was "very difficult to cut with a knife, and practically impossible to cut with a razor".[13]

Because of the size of the Witwatersrand gold fields and the large number of underground workers, it is to be expected that, if inadequate preventive measures against the inhalation of dust were taken, a huge crude prevalence of silicosis would occur.[14] Even so, and despite the disclaimers of certain doctors to the contrary,[15] in relative terms the prevalence of silicosis was higher on the Witwatersrand than at other metalliferous mining centres. Also, both its

incidence and fatally incapacitating development were far more rapid on the Rand gold mines than elsewhere.[16] In 1916 J. Pratt Johnson, a doctor who was more candid and outspoken than most of his colleagues, summed up the extreme dimensions of the disease on the gold mines: "The mortality from miners' phthisis [on the Rand] is unequalled in any part of the world."[17]

Johnson's chilling message suggests two of the purposes of this study. One is to explore the reasons that the Witwatersrand miners were more vulnerable to silicosis, particularly accelerated silicosis, than their counterparts at any other hard rock mining centre in the world. Another is to uncover the extent of the mortality from the occupational disease amongst both rock drillers and general miners.

By 1910 the standard South African nick-name for silicosis was the "white death".[18] The name was ironic because health officers regarded silicosis as a serious health problem for white mineworkers only. Although they did not regard Africans as being immune to the disease, its incidence, prevalence and mortality amongst Africans were viewed as being so slight as not to warrant concern. Medical opinion held that the relatively short and intermittent contracts of most African mineworkers, which usually did not last for more than "seventeen to eighteen" months,[19] did not give the black workers the "same opportunities [as Europeans] of contracting Miners'

Phthisis".[20] Also, health officers claimed that, after the African had returned to their rural homesteads, they were able "to shake off that tendency" to silicosis and "get back to normal conditions".[21] Therefore another aim of this study is to to explore the validity of this medical belief.

There are very few academic studies of the history of silicosis on the Witwatersrand. Apart from the articles and papers which I wrote twelve years ago, only four historians have tackled the problem. Most of these studies are brief surveys and lack detailed analysis of the many complex issues.[22] Another defect of these studies is the organisation of information. The subject is complex requiring the exploration of many themes which provide solutions to a large number of apparently unrelated issues. When an attempt is made to discuss these themes in parallel, the strands of the argument remain separate and are not unified in a sythesis of inextricably related issues and events. For this reason I have chosen a thematic approach. Chapters two to nine deal in depth with significant themes so that in the final chapters, without the need for digression, the connections between the themes can be woven together.

Apart from silicosis, a unifying theme throughout the discrete sections is the perceptions of miners of their vocation in general, and of this occupational disease in particular. We must use historical data to interpret the experiences of workers. By exploring

the perceptions of miners we gain an understanding of their needs and feelings: their fear of the disease; their need to distance themselves from the realities of disablement and death; and their desires for job security and for danger pay.

The scope of this study does not permit an intensive exploration of the debate as to whether the Transvaal state dominated the mining houses or whether the reverse was true.[23] All the same, another unifying theme in this project is the connection between the state and the gold mining industry.[24] We examine this relationship under three consecutive, but distinct, governments: first, the South African Republic under President Paul Kruger in the era 1886 to 1899; second, the British administration's crown colony government from 1902 to 1907; and third, responsible government, with Louis Botha and Jan Smuts at the helm, under Het Volk's rule during the period 1907 to 1910. Such analysis will enable us to understand the responses of the state and the mining houses to the prevalence of and the mortality from silicosis.

In 1983 David Yudelman argued that during the period 1902 to 1936 there was a "growing symbiosis" between the state and the industry. Also, both before and after Yudelman published his work, Pieter C. Grey, Peter Richardson and Alan Jeeves provided supportive evidence for this author's hypothesis.[25] We will therefore test whether this state-capital alliance, the theory posited by all these historians, helped

shape the reactions of the various Transvaal governments and the mining houses to the problem of the occupational disease, silicosis, which manifested itself on a huge scale on the Witwatersrand gold mines.[26]

In addition to a thematic approach there is a need for periodisation. As silicosis is a slow-developing disease, its manifestation in an accelerated form on the Witwatersrand in 1901 was not the result of mining techniques then in use. Rather, at that time the huge crude prevalence of silicosis on the Reef reflected the existence of excessive dust levels on the Witwatersrand gold mines, which had prevailed for a number of years before the turn of the 20th century. This explains why the starting point for this study is 1886, when gold was discovered on the Rand. Until 1910 this occupational health hazard was an issue confined to the Transvaal. With Union in 1910 the Botha-Smuts ministry manipulated the issue of silicosis so that the financial burden of compensation would be borne not by the Transvaal alone, but by the new state, the Union of South Africa. In 1911 legislation for the award of compensation heralded a new era in the history of silicosis on the Witwatersrand gold mines.[27]

Through the detailed exploration of an occupational disease, silicosis, new insights are gained into the medical and health, demographic, socio-economic, and political impact of the mining

industry on the fabric of Transvaal and ultimately South African society. Unlike several of the studies of silicosis on the Witwatersrand mentioned in the review which follows, my study does not set out to describe, but rather to interpret the available data and to solve the many problems that they pose.

This study investigates a number of unresolved issues which require solutions that differ from those offered in the historical accounts published in the reports of various health officers and of successive commissions of enquiry up to 1937. It is these historical narrations that most of the popular and academic studies have slavishly followed. Some of the fresh issues on which my study focuses include the nature of management's mining methods on the Witwatersrand during the eras both before and after the Anglo-Boer War. The study questions the orthodox view that technology, particularly machine drills, was solely responsible for promoting the incidence of accelerated silicosis amongst miners. I therefore examine labour intensive practices, peculiar to the Witwatersrand gold mines, to determine the extent to which they were liable for creating conditions under which general miners, as distinct from rock drillers, could also contract a rapidly progressive form of the disease.

I tackle the question of why more than one-third of the overseas miners died from the disease abroad and not on the Witwatersrand. In so doing I explore

the reasons that most professional miners chose to remain migrants rather than become permanent Transvaal residents. Further, the study analyses the processes by which the skills of the Witwatersrand miners were fragmented and it shows how the miners' fears of job displacement by semi-skilled African mineworkers promoted their demands for the extension of the colour bar and the legal prescription of a fixed ratio of white to black mineworkers. Even so, this study will show that an additional reason that strongly reinforced the growth of miner militancy, which characterised the period 1902 to 1910, was the vulnerability of miners to a preventable occupational disease, silicosis.

I also explore the reasons for the silence until 1910 in the Transvaal of the public, the press and the medical profession concerning the ravages of the disease amongst the professional miners. Next, I question the conventional wisdom that Alfred Milner, the Governor of the Transvaal, by appointing in 1902 a commission of enquiry into the disease, acted with promptitude to remedy the problem of silicosis. Finally, I re-examine the claim of orthodox historical summaries that during the period 1902 to 1910 the state and the mineowners were not able to reduce the prevalence of and mortality from the disease for reasons beyond their control, including the miners' so-called wilful neglect for their health.[28]

These are the details of the scope of the study. Chapters two to nine deal in depth with significant themes. First, I examine the disease itself, the mining and medical precautions against the disease and the age-old disregard by governments, health officers and employers for silicosis in its chronic form. Then follows an analysis of the industrialists' need to reduce working costs, the development of mass-production technologies and the resort by management to "speeding up". Although modern technology helped the mineowners to increase the industry's productivity, it will be argued that their labour-intensive practices were also important in increasing dust densities underground.

The next set of themes, those of job security, which was emeshed with African competition and the institution and extension of the colour bar, and of wages, throw light on the emergence of the concept of danger pay. The miners' needs for job and wage security together with the threat of silicosis help explain their militancy, particularly in the mine-wide strike of 1907.

Chapter nine deals specifically with underground health conditions. Chapter ten describes the realisation of the existence of the new form of the disease, accelerated silicosis, and the establishment of the Weldon Commission, the first commission on silicosis in the Transvaal. Chapter eleven discusses

the failure of the state and of the industry to implement change. Finally, chapter twelve explores the prevalence of and mortality from silicosis amongst both white and black mineworkers and it analyses the responses of each of these sections of the workforce to the ravages of the occupational disease. In chapters ten, eleven and twelve a synthesis is offered of the themes and findings of the previous chapters.

As Sebastian Valentyn van Niekerk, the Medical Inspector of Mines, explained in 1914, all underground workers on the Witwatersrand gold mines were vulnerable to silicosis:

> Miners' Phthisis is a disease which affects all classes of underground workers from the very humblest to those in the highest positions, not even the managers themselves being safe.[29]

But miners, particularly rock drillers, were more prone to the disease than the other underground workers.[30] It is therefore clear that this study requires a clear and unambiguous use of the terms "mineworkers", "miners" and "artisans", as their indiscriminate and interchangeable use has led to mistaken or distorted historical findings.

"Mineworkers" is the generic term for wage-earners on a mine. In the past it was customary for specialists in South African history to apply the term solely to white wage-earners on the mines. Currently it is also used to denote the African workforce, but together with suitable adjectives which

serve to distinguish this racially distinctive body from its white counterpart. The adjectives, "black" and "white", describe only the race of the job holders; their occupational class fication, even if it is only a linguistic one, is identical for both racial groups. Contemporary colloquialisms for African mineworkers included "kaffirs", "niggers" and "boys" The phrase "native labour" was, until fairly recently, a standard expression used to denote the black workforce on the mines.

During the 19th century and the first half of the 20th century African mineworkers were officially called "natives", "labourers", and "boys"; and the Chamber of Mines used these official tags too.[31] The state and management also referred to black mineworkers as "Coloureds".[32] But this was an ambiguous term. Sometimes it denoted only Africans. On other occasions, however, it was used as a generic term for all mineworkers who were not classified as whites and therefore embraced the Indians and coloured workers - and later the Chinese. Instead of "Coloureds", this study uses the term "non-whites" as a collective noun for Africans, Indians, Chinese and coloured persons. "Non-whites" is used purely as a descriptive term.

The coloured workers, generally referred to on the gold mines as "Cape boys",[33] were a racial group comprising persons of mixed descent. The Indian and coloured mineworkers, both singly and combined,

constituted only a tiny proportion of the total gold mining workforce. In contrast to Africans, the vast majority of whom were migrant workers, Indians and coloured persons were often, though not always, freely employed without indentures. Also, they were usually, like whites, considered permanent members of the workforce.[34] Their role and relative importance on the gold mines has not yet been researched. For these reasons, unless otherwise noted, the calculations in this study, which are based on contemporary statistics, refer only to African mineworkers.[35]

Both officially and colloquially, contemporaries referred to the white wage-earners on the gold mines simply as "mineworkers", "workers", "workmen" and "men".[36] These nouns had unambiguous European connotations, just as blackness was implicit in the terms "natives", "labourers", and "boys". As is the custom in similar historical works, this study follows contemporary convention with respect to white mineworkers. The terms "workmen" and "mineworkers", when used on their own and without racial qualification, denote wage-earners on the mines of European descent. In this way white wage-earners are also distinguished from the salaried "class" of white mine employees. The latter comprised the clerical, professional and technical staffs and included draughtsmen, doctors, engineers, assayers, electricians, surveyors, samplers, managers, foremen, mill and cyanide overseers, mine captains and shift

bosses.[37] In this study the term "mineworkers" embraces a wide spectrum of wage-earners on the mines: the skilled professional miners and the nominally semi-skilled gangers together with the skilled artisans and the semi-skilled machine operatives:[38]

"Mineworkers" is a convenient word for conveying general quantitative data, such as average numbers, wages, percentages and ratios. It is also a collective noun which can be helpful in differentiating workers on the mines from bodies of workers in other industries, for instance railway workers. In other respects, however, its use is limited. As the term "mineworkers" encompasses different categories of workers each with distinctive interests and goals, it cannot be used to convey subtle connotations or qualitative criteria.

The word "mineworkers" should not be used interchangeably with "miners", as is common practice.[39] Professional miners constituted a discrete category of workers comprising approximately one-third of the total white workforce of the gold mines. Miners consisted of two groups. The first and much larger group comprised developers and stopers who were usually supervisors of Africans. The second smaller group consisted of timbermen, plate layers, pipe fitters and pump minders, who comprised approximately 10 per cent of the white underground workforce.[40] Although most specialist pitmen, as the smaller group of miners was collectively called, possessed blasting certificates

and had the necessary skills to do developing and stoping, management tended not to regard them as "miners". Instead, it usually, but not always, reserved the term "miners" for supervisors, who comprised approximately 50 per cent of the underground workmen.[41] Therefore when the term "miners" was sometimes extended to include the specialist pitmen, they constituted approximately 60 per cent of the underground workmen. Because the terms of service of most miners differed markedly from the rest of the white workforce, miners had few common interests with other underground workmen and even less with surfacemen.[42]

Social scientists who confuse miners with other workmen, particularly underground operatives and artisans, or who use the term miners as a synonym for mineworkers or workmen, are not entirely to blame for this vagueness: contemporary commentators did not always avoid this pitfall either. Often they casually referred to workmen of all descriptions on the mines by using miners as a generic term.[43] It is therefore not surprising that a recent biography of an engine driver on the mines, is confusingly and incorrectly entitled *Letters of a South African Miner*.[44]

Apart from the necessity for appraising the qualitative differences between miners and other semi-skilled and skilled artisans employed on the mines, there is another important reason, as we have noted, for observing a strict definition of miners as

a distinctive group. Other workmen - even those who worked on a semi-permanent or permanent basis underground - were not as vulnerable to silicosis as were miners; and when the disease occurred in such workers, it usually developed slowly rather than in an accelerated way. If we are therefore to understand why the disease, particularly in its rapidly progressive form, was so prevalent amongst miners, they have to be carefully distinguished from the remainder of the underground workforce.

As we have noted, I have created my own approach to exploring the many themes asociated with the history of silicosis on the Witwatersrand. Neither general histories of occupational diseases nor specific studies on silicosis have provided me with a suitable model for my study. Even so, a number of these studies raise important issues, which have helped me in directing my focus and in solving problems.

As regards occupational medicine in the Western world, two general histories, those of Donald Hunter and George Rosen, provide valuable background information, as does the history of the pneumonconioses, edited by the Air Hygiene Foundation of America. But all these studies have the same limitations: their focus is almost entirely medical; their historical sweep is too broad to be definitive; and the methodology is purely descriptive.

The 1985 essays, edited by Paul Weindling, which deal with the history of selected topics in occupational health care, are multi-disciplinary and provide valuable insights. But as most of the essays explore only one or two aspects of occupational health, they are unsuitable models for this study.

All the essays of Vicente Navarro are written from a materialist standpoint. So, too, are the contributions of other social scientists which Navarro has edited, either alone or together with Daniel Berman. Such studies challenge the practice of medicine in the Western world today and explore the relationship between the medical profession and society. Although none of the monographs deals with silicosis specifically, the questions which they raise are pertinent to this study.

Silicosis on the Witwatersrand gold mines was a serious problem for both the industry and the South African state for the first half of the 20th century. For instance, during the period 1902 to 1925 alone silicosis was the subject of nine legislative acts,[45] six commissions of enquiry and ten parliamentary select committees.[46] Also, during the period 1912 to 1919 the Miners' Phthisis Prevention Committee, which was jointly funded by the South African government and the Chamber of Mines,[47] presented four major reports.[48] It is therefore surprising that only a handful of academic historians have found the implications of the disease to be sufficiently

important to warrant specific study.

The paucity of research on a significant historical subject is not the sole problem. Another cause for concern is the inadequacy of the few studies which focus on the problem of silicosis. Their brevity is not the reason for my dissatisfaction with them. Rather, I find them to have methodological weaknesses. Also, all the studies are flawed by the writers' superficial understanding of the complexities of dust-induced diseases in general, and of silicosis in particular. We first survey the single-theme studies of Pieter Grey and Brían Kennedy and then examine the multiple-theme journal articles by Gillian Burke and Peter Richardson and by H. J. van Aswegen.

In 1969 Grey wrote a doctoral thesis on the development of the mining industry from 1902 to 1910. He relegates his treatment of silicosis to part of a chronological chapter devoted to health conditions on the mines. In this small section on silicosis Grey focuses on the responses of the state and the mining industry to the problem.[49] Consequently his discussion of the disease does not explore its complex socio-economic and political ramifications. Also, although his research is thorough, it is based mainly on official sources, which obviously have an inherent bias. In brief, Grey uncritically assembles a quarry of unevaluated empirical data.

Kennedy's essay, published in 1984 in *A Tale of Two Mining Cities*, is entitled "The Conquest of Phthisis". His theme is therefore self-explanatory. His discussion provides several useful insights, particularly those concerning the prevention of the disease in Broken Hill, the Australian mining centre to which he compares the Witwatersrand. Because of Kennedy's unfounded assumption that the disease was "conquered" during the period which he explores, he exaggerates the efforts made by the Transvaal governments, the medical profession and the gold mining industry to control the disease in South Africa. Kennedy also concludes that the mortality from silicosis amongst Africans increased during the period 1902 to 1910.[50] As we will later show, however, the source for his epidemiological data, the records of the Medical Officer of Health for Johannesburg, is unreliable.

The 1978 article by Burke and Richardson on the "migration" of the disease between Cornwall and the Witwatersrand between the 1890s and 1918 is analytical and explores a number of significant themes. It nevertheless lacks depth of research. Many of its premises, including those which concern the pathology of the disease, mining techniques and preventive measures, are unfounded. Consequently incorrect conclusions abound.

Van Aswegen's recent article, published in 1989, has different defects. First, because it is based on too few sources, which are of limited value, his treatment of significant themes is superficial. For instance, van Aswegen unquestioningly accepts the historical summaries, given in government publications and those written by health officers, concerning the introduction of dust preventives. Consequently he both exaggerates the initial concern shown by the state and the industry towards the occurrence of the disease and overestimates the benefits and quality of the remedial measures taken. Second, in a single paper he produces several chronological surveys of different aspects of the problem, without providing any implicit or explicit connections between them. We are therefore left in doubt as to the purpose of his exercise.

A number of studies specifically concerned with African mineworkers,[51] amongst whom mortality rates were excessively high, investigate the medical care of these workers.[52] But most general academic studies of the mining industry seldom tackle health issues. It is astonishing that three fairly recent major works, those of Frederick Johnstone,[53] in 1976, of Robert Davies,[54] in 1979, and of David Yudelman,[55] in 1983, pay no attention whatsoever to the problem of silicosis. Yet these histories intensively explore the links between white labour and the gold mining industry from its pioneering days until modern times.

Most of the recent popular histories of the mining industry do not mention the spectre of silicosis, which for so many years provoked distress amongst miners.[56] This is not surprising as most of these histories are simply public relations exercises.[57] John Lang's recent institutional history of the Chamber of Mines does not, however, shirk the issue. Even so, his treatment of the disease is scanty: he does not give it the prominence which it warrants and to which the Chamber's archival records after 1911 attest.[58]

Silicosis is a theme in most academic studies which have investigated the origins and growth of labour organisations. Even so, most historians, including myself, have underestimated the importance of the disease. For instance, Francois Grobler, David Ticktin and Karen Thorpe fail to connect the growing militancy of miners with their dread of the disease. Also, the realisation in 1912 that all underground workers shared a common danger underpinned the growing alliance between the different sections of white labour. Silicosis was as important, if not more so, than abstract socialist concepts, in helping to promote the unification of organised labour on the mines. The alliance found concrete expression in the general strike of 1913. Of course the general strike was prompted by a wide variety of worker grievances.[59] Even so, as Lord Gladstone, the Governor General of South Africa, perceptively understood, the catalyst in

the general strike was the "growing realization...in the last two years" by all underground workers that they shared the "frightful risks from the ravages of Miners' Phthisis to which their work exposes them".[60]

Histories of organised labour, including those by Ernest Gitsham and James Trembath in 1926 and by Ivan Walker and Ben Weinbren in 1961, as well as the biography of Bill Andrews written by R. K. Cope, have a credibility which is out of proportion to their contribution. Their ethos derives from a belief that active trade unionists know more about the inner workings of organised labour than do "outsiders". While this may be true of current events, the assumption has less validity with respect to historical events, when such writers have to draw on memories. Oral history can, of course, provide valuable insights, particularly when there is no documented record of events. Oral history can also often reflect perceptions in a more significant way than do many written accounts. But reminiscences have obvious limitations which relate to clarity of memory. Although Trembath, Walker and Andrews participated actively in organised labour during the first decade of the 20th century, they recounted the events during this period many years later.

With respect to the topic of miners in general, and to silicosis in particular, the limitations of these retrospective accounts by trade unionists are even more marked. The writers were all artisans and

not miners: both Gitsham and Walker were typographers, who did not therefore work on the mines and had no personal acquaintance with mining conditions. Trembath accumulated an enormous collection of press cuttings on labour activities throughout South Africa, which cover the period with which this study deals. But Trembath was based in Kimberley and did not have first-hand knowledge of conditions on the Witwatersrand at this time.

Andrews's account - through his biographer Cope - of events on the mines has more authenticity. From 1907 to 1912, as organiser of the Amalgamated Society of Engineers, Andrews had the opportunity to meet and talk with many miners, as distinct from the mineworkers whom he recruited as members of his own organisation.[61] Also, before his election as organiser of his union, Andrews spent many years as a fitter in the mines' workshops. Even so, when Cope compiled the biography, Andrews was already an old man. Some of his recollections, including those concerning the Crown Reef strike in 1902, are anachronistic.[62] Similarly, his reminiscences of the threat of silicosis are hazy.[63]

More important, the writers of these books lacked access to the records of the Transvaal Miners' Association and could not verify their accounts. Nor could any pioneer miners reminisce: miners' oral accounts of their own experiences would have assisted the trade union writers to compile their studies. By

1926, when Pitsham and Trembath published the first trade union history, all the pioneer miners, who had stuck to their calling, were dead: most had perished from an occupational disease, silicosis.

The present-day South African Mine Workers' Union, the successor to the Transvaal Miners' Association, founded in 1902, claims that it does not possess any records of the pioneer union. As access to this valuable source is clearly impossible, we have to look in many other repositories for information concerning the early-day overseas miners.

A valuable source for the experiences and perceptions of miners of their profession in general, and of silicosis in particular, is the evidence of miners given before many government commissions and parliamentary select committees during the period 1897 to 1914. The evidence of numerous miners to the Mining Industry Commission during 1907 to 1908 is especially valuable, as it provides important insights into miners' terms of service and underground working conditions both before and after the Anglo-Boer War. Similarly, the testimony in 1907 of Thomas Mathews and Mathew Trewick, officials of the Transvaal Miners' Association, to the Mining Regulations Commission reflect accurately the shocking conditions and the excessive dust levels which existed on the Witwatersrand gold mines.

Both Mathews and Trewick were industrious and responsible miners. More important, they were "exceptionally well-informed".[64] After leaving his birth-place, Cornwall, during the 1880s and the 1890s Mathews worked in a variety of metal mines in the western states of America. In Montana he was appointed Commissioner of Mines and Mining and was elected from 1892 to 1894 as Speaker of the House of Representatives.[65] On returning to Cornwall Mathews studied at the Camborne School of Mines and came to the Witwatersrand in 1897 as a graduate.[66] Similarly, before emigrating to the Transvaal, Trewick had had seventeen years of experience in coal mining in Northumberland, where he had been appointed as a local colliery inspector.[67] Both these officials of the Transvaal Miners' Association had the experience and expertise to compare objectively conditions on the Witwatersrand with those in many other mining centres.

Unlike most other chairmen of commissions,[68] Andries Stockenstrom, who chaired the Mining Industry Commission, allowed the evidence of an unlimited number of witnesses. The eagerness with which many miners, who were not union officials, volunteered to testify, showed their desire for improved mining conditions: most miners confessed that they were frightened of contracting silicosis. They were prepared to risk victimisation by management,[69] to give up valuable working time, to forgo a day's pay

and to face the "ordeal" of "cross-examination in much the same way as a guilty prisoner".[70] For purposes of this study, the approximate 1 600 pages, which comprise the minutes of evidence of the Mining Industry Commission, are worthy of painstaking scrutiny.

Like Mathews and Trewick, most professional miners came from Great Britain.[71] Approximately 50 per cent of the miners had originally learned their vocation in Cornwall and had a thorough training in hard rock mining.[72] Many miners had additional expertise, which they had acquired abroad. Consequently most miners had a sound yardstick, based on experience, against which to measure both health and work conditions on the Rand. This was the basis of their testimony before all the commissions and select committees, which we have noted. We must acknowledge the validity of miners' perceptions in the same way as did M. H. Coombe, the manager of the State Min , in 1906:

> It is the common belief amongst miners, and I am with them there, that this is the most unhealthy camp, from a miner's point of view, that can be found, not even excepting the Australian Broken Hill silver mines.[73]

As we have seen, Cornwall provided approximately 50 per cent of miners on the Witwatersrand.[74] From no other county in Britain did miners emigrate to the Transvaal on such a large scale. Cornwall has another dimension of importance, particularly for social scientists specialising in the history of the

Witwatersrand gold mines. No British community was as intimately involved with the activities of its mining kin abroad as the district of Redruth. Local newspapers, published in west Cornwall, maintained and reinforced the links between the west county and far-flung mining communities. The press carried regular columns and articles, which covered both trivial and significant events, concerning the personal and communal involvements of Cornishmen abroad. Of course the newspapers devoted a great deal of attention to the state of trade abroad and the availability of jobs. Even so, many items concerned the intimate details of Cornishmén abroad: the birth of children, marriages, funerals and felonies. News coverage also focused on happenings at overseas mines. Such commentaries ranged from accidents, promotions and presentations to social and recreational activities, including sporting events such as soccer and wrestling.[75]

This type of journalism was unique to Cornwall and occurred in no other mining centre in Great Britain.[76] Consequently Cornwall possesses a valuable repository of source material which illuminates the historian's perceptions of the aspirations and daily activities of migrant miners abroad: of Cornish miners in particular, and of British miners in general.

But Cornish newspapers constitute only one of many important documentary sources concerning the history of the Witwatersrand miners. Of equal

importance for the purposes of this study are contemporary Cornish medical records and reports, which are also unique. The number of returned Witwatersrand miners who died in the "rapidly filling graveyards" of Cornwall is well documented.[77] This was not so in the other mining centres in Britain, as the Home Office, in 1912, confirmed:

> The Secretary of State understands that members of the Royal Commission which is now making an enquiry with regard to metalliferous mines and quarries in this country have heard of deaths from Miners' Phthisis in districts in the North of England among men who have returned from the Transvaal, but that it would be also impossible to obtain statistics, as the cases are scattered among a large population.[78]

Statistical information was also defective for the iron and colliery districts of South Wales.[79]

During the period 1896 to 1907 approximately 95 per cent of professional miners on the Witwatersrand were of overseas extraction; and most of them had been born in Britain.[80] Many miners who contracted silicosis in its rapidly progressive form on the Witwatersrand returned home to die. On the basis of statistics compiled in 1906 by Louis Godfrey Irvine and Donald Macaulay, two Transvaal mine medical officers who showed a marked interest in the occupational disease, the Mining Regulations Commission estimated that "at least one-third (and probably more) of the disabled miners", did not die on the Reef. It is for this reason that the Cornish death rate figures for silicosis are invaluable.

From 1907 medical officers of health in both the rural and urban districts of Redruth, the major Cornish tin mining centre, kept separate records of the mortality from silicosis amongst rock drillers who had worked only on the Witwatersrand.[81] Miners from Redruth comprised approximately one-third of the overseas contingent of miners on the Witwatersrand. Therefore in order to obtain more comprehensive data on the death rate from silicosis, which so severely affected migrant miners who worked on the Witwatersrand, the Redruth mortality statistics can be applied, even if cautiously and tentatively, to other United Kingdom and overseas mining districts.

For the period 1902 to 1910 there are no official data for the mortality from silicosis of miners on the Witwatersrand. In fact, there are "no accurate occupational death-rates" at all.[82] Charles Lane Sansom, the Witwatersrand Medical Officer of Health from 1902 to 1907, compiled figures for the mortality amongst mineworkers from lung diseases, which Irvine and Macaulay used for their 1906 and 1907 investigations. Even so, the research doctors found the statistics to be unsatisfactory:

> The data obtained from the Registrar [of the Witwatersrand Medical Officer of Health] do not permit a separation to be made between the underground and surface employees on mines, or amongst the former to distinguish rock drill men from general miners. This is unfortunate and should be remedied.[83]

Charles Porter, the Johannesburg Medical Officer of Health, was a member of the Mining Regulations

Commission to which Irvine and Macaulay presented this testimony. Had the mortality data from the Johannesburg municipality been of any value to the commissioners, they would have included them in their statistical conclusions.[84] For these reasons this study does not use the Reports of the Johannesburg Medical Officer of Health. They are of no value for the period under review.[85]

For the period 1902 to 1910 the Transvaal Archives Depot possesses no epidemiological or demographic data on silicosis whatsoever.[86] Even Sansom's statistics are missing. The documents with the information were either lost, "mutilated" or destroyed.[87] The disappearance of these data was discovered in 1912, when the Minister of Mines, F. S. Malan, sent an urgent telegram to Pretoria requesting them for presentation to parliament.[88] Neither the Department of Mines nor the Department of Interior could "trace any papers on this subject" for the period 1903 to 1909.[89]

Likewise, the archives of the Chamber of Mines have no statistics for incidence, prevalence and mortality data from silicosis.[90] In compiling epidemiological data I therefore rely on accurate statistics compiled after 1914. Also, I base my estimates on fragments of contemporary evidence: they include both the Cornwall figures and the statistics compiled by the private consulting mining engineer, E. J. Moynihan, which he published sporadically in

Transvaal newspapers between 1910 and 1913.

There is very little general information on silicosis in both the Transvaal Archives Depot and in the Transvaal Colonial Office files in the Public Record Office: only a handful of despatches pertain to the problem. For the period 1902 to 1903 the duplicates in the Public Record Office provide a more comprehensive source of information than their counterparts in the Transvaal Archives Depot. This is because they include the enclosures which the Colonial Secretary, Joseph Chamberlain, sent to Alfred Milner, the Governor of the Transvaal. The enclosures consisted of letters and published articles written by British doctors and mining personnel. They were concerned about the mortality from silicosis amongst miners who had returned to Britain during the Anglo-Boer War and suggested to Chamberlain that dust precaution measures be adopted on the Witwatersrand gold mines.

On the arrival of the documents in South Africa, the Transvaal administration separated the enclosures from the covering letters placing them in separate files. The files containing the covering letters are available in the Transvaal Archives Depot. But the enclosures, like all the documents on silicosis for the period 1903 to 1909, have been "lost".[91] The missing file, SF 34, contained the Colonial Office enclosures as well as most other information pertinent to the appointment, in 1902, of the Weldon Commission

on silicosis in the Transvaal.

Apart from the despatches, there are fragments of useful material in the Transvaal Archives Depot. I found a few on the computer under "miners' phthisis". Most are not accessed so simply, but are scattered throughout the hundreds of files which comprise the archives of the Secretary of Mines in both the Transvaal Archives Depot and the Central Archives Depot.

To find material relevant to the health of black and white mineworkers in general, and to silicosis in particular, the researcher in the Public Record Office must scrutinise almost every file in the Transvaal Colonial Office section. Similarly, in the Transvaal Archives Depot the historian can find isolated but significant pieces of evidence only by laboriously searching through virtually all the official files.

Milner, Chamberlain and Patrick Duncan, the Colonial Secretary of the Transvaal and the colony's acting Lieutenant-Governor in 1906, spoke publicly about the problem of silicosis. But they did not mention it in their private correspondence.[92] Likewise, neither the published letters of Jan Smuts, who became the first Union Minister of Miner in 1910, nor those of J. Percy FitzPatrick, a director of the Corner House, mention the disease.

I found only a few relevant documents in the archives of H. Eckstein and Company (housed in the

Barlow Rand Archives), so confirming what the librarian had initially told me, that "there was practically nothing" on silicosis. The same was true of the archives of the Transvaal Chamber of Mines. Apart from the files concerning the establishment and building of Springkell - the sanatorium for miners disabled by silicosis - the problem of the occupational disease did not warrant correspondence by the Chamber.[93]

For the period 1902 to 1910 the dearth of primary source information concerning silicosis in these important repositories does not in any way diminish the importance of the subject. Rather, the absence of commentary is significant. In "Silver Blaze" Sherlock Holmes wanted to know why the dog did not bark, when it could well have been expected to do so. In a similar way the silence of these important people and institutions both whetted my curiosity and provided clues. Silicosis was not a high-profile disease. Nor was it a contagious one which placed the entire community at risk. The silence of all these parties confirmed that they were totally unconcerned with and indifferent to a serious health problem, which concerned only members of the working class.

The private papers of John X. Merriman, Prime Minister of the Cape Colony before Union, are a valuable source of information concerning silicosis. In 1912 Merriman was a member of the first Parliamentary Select Committee on silicosis.[94] As

important, his nephew, Richard Barry, manager of the Nourse from 1911 to 1916, was one of the few mine managers who initiated effective dust precaution measures on his mine. In 1911, for instance, C. J. N. Jourdan, an inspector of mines, in a paper presented to the South African Institution of Engineers, recommended the universal adoption on the mines of Barry's system for wetting the siliceous dust.[95] Barry's regular and intimate correspondence with his uncle provides an important source for the inner workings of the gold mining industry, particularly with respect to the problem of silicosis.

The South African medical journals, the *South African Medical Record*, published from 1902, and the *Transvaal Medical Journal*, started in 1905, provide important insights into the practice of medicine in the Transvaal. They contain little information on occupational medicine in general, and on silicosis in particular. With respect to silicosis the two medical journals published in Britain, namely the *British Medical Journal* and the *Lancet*, are more useful. They contain articles written by Sir Thomas Oliver and John Scott Haldane, two doctors concerned with the prevention of occupational diseases. The prevalence of and mortality from silicosis amongst the Witwatersrand miners distressed them: they publicised the disease and alerted the public to the need for precautions.

Between 1902 and 1910 singularly few Transvaal doctors wrote articles on silicosis. On the whole the published periodical contributions of doctors between 1902 and 1916 are disappointing. The articles, with few exceptions, are compressed summaries of official commission reports.

In deploring the gravity of the disease and in advocating the need for preventive measures against dust, the medical literature, like the government reports, briefly and uncritically reviewed the circumstances of the past which had created the present serious situation. The motto of the writers seemed to be based literally on the popular quotation of the 19th century British poet and diplomat, Robert Bulwer Lytton:

> Not to the past, but to the future looks true nobility; and he who seeks for true repentance for the past must woo the angel Virtue in the future.[96]

With few exceptions, South African doctors, commissioners and investigative parliamentarians refused to acknowledge the past liability of the various Transvaal governments and the mining industrialists for condoning the existence of dust conditions which by 1910 had created a problem of great magnitude - one equivalent to a "national calamity".[97] When these parties made value-judgements as to who was liable for the disease, they blamed only the miners for their lack of responsibility in observing precautions.[98] They continued to do so even

after 1912, when the Medical Commission showed clearly that all underground mineworkers, irrespective of whether or not their jobs created dust, were prone to silicosis.[99]

Two original contributions by doctors on silicosis amongst African mineworkers are valuable. In 1906 the Witwatersrand Native Labour Association sent its medical officer, George Albert Turner junior, to Portuguese East Africa, south of Latitude 22° South, to investigate and report on the prevalence of pulmonary tuberculosis and other lung diseases amongst the Africans in their "kraals".[100] Turner concluded that silicosis was a minor health risk for African contract workers.[101] This finding, based only on cursory observations and perfunctory physical examinations, both confirmed current medical assumptions and established a theory which, until the present, has held unchallenged sway. Scepticism concerning the theory prompted medical research with animals during the 1930s. But the results of the investigations were inconclusive.[102]

The second important contribution in this field is the doctoral thesis of the Medical Inspector of Mines, Sebastian Valentyn van Niekerk, submitted to the University of Aberdeen. Much of the study is based on the report and findings of the 1912 Medical Commission on silicosis, of which he was chairman. Its value lies in the occupational, epidemiological and demographic features of his clinical investigation

of twenty-four cases of silicosis amongst African mineworkers. Also, the reasons for van Niekerk's personal objections to investigating the prevalence of silicosis amongst Africans throw light on the importance contemporaries attached to Turner's theory.

Mention must be made of the paper, "Safety Measures in Mining", read in 1906 by D. Macaulay and L. G. Irvine to the Chemical, Metallurgical and Mining Society of South Africa. The second part of their paper is an original investigative study of silicosis, which emphasises its epidemiolgical severity on the Witwatersrand. The two doctors up-dated the 1906 death-rate figures and in 1907 Irvine presented the paper as testimony to the Mining Regulations Commission, an important health commission, which its title does not indicate. The implications of the findings of the two doctors were serious. The commission immediately sent a directive to the Minister of Mines, Jacob de Villiers, urging him to amend the dust precaution regulations so that they would be more effective.[103]

The *Journal of the Chemical, Metallurgical and Mining Society of South Africa* is a major source for this study. Founded in 1894 the society, as its name suggests, attracted as members assayers, chemists, metallurgists and mine managers most of whom were directly or indirectly connected with the mining industry. The monthly seminars were topical and

significant. During the period two papers fully and directly addressed silicosis. Other topics concerned preventive measures, including ventilation.

For purposes of this study both the papers and their discussion, which the journal published verbatim, are invaluable. Discussion of each topic often occupied several meetings. Sometimes the members gave impromptu comments. But often they carefully prepared their arguments in writing as formal presentations. Through the cut and thrust of critical debate, and in an atmosphere where members were encouraged to give expression to personal opinions, without the constraints of professional, managerial and industrial affiliations, we find a wide variety of public socio-economic responses to the problems associated with silicosis.

The *Journal of the South African Institution of Engineers* has a format similar to that of the *Journal of the Chemical, Metallurgical and Mining Society of South Africa*. The papers in the engineering journal are more technical, as are the debates. Even so, the personal opinions of the society's members, comprising mine managers and engineers, provide valuable insights into management's attitude to the disease and to its prevention.

The minutes of the Association of Mine Managers are also a valuable source of evidence. As a body, the mine managers expressed concern for the health and

welfare of miners. In 1901 the organisation was responsible for revealing the mortality from silicosis and for instituting preventive measures voluntarily.[104] Also, the Association initiated the establishment of Springkell, a sanatorium for silicotic miners.[105]

Committee decisions could not, and did not, bind individual mine managers to implement them. The mine managers' corrective resolutions, which committed them to eradicate silicosis, more often than not conflicted with issues involving individual self-interest and the economic constraints of running the mines at a profit. An examination of the minutes of the Association of Mine Managers in combination with the discussions of the Chemical, Metallurgical and Mining Society and those of the Institution of Engineers reveal clearly the many contradictory attitudes of management to the problem of silicosis.

William Cullen, the General Works Manager of the British South Africa Explosives Company, was an energetic member of the Chemical, Metallurgical and Mining Society and was several times elected as its president. In addressing a problem associated with silicosis, in 1905 Cullen's presidential speech urged the members to adhere to the "aims" of the organisation. "The truth must out," he said, "[even if] the publication of unpalatable facts may or may not suit the policy of the government and the mining houses."[106] By piecing together the fragments of

evidence garnered from so many sources, this study, too, hopes to tell the "truth" about silicosis during the period 1886 to 1910.

Notes

[1] *Transvaal Leader*, 23 Aug. 1910, letter from "A Miner".

[2] *Mining Journal*, 17 Feb. 1912, p. 160, "Miners' Phthisis: The Penalty for Neglect".

[3] *GNEAR...30 Dec. 1901*, pp. 10-11.

[4] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. vii-viii, par. 10.

[5] UG 19, 1912, p. 36, Table 1, "All Miners".

[6] UG 19, 1912, p. 13, par. 32.

[7] UG 19, 1912, p. 40, Table III.

[8] *JCHMS*, Jan. 1907, p. 230, "Miners' Phthisis on the Bendigo Field".

[9] *Mining Journal*, 17 Feb. 1912, p. 159, "Miners' Phthisis: The Penalty for Neglect".

[10] See below, chapter 2.

[11] See, for instance, BRA, HE, v. 258, file 154M, R. G. Nesbitt to F. Oats, 15 July 1902, F. Hiehens [sic] [Hichens] and A. E. Pennewan [sic] [Permewan] to F. Oats, 21 July 1902, C. S. Jago to F. Oats, 22 July 1902.

[12] Van Niekerk, p. 62.

[13] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 136, q. 1 332, evidence of Dr W. C. C. Pakes.

[14] A "crude prevalence" of silicosis may be defined as the total number of cases of the disease at one time without categorising them according to their degree of seriousness.

[15] See, for instance, *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 240, evidence of Dr L. G. Irvine.

[16] J. Pratt Johnson, p. 334.

[17] J. Pratt Johnson, p. 334.

[18] The nick-name seems to have been coined by the *Transvaal Leader*, when it commented on the

findings of the Mining Regulations Commission which were published in August 1910. See *Transvaal Leader*, 11 Aug. 1910, editorial. From then on the nick-name became a standard expression.

[19] This was the case with Africans who came from Portuguese East Africa, south of Latitude 22° South. After the Anglo-Boer War they contracted with the Witwatersrand Native Labour Association for twelve months but usually stayed for six months longer. See SC 2, 1913, pp. 114-115, qq. 1 168-1 169, evidence of Dr G. A. Turner. Africans who came from the Transvaal and the rest of British South Africa undertook shorter contracts which varied from three to eight months. See *Rand Daily Mail*, 3 April 1903, "Labour Association"; SC 9, 1913, p. 200, q. 1 424, statement of C. H. Spencer presented by J. G. Lawn.

[20] SC 2, 1913, p. 117, q. 1 208, evidence of Dr G. A. Turner.

[21] SC 2, 1913, p. 117, qq. 1 213-1 214, evidence of Dr G. A. Turner. See also van Niekerk, pp. 30-31, 284-286.

[22] In his 1969 doctoral thesis on the development of the gold mining industry from 1902 to 1910, Grey devoted twenty-five pages - part of a chapter on health conditions on the mines - to silicosis. Ten years later, in 1978, Burke and Richardson, from a materialist standpoint, examined the "migration" of the disease between Cornwall and the Witwatersrand. Their article is twenty-four pages in length and their time span is from the 1890s until 1918. More recently, in 1989, van Aswegen contributed a descriptive survey paper of twenty-six pages. Based largely on official published sources, van Aswegen's paper spans the period 1886-1920.

[23] The controversy, initiated by contemporaries, concerning the relationship between Milner and his successors and the mineowners, is still endlessly debated. Denoon argues that the Randlords in pursuit of profits dominated Milner. See, for instance, Denoon, "The Transvaal Labour Crisis, 1901-6", pp. 481-494 passim, "'Capitalist Influence' and the Transvaal Government during the Crown Colony Period, 1900-1906", pp. 301-331 passim, "Capital and Capitalists in the Transvaal in the 1890s and 1900s", pp. 111-132 passim. The radical historians, Wolpe, pp. 429-430, and Legassick, pp. 260-261, support Denoon's argument, but from a different perspective. Against this, other historians contend that the Governor of the Transvaal manipulated the mineowners for his own political ends. See, for instance, Mawby, pp. 387-415 passim, who argues that Milner used the mining industry to achieve his political goals.

[24] This study takes up the debate in several chapters where reference is also made to other relevant literature, including the revisionist article by Marks and Trapido.

[25] In his review article of Yudelman's study, Davenport, o. 96, indicates clearly that Yudelman's theory is not entirely original.

[26] Cf. van Aswegen, p. 55, who claims that silicosis is an industrial disease "peculiar to the gold mines of the Witwatersrand". As we shall show later, in chapters 2 and 3, such an assertion is fallacious.

[27] Union *Statutes*, 1911, no. 34.

[28] See for instance, Irvine et al, p. 9; Payne et al, p. 8; and *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 2.

[29] Van Niekerk, pp. 26-27.

[30] UG 19, 1912, pp. 14, 15, 18, pars. 27, 31, 42, 43, 44; van Niekerk, p. 29.

[31] See, for instance, PRO, CO, 291/57, despatches, Milner to Chamberlain, 23 May 1903, enclosure; *Transvaal Government Gazette*, 1906, p. 519, "Coloured Labourers Health Regulations"; *Final Report of the Mining Regulations Commission*, 1910, v. 1, pp. 55-56; and *TCMAR*, 1896, p. 272.

[32] See, for instance, *Transvaal Government Gazette*, 1906, p. 519, "Coloured Labourers Health Regulations"; and *TCMAR*, 1908, p. 423.

[33] See, for instance, TG 2, 1908, p. xlv.

[34] See, for instance, TG 2, 1908, pp. 879, 987, 1 456, 1 494, qq. 12 672-12 682, 20 967, 21 597, evidence of W. T. Anderson, J. Davies, J. Monroe and W. W. Mein.

[35] The role of Indians and coloured persons on the diamond and gold mines is a potentially fruitful area for future historical research.

[36] *Final Report of the Mining Regulations Commission*, 1910, v. 1, pp. 65-87 ; sim.

[37] TG 2, 1908, pp. 108, 137, q. 769, evidence and annexure of L. J. Reyersbach. See also *GMEAR...30 June 1905*, Table 7.

[38] A tiny group of unskilled white labourers was also employed on the gold mines. Most of these labourers performed surface tasks. Management undertook experiments with white unskilled labourers in underground occupations both before and after the Anglo-Boer War. With the notable exception of F. H. P. Creswell, most engineers and managers found it unprofitable to employ them. See Katz, *A Trade Union Aristocracy*, pp. 78, 82-83, 117-119.

[39] See, for instance, Ticktin, pp. 259-260;

Mawby, p. 289; and Cammack, *Class, Politics and War: A Socio-Economic Study of the Uitlanders of the Witwatersrand, 1897-1902*, pp. 24-25. Much of Cammack's evidence for the living conditions of miners is based on the letters of John Cockerill, who spent a mere six months, from May to October 1902, as a rock-drill learner. He spent the remainder of his three-year stay on the Witwatersrand working for an independent contractor doing diamond-drill boring. See John Cockerill, letters, passim. Likewise, Thorpe's analysis of the early strikes on the mines of the Witwatersrand, particularly of the 1907 miners' strike, has major weaknesses because she does not distinguish miners from artisans. For instance, she classifies, p. 7, W. H. Andrews, a fitter, as a miner.

[40] TCMA, file A1(b), consulting engineer of the Johannesburg Consolidated Investment Company to Secretary of the TCM, 4 Sept. 1907.

[41] Calculations based on TG 2, 1908, pp. 83-84, annexure E, evidence of H. Weldon; *GMEAR...30 June 1905*, Table 6; and BRA, HE, v. 134, S. Evans to F. Eckstein, 11 Dec. 1905.

[42] For details, see below, chapters 5 and 6.

[43] See, for example, *The Mining Industry*, 1897, pp. 46, 119-120, evidence of S. J. Jennings and C. S. Goldmann.

[44] Hannan, *Letters of a South African Miner, 1898-1904*, pp. 9, 13.

[45] Katz, "Silicosis on the South African Gold Mines", p. 241, n. 204.

[46] Du Toit et al, p. 4. In addition to those mentioned by du Toit et al, two other commissions must be listed: the *Report of the Miners' Phthisis Commission, 1902-1903*; and the *Final Report of the Mining Regulations Commission*, 1910.

[47] TCMA, file M26, Secretary of the Minister of Mines to Secretary of the TCM, 18, 28 Oct. 1912.

[48] *Preliminary Report of the Miners' Phthisis Prevention Committee*, 1912; *Interim Report of the Miners' Phthisis Prevention Committee*, 1913; *General Report of the Miners' Phthisis Prevention Committee*, 1916; *Final Report of the Miners' Phthisis Prevention Committee*, 1919.

[49] Grey, pp. 300-325.

[50] Kennedy, *A Tale of Two Mining Cities*, p. 64.

[51] Julie Baker recently submitted a doctoral thesis concerning the health of African mineworkers. As I was in the final stage of my study, unfortunately I was unable to consult it. See Baker, Julie, "The

Silent Crisis: Black Labour, Disease and the Economics and Politics of Disease on the South African Gold Mines 1902 to 1930", unpublished Ph.D. thesis, Queen's University at Kingston, 1989. For the same reason I could not consult the recently published full-length study by Randall M. Packard, which specifically investigates tuberculosis amongst African mineworkers. Packard's work is entitled *White Plague, Black Labour: Tuberculosis & the political economy of health & disease in South Africa*, London: 1990.

[52] See, for instance, Jeeves, *Migrant Labour in South Africa's Mining Economy*; and van Onselen. In his article, referred to in the bibliography, surprisingly Packard does not note the main reason which underpinned periodic medical inspections. The inspections enabled medical officers to identify tuberculosis amongst black mineworkers who were then withdrawn from service. This step was a medical precaution intended to prevent Africans from infecting both black and white mineworkers with tuberculosis, which promoted the development of the fatal form of "complicated" silicosis, medically termed progressive massive fibrosis.

[53] Johnstone, pp. 56, 99, mentions "miners' phthisis" in passing.

[54] Davies, *Capital, State and White Labour in South Africa, 1900-1960*.

[55] Yudelman, p. 71, in passing notes inaccurately that in 1907 the "ravages of miners' phthisis were just beginning to become obvious".

[56] Rosenthal, p. 344, pays scant attention - one paragraph - to the disease; and Cartwright, *The Gold Miners*, pp. 274-275, glosses over the implications of the disease and focuses almost entirely on the "victory" of its conquest. Wheatcroft does not mention the occurrence of the disease.

[57] See Cartwright, *The Corner House*, *Golden Age*, *Gold Paved the Way*. Likewise, the centenary celebration volumes of Anthony Hocking and Paul Jonnson make no reference to the disease.

[58] Lang, pp. 224-225, 340-341.

[59] Katz, *A Trade Union Aristocracy*, pp. 321-379.

[60] PRO, CO, 551/42, despatches, Gladstone to Harcourt, 20 July 1913. I thank Baruch Hirson for drawing my attention to this reference.

[61] Katz, *A Trade Union Aristocracy*, pp. 176-182 passim.

[62] Cope, p. 65.

[63] Cope, p. 36.

[64] Mann, p. 197, made this assessment of Mathews.

[65] TG 2, 1908, p. 386, q. 4 081, evidence of T. Mathews.

[66] TG 2, 1908, p. 428, q. 4 571, evidence of T. Mathews; Walker and Weinbren, p. 293.

[67] *Final Report of the Mining Regulations Commission*, 1910, v. 2, pp. 8, 34, evidence of T. Mathews and M. Trewick.

[68] TAD, MM, 1395/06, 22 May 1906, "Deputation from the Transvaal Miners' Association".

[69] TAD, MM, 1395/06, 22 May 1906, "Deputation from the Transvaal Miners' Association".

[70] *JCMMS*, April 1906, "Safety Measures in Mining", p. 257, discussant J. A. Wilkinson.

[71] Chapter 5 quantifies in depth the origins of miners.

[72] Calculations are based on the following: *GMEAR...31 June 1907*, p. 13; Ticktin, p. 3, quoting *South African Review*, 26 Nov. 1909, p. 26; *ARTCM*, 1900-1901, p. 63 and *ARTCM*, 1904, 1907, 1909, pp. 492, 379, 370, "Distribution of White Employees"; CAT, Boksburg Branch Minutes, 20 March 1912-30 Nov. 1915 passim; *JCMMS*, Oct. 1906, "Safety Measures in Mining", pp. 114-115, discussant J. Yates; and BRA, HE, v. 134, S. Evans to F. Eckstein, 11 Dec. 1905.

[73] *JCMMS*, Oct. 1906, "Safety Measures in Mining", p. 114, discussant M. H. Coombe.

[74] See below, chapter 6.

[75] See *Cornubian*, 1892-1913 passim: and *West Briton*, 1892-1913 passim.

[76] This was particularly true of the *Cornubian* and the *West Briton*, published at Redruth and Truro. The *Cornishman*, published at Penzance, covered foreign events concerning emigrant's activities only when they had national importance. In contrast, the *Barrow Herald* and *Barrow News*, published at Barrow-on-Furness, the *Dalton News*, published at Dalton-on-Furness, and the *Newcastle Daily Chronicle* and the *Newcastle Weekly Chronicle*, published at Newcastle-upon-Tyne, did not carry parochial news items concerning emigrant miners from these districts.

[77] *Cornubian*, 10 July 1913, "The Rand Strike".

[78] UG 19, 1912, p. 21, par. 56.

[79] UG 19, 1912, p. 21, par. 56.

[80] See below, chapter 6, for calculations and quantification data.

[81] Cd. 7476, 1914, pp. 139, 147-148, Table 1.

[82] *JCMMS*, Dec. 1906, "Safety Measures in Mining", p. 172, reply to discussion.

[83] *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 240, evidence of Dr L. G. Irvine. See also *JCMMS*, Dec. 1906, "Safety Measures in Mining", pp. 172-173, reply to discussion.

[84] *Final Report of the Mining Regulations Commission*, 1910, v. 1, pp. 33-39 passim. See also van Niekerk, p. 27.

[85] Cf. Kennedy, *A Tale of Two Mining Cities*, p. 64, who uses the statistics from 1904-1915 to show an increase in silicosis deaths amongst African mineworkers. From 1909 Porter ensured that the annual death-rate returns were accompanied by relatively specific occupational indicators. Also, his report for 1909 to 1911 included a separate statistical schedule which listed deaths and their causes amongst the Africans employed on the mines situated in the Johannesburg municipality. See *SAMR*, 10 Aug. 1912. pp. 326-330, "Report of the M.O.H. for Johannesburg 1909-1910 and 1910-1911"; *BMJ*, 5 Oct. 1912, p. 905, "South Africa". Even so, both the Medical Commission of 1912 and van Niekerk in his doctoral thesis of 1914 found them to be of dubious value. See UG 19, 1912, p. 21; van Niekerk, p. 27.

[86] *Union House of Assembly Debates*, J. X. Merriman, 16 June 1913, col. 3545.

[87] *TNJ*, July 1912, p. 254, editorial.

[88] CAD, MNW, file MM 1505/1912, F. S. Malan to Secretary of the Department of Mines, 18 May 1912, telegram; *Union House of Assembly Debates*, W. Madeley, 21 May 1912, cols. 2810-2811.

[89] CAD, MNW, file MM, 1505/1912, memorandum, 21 May 1912.

[90] BRA, HE, v. 258, file 154M, Memorandum by W. Gemmill, 7 Oct. 1910, "Provision for Miners' Phthisis Sufferers", p. 3.

[91] CAD, MNW, file MM, 1505/1912, memorandum by R. Vaughan, 21 May 1912.

[92] The association of Milner and Chamberlain with the occurrence of the disease is perhaps rather more obvious than that of Duncan. Even so, it should be noted that in 1910 Duncan represented the working-class constituency of Fordsburg in the Union House of Assembly and soon established a reputation for showing sympathy to the "White Labour Policy" of the South African Labour Party. At this time the

problem of the disease and the need for the compensation of miners disabled by silicosis were issues which were beginning to make headline news in the South African press. It is therefore surprising that only one private letter in the Duncan Papers concerns the problem of silicosis. Duncan received it in 1936, when he was Minister of Mines. See Duncan Papers, A5. 3.1, G. E. Barry to P. Duncan, 27 Jan. 1936. For biographical details on Duncan, see "Introduction" to the inventory of the Duncan Papers, p. ii; and Katz, *A Trade Union Aristocracy*, pp. 461-462.

[93] Of course there is much information on the subject after 1911, when legislation obliged the mines to pay compensation to disabled miners.

[94] SC 10, 1912.

[95] Jourdan, pp. 48 ff.

[96] The Victorian quotation was popular in South Africa. See, for instance, *JSAIE*, Jan. 1912, "The Prevention of Dust in Development Drives of Mines during Drilling Operations, p. 141, discussant K. Austin.

[97] Merriman Papers, correspondence, 1912, no. 98, memorandum on "Mine Accidents".

[98] See, for instance, Jourdan, p. 242; Macaulay and Irvine, p. 300; and *Final Report of the Mining Regulations Commission*, 1910, v. 1, p. 41.

[99] See, for instance, Irvine et al, p. 9; UG 40, 1913, pp. 149-150, Mines Medical Inspector's Report; Watkins-Pitchford, "The Industrial Diseases of South Africa", p. 37; and SC 10, 1912, p. viii, memorandum by J. X. Merriman.

[100] The omission of this important source in the study of Packard is surprising.

[101] Turner, *Report on the Prevalence of Pulmonary Tuberculosis and Allied Diseases in the Kraals of the Natives of Portuguese African Territory, South of Latitude 22°...*, p. 17.

[102] Beadle et al, pp. 473-477.

[103] *Final Report of the Mining Regulations Commission*, 1910, v. 1. pp. 281-282.

[104] See, for instance, Council minutes of the AMM, 5 Aug. 1901; and Monthly minutes of the AMM, 19 Aug. 1901.

[105] See, for instance, Monthly minutes of the AMM, 13 Sept. 1908.

[106] *JCMMS*, July 1905, p. 7, "Presidential Address".

CHAPTER 2

MEDICAL ASPECTS OF SILICOSIS: AN HISTORICAL PERSPECTIVE

> "The dust and stones fall upon the lungs, so that men have lung disease, breathe with difficulty and at last take consumption."---Dr G. E. Loehniss, 1690.[1]

> "But behind this great industrial development [the gold mining industry] there has lain another and sinister reality, that of miners' phthisis. It is the shadow that has accompanied that spectacular advance - the 'black care' which has sat always behind the horseman...'Miners' phthisis' and 'silicosis' are used as equivalent terms."---Dr L. G. Irvine and Dr A. Mavrogordato, 1929.[2]

In 1863 Edward Williams, a physician from Wales, in evidence to the Kinnaird Commission on metal mining in Britain, said:

> I do not recollect that I ever attended them [miners] for the miners' asthma...and I think that they do not seek any advice from, perhaps, the misfortune that we [doctors] cannot give them much assistance. It is a thing which goes on...I never recollect being called in particularly to a case of miners' asthma...The men think that there is no remedy; it is a thing which they think that they have to submit to, and they submit to it with the best grace they can, and they

drag on their existence as well as they can.[3]

The testimony of Dr Williams reveals his perception of miners as fatalists. He believed they faced the prospect of contracting occupational lung disease with its accompanying spider of premature death with stoicism. This was not an idiosyncratic view but one that was typical of his profession. The opinion was so pervasive that it took root in the mystique and mythology attaching, in particular, to the metalliferous miners of Cornwall.[4] This chapter does not test the validity of the assumptions of Dr Williams, for the theme is explored later. It rather attempts to provide an historical context for the changed medical attitudes towards patients suffering from pneumoconiosis. Present-day doctors do not regard miners as being fatalistic towards the disease. Instead, they acknowledge that their patients express feelings which range from resentment to pessimism.[5]

Also, this section provides an historical overview of the origins, causes, development and progression of silicosis; and it further attempts to explain its association with the infectious disease, tuberculosis of the lungs, which it analyses in detail. A great deal of research undertaken after World War 11 has considerably enhanced medical knowledge of silicosis; and the need for a current understanding of this complex and slow-developing lung illness is well illustrated by two recent articles dealing with its manifestation during the late 19th

and early 20th centuries in the Transvaal - and in Cornwall. In both these articles the writers obscure the reader's comprehension of the disease by locating their discussions in out-of-date definitions and outmoded research findings.[6] The result is that many of their conclusions and judgements, particularly those which pertain to incidence, prevalence and mortality data of the disease in the past, are highly questionable. Also, their misunderstanding of the pathology of the disease has resulted in their over-emphasising relatively unimportant issues and their dismissing other significant features of the disease, particularly the morbid potential of silicosis in its simple form.

Because the meanings of medical terms have changed, it is often difficult to quantify data regarding diseases of the past. This is particularly true of silicosis because of its still controversial association with tuberculosis of the lungs. It is therefore hoped that the portion of this chapter devoted to terminology will better equip the reader to understand and interpret contemporary medical reports and statistics which are quoted in subsequent chapters.

The chapter also focuses on the occurrence of pneumoconiosis in industry and mining in Britain, the original home of most of the coal and hard rock miners who migrated to the Witwatersrand gold fields. Although poor health conditions existed in the

majority of the metalliferous mines in Missouri, Colorado, Utah, California and Nevada before World War 1, few American doctors were familiar with or interested in work done on the subject of pneumoconiosis in Europe, in Britain and in its overseas possessions.[7] Also, the subsequent research findings of the American doctors had minimal impact in their own country; it was not until 1929 to 1930 that the federal government and American industry turned their attention to the problem.[8] For these reasons the history of silicosis in the USA has been omitted.

This chapter deliberately draws a mono-causal link between the industrial revolution and the proliferation of occupational diseases. But the link is established merely for a survey and to locate the pneumoconioses in a chronological context. The complex relations between the social, economic and political aspects of occupational disease and of industrial medicine are discussed later. The chapter does, however, focus on the important contributions of South African doctors in advancing medical knowledge of the disease, with particular reference to its pathology.

Silicosis is classified as one of the forms of pneumoconiosis, which is the generic term for a group of lung diseases associated with dusty occupations. These diseases arise almost exclusively from industrial pursuits and are therefore preventable. Also, in their simple form the diseases are not

infectious so that they cannot be communicated from one person - or animal - to another.[9]

Pneumoconiosis, meaning "dust-containing-lung", is an 1874 abbreviation of the word pneumonokoniosis, a term coined in 1866 by a German pathologist, Friedrich von Zenker.[10] Today the word does not merely describe dusted lungs, as its author, Zenker, intended, when he found these organs impregnated with coal dust. Instead, it specifically denotes all forms of dust-inhaled diseases. Although the term pneumoconiosis has morbid connotations, there is a minority of diseases under its umbrella which are, however, benign. This applies to stannosis and baritosis which are produced by tin dust and barium dust. In these forms of pneumoconiosis the inhaled dust causes minimal damage to the lungs; the slight tissue changes which result are visible only on X-rays.[11] It therefore follows that only some dusts - and silica is one of them - are harmful.

Except in the case of persons who suffer from asthma, most inhaled dusts are innocuous: they produce only pigmentation or slight chronic inflammatory reactions in the lungs of human beings - and of animals. Certain forms of mineral dust which are non-toxic in the ordinary sense of the word are, however, fibrogenic. This means that if they are inhaled continuously and over long periods they are able to breach the "magnificent" defence mechanisms of the lungs and to penetrate the deeper layers of lung

tissue where they remain and cause damage.[12] All foreign substances entering the body act as irritants; and in most cases irritation is followed by the formation of fibrous tissue.[13] Nature aims at limiting the hurtfulness of foreign bodies that cannot be absorbed or removed by physiological processes;[14] it does so by surrounding them with a capsule of fibrous or scar tissue.[15] This happens to an intense degree in the lungs. Here the dust particles provoke the growth of fibrous connective tissue, called fibrosis, in excess of that which is normally present. These fibrogenic responses are not, however, identical and vary according to the kind of dust irritant.[16]

Similarly, each of the pneumoconioses is terminologically linked to the particular dust or industry or process which causes the fibrosis. Thus silicosis, coalworkers' pneumoconiosis, berylliosis and asbestosis and silicatosis are caused by silica, coal dust, beryllium, asbestos and other silicates.[17] These are all potentially morbid diseases, as is mixed-dust pneumoconiosis, which results from the inhalation of normally inert dust mixed with small amounts of silica.[18] Because exposure to fibrogenic dusts is almost exclusively occupational, it should be stressed that the pneumoconioses are man-made diseases.

Men have contracted pneumoconiosis ever since they first began to manipulate the products of their environment; and it has been suggested that

palaeolithic miners died of the oldest occupational disease - silicosis.[19] Physicians in Greek and Roman times did not distinguish between various forms of respiratory diseases. Nevertheless, they drew a general connection between lung disorders and dust inhalation.[20] Workers also did so; and there is evidence from Roman times right through till the middle ages that miners wore veils as a precaution against inhaling dust and noxious fumes. Although occupational dust had not yet been identified as the prime cause of incapacity, workmen were aware that it damaged their lungs and that they ran the risk of premature death as a consequence.[21]

After the advent of the industrial revolution the pneumoconioses proliferated. With the industrial revolution's technological advancements, mass-production techniques and sophisticated machinery, driven by powerful sources of energy, massive amounts of dust were generated in the workplace. As a result the prevalence of the pneumoconioses, the overall number of old and new cases in a single period, grew rapidly. The incidence of the disease, namely the occurrence of new cases in a single period, also accelerated. Equally important, in many workers liable to dust the onset, development and progress of the disease took place at much earlier stages in their working lives than had been the case before. Indeed, by the beginning of the 19th century large numbers of workers were handicapped or died from

dust diseases at a relatively young age. British workmen gave these industrial diseases graphic names which they perceptively associated with their trades and occupations; and "potters' rot", "grinders' rot" and "miners' decline" are but a few examples of the morbid terms they coined. Invariably these names illustrated pulmonary disorders which resulted from the impairment of lung function.[22]

The main function of the lungs is the exchange of gases which are essential for the life processes. Also, in the process of gas exchange the body forms and uses many substances which maintain and stabilise its metabolism. Hence any damage to a person's lungs vitiates their performance and undermines the healthy existence of the individual.[23] More specifically, in some forms of pneumoconiosis, and particularly in the case of silicosis, the fibrotic nodules obliterate many of the air sacs and blood capillaries, so effectively diminishing both the amount of aerating surface in the lung and the amount of blood brought into contact with the air.[24] With silicosis this damage, for reasons not yet fully explained,[25] produces characteristic symptoms: shortness of breath, exacerbated by exercise or exertion, and an irritative cough.[26] If the fibrosis becomes extensive, gas exchange becomes increasingly difficult; and when there is no longer sufficient ventilation, the right side of the heart may dilate from the strain. This condition, known as *cor pulmonale*, is always disabling

and poses a serious threat to life.[27]

It has earlier been suggested that silicosis has a long and traceable history dating back to palaeolithic miners. The disease, however, is not related solely to mining; man has been exposed to silica dust in many other occupations. The development of technology, particularly during the 18th and 19th centuries, with an accompanying expansion of dust levels in the workplace, increased the prevalence and incidence of silicosis throughout the Western world. This prompted research, which was aided by the development of the microscope. During the 1860s, when Zenker was publishing his pathological findings on pneumoconiosis, a few investigators, notably Edward Headlam Greenhow in Britain and Adolf Kuessmaul in Germany, identified both chemically and microscopically the dusts found post mortem in the lungs of workers.[28]

Once scientists had correlated specific dusts with the occupations of deceased workers, it then became customary for them to name each form of dusty lung after the responsible dust or occupation.[29] Four years after Kuessmaul had affirmed the presence of silica in the lungs, Visconti, in an article published in a French medical journal in 1870, devised the term silicosis, so distinguishing this dust disease from the other pneumoconioses. Silicosis has for its root the Latin word, *silex*, meaning a flint. The derivation aptly describes the mineral character of

the silica (or quartz) dust.[30] Silicosis becomes established in men and in animals when more or less pure silica dust, or mixed dust in which silica is found in varying proportions, is inhaled over continuous periods.[31]

The cause (or aetiology) of silicosis - the inhalation of silica dust - appears to be simple, but there are many complex variables. The onset of the disease depends on the duration of dust exposure, the percentage of silica in the dust and the intensity of the dose. These factors are also contingent on the nature, shape and size of the dust particles. Together the variable dust elements constitute the respirable fraction, which is discussed later.[32] Also, the minimum amount of dust exposure necessary to cause the disease remains unspecified.[33] Similarly, the development of the disease is complex and is even today the subject of controversy. A discussion follows of the two forms of the disease: first, simple silicosis; and second, progressive massive fibrosis. It should here be noted that the term progressive massive fibrosis has replaced the earlier and less scientific names for this form of the disease: in the 1920s it was called "infective silicosis",[34] while its later loose variant was "complicated silicosis".[35]

In simple silicosis scar tissue or fibrosis usually develops slowly. In the early stages of the disease only isolated nodules form and, if the individual is removed from dust exposure, the disease

is unlikely to progress further. But once nodules replace lung tissue - and this always occurs in silicosis - there is permanent lung damage however slight.[36]

There are three different kinds of simple silicosis. Each has a distinctive tissue reaction, which is the product of a specific kind of dust dose.[37] First, chronic silicosis, with discrete nodules of approximately five millimetres in diameter, usually results from moderate dust exposure over a period of twenty to forty years. Second, as studies of sandblasters have shown, an increased particle dose can produce the less common form of accelerated silicosis within five to fifteen years.[38] Finally, heavy concentrations of dust during a period of two to five years may cause diffuse silicosis. Therefore the number of silicotic nodules is related to the duration and intensity of the dust dose.[39] Eventually the whole of the lungs may become packed with nodules and in places they may even be confluent.[40]

Although the relationship between dust dosage and individual response is clearly important, current medical science has not, as yet, been able to define it precisely. Even today it is not clear, for instance, whether short intermittent periods of exposure to intense dust concentrations are as dangerous as, or more dangerous than, exposure to lesser dust concentrations over a prolonged and continuous period.[41] There is, in addition, an

anomalous aspect relating to the onset of the disease. After inhaling significant amo ' of dust during a relatively short time, a worker may have a latent period of several years before signs and symptoms of the disease develop.[42]

These features of the disease are relevant in assessing the views of South African and British doctors on the incidence of silicosis amongst black migrant mineworkers during the first three decades of the 20th century. Doctors concedeu that Africans were exposed to intense dust concentrations of short duration. Yet medical opinion almost unanimously held that, in contrast to white miners, there was a markedly low incidence of a disabling form of silicosis amor gst black underground mineworkers. Doctors attributed this to the short contracts of African mineworkers, which were also intermittent. They believed that after each contract an African mineworker had a chance to recover his health: he had a "holiday" at his "kraal" before resuming work on the mines.[43] This study cannot make judgements on the validity of the medical knowledge of that time. Using other criteria, however, including the medical profession's own dependence on the continued prosperity of the gold mining industry, this study questions the accuracy of the contemporary physicians' estimates for the low incidence of a handicapping form of silicosis amongst African mineworkers.[44]

The onset of the disease is usually slow and insidious and its advance is progressive. While the individual may not necessarily suffer premature death, the replacement of "soft and spongy" lung tissue by "hard unyielding" nodules is often crippling.[45] One would expect the degree of disability to be linked with the amount of fibrosis, which is currently radiologically graded in South Africa as slight, moderate or marked.[46] But this is not the invariable pattern: often the severity of the symptoms has no direct relation to the number of nodules.[47] Generally speaking, chronic fibrosis may cause in some workers little or no incapacity. But chronic silicosis may be so pronounced in other workers that they are unable to perform jobs that require any effort or exertion.[48] Also, the disease is progressive. Removal from dust exposure may significantly retard the progression of the disease. Even so, this medical control does not necessarily halt it.[49]

Unlike their British counterparts who were investigating the disease in Cornwall at the same time, Transvaal doctors in 1902 to 1903 seldom found that the silicosis they observed was accompanied by tuberculosis.[50] But by 1906 physicians on the Witwatersrand were acknowledging that the manifestation of silicosis appeared to have altered. By this date, in contrast to the "dry" form of silicosis which had predominated earlier,[51] they found many cases that were "complicated" by tuberculosis.[52]

This did not necessarily mean that their earlier finding of a fatal but "uncomplicated", or "uninfective", silicosis had been incorrect. Simple but accelerated silicosis may be as potentially lethal as progressive massive fibrosis.[53]

Significantly, from this time the majority of British and Transvaal health officers incorrectly viewed simple silicosis as a relatively minor illness.[54] Doctors did not consider most cases of simple silicosis, particularly those in the early stages, to be severely incapacitating or fatal.[55] Doctors held that the significance and potential danger of simple silicosis lay in its capacity to pave the way for the onset of pulmonary tuberculosis. They were correct in their view that "infective" (or "complicated" silicosis) - progressive massive fibrosis - was a major threat to a worker's health and life.

In 1927 Dr Wilfred Watkins-Pitchford,[56] the retired Director of the South African Institute for Medical Research and the former Chairman of the Miners' Phthisis Medical Bureau,[57] summed up the prevailing medical views as follows:

> In at least the very great majority of cases, the final and determining cause of the appearance of the disease in the Rand gold miner, even in its "simple" form, has been a tuberculous infection, rather than a surpassing of the capacity of the lung harmlessly to incarcerate quartz particles...
>
> The agency which converts the liability into a manifestation of the disease, in any

particular case, is usually a tuberculous infection.[58]

Progressive massive fibrosis seems to be an addition to, rather than a development of, simple silicosis, and a dust-laden lung is a necessary precursor for its onset.[59] At what stage of simple silicosis this may occur is uncertain, but some doctors believe that it is more likely if fibrosis is accelerated or diffuse rather than chronic.[60] Against this, other physicians believe that tuberculosis can be superadded to simple silicosis at any stage in a worker's career: from even before dust lesions appear on X-rays to a late juncture of chronicity.[61]

The latter rationale would explain the relatively lengthy working lives of numerous gold miners in Australia, for instance. During the early part of the 20th century Australian miners worked under less severe conditions of dust exposure than those which generally prevailed on the Witwatersrand mines. They plied their trade for approximately thirty to thirty-five years, that is until they reached an average age of just under or just above fifty years. Therefore many of them developed progressive massive fibrosis, in which tuberculosis was the predominant element, at a relatively late stage in their careers.[62]

When simple silicosis changes to progressive massive fibrosis, the nodules proliferate rapidly and enlarge: they are usually more than two and a half centimetres in diameter, but sometimes reach the size

of golf or cricket balls.[63] As the condition progresses, the nodules lose their regular pattern, structure and concentric shape. They form dense "stoney" mattings containing obliterated blood vessels and air-vesicles: normal remnants of lung structures are barely found within them. In advanced cases these fibrous masses may even distort the shape of the lungs.[64] Clearly progressive massive fibrosis is always disabling and is often rapidly fatal.[65] Also, like simple silicosis, it is progressive: withdrawal from dust exposure does not necessarily halt its advance.[66]

Apart from silicosis, progressive massive fibrosis also occurs in other forms of pneumoconiosis: it is, for example, found in the lungs of haematite workers and colliers.[67] Consequently progressive massive fibrosis has been the subject of much research, but its aetiology has not yet been precisely elucidated.[68] Some doctors attribute its manifestation solely to infection, specifically by tuberculosis.[69] But others suggest that it may be caused by any of the following three factors either singly or in combination: tuberculosis; massive retention of silica dust; and bronchial obstruction with partial lung collapse.[70]

The evidence indicates a strong but not exclusive association between the tubercle bacillus and progressive massive fibrosis.[71] As a result, from early times medical controls in dusty occupations

throughout the Western world have insisted on the removal of a worker with tuberculosis from the source of dust. This is to prevent the person from infecting his fellow workers. The medical precaution obviates the possibility of other workers already prone to, or affected by, simple silicosis from developing progressive massive fibrosis.[72] Apart from the risk of tuberculous exposure on the industrial floor, workers with simple silicosis also face considerable danger if they are exposed to tuberculous infection in their living quarters. In conditions of dust density at the workplace they were particularly liable to contracting tuberculosis and to developing, as a consequence, progressive massive fibrosis.[73]

Clearly the onset of tuberculosis in a case of simple silicosis is highly dangerous. It is important, however, to dispel the popular but outmoded misconception that tuberculosis is the inevitable and immediate cause of death in progressive massive fibrosis. This was a mistaken view held by doctors during the first two decades of the 20th century: and is one to which certain historians currently subscribe.[74] Of course tuberculosis may in some cases be the primary cause of death: more particularly this depends on the virulence of the bacillus in relation to the immunity and resistance of the patients. But in most instances, particularly those which involve urbanised workers, who have an acquired childhood immunity to tuberculosis, the disease has a fatal

outcome for another reason: it leads to the onset of progressive massive fibrosis. It is this condition which destroys the arterial pulmonary system. Consequently heart failure, or more specifically, *cor pulmonale*, causes death.[75]

As silicosis is generally a chronic disease, the principal symptoms manifest themselves relatively late in the life of a worker; X-rays of the chest are not usually positive until an individual has been exposed to twenty years of respirable silica. This would of course be in conditions of moderate dust density. In such cases life expectancy is estimated at forty years after first exposure. Heavier exposure in confined spaces accompanied by the absence of dust preventive measures - conditions under which Witwatersrand miners worked both before and after the Anglo-Boer War - leads to a more rapid and earlier onset of the disease. In cases of simple silicosis the earliest symptoms are cough and expectoration. As the disease establishes itself more firmly, these symptoms are accompanied by shortness of breath on effort.[76] If tuberculosis supervenes, cough and expectoration increase. The infection may also lead to characteristic chest pain, the coughing of blood and weight loss. Tuberculosis, as has been noted, seems to be a common occurrence when fibrosis is accelerated or diffuse.

In accelerated silicosis the major features are identical to those which are present in chronic

silicosis. But the chest X-rays become positive far earlier - within four to eight years of first silica exposure. In such cases deterioration is rapid and may be fatal within ten years of first exposure.[77] These characteristics of accelerated silicosis, as sometimes happens with modern-day sandblasters and foundry workers, are strikingly similar to the descriptions of silicosis in rock drillers on the Transvaal gold fields eighty years ago.

During the period 1892 to 1912 most rock drillers on the Witwatersrand, as well as general miners who were also exposed to intense dust doses, undoubtedly contracted accelerated silicosis. Present-day descriptions of the pathology and presentation of this form of silicosis are consonant with early Transvaal medical investigations. These contemporary studies confirm that large numbers of miners were severely disabled or died within five to fifteen years of first dust exposure. From 1902 until 1912 doctors estimated that the average life expectancy of Rand rock drillers was 35,5 years; their average working lives were a mere 6,49 years.[78]

The symptoms described by pioneering doctors in the field of occupational medicine correspond with those seen today. In 1905 Sir Thomas Oliver, a physician from Newcastle-upon-Tyne and a world authority on industrial medicine, maintained that shortness of breath was the only sign of "noteworthy fact" in diagnosing silicosis in its early stages.

For him the symptom indicated that a Witwatersrand miner was becoming "lunged".[79] Also, in 1912 two full-time Witwatersrand mine doctors, Louis Godfrey Irvine and Andrew Hutton Watt, graphically described the progression of the disease:

> As the disease progresses to the stage of *advanced fibrosis* the cardinal symptom of shortness of breath becomes more urgent and distressing; the irritative cough more frequent; expectoration may become more copious, but is still in most cases slight. The patient becomes unable to work, he loses flesh, his narrow shrunken rigid chest may scarcely expand at all even on forced inspiration, the shoulders are hunched, the chest appears to be practically fixed in the position of extreme expiration. The lips are bluish, the expression anxious, the pulse rate is accelerated, and the right side of the heart dilates under the strain.[80]

At the turn of the 20th century physicians diagnosed the disease almost exclusively by clinical methods.[81] Doctors relied heavily on symptoms, physical and stethoscopic examinations, and chest measurements for inhalation and exhalation - crude and inaccurate criteria by today's standards.[82] Sometimes, if tuberculosis was suspected, sputum was tested for evidence of the tubercle bacillus.[83] Radiography was seldom used as a diagnostic tool.[84] This was so despite the production of high quality plates exhibiting shadow and fine detail shortly after William Conrad Röntgen's discovery of X-rays in 1895.[85] Apart from the high costs of the equipment and plates, conservative members of the medical profession were sceptical about the use of X-rays, which they viewed as merely an expensive adjunct for confirming

what they considered to be accurate clinical diagnoses.[86] In 1912, when sliding scales for compensation based on the degree of disability were introduced for gold miners in South Africa, radiography was used only in doubtful clinical cases.[87]

As increasing numbers of doctors equipped themselves to handle the apparatus and developed specialist skills in interpreting the images, the medical profession confirmed the importance of radiography.[88] The efficacy of X-rays in diagnosing tuberculosis and silicosis before these diseases could be clinically determined made it especially valuable; and in 1914 the Johannesburg medical fraternity agreed that opinions based on simple clinical examinations were "an example of the dogmatism born of a complete ignorance of the subject" of radiography.[89]

In 1916 at the newly founded Miners' Phthisis Medical Bureau the use of X-rays in all first-time and periodic medical examinations of white miners became standard practice.[90] Health officers today are convinced that a definitive diagnosis of silicosis requires X-ray appearance together with an occupational history evincing prior dust exposure.[91] Also, present-day health officers, when they stress the necessity for radiography, often quote the views of pioneering South African doctors.[92] In 1916 the medical report of the Miners' Phthisis Prevention Committee firmly laid the foundation for modern

standard practice:

> We are fully of opinion that the radiographic appearances in cases of silicosis afford the most reliable *single* piece of evidence, in establishing the existence and the actual stage of the disease in any particular case, in determining the presence or absence of tuberculosis, and in differentiating cases of early silicosis from cases of commencing tuberculosis.
>
> Radiographic examination is of particular value in the case of natives, in whom ordinary clinical examination is often difficult and may be misleading.[93]

Despite this statement, white miners were the sole recipients of routine radiography. Only a small number of black mineworkers was examined by X-rays. In isolated instances and for purposes of compensation, the Miners' Phthisis Medical Bureau X-rayed Africans, in whom cases of simple silicosis and progressive massive fibrosis were suspected. This occurred after Africans had been clinically checked at preliminary and periodic examinations.[94] Later, in 1926, the Chamber of Mines instituted annual radiographic examinations, but only for the "long-service natives".[95] Thus unlike the case of white miners, thirty years elapsed before routine radiography was introduced for African mineworkers.[96] This racially discriminatory practice blemishes an otherwise important and frequently quoted precedent.

Silicosis, as earlier noted, is an irreversible condition for which there is no cure. As was recognised in the early part of the 19th century, the only way of halting the advance of the disease was for

workers to "renounce their work".[97] Even after such drastic medical intervention physicians conceded that they had little else to offer. At best they could try to alleviate the patient's condition with medication for the symptoms and hygienic care.[98] Modern medicine can do little more. It stresses early diagnosis of the disease, followed by the removal of the worker from the dusty atmosphere and resettlement elsewhere, before disablement occurs.[99]

In 1912, when compensation was first introduced in South Africa, it was compulsory for miners, who were positively diagnosed as having silicosis, to stop working underground after they had been compensated. But this is no longer the law today.[100] Apart from the instant and permanent withdrawal of mineworkers in whom tuberculosis is diagnosed, since 1917 miners with simple silicosis have been allowed to resume underground work if, after re-examination, they are found to be fit to do so. They are allowed to work underground until the disease has advanced to a legally prescribed stage. They are then withdrawn from service and are given additional compensation, lest the progression of the disease causes disability.[101]

There are two reasons for the changes. First, because the disease is progressive, removal from dust exposure does not guarantee to stop its advance.[102] Thus it seems pointless to deprive a worker of his job when the pathological changes to the lung, which are

constantly monitored, will inevitably progress to another stage, but one which as yet causes no disability. The second reason has significant implications and relates to the vicious circle in which silicotic victims are trapped. On the one hand, in continuing to work under controlled dust conditions silicotics run the risk of their disease advancing. On the other, their removal from dusty work may expose them to an equal, if not more dangerous, health risk. This is because silicotic patients, who seldom find alternative work at equivalent wages, invariably experience reduced earnings, with an accompanying fall in nutritional and housing standards: under these reduced living conditions they face the possibility of tuberculous infection and the onset of progressive massive fibrosis. Therefore, as research has shown, compulsory withdrawal of workers from their jobs in the early stages of silicosis may ironically be more harmful to their health than continuing to work.[103]

During the first two decades of the 20th century early-stage silicotic miners on the Witwatersrand would have preferred the option of continuing to work rather than face the bleak prospect of "starvation" for themselves and their families: they could not subsist on their compensation awards of £8 per month, which were given for one or two years depending on their stage of disability.[104] Also, as there was virtually no secondary industry in South Africa, most silicotic miners could not find alternative

employment. Former miners, devoid of other skills and lacking the capital necessary for farming or business enterprises, became all too often dependent on the charity of their friends.[105] Immigrant miners, who lacked the means to return home, often remained on the Reef. There, together with South African born silicotics, they swelled the ranks of the landless unemployed Afrikaners.[106] Others resorted to petty crime.[107] Such activities and the wretched destitution of these workers made headline news in the press - even in those newspapers which the mineowners controlled.

As has been noted, the reduced living standards of silicotics makes them vulnerable to pulmonary tuberculosis. During the early decades of the 20th century, however, the disease was an even more ominous threat than it is today: at that time tuberculosis was highly prevalent in the white urban centres of South Africa.[108] The infection so dangerously affected miners that in 1912 the Miners' Phthisis Board estimated the life expectancy of a silicotic miner who also had tuberculosis - he had progressive massive fibrosis - at no more than six months on average.[109]

Complicated employment issues still confront silicotic workers today.[110] But the choice of continuing to work under "controlled conditions" is, at best, a limited answer to a complex mesh of socio-economic problems. In such a solution, based partly on both compassionate and pragmatic

rationales,[111] there is a touch, too, of expediency.

Current drug treatment is directed entirely at infectious complications. Certain modern compounds may alleviate symptoms and retard infection so prolonging life. But medicinal therapy remains largely futile. When tuberculosis supervenes on silicosis, anti-tuberculosis drugs are used. Apart from sterilizing the sputums of infected patients, little else can be done. In contrast to the success of drugs in controlling the simple form of pulmonary tuberculosis, they exert minimal curbs on the infection in progressive massive fibrosis.[112]

The association of tuberculosis with silicosis is a complex problem which has puzzled the medical profession for many years. Repeated studies in epidemiology, pathology and experimental medicine have demonstrated their frequent co-existence but the reason is not yet understood.[113] Long before Zenker coined the term pneumonokoniosis for the lung condition observed in miners and stoneworkers, both workmen and physicians had been well aware that a number of occupations gave rise to lung disorders which led to premature death. Georg Bauer, more well known under his *nom de plume*, Georgius Agricola, in his famous treatise on mining - *De Re Metallica* - vividly described the terminal features of the miners' condition which so closely resembled tuberculosis.[114]

He recorded:

> The constant dust enters the blood and lungs producing that difficulty of breathing which the Greeks call asthma. When the dust is corrosive it ulcerates the lungs and produces consumption.[115]

Tuberculosis has been recognised since ancient times. It has always been identified with the market place and "civilized" urban ways of life in which overcrowded living conditions and low nutritional standards provide ideal conditions for its proliferation. So, too, it flourished in the new industrial complexes of 18th and 19th century Europe.[116] Physicians have named tuberculosis the "poor man's disease".[117] The medical interpretation is simply that tuberculosis makes greatest headway amongst those people whose standard of living is low and whose resistance to the germ is correspondingly feeble. The disease is infective and is caused entirely by the activity of the bacillus of tuberculosis, or the tubercle bacillus, today known as *Mycobacterium tuberculosis*. Unless the specific germ is present, the disease cannot occur. While it may affect any part of the body - the lungs, lymph glands, spleen and bones - in this summary only the lungs are discussed, as other forms of tuberculosis are not associated with dust diseases.[118]

The germs are spread largely by spitting or sneezing. Once released into the atmosphere they are usually inhaled and are sometimes ingested. Although they thrive only in animal or in human tissues, they

are resistant and may remain viable for many months in dust or on articles in daily use. The contagiousness of tuberculosis, well known since its earliest manifestation, has always resulted in health officers insisting on the observance of hygienic habits to curb its spread. During the late 19th century and the early decades of the 20th century the study of bacteriology predominated. The focus on this new medical science coincided with an era in which tuberculosis was alarmingly prevalent, with corresponding high death rates, particularly amongst the industrial classes.

During the 19th century the emphasis on bacteriology and the prevalence of tuberculosis help explain the medical profession's commitment to improve sanitation in the workplace before embarking on other occupational health precautions.[119] The anxiety of health officers with regard to spitting is understandable; and today one of the aims of drug therapy is the care and control of sputums of infected persons.[120] A concern with spitting - indeed it verged on the obsessional - underpinned the contemporary medical profession's determination to curb tuberculous contagion.[121]

On the Reef, before 1912, it was commonly, if not optimistically, believed that the tubercle bacillus could not survive the acidity in the Witwatersrand gold mines.[122] In its interim report of 1913 the Miners' Phthisis Prevention Committee, however,

dispelled the misconception; the committee reported that 15 per cent of sputum samples taken underground contained living bacilli.[123] This finding evoked anxiety not only amongst doctors but also amongst the informed public, including miners.[124] It proved that miners were exposed to a double risk of infection: from the workplace itself; and from their fellow workers, one-sixth of whom were estimated to have active tuberculosis.[125]

African and Afrikaner adolescents and young males were particularly susceptible to tuberculous infection. Their rural backgrounds did not equip them with an acquired childhood immunity. Also, in the towns they had insufficient resistance to withstand the environmental stresses of poor living conditions and physically debilitating mine work. Hence they experienced a high morbidity and mortality from simple tuberculosis when they came to work on the gold mines.[126] In these "unseasoned" populatior , simple tuberculosis often occurred in a rapidly progressive form, which frequently had a fatal outcome.[127] Therefore rural Afrikaner and African mineworkers often died from simple tuberculosis: they died before simple silicosis had sufficient time to become established.

Apart from its contagiousness, laboratory experiments have shown that there are different strains of tubercule bacilli with varying degrees of virulence, which produce the disease. Whatever the

virulence of the strain the early changes are the same and the initial lung lesions produced are similar. These comprise clumps of tuberculous cells called the tubercle. But the course of the disease alters or is modified by a number of circumstances. For example, when a moderate number of mildly virulent strains of bacilli become implanted in the lungs of an individual of average resistance, the disease will usually follow an orderly progression which ends in complete healing. This is because the body is sufficiently healthy to combat the germs. Such a person does not normally develop active tuberculosis and consequently does not pass on the infection. In contrast, numerous bacilli of high virulence in a person whose resistance is low may cause changes which fail to produce a healing reaction and which result in permanent lung damage. Such lowered resistance may be caused by a number of factors, including poor living conditions, stressful work and, importantly, the presence of silicosis.

When healing occurs, the tubercles may be absorbed and disappear, or they may become encapsulated by tissue and appear as scars. The scars frequently enclose a latent form of tuberculosis, so-called because living bacilli are walled up in the scar tissue. Although the presence of these bacilli will cause a tuberculin test to be positive, they will usually remain quiescent if the individual leads a healthy life. But should his health and life-style

alter detrimentally, the bacilli may be reactivated and the tuberculosis will flare up and spread. Changed circumstances may include: lowered socio-economic standards; a heightened stress level which may be induced by environmental strains or accelerated physical labour demands; a serious infection, such as pneumonia; and finally, and importantly, lung damage, especially pneumoconiosis.

Significantly, when reactivated tuberculosis occurs in a silicotic lung, its progression usually differs from a primary tuberculous infection; it invariably runs a serious course. Also, the dormant bacilli tend to die in the course of time. The chances of their reactivation are therefore considerably higher in younger rather than older men.[128] This fact partly explains the markedly high number of Witwatersrand miners in the prime of life who were afflicted by progressive massive fibrosis in which tuberculosis was an important element. In such cases open or communicable tuberculous infection may have co-existed with the silicosis.[129] Alternatively, extensive fibrosis could have caused the reactivation of latent tubercles.

Before Robert Koch in 1882 identified the bacillus responsible for tuberculosis, the disease had a variety of names. As the striking symptom in tuberculosis is the wasting away of the body, the Greeks called it *phthisis*; and consumption and decline were subsequent synonyms. Phthisis was also a major

symptom of industrial dust diseases. It was, therefore, logical to link the term to the occupation, hence miners' phthisis, stonemasons' phthisis or potters' phthisis.[130]

During the latter part of the 19th century the science of pathology grew through the use of the microscope, which aided Koch's identification of the tubercle - the clumps of tuberculous cells - and his subsequent discovery of the tubercle bacillus.[131] But the concentric fibrotic nodules of simple silicosis, found in post-mortem examinations of the lungs of workers who had plied dusty trades, closely resembled the tubercle. The similarity was so marked that Koch designated it the pseudo-tubercle, a term also deployed in the 1920s by Anthony Mavrogordato of the South African Institute for Medical Research.[132] Although pathologists gradually drew distinctions between "dust phthisis" and "tubercular phthisis" on the basis of whether or not there was a history of occupational dust exposure,[133] most doctors viewed tuberculosis as an integral part of silicosis and continued to call it miners' phthisis.[134]

This was the term which Transvaal doctors adopted at the beginning of the 20th century.[135] Few disputed that the debilitating condition which developed was established by an initial silicosis;[136] but silicosis was reserved for delineating only the original damage and the progressive tissue responses. In other words the term silicosis was restricted to defining the

pathology of the disease;[137] miners' phthisis was the name given to its complex clinical manifestation.

At the beginning of the 20th century the South African medical profession agreed that the name miners' phthisis lacked precision. But they also believed that it aptly summed up the complicated character of the disease.[138] Although they later conceded that it was an unscientific title, they retained it in the belief that tuberculous infection, or phthisis, as the infection was often colloquially called, was the predominant factor in most cases of pronounced disablement and premature death in miners.[139] By adhering to their choice they laid the basis for its legal recognition in 1911.[140] In 1914 Sebastian Valentyn van Niekerk, the Medical Inspector of Mines, gave the reasons for the South African doctors' preference:

> We know now that these bacteria are abundantly met with underground, and the disease they produce and Miners' Phthisis is so interdependent, in my opinion, that I have been wont to say that, by eliminating the causes of Pulmonary Tuberculosis,...we may consider the Miners' Phthisis battle half won, and *vice versa*.[141]

Eventually the South African doctors were swayed by their British colleagues, who preferred the name silicosis of the lungs.[142] During the 1920s silicosis became the preferred term in South Africa. But until 1950, both in South Africa and internationally, miners' phthisis was retained and used interchangeably with silicosis because of legal precedents. In 1927

Wilfred Watkins-Pitchford explained this anomaly:

> The term miners' phthisis, if it has any precise meaning, refers only to silicosis when associated with an active tuberculosis; its retention in the title of the South African Act [of 1925], and in the title of the [Miners' Phthisis Medical] Bureau which administers its medical provisions, is indeed merely a historical relic.[143]

Finally, at the Third International Conference of Experts on Pneumoconiosis held in Sydney in 1950, the delegates agreed that in future the terminology of pneumoconiosis would take the form of naming the dust or occupation to which the worker was exposed.[144] At this late date in the 20th century the international medical profession formally abandoned the use of the term miners' phthisis.

The changes in international nomenclature for silicosis indicate that an understanding of contemporary disease terminology is vital if one is to interpret the medical reports and the health statistics of the past. This is particularly necessary in the case of South Africa where until 1920 medical terminology was often peculiar. In 1912 the medical commissioners on miners' phthisis reported that "the classification of the causes of death is not uniform with that employed in other countries".[145]

As has been noted, during the first two decades of the 20th century silicosis and miners' phthisis had, in the Transvaal, specific clinical and pathological meanings. Likewise, the word phthisis, too, presents difficulties. Only a few medical

practitioners used pulmonary tuberculosis to denote this specific illness in the scientific way which Koch had made possible by his identification of the tubercle bacillus.[146] Instead, the majority of doctors preferred the old-fashioned and colloquial term for this infection, namely phthisis.[147] In contrast to Britain, where phthisis unambiguously came to mean simple pulmonary tuberculosis, its use in South Africa was rather more vague, for here it carried both specific and general connotations.[148] Of course phthisis was frequently used to denote simple pulmonary tuberculosis. But in other circumstances it was the rubric for many lung diseases which were thought to possess a tuberculous element.[149] Consequently in South Africa mortality statistics for phthisis often indicated non-tuberculous diseases, including bronchitis and emphysema, and simultaneously embraced both simple pulmonary tuberculosis and "infective" silicosis - or miners' phthisis - as progressive massive fibrosis was then clinically termed.[150] Clearly the generic use of the word phthisis poses problems for the investigator of the Witwatersrand's incidence, prevalence and mortality data for both simple pulmonary tuberculosis and progressive massive fibrosis. Also, even if occupational indicators showing dust exposure accompany mortality statistics under the umbrella of phthisis, the researcher cannot definitively conclude that this rubric denoted progressive massive fibrosis. Possibilities may be inferred; but at best

these can be only tentative.

Apart from its lack of precision, the meaning of the term phthisis gradually changed with time. In Britain, as has been noted, and "elsewhere" it was merely modified and refined to denote unambiguous simple pulmonary tuberculosis. But in South Africa its meaning changed rather more dramatically.[151] As silicosis became publicised in the Transvaal, laymen abbreviated miners' phthisis to phthisis and doctors followed suit. This practice caused confusion "in the public mind" as there was an interim period during which phthisis was a synonym for both silicosis and pulmonary tuberculosis.[152] In 1910 an inspector of mines clearly demonstrated the anomaly:

> The disease to which miners are particularly subject on these fields is that of consumption, phthisis or tuberculosis, - call it by any of these names. This disease is aggravated and, perhaps in the first instance, brought about by silicosis, to which the erroneous name of phthisis is generally applied by the uninitiated.[153]

The imprecision persisted, however, for nearly a decade. Finally, legislation in the early 1920s compelled doctors to register diseases in a more uniform way and in internationally accepted medical language.[154] Clearly an accurate historical interpretation of contemporary medical data in South Africa and in other parts of the world requires a close scrutiny of the data's terminological contexts.

Several historians of southern Africa have reached the mistaken conclusion that phthisis always

meant so-called miners' phthisis.[155] On the basis of contemporary statistics for phthisis, derived from reports of medical officers of health and the census, some research workers have further concluded that official figures understated the prevalence of silicosis on the gold mines.[156] Although the hypothesis is plausible, particularly in the case of African mineworkers, other kinds of substantive evidence rather than such imprecise data are needed to warrant this finding.

It should also be stressed that official death rates and the census are far from being good indices of the actual prevalence of occupational disease. The diagnosis of death frequently referred to the immediate cause or terminal condition, including heart disease and pneumonia; more often than not phthisis was blamed. The cause of death seldom cited the primary disease, the more especially if this was a chronic condition of long standing, as in the case of silicosis.[157] Unless an autopsy had been performed, a procedure against which white workmen had "a natural prejudice",[158] even hospitals seldom recorded miners' phthisis as a cause of death.[159]

As we have seen, the general consensus amongst South African and British doctors was that an initial silicosis exposed the lungs of workers to tuberculous infection, the agent which was held to be the cause of death. At the turn of the 20th century South African physicians called this "mixed" or "complicated"

clinical manifestation of progressive massive fibrosis "infective" silicosis.[160] Twenty years later, after extensive investigations, they coined a more scientific clinical term - silico-tuberculosis.

In clinical terms only, this form of progressive massive fibrosis was predominantly found amongst white miners.[161] Doctors identified the tuberculous infection, in its open form, through the disease's typical symptoms of weight loss, night sweats and sputum streaked with blood; positive sputum tests, X-rays and post-mortem findings could invariably verify dubious diagnoses.[162]

By the early 1920s, however, an opposed school of thought emerged in South Africa. It challenged the silico-tuberculosis theory by arguing that silicosis was tuberculous in origin. It held that the presence of the tubercle, whether active or quiescent, was the necessary precursor for the establishment of silicosis.[163] Tuberculo-silicosis was the name given to this clinical condition, which pathologists found to be more prevalent amongst African mineworkers than whites.[164]

To settle the controversy over which disease came first - silicosis or tuberculosis - in 1926 Frank William Simson of the South African Institute for Medical Research undertook research with animal experiments. Together with his colleague, Archibald Sutherland Strachan, he showed that that there was a

type of silicosis which from the beginning could be produced with an accompanying element of tuberculosis, but which created no detectable signs of the infectious disease in the guinea pigs while they were alive. Equally significantly, the two pathologists also demonstrated that a form of silicosis could be produced which from first to last had no element whatsoever of tuberculosis: dust alone could cause both simple silicosis and progressive massive fibrosis.[165] Although there are counter-arguments which dispute the validity of the conclusions of the latter experiment, many authorities currently accept this oft-quoted finding; they support the view that a tuberculous element need not be present in either simple silicosis or progressive massive fibrosis, both of which may be potentially fatal.[166] Although tuberculosis is frequently associated with progressive massive fibrosis, in view of the current debate it seems difficult to state incontrovertibly that silicosis predisposes to tuberculosis, and that progressive massive fibrosis with tuberculosis is necessarily silico-tuberculosis.[167]

The controversy about the association of tuberculosis with silicosis is not merely of historic or academic medical interest. Whether silicosis is conducive to tuberculosis, and which comes first, silicosis or tuberculosis, are questions which have everywhere influenced administrative procedures for awarding compensation in cases of silicosis.

Governments and insurance companies have based their decisions on principles similar to those set out in Britain in 1906. In that year a British departmental committee was appointed to recommend additional industrial diseases for inclusion under the Workmen's Compensation Act. In reaching its decisions the overriding proposition of the committee was that the disease should be "so specific to the particular employment that its causation by the employment can be established in individual cases". An industrial disease was then seen "as a disabling disease which arises out of, and in the course of, employment in an industry".[168] Simple silicosis meets these requirements: associated tuberculosis, however, caused problems as it is not a disease *specific* to any industry.

Guided by the medical belief that an element of tuberculosis was always present in "complicated" silicosis, the South African state solved the problem in 1912 by awarding compensation for progressive massive fibrosis, which was then legally termed "miners' phthisis in a marked degree".[169] The difficulty of diagnosing typical silicosis, either clinically or by X-rays, when it was concealed or masked by tuberculosis, prompted the award later, in 1916, of compensation for simple tuberculosis. But an important condition of the award in the case of gold miners was that evidence should be lodged of a two-year period of exposure to silica.[170] Compensation

for simple tuberculosis, applicable solely in South Africa to the gold mining industry, was virtually unique to South Africa.[171] The clause still applies in South Africa today, although it no longer seems to have a sound medical or scientific basis: it is retained only because of legal precedent.[172] Some present-day health officers argue that it should be retained, however, as numerous African mineworkers contract tuberculosis every year, a phenomenon which many epidemiologists regard as being occupationally related.[173]

There is no uniform international approach to compensation for tuberculosis in conjunction with silicosis. Also, the controversial association of the two diseases with each other has led to the enactment of anomalous and sometimes illogical measures. Before World War 11, for instance, in Ontario legislation went through two phases. At first compensation was awarded to "tuberculous-complicated" silicosis, but only if open or communicable infection could be demonstrated. Later, under recession, the laws were amended in rather a fickle way. Those in whom disabling but simple silicosis was diagnosed, were better compensated than those who had silicosis "complicated" by tuberculosis. This was the case despite progressive massive fibrosis being as potentially serious, if not more so, than simple silicosis. In contrast, since 1918 British legislation has been consistent and less capricious.

Whenever silicosis is "complicated" by tuberculosis, the condition is always compensated. British health officers view the disease as being totally disabling, regardless of the stage of fibrosis and of the disease h comes first.[174]

A close association also exists between the progressive massive fibrosis of coalworkers and tuberculosis.[175] This finding stems from epidemiological studies and research, conducted after World War 11, particularly in Britain and West Germany, on the health of colliers.[176] Unlike the early 1930s and earlier, when coalworkers were thought to suffer minimally from dust disorders,[177] after 1945 reassessments showed clearly that coal dust posed a marked health threat to workers in the industry. In addressing problems stemming from coalworkers' pneumoconiosis, health officers involved in recent projects often find solutions which derive from earlier experiences with silicosis. Apart from the medical data they supply, silicosis studies also provide a wide variety of valuable socio-economic insights, which apply to coalworkers' pneumoconiosis.

This was not the case at the turn of the 20th century. Remedial measures applied to hard rock mines often stemmed from their earlier adoption in the coal mining industry. It is against this background that we must examine 19th century attitudes to dust diseases in mining. We must turn to Britain if we are to understand the health conditions on the

Witwatersrand gold mines, after the metal had been discovered in 1886. Britain was the birthplace of most of the Witwatersrand gold miners; and the metal mines of Cornwall provided much of the expertise for the industry's initial technology.[178] Also, both before and after the Anglo-Boer War, most of the physicians on the Witwatersrand were of British stock. They brought with them their training and knowledge, as well as British attitudes to medicine in general, and to occupational health in particular. As important, most of these doctors belonged to the British Medical Association. Their receipt of the society's journal enabled them to keep abreast with current medical trends in Britain, Europe and elsewhere.[179]

In Britain, by the mid-19th century, appalling working conditions on the mines, with an accompanying high mortality from lung diseases and accidents, evoked strong social pressures for reform. The coal mines, in particular, were the subject of beneficial "hygienic" regulation; and by the beginning of the 20th century the occupation of coal mining was declared "now a healthy and comparatively safe one".[180]

By the end of the 19th century, too, the medical profession's curiosity concerning dust diseases had for several reasons dwindled almost to vanishing point. According to Sir Thomas Oliver, the renowned occupational disease specialist, one of the reasons was the growth and prestige of bacteriology with its

focus on the germ causes of disease. In 1903 he declared:

> Speaking generally, I may safely say that there has been comparatively little written upon pneumoconiosis since the discovery of the tubercle bacillus by Koch [in 1882]. The bulk of prevailing medical and pathological opinion upon miners' phthisis was formed anterior to Koch.[181]

Another significant reason for the loss of medical interest in dust diseases was the "disappearance" and "rarity" of colliers' phthisis. Dr John Tatham, the statistical expert in the Registrar's Office, attested to this phenomenon in 1902 when he blithely wrote: "As a class colliers compare favourably with men in most other occupations on the score of health."[182]

Tatham's testimony also strongly suggests that one of the reasons for the medical profession's earlier focus on dust diseases, particularly coalworkers' pneumoconiosis, or "black lung" and anthracosis, as it was then termed, stemmed from the significant contribution of the coal mining industry to the economic strength of Britain. In 1851 the number of coal miners was 216 000; and within forty years it had more than doubled to 517 000.[183] By 1905, in the Newcastle district alone, 25 000 miners were responsible for the production of nine million tons of coal.[184] This constituted 22 per cent out of the national annual output of approximately 200 million tons.[185] Coal mining was of national importance for another reason as well. By 1911 it employed, in one way or another, nearly one-tenth of the adult male

population.[186] The industry's magnitude suggests that, had a high mortality, particularly from pneumoconiosis, persisted amongst its many employees, doctors would have continued with research on dust diseases: self-interest, curiosity and social pressures would undoubtedly have had this result.

During the same period, and in contrast to the coal mines, the tin mining industry was relatively unimportant in the national economy of Britain.[187] This could explain the indifference, and even negative attitudes, of the state and of the medical profession to the welfare of tin miners. In contrast to the coal mines, lung disease persisted on the tin mines after 1850. Also, after 1880 it caused a considerably higher mortality than before amongst relatively young men. Apathy towards the fate of tin miners was, indeed, marked in Britain.

During the period when research on dust diseases was most intense, in 1861 and 1862 Edward Headlam Greenhow, the first English medical factory inspector, presented two reports, which demonstrated the high prevalence of respiratory illness amongst metal miners. Greenhow's disturbing findings probably influenced the appointment in 1862 of a commission under Lord Kinnaird to investigate the health conditions on metal mines. A research officer of considerable standing, Greenhow was convinced - although he did not yet have conclusive proof - of the damaging properties of occupationally produced dust;

and his reports reflected this belief.[188] His premise was reinforced by the statistics of the first superintendent of government statistics, Dr William Farr, whose figures for lead, tin and copper miners reflected an excessive mortality from lung diseases.[189]

But in 1864 the Kinnaird Commission's report ignored both Greenhow's evidence and the sinister statistics of Farr.[190] Likewise, the commission dismissed the evidence of miners who asserted that dust was the cause of their illness.[191] Instead, its conclusions were guided by the views of most medical experts who attributed a subsidiary role to dust in provoking lung disease. Noting that the disease rate amongst coal miners was then on a par with that of metal miners, the Kinnaird Commission, as with the coal mining investigations, recommended similar "hygienic" improvements.[192]

Ironically, by the mid-1860s two doctors found proof of the causative role of silica dust in occupational lung disease. The one physician was Greenhow. The other was Thomas B. Peacock, from St Thomas's Hospital and the principal medical investigator for the Kinnaird Commission, who had been, during his earlier investigations, not at all sure about the relationship.[193] Neither doctor was given the opportunity to make the proof public: there were no more commissions on metal mining for the remainder of their lives. Forty years passed before

the next official investigation; this was the important Haldane Commission of 1902.[194]

During the interim period the Metalliferous Mines Act of 1872 incorporated most of the recommendations of the Kinnaird Commission. These measures, *inter alia*, prohibited children under twelve from working underground; and they banned the use of vertical and overhanging ladders.[195] These improvements and other "hygienic" provisions certainly helped eliminate some of the more glaring malpractices in the Cornish tin mines. But the remedial measures were offset by the continued "excessive mortality at all ages" from lung disease.[196]

As has been noted, British and European research on dust disease virtually ceased during the last three decades of the 19th century. Such lack of interest, coupled with the coinciding decline of Cornish tin mining, had unfortunate repercussions for the tin miners. Owing to overseas competition, accompanied by a falling price in the metal, after the early 1860s Cornish tin mining declined from its former pre-eminence.[197] There were minor upturns, but the number of mines steadily dwindled.[198] The decline was reflected in the emigration of thousands of males; they were unable to find alternative employment in Cornwall and were therefore obliged to find mining jobs elsewhere in Britain or overseas.[199] Also, there were fewer active miners in Cornwall itself. In 1873, when the mining population had already been halved,

there were 26 500 surface and underground workers. By 1878 the number had decreased to 17 380; and in 1898 a mere 2 749 men and boys were still registered as mineworkers.[200]

In 1871 when Charles Barnham, a physician from Truro and a former medical investigator for the Kinnaird Commission, presented a significant paper to the British Medical Association, it evoked no response. The apathy persisted despite his exposure of the high incidence of lung disease among Cornish miners and his suggestion of the causative role of dust.[201] Apart from a brief two-year boom, from 1870 to 1872, the tin mining industry was in decay and of minimal account in the national economy.[202]

Tin's relative economic unimportance seems to explain the lack of interest of both the government and the medical profession in the prevalence of lung disease in the tin mines. This was so in 1892 when neither party followed up the findings, which the superintendent of government statistics, Dr William Ogle, submitted to the Labour Commission. His comparative occupational tables for the years 1880, 1881 and 1882 revealed that, unlike the case of colliers, there had been no decrease in the incidence of lung disease amongst tin miners since Farr had, thirty years earlier, quoted his statistics to the Kinnaird Commission.[203] In addition, tin miners - and, not surprisingly, potters, whose mortality was marginally lower - *"had the highest death-rate from*

disease of the lungs of any occupied class in the United Kingdom".[204] For tin miners the average death rate was three times higher than that of coal and ironstone miners; and in the age groups twenty-five to thirty-five, thirty-five to forty-five, and forty-five to fifty-five, their mortality was three, four and a half, and two and a half times greater.[205]

Unlike Farr's computations which remained unpublicised, the "oft-quoted statistics" of Ogle, presented under similar circumstance, seem to have been widely circulated. They were certainly common knowledge amongst doctors and mining inspectors.[206] Although health officers understood the implications of the figures, they apparently lacked the resolve or inclination to heed Ogle's warning that "diseases of the respiratory organs other than phthisis [tuberculosis]" were responsible for the high death rate amongst a particular occupational group.[207]

The public also disregarded this aspect of Ogle's report. Despite the "excessive mortality in all ages" which silicosis caused,[208] the chronic lung disease lacked the spectacular ingredients to provoke popular indignation and sympathy for what was essentially a working class problem. Its lack of dramatic impact contrasted strongly with the intense feelings aroused by "phossy's jaw", an occupational disease that caused agonising pain and disfigurement in young girls and women. Its hideous spectacle evoked public outrage which led in the early 20th century to the world-wide

abolition of phosphorous in the match making industry.[209]

The indifference of health officers was reflected in other ways too. Mining inspectors overlooked deteriorating dust levels, the results of which were evident in the registrar's annual mortality and disease statistics for 1892 and 1893.[210] But the alarming rise in the death rate for tin miners in the forty to fifty-five year age group,[211] as these figures strikingly demonstrated, evoked no public or official response. Despite the considerable medical knowledge about the causative link between dust and lung disease, health officers were diffident about drawing obvious conclusions concerning the prevalence of disease and mortality on the tin mines. The state did nothing; and safety measures remained conspicuously absent.[212] It is scarcely surprising that Cornish tin miners seemed oblivious of the increased dangers of their calling.

These heightened dust levels were in part a reflection of the depressed condition of the Cornish tin mining industry. Its deteriorating economic position, accelerated by Australian competition during the 1870s, necessitated the introduction by the mineowners of labour-saving devices and the application of new technologies. Such measures were designed to prevent the closure of additional mines and to revitalise the industry.[213] Ironically, both the introduction of mechanised rock drills during the

1870s and the substitution of dynamite for gunpowder seriously endangered the lives of miners at an earlier age than that to which they were already accustomed.[214]

Minor strikes erupted amongst tin miners in Cornwall during the 1890s.[215] This was a new form of resistance amongst the Cornish miners who until this time, in contrast to the colliers, had a history of feeble union organisation:[216] Cornish miners had tended to avoid potential conflict by emigrating.[217] Although the disturbances coincided with the publication in the 1890s of the registrar's mortality statistics, the strikes were linked to discontent with hours and wages,[218] grievances which reflected the changed status of Cornish miners from that of "handicraft" workers to wage labourers.[219] The evidence does not show that the dissatisfaction of miners was explicitly related to health and safety conditions.[220] Inured to shortened lives and long accustomed to the presence of dust, Cornish miners were not aware of - nor were they alerted to - the morbid threat posed by increased levels of dust.

Simultaneously, beginning in 1892, the concerted industrialisation of the Witwatersrand gold mines began. Cornwall braced itself for "another rush of emigration", this time to Johannesburg, which became styled "the Cornishman's El Dorado".[221] The news of the "LAND OF TRANVAALIAN GOLD" also spread to other mining centres;[222] and colliers from all parts of the

British Isles departed for "remuneration never dreamt of by them at home".[223] They were accompanied by lead miners from Northumberland, Durham, Cumberland, Westmoreland, Yorkshire and Lancashire and as far afield as the Isle of Wight.[224] Also, Cornish miners did not go unprepared. Before leaving, many of them equipped themselves to handle the mechanised rock drills.[225] These drills had first been introduced on the Witwatersrand in 1889, within three years of the discovery of gold;[226] and by 1896 approximately 1 115 were in use.[227]

Coal and ironstone miners who had for thirty years enjoyed "considerable immunity from pulmonary consumption" and "respiratory disease" seemed blithely unaware of the inherent dangers to their health and lives in gold mining.[228] Cornish miners, inured to such occupational perils, were fatalistic about their future. In 1863 a surgeon giving evidence before the Kinnaird Commission affirmed his knowledge of the prevalence of "miners' disease" and explained why miners did not consult him:

> Because I think that they look upon it as part and parcel of their business. A man embarks as a miner, and he looks forward to the day when he will have the disease; he expects it, - he reconciles himself to it, - and puts up with it.[229]

Dr E. Williams's perceptions of miners' attitudes were confirmed by the views of D. Buzza, an elderly Cornish miner. Having outlived most of his contemporaries, Buzza expressed a hint of satisfaction, if not pride,

at beating the odds:

> Can you go up the ladders now? - Very badly; nothing like what I used to do; but then there is one thing besides this, my age is great; I am an old man for a miner, 60 years of age; I was 60 last February.[230]

Notes

[1] De Fenton, p. 223.

[2] Irvine and Mavrogordato, pp. 163-164

[3] Cd. 3389, 1864, p. 469, qq. 20 097-20 101, evidence of Dr E. Williams.

[4] Jenkin, p. 260.

[5] Cole, p. 112.

[6] All such errors are referred to in the relevant footnotes. See van Aswegen, pp. 1-21 passim; and Burke and Richardson, pp. 151-155. The identical article, by the latter two historians, appeared in another publication one year later, but under a different title. Only their article published in the *Journal of African Studies* is referred to in this study. See also Richardson, "Miners' Phthisis in the Transvaal Gold Mining Industry", where these errors are repeated.

[7] UG 19, 1912, p. 6, n; AHFA, p. 45.

[8] AHFA, p. 51.

[9] Webster, "Lectures on Pulmonary Pathology", pp. 55-56.

[10] Holt, p. 1; Hunter, p. 1 034; Rosen, p. 392, n. 81.

[11] Webster, "Lectures on Pulmonary Pathology", p. 55.

[12] Green et al, pp. 479-480. See also Green, pp. 691-703 passim.

[13] Engelbrecht, p. 396.

[14] For detailed discussions of the anatomy of the lung and these physiological processes, see Warwick and Williams, pp. 714-716, 744, 1 202-1 204; Spencer, v. 1, pp. 59 ff; Robbins, p. 784; Bertalanffy, pp. 19-30; Bouhuys and Gee, pp. 1 378-1 388; Rippey, pp. 13-16, 43, 146-147.

[15] Ziskind et al, p. 647.

[16] R. A. Steele, p. 21; Miller, p. 5. See also Farber and Wilson, pp. 36-38.

[17] Some medical authorities prefer the term beryllium disease to berylliosis. See Orenstein and Webster, p. 1.

[18] Webster, "Lectures on Pulmonary Pathology", p. 55; Bouhuys and Gee, p. 1 386.

[19] Collis, p. 2. See also Rosen, pp. 3-7; and Hunter, pp. 954-955;

[20] Collis, p. 3; de Fer p. 223.

[21] Collis, p. 3; de Fenton, p. 223. See also Rosen, p. 60.

[22] Hunter, p. 954; Rosen, p. 366.

[23] Warwick and Williams, p. 172; Weibel, p. 1.

[24] R. A. Steele, p. 21; Green et al, p. 482; Reid, p. 5.

[25] Orenstein and Webster, p. 5.

[26] West, pp. 1 333-1 334.

[27] R. A. Steele, p. 26; West, p. 126; Bouhuys and Gee, p. 1 381.

[28] Sayers and Lanza, pp. 5-6.

[29] Oliver, *Diseases of Occupation*, pp. 344-345.

[30] Free silica is the correct scientific term. I use silica or quartz for reasons of brevity.

[31] Hunter, p. 954; Worth and Schiller, p. 812.

[32] See below, chapter 3.

[33] Hugh-Jones, p. 38.

[34] See, for instance, Watkins-Pitchford, "The Silicosis of the South African Gold Mines", pp. 109-139 passim.

[35] See, for instance, Hurwitz passim. Hurwitz qualifies his use of the term by defining it, p. 123, as "complicated silicosis with progressive massive fibrosis".

[36] Webster, "Lectures on Pulmonary Pathology", p. 58.

[37] Ziskind et al, pp. 648-649.

[38] Bailey et al, pp. 115-125.

[39] An "acute" form of simple silicosis is also currently identified. It results from exposure to heavy concentrations of respirable silica in enclosed spaces. Under such conditions the disease develops so

rapidly that clinical symptoms appear after only one to three years. See Ziskind et al, p. 651; and Hunter p. 963. Although scanty, the impressionistic evidence of Tranvaal doctors at the turn of the 20th century suggests the rare occurrence of this form of the disease on the Witwatersrand. See *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 27, 56-57, qq. 176-177, 403-410, evidence of Dr. W. D. Frazer and Dr R. G. Ralston. See also Irvine p. 223; and Oliver, "An Address on Rand Miners' Phthisis...", pp. 919-920.

[40] E. J. King, p. 16.

[41] Beadle et al, pp. 473-477 passim.

[42] Orenstein and Webster, p. 4.

[43] *Evening Chronicle*, 9 July 1913, "The Holiday Cure". See also Irvine and Macaulay, p. 365; and Oliver, *Diseases of Occupation*, p. 285.

[44] See, for example, van Niekerk, pp. 30-31.

[45] R. A. Steele, p. 26; Oliver, *Diseases of Occupation*, p. 280.

[46] Webster, "Lectures on Pulmonary Pathology", p. 58.

[47] Orenstein and Webster, p. 2.

[48] R. A. Steele, p. 26.

[49] Orenstein and Webster, p. 6.

[50] Cd. 2091, 1904, p. 10.

[51] Irvine, pp. 225-226.

[52] *TMJ*, Feb. 1906, p. 210, "Health Regulations for Mines".

[53] Following the consensus of medical opinion in the 1920s, Burke and Richardson, p. 152, as does van Aswegen, p. 1, incorrectly view all cases of simple silicosis as non-terminal.

[54] Sir Thomas Oliver was one of the few experts who did not agree with the majority opinion. See, for instance, *BMJ*, 6 Jan. 1912, "British Medical Association", 4 April 1914, "Gold-Miners' Phthisis".

[55] Macaulay and Irvine, pp. 208-209; Irvine and Watt, p. 38. Cf. Malan, p. 355, who incorrectly states that as early as the 1920s the medical profession accepted that "a silicosis without infection could in itself progress to a stage where it caused incapacity and ultimate death".

[56] For biographical details on Dr W. Watkins-Pitchford, see *SAMR*, 20 June 1926, pp.

284-285, "Retirement of Dr. Watkins-Pitchford".

[57] Malan, pp. 32-33.

[58] Watkins-Pitchford, "The Silicosis of the South African Gold Mines...", pp. 112, 113.

[59] E. J. King, p. 26.

[60] Ziskind et al, p. 652.

[61] See, for instance, Hurwitz, p. 127.

[62] *JCMMS*, Jan. 1907, pp. 230-231, "Miners' Phthisis on the Bendigo Field"; *BMJ*, 28 Sept. 1907, p. 839, "Miners' Phthisis in Bendigo". See also TG 2, 1908, pp. 324, 515, qq. 3 097, 6 028, evidence of S. S. Crowle and E. Moore

[63] Webster, "Lectures on Pulmonary Pathology", p. 58; Hurwitz, p. 118.

[64] R. A. Steele, p. 26; Hurwitz, p. 118.

[65] E. J. King, pp. 26, 30.

[66] E. J. King, p. 26; Ziskind et al, p. 660.

[67] Spencer, v. 1, p. 392; E. J. King, p. 26.

[68] Hurwitz, p. 123; R. A. Steele, p. 27.

[69] Ziskind et al, pp. 651-652.

[70] Webster, "Lectures on Pulmonary Pathology", p. 58; E. J. King, pp. 26-30.

[71] Cf. Burke and Richardson, p. 152. They coin a confusing term "phthisis" to define progressive massive fibrosis.

[72] Union, *Statutes*, 1912, no. 19, section 25; *TMJ*, 1 Sept. 1905, editorial; Macaulay and Irvine, p. 299; Rae, p. 37.

[73] Katz, "Silicosis on the South African Gold Mines", p. 206; Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 135.

[74] Burke and Richardson, p. 152, repeat this mistaken view. See, too, van Aswegen, p. 1, who also implicity draws the wrong inference.

[75] Hurwitz, pp. 92, 99, 103, 127; Ziskind et al, p. 652; West, p. 126. This may also occur in coalworkers' pneumoconiosis. See Heppleston, "The Morbid Anatomy of Coalworkers' Pneumoconiosis", p. 81.

[76] Ziskind et al, p. 652. The following give reasons for shortness of breath and cough: Orenstein

and Webster, p. 5; Farber and Wilson, p. 42; Leathart, pp. 83, 91; Gilson, pp. 318, 320; Heppleston, "Emphysema in Relation to Dust", pp. 312-314; and Ulmer, p. 331.

[77] Ziskind et al, p. 652. See also Ehrlich et al, p. 704.

[78] *Report of the Miners' Phthisis Commission, 1902-1903*, p. viii, pars. 9, 10; UG 19, 1912, p. 18, par. 40. See also Merriman Papers, correspondence, 1912, no. 112, "Amendments to Miners' Phthisis Bill", 5 Feb. 1912.

[79] Oliver, "An Address on Rand Miners' Phthisis...", p. 920.

[80] Irvine and Watt, p. 36. See also Kennedy, *A Tale of Two Mining Cities*, p. 61, who quotes part of this extract.

[81] Irvine, pp. 225, 226.

[82] Hurwitz, p. 141.

[83] Irvine, pp. 228-229. The term, *tubercle bacillus*, was originally given to the micro-organism that causes tuberculosis. But this has now been changed: the term in use today is *mycobacterium tuberculosis*.

[84] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 56, qq. 396-399, evidence of Dr R. Ralston; Watt, p. 5.

[85] Hurwitz, p. 1. See also Brecher and Brecher, p. 5.

[86] Van Niekerk, pp. 143-146; Watkins-Pitchford, "The Industrial Diseases of South Africa", p. 41.

[87] Irvine and Watt, p. 38.

[88] Thornton, pp. 262-266; Jordan, pp. 527-529; Morton, pp. 929-931; Irvine and Watt, p. 38.

[89] *MJSA*, May 1914, p. 222, editorial.

[90] *MJSA*, June 1916, pp. 187-188, editorial.

[91] Hurwitz, p. 141; Ziskind et al, pp. 653-657; Rae, p. 31. In extremely complex cases definitive diagnoses can be obtained only from biopsies. See Ziskind et al, p. 657.

[92] See, for example, Rae, p. 31; and Meiklejohn, "The Clinical and Epidemiological Aspects of the Role of Tuberculosis in Pneumoconiosis", pp. 326-327. See also Kennedy, *Silver, Sin and Sixpenny Ale*, p. 150, who shows the influence of South African diagnostic methods in Australia.

[93] *General Report of the Miners' Phthisis Prevention Committee*, 1916, p. 103. The medical survey in the 1916 report was also published in full as a two-part article in a medical journal. See Watt et al, *MJSA*, Aug. 1916, pp. 1-9, Sept. 1916, pp. 15-22.

[94] CAD, MNW, file MM 3629/20, W. Watkins-Pitchford to Secretary of Mines, 21 Dec. 1920; *MJSA*, April 1918, pp. 161-162, editorial; Birdwood, pp. 4-7. See also Katz, "Silicosis on the South African Gold Mines", pp. 215-217.

[95] Irvine and Mavrogordato, p. 166.

[96] Cartwright, *Doctors of the Mines*, pp. 130-131.

[97] Oliver, "Gold Miners' Phthisis and some of the Dangers to Health incidental to Gold Mining in the Transvaal", p. 1 679. See also Irvine, p. 235.

[98] Oliver, "Gold Miners' Phthisis and some of the Dangers to Health incidental to Gold Mining in the Transvaal", p. 1 679; van Niekerk, pp. 269-271; Irvine, p. 235.

[99] Rae, pp. 36-37.

[100] Union, *Statutes*, 1912, no. 19, section 26. The Miners' Phthisis Allowances Act, no. 34 of 1911, provided provisional compensation for miners. It was systematised by the act of 1912. For details of legislation in 1963 and 1972, see Katz, "Silicosis on the South African Gold Mines", pp. 223 ff. and p. 242, n. 223.

[101] Union, *Statutes*, 1917, no. 44, "Provision of Act No. 14 of 1916, amended by section *thirty*, subsection (2).

[102] Orenstein and Webster, pp. 6, 13.

[103] Meiklejohn, "What Advice should be Given to a Person in the Early Stages of (a) Silicosis...", p. 569. The problem of alternative employment for coalworkers in Britain is thoroughly discussed by White, pp. 60-65 passim.

[104] Union, *Statutes*, 1912, no. 19, section 21(a), 1916, no. 29, section 2(1)(a). See also Katz, "White Workers' Grievances and the Industrial Colour Bar", p. 138.

[105] *Evening Chronicle*, 22 May 1914, "Phthisis Victim's First Stage"; *Union House of Assembly Debates*, W. Madeley, 13 May 1913, col. 2370.

[106] SC 4, 1914, pp. 11, 226-240, qq. 42-47, 1 275-1 331, evidence of H. W. Smythe and of J. L. van der Merwe and W. Wybergh.

[107] SC 4, 1914, p. 92, q. 541, evidence of J. Hindman.

[108] UG 34, 1914, pp. 17-21.

[109] UG 13, 1913, p. 9; TCMA, file M21(d), memorandum no. 26, by W. Gemmill, 31 Jan. 1913.

[110] White, pp. 60-65.

[111] Orenstein and Webster, pp. 13-14.

[112] Ziskind et al, p. 653; McCallum, pp. 53-54.

[113] Webster, "Lectures on Pulmonary Pathology", p. 58; Bailey et al, pp. 115-125.

[114] De Fenton, p. 224.

[115] This quotation is cited in Meiklejohn, "The Clinical and Epidemiological Aspects of the Role of Tuberculosis in Pneumoconiosis", p. 325; and in Rosen, p. 59.

[116] Collins, p. 780; AHFA, pp. 7-10.

[117] TCMA, file T13(c), Francis Aitken, "Transvaal Miners' Phthisis Sanatorium", in circular 176/15, 4 Dec. 1915.

[118] The discussion of tuberculosis is based on the following works: Robbins, pp. 411-415 passim; Rippey, pp. 13-16, 43, 146-147, 174; AHFA, pp. 114-129 passim; and Collins, pp. 780 ff.

[119] TCMA, file T13(c), Francis Aitken, "Transvaal Miners' Phthisis Sanatorium", in circular 176/15, 4 Dec. 1915; Rosen, pp. 381-382, 398, 416-417. See also, for example, Macaulay and Irvine, p. 300.

[120] AHFA, p. 115.

[121] TCMA, file T13(c), Francis Aitken, "Transvaal Miners' Phthisis Sanatorium", circular 176/15, 4 Dec. 1915; *Rand Daily Mail*, 13 Jan. 1911, "Where Disease Lurks"; Merriman Papers, correspondence, R. Barry to JXM, 5 July 1914.

[122] TCMA, file T13(c), Francis Aitken, "Transvaal Miners' Phthisis Sanatorium", in circular 176/15, 4 Dec. 1915; van Niekerk, p. 53.

[123] *General Report of the Miners' Phthisis Prevention Committee*, 1916, p. 182.

[124] TCMA, file T13(c), Francis Aitken, "Transvaal Mirers' Phthisis Sanatorium", in circular 176/15, 4 Dec. 1915; van Niekerk, p. 53; *Evening Chronicle*, 11 Aug. 1913, "The Dust that Slays".

[125] *Evening Chronicle*, 11 Aug. 1913, "The Dust that Slays".

[126] TCMA, file T13(c), Francis Aitken, "Transvaal Miners' Phthisis Sanatorium", in circular 176/15, 4 Dec. 1915.

[127] Collins, pp. 782-783; SAIMR, pp. 151-154. See also Packard, p. 198.

[128] Allison, p. 135.

[129] Orenstein and Webster, p. 4.

[130] AHFA, p. 7; Rosen, pp. 223, 232, 360.

[131] Rosen, p. 232.

[132] AHFA, p. 10; Mavrogordato, "Contributions to the Study of Miners' Phthisis", pp. 17-18.

[133] Oliver, "Dust as a Cause of Occupational Disease", p. 273.

[134] Oliver, "Dust as a Cause of Occupational Disease", p. 273. While Oliver believed that in some cases of silicosis infection by tuberculosis might occur, he did not agree that silicosis predisposed to tuberculosis.

[135] W. C. C. Pakes was one of the few doctors who argued against the use of the term miners' phthisis. See *JCMMS*, March 1903, "Miner's [sic] Phthisis: Some Notes and Suggestions", p. 241, discussant Dr W. C. C. Pakes.

[136] Irvine, p. 222; *JCMMS*, March 1903, "Miner's [sic] Phthisis: Some Notes and Suggestions", p. 241, discussant Dr W. C. C. Pakes; Irvine and Watt, p. 33; Watt et al, pp. 276-277 ff.

[137] Irvine and Watt, p. 33; Watt et al, pp. 276-277 ff.

[138] Irvine, p. 222.

[139] Watt et al, p. 2; Watkins-Pitchford, "The Industrial Diseases of South Africa", pp. 46-49; van Niekerk, pp. 53-54.

[140] Union, *statutes*, 1911, no. 34.

[141] Van Niekerk, pp. 53-54.

[142] *BMJ*, 11 Nov. 1916, "Reviews".

[143] Watkins-Pitchford, "The Silicosis of the South African Gold Mines...", p. 111.

[144] Orenstein and Webster, p. 1.

[145] UG 19, 1912, p. 21.

[146] See, for instance, van Niekerk, pp. 59-61.

[147] See, for instance, *TMJ*, March 1911, "Medical Matters in Parliament"; *JCMMS*, March 1903, "Miner's [sic] Phthisis: Some Notes and Suggestions" p. 241, discussant Dr W. C. C. Pakes.

[148] Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 135.

[149] Tatham, pp. 134-135.

[150] See, for instance, UG 19, 1912, p. 22, par. 60.

[151] Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 135.

[152] Brock, p. 122.

[153] CAD, MNW, file MM, 1106/10, Assistant Inspector of Mines to Deputy Inspector of Mines, Krugersdorp, minute by I. M. K., no. 299/10, [April] 1910.

[154] *MJSA*, Feb. 1919, p. 374, "Hospital Statistics".

[155] Butler, p. 95; Kennedy, *A Tale of Two Mining Cities*, pp. 49-70 passim; Grey, p. 192; van Onselen, p. 49. Cartwright, *Doctors of the Mines*, pp. 23, 138, 174, distinguishes silicosis from tuberculosis, but he is not always consistent in his use of terminology.

[156] Burke and Richardson, pp. 163-165; Burke, "Disease, Labour Migration and Technological Change: The Case of the Cornish Miners", p. 79; Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", p. 145.

[157] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 24, 26, qq. 131, 161, evidence of Dr W. R. Rogers.

[158] *Star*, 12 Nov. 1902, "Miners' Phthisis".

[159] UG 19, 1912, p. 12, par. 20.

[160] Watkins-Pitchford, "The Silicosis of the South African Gold Mines...", p. 115; Irvine, p. 228.

[161] Watkins-Pitchford, "The Silicosis of the South African Gold Mines", pp. 115-116. See also Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 111.

[162] TCMA, file M23(a), W. Watkins-Pitchford to Secretary of the TCM, 31 Dec. 1915, file T13(c), Francis Aitken, "Transvaal Miners' Phthisis

Sanatorium", in circular 176/15, 4 Dec. 1915; AHFA, p. 30; Irvine and Watt, pp. 35-36; van Niekerk, pp. 66-68; Hurwitz, pp. 124, 128; Collis, p. 31.

[163] *BMJ*, 3 Jan. 1931, p. 28, "Silicosis in South Africa"; Watkins-Pitchford, "The Silicosis of the South African Gold Mines", pp. 115-116. Mavrogordato, "Contributions to the Study of Miners' Phthisis", pp. 10, 13; Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 132.

[164] *BMJ*, 3 Jan. 1931, p. 28, "Silicosis in South Africa"; Watkins-Pitchford, "The Silicosis of the South African Gold Mines...", pp. 115-116; Mavrogordato, "Contributions to the Study of Miners' Phthisis", pp. 10, 13; Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 132.

[165] Simson and Strachan, pp. 367-406 passim.

[166] Some doctors also believe that on rare occasions the silica particles and the tubercle bacilli gain entrance to the lungs simultaneously. See Gloyne, p. 217.

[167] The historians, Burke and Richardson, categorically state, pp. 151-152, that silicosis predisposes to tuberculosis.

[168] Watkins-Pitchford, "The Industrial Diseases of South Africa", p. 34.

[169] Union, *Statutes*, 1912, no. 19, section 21(1)(b).

[170] Union, *Statutes*, 1916, no. 44, sections 8, 9(e).

[171] AHFA, pp. 7-43 passim. Similarly, prior to World War II provision was made in Germany under the National Health Insurance Acts for the compensation of silicotics who exhibited signs only of tuberculosis. See AHFA, p. 11.

[172] *Financial Mail*, 18 Aug. 1978, pp. 596-597, "Tuberculosis and the Gold Mining Industry".

[173] For a more detailed discussion on compensation for simple silicosis, see Katz, "Silicosis on the South African Gold Mines", p. 255; and Katz, "Silicosis of the South African Gold Mines; Incidence and Prevalence; Compensation; 1902-1978", p. 77.

[174] AHFA, pp. 13-105 passim. Since 1973 compensation has been awarded in South Africa to those who, in the course of work on the gold mines, contract chronic obstructive lung disease, including emphysema and chronic bronchitis. For a discussion of their incidence and prevalence, see Katz, "Silicosis of the

Witwatersrand Gold Mines; Incidence and Prevalence; Compensation; 1902-1978", p. 80; *National Research Institute for Occupational Disease, Annual Report*, 1975 and 1976, p. 16.

[175] Rae, p. 37; McCallum, pp. 51-53; Heppleston, "The Morbid Anatomy of Pneumoconiosis", pp. 80-81; Allison, pp. 132-136. Cf. Burke and Richardson, p. 150, who state that only silicosis "predisposes" to tuberculosis.

[176] Heppleston, "A Review of Pneumokoniosis and Dust Suppression in Mines", p. 571; Rogan, pp. 9-10.

[177] Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 124.

[178] Although a large number of mine managers and engineers came to South Africa from the USA, many of them were undoubtedly of original Cornish stock or had been influenced by Cornish expertise. See *Mining Journal*, 12 July 1902, p. 963, "Cornwall and Devon". Waves of emigrants from Cornwall to metal mines in all parts of the globe date from the beginning of the 19th century. See D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 13, 20-21.

[179] See, for instance, *TMJ*, May 1908, pp. 246-253, "Presidential Address - British Medical Association, Transvaal Branch, 1908".

[180] Oliver, "An Address on Rand Miners' Phthisis...", p. 190.

[181] Oliver, "A Discussion of Miners' Phthisis", p. 572. See also Rosen, pp. 381-382, 397-399.

[182] Tatham, p. 158. See also Rosen, p. 380.

[183] Rosen, pp. 166-167.

[184] Oliver, "An Address on Rand Miners' Phthisis...", p. 920.

[185] Nef, v. 2, pp. 18-22 passim

[186] Court, p. 233.

[187] According to Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", p. 13, it became contemporary practice to use the generic term, tin, for all metalliferous mining in Cornwall. Accordingly, I also follow the practice.

[188] Rosen, p. 365.

[189] Rosen, pp. 213-214, 220.

[190] It is beyond the scope of this survey to investigate the reasons that the Kinnaird Commission overlooked the thought-provoking evidence of Greenhow and Farr. Rosen's explanation for the subsequent

neglect of Farr's statistics is apologetic and superficial. He contends, p. 213, that the reason that Farr's statistics were overlooked was that "they were entombed in government reports and blue books, removed from the cognizance of physicians and laymen".

[191] Collis, p. 5; Hannan, *Travels and Heartaches of a Mining Family*, pp. 64-65.

[192] Hannan, *Travels and Heartaches of a Mining Family*, pp. 66-67; Cd. 2091, 1904, p. 5, table 1; Rosen, pp. 364-365.

[193] Oliver, "A Discussion of Miners' Phthisis", p. 568; Rosen, pp. 362-372 passim, 380-381; Collis, pp. 5-6; Hannan, *Travels and Heartaches of a Mining Family*, pp. 66-67, 99-100.

[194] Cd. 2091, 1904.

[195] D. B. Barton, *Essays in Cornish Mining History*, v. 1, p. 55.

[196] Tatham, p. 162. See also Rosen, p. 243.

[197] For a meticulous genealogical account of the history of tin mining during this period, see D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 124-235.

[198] Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", p. 30; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, p. 173.

[199] D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 13, 20-21, 24, 48-49, 59; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 134, 147, 175; Hannan, *Travels and Heartaches of a Mining Family*, pp. 56-58.

[200] D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 133-134, 175. See also Cd. 2091, 1904, pp. 5, 13.

[201] Rosen, pp. 220-223; D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 26-27. For biographical details on Dr Charles Barnham, see Hannan, *Travels and Heartaches of a Mining Family*, pp. 65-66.

[202] Ashworth, p. 79; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 152-153.

[203] Cd. 2091, 1904, pp. 5, 6; *Report of the Miners' Phthisis Commission, 1902-1903*, p. 88, q. 802, evidence of A. R. Sawyer.

[204] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 8, q. 3, evidence of Dr F. Napier.

[205] Cd. 2091, 1904, p. 5.

[206] Irvine, pp. 221-222; *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 8, 88, qq. 3, 802, evidence of Dr F. Napier and A. R. Sawyer.

[207] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 88, q. 802, evidence of A. R. Sawyer.

[208] Tatham, p. 162.

[209] Hunter, pp. 127, 360-387, gives details of this disease.

[210] Cd. 2091, 1904, p. 5.

[211] Cd. 2091, 1904, p. 5.

[212] Cd. 2091, 1904, p. 26.

[213] D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 56, 57, 66; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 126, 187-190, 214-218. It must be presumed that employers found it economical to substitute capital for labour in certain mining processes. This aspect needs research.

[214] D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 173-175; Cd. 2091, 1904, p. 26. Mechanisation occurred mainly in development and not in production. See Cd. 2091, 1904, p. 10. Consequently it was not as intensive as some writers imply. See, for example, Burke and Richardson, pp. 156-158. Burke and Richardson correctly note the significance of dynamite and machine drilling in increasing dust levels. But they overstate, as does Burke, "Disease, Labour Migration and Technological Change: The Case of the Cornish Miner", p. 79, the widespread use of rock drills.

[215] Michell, p. 198; Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", pp. 29, 70; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 148 ff., 207.

[216] Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", pp. 29, 70; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 148 ff.

[217] D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 46-48.

[218] D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 50, 59, 61; D. B. Barton, *A History of Tin Mining and Smelting in Cornwall*, pp. 148 ff; Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", pp. 29, 70.

[219] Burke, "The Cornish Miner and the Cornish Mining Industry 1870-1921", p. 30.

[220] Dissatisfaction with health conditions as a cause of strikes has not been specifically investigated and research is indicated.

[221] *Cornishman*, 5 June 1902, "Mining Notes". See also Rita Barton, p. 163.

[222] Du-Val, p. 17.

[223] Du-Val, p. 15. See also Katz, "Silicosis on Witwatersrand Gold Mines with particular reference to the Miners' Phthisis Commission of 1902 to 1903", p. 7.

[224] Oliver, "An Address on Rand Miners' Phthisis...", p. 919; *Report of the Miners' Phthisis Commission, 1902-1903*, Appendix A, table 2; ibid., p. 58, q. 414, evidence of Dr E. A. Miller. See also Rosen, p. 219.

[225] Cd. 2091, 1904, p. 17. See also *JCMMS*, April 1903, "Miner's [sic] Phthisis: Some Notes and Suggestions", pp. 245-246, discussant T. L. Carter.

[226] *Report of the Council of the Association of Mine Managers*, 1893, "Presidential Opening Address".

[227] *TCMAR*, 1904, p. lxiii, Exhibi no. 14.

[228] Oliver, *Diseases of Occupation*, p. 267.

[229] Cd. 3389, 1864, p. 469, qq. 20 097-20 100, evidence of Dr E. Williams.

[230] Cd. 3389, 1864, p. 24, q. 1 680, evidence of D. Buzza.

CHAPTER 3

PREVENTIVE ASPECTS OF SILICOSIS: AN HISTORICAL PERSPECTIVE

> "We venture to express our conviction that there is no reason why work underground in whatever kind of mine, should not be a perfectly healthy employment; the work itself is thoroughly wholesome both to body and mind, and the special dangers, whether to health or to life and limb, associated with different kinds of mining, are such as, if recognised and faced, can be avoided provided that both employers and employed will co-operate in bringing this end about."---Haldane Commission, 1904[1]

> "If this is so [that mining can be perfectly healthy employment], then I think the title which Drs. Irvine and Macaulay might have chosen for this paper would be 'The Prevention of a Preventible [sic] Disease'."---Dr J. C. McNeillie, 1906.[2]

At the turn of the 20th century preventive medicine was in its infancy - an evaluation which many current doctors still apply to this branch of medicine today. Preventive medicine was at that time narrowly focused on sanitation and "hygiene", which constituted a branch of public health; and its major object was to combat germs. An extract from an article in the

Lancet of 1905 provides an apt illustration:

> When in the future the historian sits down to record the progress of preventive medicine throughout the world, the record will consist very largely of an account of the diffusion of English sanitation and ideas through the instrumentality of her Crown and self-governing colonies. Other nations, however, will also justly claim their share. For instance, the story of the battle fought by the Americans in Havana against, not the Spaniards, but, the stegomyia fasciata, the bearer of yellow fever, is an excellent example of hygienic progress made immediately after the war.[3]

Such views gained strength from the sanitary and hygienic provisions applied to the coal mining industry of 19th century Britain. Such remedial measures successfully reduced the high mortality rates, particularly from lung disease, which were prevalent amongst coalworkers during the first half of that century. It was comparatively easy for the state to prescribe prophylactic provisions in a single industry. But the protection of workers from silica dust was a more complex problem, which sanitary and hygienic measures alone could not curb; and the task of introducing remedial legislation to reduce silicosis was exacerbated by the vast array of silica-producing industries, including certain kinds of hard rock mining.

This chapter shows that silica is ubiquitous and therefore an important explanation for the widespread occurrence of silicosis. It also demonstrates how the different forms of silica vary in their potential for causing disability and death.

The chapter further discusses a number of theories accounting for the toxicity of silica and explains some of the complexities of dust. In so doing it focuses on the origins and development of non-medical technologies used for suppressing and diluting dust, and it examines medical controls to reduce both the incidence and prevalence of silicosis. These preventive measures illustrate the successes of doctors. But they also show the limitations of conventional medicine in the field of preventive medicine in general, and of occupational health in particular.

Vicente Navarro perceptively warns that industrial illnesses cannot be viewed solely as the logical and inevitable outcome of industrialisation. Such a mechanistic interpretation ignores the process of industrialisation and its relationships with those forces which have helped to mobilise capitalist societies.[4] Therefore although some of the complex social, economic and political responses to industrial disease are noted in this section, they will be examined in detail only in later chapters.

This section also shows how the Miners' Phthisis Prevention Committee, the South African Miners' Phthisis Medical Bureau and the South African Institute for Medical Research helped create a world-wide awareness and understanding of the problems of silicosis. These are achievements which many overseas authorities have acknowledged.[5] The decision

by the International Labour Organisation to hold the first international conference on silicosis in Johannesburg in 1930 was a logical consequence of such pioneering work. At this conference, attended by representatives of many countries, South African medical and mining experts presented nineteen of the twenty-six papers.[6]

The element silicon is widely distributed in nature. It comprises approximately 28 per cent of the earth's crust and is second in abundance only to oxygen.[7] Although silicon is found in practically all rocks and in many sands, clays and soils, it does not occur in a pure form. When combined with oxygen it forms a chemical chain called silicon dioxide, colloquially known as silica, which, as we have seen, causes silicosis. It may also combine with oxygen plus other elements to form more complex chains called silicates, of which asbestos is one example.[8] The ubiquity of silica is therefore one reason that it is a major health hazard.

It is believed that the crystalline structure, peculiar to silica, accounts for its potential to cause lung damage.[9] Silica occurs in three kinds of crystal forms. The largest group constitutes the macro-crystalline forms. These include quartz, by far the most common, as well as sand, sandstone, quartzite and certain granite formations. Cristobalite and tridymite, which are uncommon but extremely dangerous varieties found in meteorites and certain volcanic

rocks, are part of this group. So, too, are coesite and stishovite, which are even rarer forms occurring in meteorite craters.[10] Although these rare macro-crystalline forms have, as yet, no industrial application, they are included here because of their experimental value to research workers in exploring the fibrogenic properties of silica. In contrast, crypto- or micro-crystalline forms, such as chalcedony, of which true flint is a form, are used occupationally. So, too, are the non-crystalline, or amorphous, kinds. Consequently silica is also found in the non-crystalline gemstone, the opal, and in diatomaceous earth, which is composed of minute skeletons of fresh-water or marine plants. Although scientists do not believe that non-crystalline forms of silica have fibrogenic properties, these silica forms may acquire them if heated and melted: such processes convert the non-crystalline forms into the macro-crystalline ones of cristobalite and tridymite, which are even more dangerous than quartz.[11]

Metal miners all over the world face the hazard of silica in two mining processes. They first encounter it when digging through the country rock to reach the deposits in the development stage of the mines. Also, as there are usually large quantities of silica in the lodes, seams and reefs in which the actual ores are embedded, exposure to silica occurs during the excavation of the ore, that is during the production stage of mining. The Witwatersrand gold

fields illustrate this point well.

When gold was discovered on the Rand, it was found in several series of beds or strata, known as reefs, of which the Main Reef was then the most important.[12] The reefs are studded with pebbles ranging in size from peas to 'hens' eggs; and this formation distinguishes them from other non-auriferous strata.[13] As these conglomerate beds resembled a type of confectionery decorated with almonds, the Dutch likened them to banket, which became their name.[14] Sixty to 80 per cent of the banket comprises quartz.[15]

The scattered pebbles, which contain grains and pellets of pyrites and the finely divided gold, also consist largely of quartz. These components are bonded together by silica; and it is in this siliceous cement, and in the pyrites, that the pin-pricks of gold are found.[16] To reach the gold-bearing banket, shafts are sunk and tunnels are driven through the prevailing rock. The rock consists principally of quartzite, a sandstone rock which has been hardened and solidified by metamorphosis; and sand-stone shale also abounds.

This geological description makes it clear that even during the haphazard first stage of mining on the Witwatersrand, from 1886 to 1892, miners were exposed to the danger of silica dust. After management in 1892 systematised the development and production processes, the risk to miners of contracting silicosis

intensified. The danger persisted until the outbreak of the Anglo-Boer War, in 1899, and was all the more serious because of the complete absence of dust preventive measures. During the post-war period, between 1901 and 1912 - and even later - the exposure of miners to excessive dust levels continued; although remedial measures were eventually introduced, they were poorly implemented.[17]

The huge number of miners afflicted by silicosis on the Witwatersrand was linked partly to the nature of the gold deposits. These were unique in the world in two major respects, namely their extensiveness and regularity.[18] Therefore the concentration of a large number of miners in a single but contained gold producing region led to a prevalence of silicosis which, in absolute terms, exceeded that of any other hard rock mining centre in the world.

But there was also another reason for the excessive prevalence of dust and hence the disease. It concerns the nature of the ores, which on the Witwatersrand are generally of low grade quality. This meant that when average grade ore of approximately 6,5 penny weights (dwts) per ton was being mined, two to three tons of rock had to be excavated and processed to extract a single ounce of gold.[19] Accordingly, the industry could be viable only if huge tonnages were milled. Also, the mineowners were determined to extract from the reefs every possible ounce of gold.[20] This led them to expand the

scale and to intensify the rate of development and production to levels which other mining centres did not attain;[21] in relative terms the monthly tonnage excavated and processed on the Witwatersrand exceeded monthly tonnages elsewhere.[22] The resort by management to such techniques released vastly increased dust densities to which miners were exposed. Despite the disclaimers of Transvaal doctors that the occurence of the disease was global,[23] the incidence of silicosis, both in absolute and relative terms, was higher and of a more accelerated kind on the Witwatersrand gold fields than at other similar mining centres.[24]

Not only metal miners are exposed to silica.[25] Coal miners also face the danger. In bituminous (or "soft") coal mining small amounts of particulate silica exist in the airborne coal dust. In the mining of anthracite, a less common form of coal, silica dust is generated in far larger quantities. In Wales and Somerset, in south-west England, and in European countries, including Germany and Russia, miners have to tunnel through siliceous sandstone rock to reach the "hard-headings" from which the anthracite deposits are hewn.[26] While the presence of silica in coal dust was widely recognised in the late 19th century and the early decades of the 20th century, only a handful of perceptive research workers linked the presence of silica particles with the lung diseases of coal miners. Like the descriptions of Bernardino Ramazzini at the beginning of the 18th century,[27] doctors'

accounts of the injured lungs, were only much later to be classified as typical examples of silico-anthracosis.[28] Therefore at the turn of the 20th century, although the majority of medical experts clearly recognised the potential of silica dust for lung injury, they generally dismissed the danger of coal dust.[29] Also they believed that coal dust had a prophylactic effect on respirable stone dust; doctors held that coal dust protected workers against lung damage, particularly by pulmonary tuberculosis.[30] This explains why, until the middle of the 20th century, pneumoconiosis was ignored on the South African coal mines.

It was only after 1930, following intensive research in Germany and Britain, that the dangers of coal dust became clear;[31] and in 1943 British legislation introduced compensation for coalworkers' pneumoconiosis.[32] There is still controversy about coal dust's potential for damage. One school contends that, irrespective of whether or not silica is mixed in the dust, coal dust on its own is damaging, and that an overload in the lungs produces fibrosis and emphysema.[33] The other school argues that coal dust is benign and that it disables colliers only when it is mixed with significant quantities of silica.[34] Both schools agree, however, that progressive massive fibrosis, with an associated tuberculosis, can develop in coal-dusted lungs. Both groups also believe that the presence of silica dust plays a greater or lesser

role in the genesis of coalworkers' pneumoconiosis.[35]

It was not in miners' lungs that silica was originally found.[36] Initially doctors identified it in the lungs of tradesmen, including potters, metal-grinders and millstone makers; and it is to non-mining dusty industries to which we now must turn.[37]

Quartz, the most abundant form of silica, is widely used in industry because it is cheap and has other valuable properties.[38] Its peculiar crystalline structure enables it to resist fracture or cleavage, so endowing it with a required industrial hardness.[39] Also, it is of great importance in the refractory industries because of its high melting temperature, its low co-efficient of expansion and its inertness at high temperatures.[40] Workers are therefore exposed to silica in a wide variety of industrial occupations.

As quartz is a major component of sand, sandstone, flint and quartzite, numerous jobs involving their use pose a health hazard. Workers come into direct contact with quartz in sand-blasting, in any tasks involving the use of concrete and mortar, and in the manufacture of certain types of abrasives and scouring powders. Apart from their use in roads, railways and building construction, sandstone and quartzite are used to manufacture stonework. In the masonry industry, and particularly in the production of monumental masonry, the cutting, cleaning and

polishing processes release dangerous amounts of dust in close proximity to the craftsmen. Flint dust is equally hazardous. "Glass sand", or "potter's flint", derived from grinding or crushing calcined flint pebbles, is highly prized in the manufacture of optical glass and pottery products, including china, earthenware, tiles and ceramics. Vitreous silica, or "quartz glass", which is obtained in a similar way and is used for making precision instruments, also falls into this category. These powders are highly sought after because of their purity which does not stain the finished products. The production of these powders, however, poses serious health risks.

Under certain circumstances tiny quantities of silica dust may also cause silicosis. This occurs, for instance, when silica is superheated. As has been noted, the grinding of flint pebbles for pottery glazes is a dangerous occupation. So, too, is the cleaning of pottery after it has been kiln-fired at high temperatures. Under the very high temperature conditions, the silica in the clay is converted into cristobalite and tridymite. Although the level of respirable silica is relatively low, the loose dust on the utensils is highly fibrogenic.

This also happens with industrial ganister. Ganister, a variety of sandstone-shale, can withstand high temperatures. As a result in the past it was often mixed with clay to make bricks for furnaces in iron and steel foundries. Under normal conditions

ganister dust is potentially hazardous. But its silica content, as with flint, becomes specially harmful when subjected to high temperature conditions. Early medical works frequently refer to the dusted lungs of ganister workers and potters; and their indisposition - silicosis - was termed "ganister lung" or "potter's rot".[41]

The possibility of dust exposure is not limited to those who work in confined spaces. Risky outdoor operations include sand-blasting and the quarrying of granite, quartz, sandstone and slate. Quarrying also involves related tasks such as hewing, cutting and shaping blocks of stone. As a result there was a high incidence of silicosis amongst the masons responsible for Cragleith sandstone buildings in the city of Edinburgh.[42]

The dust generated by stone crushing is also dangerous. On the Witwatersrand gold mines the prevalence of silicosis was markedly higher amongst underground workers than amongst surface hands. Health officers, accordingly, directed preventive measures almost entirely at underground conditions. Nevertheless, as early as 1910, doctors began to advocate safeguards for surface workers at crusher stations.[43] They, too, were falling prey to the "white death", but in fewer numbers than miners.[44] Later investigations proved the validity of the physicians' assertion; and in 1914 compensation for silicosis was extended to include crusher-station workers on the

gold mines.[45]

In some industrial processes the silica in the airborne dust may exceed concentrations of 60 per cent. Even so, it is interesting to observe that workers subject to these conditions do not develop silicosis. Nor do desert dwellers who are frequently exposed to dust storms. There are two reasons for this phenomenon. First, the dust particles may be too large and coarse to be inhaled. Second, if they are inhaled, the respiratory system has no difficulty in expelling them because of their physical composition. It follows that only a certain portion of the total airborne dust generated in mining or in industry constitutes a health hazard.[46]

Two major variables, suspension characteristics and size, create the toxic fraction of airborne dust: first, the dust must be able to remain suspended in the air for a reasonable length of time;[47] and second, the particles must be sufficiently tiny to fall within the respirable fraction.[48] Only those particles which are less than seven microns in diameter – a micron is 0,001 of a millimetre – constitute the respirable fraction; and they have the potential to be both inhaled and retained in the lungs.[49] The coarse particles within this fraction may be significant in causing airways diseases, including chronic bronchitis and emphysema. But they are too large to penetrate the alveoli, or tiny air sacs, situated in the deep lung tissue.[50] The particles with this potential are

even more minute. They range from one to three microns in diameter; and particles of only this size cause silicosis.[51]

While most physicians by the beginning of the 20th century agreed that silica dust caused silicosis, several years elapsed before they began to suspect that particle size was also significant.[52] Also, they had formulated simplistic and mistaken theories to explain the toxicity of silica.

Such impressionistic theories were in conflict with the new imperative - the 20th century demand for scientifically proven medical answers. The eclecticism of the 19th century medical doctors spilled over to the first two decades of the 20th century. But research-oriented doctors were gradually obliged to specialise. Even so, it was beyond the scope of the medical profession alone to solve all the numerous complex problems which occupational medicine entailed. Medical specialists had to rely on the expertise of professionals in the non-medical sciences, including engineering, chemistry and physics. Specialisation and collaboration were both compatible and desirable in the field of occupational health, particularly with regard to dust diseases.

Specialist medical doctors applied the findings of other disciplines to their own field; and their own discoveries assisted other experts to devise precautionary and preventive measures. In these

years, as in the 19th century, forensics was the starting point for many investigations. Through post-mortem examinations of the lungs, research workers from a growing number of diverse scientific fields measured and quantified silica particles and analysed their composition to explain the damaging potential of the particles.

Although silica has a low chemical activity, it stimulates severe biological activity in the lungs.[53] As important, the reaction of the lungs to silica is out of all proportion to the amount of foreign material: small quantities of silica produce large amounts of fibrous tissue.[54] This has puzzled scientists ever since silica was identified in the lungs; and at least sixty theories - physical, chemical, immunological and "macrophage" - have been advanced to explain its damaging mechanisms.[55]

At first it seemed logical to attribute the fibrogenic potential of silica to its physical properties. This was the only recognised view at the turn of the 20th century when attention in the Transvaal and Britain focused on silicosis. Doctors at this juncture believed that the hardness and sharpness of the silica particles abraded and lacerated the lung lining causing fibrosis. "The dust quarried on the Rand is about as sharp and as hard as possible," said Dr Walter Pakes, the Transvaal government bacteriologist in 1903. He added: "The sharper the particles the shorter the life of the

miner."[56] This theory seemed so plausible that scientists found it to be congruent with the pioneering findings, in 1913, of the chemist, John McCrae, that only the minute silica particles were responsible for silicosis. In 1923, however, an American doctor, Leroy U. Gardner, convincingly refuted the theory by showing that the harder and equally sharp dusts of carborundum and diamond lacked the toxicity of silica.[57] Gardner further proved that soft, amorphous silica was fibrogenic, while stishovite, with its angular crystalline structure, was not.[58]

Research into the physical properties of silica continued. In 1935 P. Heffernan, a British medical officer, tried to prove that freshly fractured quartz had greater fibrogenic potency than quartz which had aged. This was an attempt to counter the propositions of the then predominant chemical school. Although Heffernan's theory had a number of adherents for some time, it, too, has been discarded.[59] A more sophisticated physical hypothesis is the piezo-electric one, which suggests that the compression of silica particles releases a toxic electric charge. The theory has, however, been discarded in the West; and Russian scientists currently pursuing such research have had no conclusive results.[60]

During the 1920s and 1930s chemical theories held sway. A major hypothesis, developed by British

scientists, was the "solubility" or "chemical toxicity theory of silicosis". It stemmed from the findings that, although ordinary quartz has a low solubility level, powdered quartz, namely silica dust, has a high level of solubility. Prominent proponents of the chemical school included W. E. Gye and W. J. Purdy and, later, E. H. Kettle and E. J. King. From 1922 these scientists argued that the silicic acid liberated by silica dissolved in the tissue fluids of the body and caused fibrosis. This explanation was invalidated, however, when research workers found that the amounts of released silicic acid bore no correlation with the degree of fibrosis.[61] A reinterpretation of this theory in the 1950s by a British chemist P. F. Holt, emphasised the surface size of silica particles.[62] But this hypothesis cannot explain why tridymite, with its low level of silica solubility, produces more scar tissue than other forms of quartz with higher solubility rates.[63]

Immunological theories were at various stages also introduced to explain why persons working under virtually identical dusty conditions were differently affected by silica. Such theories gained strength from the fact that every person is unique: he is "characterised by his own individual umbrella".[64] Doctors assumed that the varied fibrotic responses were due to individual allergic reactions to silica in the lung tissues. This narrow focus on hereditary factors is presently not entirely discounted. But

immunological theories today place more emphasis on acquired features. These encompass a host of environmental agencies of which irritant gases, including nitrous fumes, atmospheric pollution and tobacco smoking, are a few.[65] Such agents do not directly cause fibrosis: they may affect the dust-clearing mechanisms of the lungs, alter an individual's vulnerability and predispose him to developing other lung conditions, which in turn may reduce his immunity to silicosis.[66]

It is in this dust-clearing function of the lungs that the "macrophage" theory is located. This hypothesis, which most scientists currently support, was originally propounded by A. C. Allison and his co-workers. Aided by improved techniques in tissue culture and the development of the electron microscope, much research work is still devoted to this proposition.[67]

Macrophages are free-lying tissue cells - in the lungs they are found in the alveolar spaces - and they constitute one of the body's defence lines against invading organisms.[68] Through the process of phagacytosis the cell membrane is able first to depress and engulf a foreign particle, and later to destroy it by means of released enzymes. Sometimes, however, as with the tubercule bacillus, the foreign body is resistant to the enzymes; and when the macrophage dies, the foreign body is released back into the lung tissue.[69]

In the case of silica a different reaction is believed to occur. According to doctors, after the macrophage has absorbed the silica particle, this particular foreign body alters - or destroys - the enzymes. Consequently when the macrophage dies, both the original silica particle together with the altered macrophage content are released. Fresh macrophages then congregate to attack the foreign substances; and they, too, are in turn altered and destroyed. Chronic inflammation results; and the healing process is ironically fibrosis.[70]

Despite advances in the understanding of the toxic mechanism of silica, the findings of scientists have had only limited application in the field of prevent on. A striking example,. however, was the application of the chemical "solubility" theory in Canadian gold mines. Aluminium oxide powder was released into the mines as a coating for silica dust to reduce its solubility, so stifling its fibrogenic potential. Although other dust suppressant measures accompanied the use of this compound, it was thought to have prophylactic value.[71] But experiments have since revealed the compound's weaknesses. Its use delays, but does not prevent, the onset of fibrosis in animals. Also, in numerous cases silicosis manifested itself in the animals long after they had been removed from dust exposure.[72]

Prophylactic methods have been more successful in other industrial processes, particularly those where

sandstone grinding wheels were formerly used. In such cases emery, corundum and carborundum have proved to be relatively safe substitutes.[73] Also, relatively harmless compounds have replaced flint in the manufacture of scouring powders and abrasive paper and cloth.[74] All the same, substitutions of this kind owe more to general knowledge of the dangers of silica than to any specific theory related to its disabling properties. Likewise, new immunological precautions are the result of environmental studies. While generations of scientists have gradually expanded our knowledge of the fibrogenic mechanisms of silica, their theories and findings have had only limited application in the fiel f prevention. Also, there is still no certainty that the current and popular macrophage theory provides conclusive answers.[75]

Other avenues of dust research have been more successfully applied in practice. By the beginning of the 20th century health officers, who believed that dust particles caused silicosis, did not then realise the importance of particle size. The visibility of dust clouds, accordingly, was used as a criterion for judging whether or not a working place was a dangerous zone. Such judgements were wrong because visible dust clouds are caused by particles larger than those which constitute the respirable fraction. The most dangerous dust particles - those less than 10 microns in diameter - leave no trace of haziness and are, in fact, invisible to the human eye.[76] The potency of

these particles in causing lung damage was established in 1913, as we have noted, by the research work of Dr John McCrae, a chemist at the newly founded South African Institute for Medical Research.[77] His findings reinforced the need for collaboration between medical doctors, scientists and mining engineers on the Witwatersrand.

Until then the medical profession had been responsible for most of the research on silicosis. Therefore this harnessing in the Transvaal of the expertise of members from many other professions marked a new departure. Co-operative investigations aimed at the prevention of silicosis in mining were initiated in South Africa; and the Miners' Phthisis Prevention Committee was instituted in 1911. The personnel of this body, which was jointly funded by the government and the Chamber of Mines, was drawn from both the state and the private sector.[78]

Another change occurred almost simultaneously. As we have seen, prior investigations on silicosis in Europe had centred largely on its occurrence in industry. But since then until now, throughout the British Empire and the Western world, research on silicosis has focused largely on its occurrence in mining.[79] This new emphasis was triggered largely by the disclosures of the alarming prevalence - and even notoriety - of the disease on the Witwatersrand gold mines.

With McCrae's pioneering findings research on particle size continued.[80] Simultaneously mining engineers created rudimentary 'dust-measuring instruments.[81] These, notably the Kotze konimeter, devised by the government mining engineer, Robert Nelson Kotze, were used to calculate the number of invisible but toxic particles of dust circulating in working places in the mines.[82] Also, the konimeter was useful for making snap dust measurements in dangerous localised zones. The use of the konimeter involves laborious and tedious particle counts. But it represented a major innovation as, until then, dust density had been measured by weight, an inaccurate and crude dust-calculation method.[83]

Dust measurement is an extremely complicated procedure; and it was only in the 1930s that some of its complexities began to be unravelled. An understanding of the complexities of dust resulted partly from the introduction of the sophisticated thermal precipitator which, like the konimeter, is also used for taking "on the spot" dust samples.[84] Research in this field continues today. The measurement of dust assists health officers to assess the degree of risk in the industrial environment; dust measurement helps to control the pneumoconioses as well as other dust-induced diseases.[85]

Although the measurement of dust is a significant indicator of risk in a given situation, its suppression is crucial. This is accomplished by two

inter-related methods. The "wet" methods involve the use of water at the very source of dust production. The "dry" methods are important in eliminating the remaining dust. Measures for achieving the latter objective involve the use of powerful ventilation fans, which dilute the air, and filtration units, which catch and extract most of the dangerous residual particles. Other precautions, primarily connected with blasting operations, are the removal of all personnel from highly dangerous dust zones and the imposition of restricted re-entry periods.[86] Devising and implementing these efficient techniques for dust suppression, dilution and extraction took several decades. But since 1916 the efforts and accomplishments of the South African gold mining industry were, and are, pre-eminent in the field of mining throughout the world. This is reflected by the apparent minimal incidence of silicosis on South African gold mines today.[87]

This success story was neither swift nor smooth. Current technologies for delivering water are obviously highly advanced compared with methods in use at the turn of the century. All the same, contemporary principles of suppressing dust through water, which were very early advocated by the few concerned medical doctors and other professionals associated with the gold mining industry, are identical to those applied today.[88] Similarly, this small group of reformers also counselled the

prohibition of in-shift blasting, and the introduction of prescriptive periods for the exit and re-entry of workers to blasting areas. Such measures, they correctly believed, would diminish the risk for workers of excessive dust exposure, and would simultaneously provide sufficient time for the saturation and settling of the dust.[89] Even so, from 1902 to 1914 - and later - management was generally dilatory in systematically and efficiently implementing these sound principles. Clearly during this period management's professed goal to eliminate silicosis from the mines was subordinate to its commitment to reduce working costs.[90]

Between 1916 and 1920 the mineowners finally accepted their share of responsibility for eradicating silica dust though systematic watering and the prohibition of in-shift blasting. But shortly afterwards mining and medical professionals, who were studying dust composition and movement, realised that the use of water had limitations. Although dust may adhere to drops of water, it does not necessarily cause them to precipitate and settle.[91] The minute particles in water suspension constitute a hazard as grave as dry dust. The same danger also exists if dirty or contaminated water is provided, as on evaporation the water releases dangerous quantities of dust into the air.[92] Such problems created technical difficulties and raised working costs, dilemmas which management both had to confront and to reconcile with

other priorities.

At the turn of the 20th century the small group of healt' officers in the Transvaal also advocated improved ventilation as a preventive measure for silicosis, a suggestion in line with contemporary international views. Such views held that the main function of ventilation was to keep the different parts of a mine in a "healthy" condition and "therefore free from all accumulations or mixtures of dangerous gases".[93] Also, contemporary mining engineers perceived ventilation as a source of cool air so providing workers with comfortable temperature and humidity conditions, an environment in which they could achieve maximum productivity.[94] From such considerations it is clear that health officers interpreted ventilation as a general air dilutant for the promotion of hygiene and as a source of cooling, but not as a specific dust dilutant. In coal mines numerous inflammable and noxious gases abound, unless precautions are taken to diminish their risks. Therefore in view of the primacy and economic importance of the coal mining industry in 19th century Europe, and particularly in Britain, it was logical for health officers to emphasise the gas-dilutant qualities of ventilation.

During the first half of the 19th century the mortality amongst coalworkers in Western Europe was excessively high. Lung diseases produced a large number of deaths, particularly amongst colliers in the

forty- to fify-year age group; and in poorly ventilated mine workings volatile gases caused high-profile accidents. In Britain these appalling circumstances, as we have noted,[95] prompted state intervention. In the enactment of new safety and health regulations for coal mines, in 1842, 1850 and 1855, a high priority was given to improved ventilation. But the shaping of these measures owed little to advances in medical knowledge, particularly the importance of dust, and to the influence of the medical profession. Rather, public pressure was the dominant force.[96]

To meet the new legislative standards for air purity the owners of the coal mines were obliged to supplement natural ventilation; and they frequently did so by installing mechanical fans.[97] Improved standards in hygiene and ventilation, sustained by public vigilance, trade-union vigour and a reasonably strong state inspectorate, significantly reduced coalworker mortality;[98] and by the end of the 19th century doctors regarded coal mining as a relatively safe and healthy occupation.[99] An additional reason for satisfaction was the apparent decline in the prevalence of coalworkers' pneumoconiosis, then known as colliers' phthisis or anthracosis. Doctors attributed this phenomenon solely to improved ventilation, a term which, as we have seen, also connoted hygiene. Health officers did not conclude that better ventilation had diluted the dust

...els.[100]

Also, throughout the remainder of the 19th century and during the first two decades of the 20th century dust levels on the British coal mines remained relatively low and were virtually stationary, an aspect which has been overlooked by medical historians.[101] The two major technologies responsible for increasing the dust density in hard rock mines were not transferred to the coal fields. First, because of its risk in conjunction with coal-induced gases, dynamite was not used as a substitute for gunpowder;[102] and gunpowder blasting continued to release a coarse rather than a fine dust. Second, the industry remained largely labour intensive. Power-driven machines, namely mechanised coal cutters, did not replace hand tools on a large scale until the 1920s.[103] Consequently until then, coal deposits continued to be hewn by hand; and it was at a relatively late stage in the history of mechanisation that dust levels on the coal fields increased significantly. The improved health of coalworkers can therefore be attributed to a decline in ventilation-related accidents and to improved standards in air purification, dust dilution and hygiene.

Unlike colliers, metal miners do not generally face the danger of rock-producing gases, such as methane. Nor is carbon monoxide poisoning a high risk in most hard rock mines.[104] Because of these

relatively low-risk gas-related features, minimal attention was paid to the ventilation and the hygiene of metal mines during the second half of the 19th century, the period during which exacting regulations were being applied to the coal mines. In fact, in Cornwall and on the Witwatersrand, for instance, mechanical ventilators such as fans were rarely, if ever, installed.[105] This lack of attention to ventilation was a serious omission on the part of management and mining inspectors, as during this period the introduction of mechanical rock drills and the use of dynamite generated ever-increasing dust densities.

At the beginning of the 20th century the dawning awareness of the silicosis hazard focused attention on the need for supplementary ventilation in metalliferous mines. Air analyses revealed that gold miners on the Witwatersrand were invariably subjected to nitrous fumes, which were the product of defective detonators and "bad quality explosives".[106] Witwatersrand chemists and medical doctors disagreed with each other over the role of nitrous fumes in causing silicosis: chemists argued that it was the primary cause,[107] while doctors contended that nitrous fumes were secondary to dust.[108] But it should be stressed that both these professional bodies agreed that nitrous fumes as well as dust predisposed to silicosis.[109]

Because of the relative absence of nitrous fumes in Cornish mines, British doctors, particularly John Scott Haldane, vociferously opposed the view, peculiar to South African health officers, that exposure to nitrous fumes was a subsidiary cause of silicosis.[110] By the 1920s the British doctors had shown the soundness of their theory,[111] which current research confirms,[112] that dust was the sole cause of silicosis. While the inhalation of nitrous fumes may inhibit the efficacy of the lungs in dealing with particulate matter, the fumes are neither a primary nor a subsidiary cause of silicosis.[113]

Clearly the initial thrust for improved ventilation on the Witwatersrand stemmed largely from accidents caused by gassing, and the incorrect belief in the causative role of nitrous fumes in silicosis. Even so, the provision of mechanical ventilation lagged behind "wet methods" for preventing silicosis.[114] While lack of knowledge and inadequate technology were undoubtedly contributory factors after 1920,[115] between 1902 and 1914 the primary reason for the delayed introduction of mechanised ventilators was undoubtedly its expense.

From the outset management was reluctant - in fact, refused - to embark on costly projects. The views in 1906 of D. Wager Bradford, the mine manager of the Langlaagte Deep, are all the more telling because they continued to hold good for at least a

decade longer:

> In regard to the matter of mine ventilation, this is a very big question. Forced ventilation along the reef on a large scale may mean an enormous expenditure of money. It is easy to talk glibly of its advantages, nor do I deny them, but it is an expenditure which a great many mines could [sic] very ill afford, and while I recognise the fact that this argument should not weigh too heavily in connection with health, it seems to me that it is one that should be effective.[116]

Mechanical ventilation was, and still is, enormously expensive, and the cost calculations of the mining engineers at the turn of the century were not exaggerated, as an article published in a scientific journal explained in 1906:

> When mechanical ventilation is employed, the supplying of an unnecessary amount of air becomes a serious matter from an economic point of view, as the horse power required to increase the ventilation is not in proper proportion to the increased quantity, *but to its cube*, that is, to double a given quantity, eight times as much horse-power is needed; to treble it, 27 times; and so on. The cost, therefore, rises very rapidly, and the supply of more air than is necessary is a direct waste of money.[117]

In 1974 it was estimated that out of a total annual ventilation bill of R90 000 000 the electricity costs in forty-three mines for driving fans and providing cooling power alone was R10 000 000.[118]

The gold mining industry has from many quarters received praise for its ventilation programmes which have so successfully dealt with dust.[119] But these commendations ignore the mineowners' history of cost-cutting. As businessmen, who participated in the

gold mining industry for profit, the mineowners came to the most expensive way of handling dust, namely ventilation, only after cheaper methods had been tried. Even so, for many years it was the mineowners's specialty to adopt policies based on expediency with scant regard for the "ultimate consequences".[120]

When management eventually improved and mechanised ventilation, its initiatives were not directed solely towards the dilution of dust. The exigencies of deep level mining required the solution of other ventilation problems. As gold was mined at deeper and yet far deeper levels, there was an accompanying rise in temperatures to degrees so high that workers could not cope efficiently with their tasks.[121] The problem was compounded by excessively high humidity levels,[122] the result of saturating the workings with water, in accordance with government legislation.[123] Although such unpleasant conditions caused heat-related illnesses, more important, they diminished worker productivity.[124] The mineowners' primary motive for introducing mechanical ventilation was therefore economic.[125] A side benefit was the elimination of dust.

Despite their reassuring rhetoric in 1912 that the "problem" of silicosis was "now being effectively grappled with",[126] the mineowners, a year later, privately conceded the "seriousness" of the "facts".[127] Even so, they were sluggish in

implementing dust precaution measures voluntarily and did so "most grudgingly and at the point of bayonet".[128] In this respect they were little different from their counterparts in Australia.[129]

After World War 1, slowly and often tortuously, the axiom gradually gained acceptance that employers have an obligation to provide their employees with safe working conditions. A less obvious proposition, but one equally stressed by present-day health officers and trade unions, is that workers, too, have a duty at work to conserve their health.[130] Consequently there is often a tacit agreement between management and workmen in dusty industries that each party shall observe health precautions. While the efficient provision of water and ventilation by management can reduce dust to definably controlled limits, there may in certain workings and operations still exist a residual percentage of dangerous dust.[131] Under such circumstances workers are required to comply with additional preventive rules. These include their observance of prescriptive exit and re-entry periods to blasting zones, and the wearing of protective clothing.[132]

Currently there appears to be a high degree of worker compliance in the gold mining industry. But worker laxity and resistance to prophylactic aids may often occur, as it does in other industries, unless the efficacy of remedial measures can be shown beyond doubt.[133] Dissenting responses by workmen are

particularly apparent with respect to the wearing of respirators, which they regard as an inefficient dust preventive. These subjective perceptions are not entirely lacking in validity, as scientific investigations have proved. Modern respirators are sophisticated in design and are available in a wide variety of materials. But most present-day health officers concede that they cause discomfort when worn over long periods; and some experts continue to criticise their efficacy.[134]

In 1901, when respirators were first introduced on the Witwatersrand gold fields, there was much worker opposition to wearing them. Miners were all too well aware of the obvious imperfections and even inutility of respirators - findings later upheld by the Miners' Phthisis Prevention Committee in 1913. Also, the the unwillingness of most health officers to recommend "without reservation" even the best designs then available strengthened miners' resistance to respirators.[135] Nevertheless mine managers continued to impose them on unconsenting workmen;[136] they dismissed the experiential evidence of miners as impressionistic and in "complete ignorance of science".[137] Doctors also subscribed to management's views of miners that they were ignorant;[138] and the following quotation illustrates the paternalism of both health officers and managment:

> The miner here...has been and and is careless of his life. It will probably require legislation to force him to care.[139]

In many societies where wealth and power largely coincide in the dominant social and political structures, not surprisingly we also find paternalistic value systems which tend to ignore the common humanity shared by both employers and workers. Also, there is a tendency in such societies for medical care to be skewed in favour of those who belong to the dominant classes.[140] Perhaps even more deplorable is the view that workers are responsible for their own illness: the victims are blamed for getting ill by chance and through their own fault.[141]

"Blaming the victim" strategies can be traced back to Agricola in the 16th century, when he wrote:

> The critics say further that mining is a perilous occupation to pursue, because the miners are sometimes killed by the pestilential air which they breathe: sometimes their lungs rot away; sometimes the men perish by being crushed in masses of rock; sometimes, falling from the ladder into the shafts, they break their arms, legs, or necks...But since things like this rarely happen, *and only in so far as the workmen are careless,* [italics added] they do not deter miners from carrying on their trade.[142]

These rhetorical forms persisted for the next two centuries, and by the end of the Victorian era, when silicosis became an identifiable industrial menace in the Transvaal, a chorus of victim-blaming echoed along the Witwatersrand. Implicit in this rhetoric is a denial of responsibility. Individuals in positions of authority use it as a medium for transferring to others their own liability for the occurrence of disease and their failure to prevent it.

"Blaming the victim" strategies are still invoked today. In South Africa, however, they are not directed solely at black workers, as is sometimes suggested.[143] With respect to health most black workers are in many respects far more disadvantaged than their "privileged" white counterparts. Nevertheless, white workmen are also the targets for "unjust" censure.[144] While some present-day health officers, in much the same way as their predecessors, continue to blame workers for their ignorance and carelessness,[145] others base their case on lack of worker compliance in observing precautions. Instances of genuine worker negligence certainly occur. But the ostensibly plausible reason for censure has inherent flaws: the mere provision by management of preventives does not guarantee their routine use. Workers do not readily avail themselves of preventive measures, unless they are alerted to the specific dangers in the workplace and are schooled in the use of safeguards. But many medical officers seldom take these factors into account. Instead, in the event of accidents or the incidence of occupational disease, all too often they simply blame the victims for their lack of co-operation. This kind of rhetoric conceals a refusal by many medical authorities to take responsibility for the health education of workmen.

In societies where the working class is accorded inferior economic and social status its collective actions or the actions of its individual members

invariably tend to be discredited. Nor are "intellectuals", who claim to be objective, necessarily immune or less prone to making such value judgements.[146] In a recent article a dermatologist, possibly unconsciously, typifies this tendency by both his tone and emphasis. On the assumption that preventive measures are routinely used, he first blames the workers for neglecting to take precautions.[147] Only several paragraphs later is the need for health education advocated:

> In the management of occupational skin disease, prevention is most important. It can be difficult to persuade the workforce to use precautionary measures routinely in the prevention of dermatitis; and it may not be until an outbreak of dermatitis has occurred that measures will be deployed...
>
> Both the workforce and managerial staff should be aware of potential sensitizing and irritant properties of all materials used. There is a need for continuous education about the hazards of materials used and preventive measures.[148]

Some individuals with an avocation will voluntarily choose a potentially unhealthy industrial job. But in all societies most people for economic reasons must accept work wherever the labour market can absorb them.[149] Accordingly, in South Africa many workforce novices, most of whom are black, join industrial enterprises with health risks attached. In so doing they usually have some general but hazy notion of the potential dangers. But many of these workers, particularly those employed in secondary industries, may spend their entire working lives without acquiring any further knowledge or specific

understanding of the attendant risks.[150] This applies, in particular, to occupations where the risk becomes cumulative, but only over time, and cannot be pin-pointed at any one moment.

The findings of the Erasmus Commission in 1976 confirm that South African workers are poorly catered for with respect to health and safety education.[151] This general criticism, however, does not apply to affiliates of the Chamber of Mines, which usually provide training programmes.[152] Even so, it can be argued that these particular training schemes are too compressed and intermittent to provide a thorough occupational education.[153] Also, as the programmes are totally management devised and controlled, they do not therefore necessarily meet the perceived needs of trade unions and their members. Finally, they tend to ignore the practical experience and general occupational impressions of mineworkers, whose insights may not be as inadequate as practitioners of "blame the victim" tactics would have the public believe.

The same weaknesses are apparent in the teaching seminars given to secondary-industry workers by the National Occupational Safety Association (NOSA), an organisation financed by the commissioner of the Workmen's Compensation Fund.[154] By focusing on profit incentives these training programmes tend to skew and diffuse their ostensible health objectives. As the Erasmus Commission concluded, all these limitations

indicate the urgent need for instituting a vital nation-wide project aimed at the occupational health requirements of industrial workers.[155]

In such a scheme, if the medical experience and knowledge of union-sponsored health officers could be harnessed to the expertise of professional educationists, it would in a small, but significant way, comply with Dr Henry Sigerist's call in 1943 for a community commitment to occupational health.[156] Sigerist's request did not dismiss the need for medical and technical contributions. Indeed, he stressed the need for professionals to both assist each other, and so the community, and to continue with specialised research in their various fields.[157]

As the "wet" and "dry" preventive measures for silicosis have already been detailed, we must now examine the specific medical controls of the disease. In so doing we will again observe the pioneering role of the South African doctors. In accordance with silicosis legislation passed in 1916 the South African government established a state institution, the Miners' Phthisis Medical Bureau. Its functions were to monitor the incidence of the disease and to control the award of compensation.[158] Full-time doctors in the employ of this body initiated pre-employment and periodic examinations of first-time miners.[159] These medical controls for pneumoconiosis were the precedent for modern ones; and similar measures are currently in general use throughout the Western world.[160]

The aim of the initial examination is to prevent persons with defective lungs or sufferers from lung disease, particularly tuberculosis, from entering dusty industries. Periodic examinations - these are conducted in South Africa at six-month intervals - also serve important functions. Their aim is to detect cases of simple silicosis, progressive massive fibrosis and simple tuberculosis.[161] As we have earlier noted, workers with any form of tuberculous infection are immediately - and permanently - removed from the source of dust exposure for their own sake and to stop them infecting others.[162] In South Africa black mineworkers are, and have always been, at great risk of contracting tuberculosis in the course of their work; and health officers have always seen them as being far more liable to tuberculosis than silicosis. Consequently, insofar as Africans are concerned, the importance and purpose of the periodic examinations have always been the detection of tuberculosis.[163]

Although sound in principle, until the 1940s in practice these examinations in South Africa had grave shortcomings. The reason was that the medical scrutiny of black mineworkers was inferior to that of white miners. White miners were the sole recipients of medical attention at the Miners' Phthisis Medical Bureau. Indeed, from 1916 the first-time miners, known as the New Hand Miners, were throughly checked both clinically and radiographically. This was not so with

the vast majority of African mineworkers. After 1926 the tiny group of long-service workers, that is those who had more than five years continuous experience, received thorough physical checks and were routinely X-r[illegible].[164] But the examination of African contract workers was far less exacting. Like the New Rand Miners, they were subject to the principle of initial and periodic examinations.[165] But in practice, until the middle of World War II the application of these medical controls to African mineworkers was superficial and far inferior to those applied to white miners. Consequently many cases of tuberculosis and silicosis in black mineworkers were not discovered.[166]

This was so during the 1920s - and even later. After 1920 the mining houses did, indeed, make a concerted effort to reorganise and reform their medical care of African mineworkers. Also, they appointed to the mines full-time medical officers who devoted their attention exclusively to black workers.[167] Nevertheless, the routine examinations for detecting lung diseases were deficient in time and quality: they were purely clinical, as we have noted earlier. Moreover, as Africans were obliged to strip naked for the investigations, they regarded them as being a degrading experience.[168] Although a number of health officers also recognised the deficiencies of the medical examinations, they did not explicitly challenge them. Their rationale was that the medical profession could not improve its techniques within the

constraints imposed on it by the size of the black labour force committed to its care.[169] Undoubtedly the position of mine doctors as employees of the mining industry curbed their independence as professionals and placed them in a conflictual situation. But clearly, too, during this period their allegiance to their employers outweighed their professional commitment to their patients.

Between 1914 and 1930 the medical examinations of African mineworkers continued to be perfunctory in many respects. In 1914 the Native Grievances Commissioner, H. M. Buckle, compared them to military "marches" in which the participants were inspected like soldiers on parade.[170] The analogy was still appropriate in 1930, as Dr A. I. Girdwood, the Chief Medical Officer of the WNLA, unwittingly demonstrated, when he described, with a measure of pride, his organisation's procedures in the initial examination:

> There are usually about 170,000-180,000 recruits examined annually at the W.N.L.A. central depot, and the numbers presented for examination daily vary from 300 to 1,200.
>
> A staff of six whole-time medical officers perform these examinations...
>
> [After bathing and being taught how to breath] the boys are then lined up naked, in rows of about twenty-five, before each medical officer, who carefully auscultates the chest, front and back, and a mark is made on the boy's chest on the detection of any abnormality, however slight. He is thereupon removed to a special examing room for re-examination by one of the medical officers, whose whole time is thus occupied. If considered necessary the native is detained in hospital and X-rayed; his medical and labour history are enquired into, and a bacteriological examination is

made of his sputum.

> On the completion of auscultation of each row, an inspection is made of limbs, eyes, glands and for the presence of any venereal disease, and those passed fit are sent to the depot Pass Office for registration.
>
> A medical officer, when he has become accustomed to doing this work and has developed the power of concentration, is able to examine about sixty natives per hour, but requires a break of about half an hour after two hours' work. To many it may appear impossible to examine such a number with any degree of accuracy, but it must be realised that all that is demanded of the medical officer is the detection of an abnormality and not a diagnosis of the condition. After doing this work for several years the W.N.L.A. medical officers have naturally become proficient in the use of the stethoscope.[171]

In South Africa there are other factors which weaken the principle of using medical examinations for regulating the incidence and prevalence of silicosis. Such factors do not possess overt racial implications. Rather, their shortcomings relate to the absence of legislation for the control of silicosis in general. In many other countries medical controls, similar to the ones discussed, are obligatory in all pneumoconiosis-producing industries. But in South Africa their legislative force does not extend to secondary industries; and they apply only to present-day "controlled" mines. This is also the case with the observance of "permissive" dust levels.[172]

Thousands of South African workers in dust-producing industries are at risk, some gravely so.[173] Unlike workers on the "controlled" mines, these workmen do not have the benefit of state-prescribed

medical controls and "permissive" dust levels: they rely solely on management for the voluntary introduction of such preventive measures. As the Erasmus Commission concluded and as recent medical studies have proved, the latitude given to management in implementing precautions may partly explain South Africa's poor record for safety and health in many secondary industries and in certain primary industry sectors.[174]

The conduct of medical examinations is one method of monitoring disease incidence. Also, from its inception the Miners' Phthisis Medical Bureau has provided a second valuable service, that of recording incidence, prevalence and mortality data for silicosis. Apart from their statistical importance, such records assisted technologists in monitoring the adequacy of dust controls and guided state officials in determining "permissive" dust levels. For health officers records of this kind continue to be valuable indicators of disease and dust control.[175]

The South African medical profession has always tended to underrate its contributions to solving the problem of silicosis on the gold mines. Doctors perceive the achievements of technologists as being of greater significance than their own, and one will frequently hear them express the view that ventilation "cured" the disease.[176] In 1933 Alexander J. Orenstein, a leading mine health officer and an

authority on silicosis, stated:

> Except in the matter of the elimination of the unfit, the medical fraternity cannot claim very much credit for the reduction of the incidence of phthisis [silicosis]. It is the mining engineer who, mainly through increased ventilation, has been responsible for the betterment of our conditions.[177]

Such views indicate both the medical profession's sense of frustration with preventive medicine and its diffidence in the area.

The primary objective of doctors is to promote health. In their view it amounts to prevention, cure and rehabilitation. As rehabilitation is seldom optimal, doctors place far more stress on the first two constituents, and of these prevention is their first option, as the medical adage "prevention is better than cure" testifies.[178] Ironically, this is only a rhetorical priority: medical practitioners tend to shy away from the processes of prevention, especially when the causative agents of disease are not exclusively linked with germs.

The gap between words and deeds arises from a combination of ignorance and indifference. Ignorance can be ascribed partly to the learning process. In developed countries prevention of disease usually forms a tiny component of most undergraduate teaching programmes: here the hospital, with its focus on curative medicine, provides the context for medical training.[179] This curative approach is reinforced in graduate doctors by hospital-based fiscal and state

policies.[180] Consequently where health care is equated with cure, the distribution of health services becomes skewed and favours the affluent classes.[181] As a branch of preventive medicine, the teaching of occupational health is even more inadequately taught.[182] As a result clinicians, whose practices are unconnected with the industrial floor, rarely recognise the preventive and work-centred paradigms of occupational medicine. Nor have working class problems associated with occupational health evoked a growing and substantial response amongst the medical profession.[183] Such low levels of interest are replicated in the teaching of medical students who, in turn, tend to neglect the psychological, occupational and socio-economic integers of their patients' environment.[184]

The indifference of doctors to preventive or community-based medicine can be partly explained by deficient teaching programmes at undergraduate levels. But another significant feature of these programmes is the fact that they are devised by the medical profession itself. This creates a complex situation which gives rise to another explanation for the profession's general lack of interest in preventive medicine. Radical political economists argue that in many developed countries the medical profession is linked to the dominant power bases of societies: in helping the state to shape the nature of health services doctors therefore have a vested

interest in protecting the *status quo*.[185] But, perhaps more simply, because the financial incentives are few, doctors are not much interested in preventive medicine in general, and in occupational health in particular. As the financial attractions of preventive medicine are truly limited, doctors prefer to cater for the more affluent sectors of society; it is those sectors that help them to sustain their standards of living.[186] Consequently the profession has neglected those health and care structures which cater for the working classes. This is obviously so in South Africa where black persons, who predominate among the poor, are the recipients of an inadequate health care system.[187] Another less obvious case is that of Britain, where occupational health has been excluded from the National Health Services,[188] even relatively exempt as that structure has been from overt materialistic considerations.

Preventive medicine in its wide context of community health care and development also carries political implications, which many doctors avoid in the belief that the practice of medicine ought to be value neutral.[189] This may be one reason that occupational health is not a popular speciality.[190] As intermediaries between employers and workers it is difficult for industrial doctors to avoid conflict and political involvement by maintaining a neutral stance. They are willy-nilly obliged to confront the often painful dilemma of their dual allegiance: to

vested interests who employ them; and to workers whose well-being is also their concern. The neutrality of the industrial doctor, however desirable, may therefore prove unsatisfactory, if such a posture results in one of the parties being favoured at the expense of the other.

The need for occupational health specialists to take decisions may be an every-day occurence: the reason is that issues may range from details of compensation to "safe" dust levels. Also, the difficulties experienced by doctors in making commitments are exacerbated when problems involve probable solutions rather than scientifically grounded ones. In this regard cautious specialists, who warn against active involvement, nevertheless criticise their colleagues for political "inertia" when they fail to ensure the implementation of proven preventive measures.[191]

Finally, preventive medicine demands teamwork. It is not only inter-professional but multi-professional. The application of non-medical technologies has been markedly successful in curbing the incidence of dust diseases, so challenging the medical profession's self-image of omnipotence. Even so, conventional medicine has also made important contributions in the field of pneumoconiosis. Although medical controls cannot cure silicosis, they have succeeded in reducing disability, morbidity and mortality.[192] But non-medical successes have clearly

undermined the medical profession's belief in its curative competence and have led it to question its sense of worth. Such negative perceptions need not persist if the profession can accept as one of its primary functions the provision of *medical care* as distinct from *medical cure*.[193] The acceptance of this proposition can have positive consequences. The medical profession will not need to devalue its utility in occupational medicine. Rather, it will be better able to accept and integrate the implications and limitations of its co-operative role in preventive medicine.

A community-centred approach to occupational medicine is, to a large degree, and in economists' jargon, a public good. The market provides few incentives for its provision; there is no profit in it. If it is to be provided it must be done through non-market mechanisms. Clearly there could be inefficiencies, including greater bureaucracy and dispensing with the information provided by the price system. Depending on phenomena such as political structures and social values, there is a trade-off - the improved health and safety of an entire society.

The insights of Dr Henry Sigerist on occupational medicine are as valid today as they were nearly fifty years ago. During the 1940s, when he initially aired

them, he noted:

> The prevention of occupational disease is the collective responsibility of society and all measures available - technical medical and social - must be applied to promote and protect the health of those who through their labor create the nation's goods.[194]

Notes

[1] Cd. 2091, 1904, p. 31.

[2] *JCHMS*, May 1906, "Safety Measures in Mining", p. 337, discussant Dr J. C. McNeillie

[3] *Lancet*, 29 April 1905, p. 1 144, "Public Health in the Transvaal".

[4] Navarro, "Work, Ideology and Science: The Case of Medicine", p. 16.

[5] See, for example, AHFA, pp. 28-29; and Sayers and Lanza, pp. 6-7, 40.

[6] International Labour Organisation, *International Silicosis Conference...*

[7] Ziskind et al, pp. 644-645.

[8] Ziskind et al, p. 645; Hunter, p. 954; Taggart, pp. 3:27, 3:82.

[9] Ziskind et al, p. 647; Miller, p. ; R. A. Steele, p. 32; Holt, pp. 15-16.

[10] Miller, pp. 3-4; Taggart, pp. 3:82, 3:84, 10:16; Holt, pp. 14-15; Ziskind et al, pp. 645, 647, 654; Hunter, p. 975; R. A. Steele, p. 31.

[11] Ziskind et al, p. 645; Miller, p. 5.

[12] Denny, p. 18. Denny, p. 16, argues that the term reef is a misnomer and that stratified beds is the correct title. Lang, p. 19, suggests that the term reef was coined by Australian diggers. For a full description of the reefs, see Truscott, pp. 20-23, 53, 68-117 passim; and Hatch and Chalmers, pp. 9-87 passim.

[13] Letcher, p. 6.

[14] Struben, p. 193; Truscott, p. 5.

[15] Beadle and Bradley, pp. 464-465.

[16] For a discussion of the origins of the banket, see Haughton, pp. 345-347.

[17] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. x-xii, pars. 16-27; UG 40, 1913, p. 113; Merriman Papers, correspondence, R. Barry to JXM, 21 May 1914.

[18] Grey, pp. 148-149; Goldmann, *South African Mines*, v. 1, p. XIV.

[19] In illustrating the difficulties of winning the gold, Wilson, *Labour in the South African Gold Mines*, p. 11, quotes a good description, taken from a Chamber of Mines public relations pamphlet. Levy, p. 9, also reproduces the quotation, citing both Wilson and the original source. But Wheatcroft, pp. 121-122, plagerises the quotation: he makes a few minor changes of his own, but does not quote any source.

[20] Jeeves, *Migrant Labour in South Africa's Mining Economy*, p. 7

[21] See, for instance, Cd. 2091, 1904, p. 25; and UG 19, 1913, p. 113.

[22] I have not been unable to find quantitative comparisons of monthly tonnages. But the weighty experiential evidence of inspectors of mines, mine managers and miners point to the conclusion that monthly tonnages on the Witwatersrand were higher than elsewhere.

[23] Irvine and Mavrogordato, p. 163.

[24] *Mining Journal*, 19 July 1902, p. 996, letter by H. Rickard; SC 10, 1915, p. 555, q. 3 885, evidence of E. H. Clifford and D. Wilkinson.

[25] Burke and Richardson, pp. 147-149, incorrectly suggest that only metal miners are at risk from silica.

[26] Spencer, p. 380; Hunter, pp. 980-983. Haddock, pp. 62-64, gives definitions of bituminous and anthracite coal.

[27] Oliver, *Diseases of Occupation*, p. 280.

[28] Rosen, p. 295.

[29] Oliver, "An Address on Rand Miners' Phthisis...", p. 720; Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 111.

[30] Oliver, *Diseases of Occupation*, p. 272; Mavrogordato, "Studies in Experimental Silicosis and other Pneumokonioses", p. 111; McCallum, p. 49.

[31] Between 1932 and 1949 approximately 18 000 coalworkers in South Wales alone were disabled by coalworkers' pneumoconiosis. See Heppleston, "A Review of Pneumokoniosis and Dust Suppression in Mines", p. 571.

[32] Walton and Hamilton, p. 146.

[33] Heppleston, "A Review of Pneumokoniosis and Dust Suppression in Mines", pp. 574-575.

[34] Orenstein and Webster, pp. 2, 7-8.

[35] Orenstein and Webster, pp. 2, 7-8; Heppleston, "A Review of Pneumokoniosis and Dust Suppression in Mines", p. 574.

[36] Rosen, pp. 344-345; Oliver, "Pulmonary Symptoms in China Workers", pp. 382-389 passim.

[37] Cf. van Aswegen, p. 55, who incorrectly claims that silicosis is an industrial disease "peculiar to the gold mines of the Witwatersrand".

[38] Taggart, p. 3:83.

[39] Ziskind et al, p. 645. Quartz is 7 on the Mohs's Scale. The hardest mineral, the diamond, is 10. See Taggart, pp. 3:20, 3:82.

[40] Taggart, p. 3:88; Holt, pp. 14-15.

[41] Hunter, pp. 975-979; Miller, p. 5; Ziskind et al, pp. 645-646.

[42] Collis, p. 4.

[43] BRA, HE, v. 258, file 154M, no. 18, memorandum, "Mining Regulations Committee", signed by Dr L. G. Irvine, 1910.

[44] *Evening Chronicle*, 11 August 1913, "The Dust that Slays".

[45] Union, *Statutes*, 1914, no. 29, Schedule.

[46] Muir, p. 67.

[47] Roberts, "Dust in Mine Air", p. 110, discusses the complexity of airborne particles in suspension.

[48] Muir, pp. 60-67 passim.

[49] Webster, "Lectures on Pulmonary Pathology", p. 56; Ziskind et al, p. 646; Green et al, p. 80.

[50] Muir, p. 67; Ulmer, pp. 328-335 passim.

[51] Webster, "Lectures on Pulmonary Pathology", p. 56; Ziskind et al, p. 646.

[52] UG 19, 1912, pp. 3-4, par. 4. See also, *JCMMS*, April 1903, "Miner's [sic] Phthisis: Some Notes and Suggestions", p. 246, discussant T. L. Carter.

[53] Miller, p. 1.

[54] R. A. Steele, p. 21.

[55] Kitto, p. 42.

[56] *JCMMS*, Aug. 1903, "Some Mine Gases: Their Toxicology and possible connection with Miners' Phthisis", pp. 40-41, discussant Dr W. C. C. Pakes.

[57] Ziskind et al, p. 647; Miller, p. 2; Heppleston, "A Review of Pneumokoniosis and Dust Suppression in Mines", p. 574. Cf. Thorpe, p. 226, who incorrectly states that silica particles lacerate the lungs.

[58] Ziskind et al, p. 647. Cf. Burke and Richardson, p. 151. They describe quartz dust as "needle-like". This influences the reader to believe that the outdated physical theory currently explains the harmfulness of silica. The reader draws the inference despite the fact that the historians later, p. 154, correctly show that the theory has been invalidated.

[59] Webster, "Lectures on Pulmonary Pathology", p. 57; Holt, p. 134.

[60] Webster, "Lectures on Pulmonary Pathology", p. 57; Holt, p. 134.

[61] E. J. King, pp. 19-22; R. A. Steele, p. 34; Miller, p. 6. See also H.R.D., p. 499.

[62] Holt, pp. 26, 138-141.

[63] Miller, pp. 6-7.

[64] Green, p. 692.

[65] Green, pp. 698-699.

[66] Green, p. 702; Shapiro, pp. 315-316.

[67] Webster, "Lectures on Pulmonary Pathology", p. 58; Ziskind et al, p. 647; Miller, pp. 9, 11-12; R. A. Steele, p. 28.

[68] Green et al, p. 482.

[69] Warwick and Williams, p. 1 202; Robbins, p. 784; Bertalanffy, p. 21; Rippey, pp. 13-16, 43, 146-147, 174; Bouhuys and Gee, pp. 1 379-1 380.

[70] Ziskind et al, p. 647; Miller, p. 12.

[71] E. J. King, p. 20.

[72] Webster, "Simson Memorial Lecture", pp. 359-360. See also Hunter, p. 1 008, who states that the use of aluminium oxide powder in the manufacture of pottery has produced inconclusive results.

[73] AHFA, p. 18.

[74] Hunter, pp. 999-1 000; Ziskind et al,

pp. 645, 661.

[75] Ziskind et al, p. 648.

[76] Webster, "Lectures on Pulmonary Pathology", p. 56.

[77] McCrae, pp. 117-122; Watkins-Pitchford, "The Industrial Diseases of South Africa", p. 38.

[78] UG 49, 1912, p. ix; TCMA, file M26, R. N. Kotze to Secretary of the TCM, 8 Dec. 1911.

[79] Ziskind et al, p. 659, note that current work on lung function disability and its relationship to silicosis has involved only miners, quarrymen and foundry workers.

[80] Watkins-Pitchford and Moir, pp. 207-230.

[81] TCMA, file M27, circular 101/15, 26 August 1915.

[82] Moss, p. 83.

[83] AHFA, p. 27.

[84] Hildick-Smith, p. 295. Roberts, "Dust in Mine Air", pp. 118-122, gives a useful comparison of these instruments.

[85] Enterline and Jacobsen, p. 364.

[86] Irvine and Mavrogordato, pp. 165-166; Quilliam, pp. 46-55 passim.

[87] Katz, "Silicosis on the South African Gold Mines", pp. 226-228.

[88] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 9-10, 90, statements of Dr F. Napier and A. R. Sawyer.

[89] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 9-10, 90, statements of Dr F. Napier and A. R. Sawyer.

[90] Merriman Papers, correspondence, R. Barry to JXM, 16 Feb. 1918; J. Pratt Johnson, pp. 334-336; *JSAIE*, Oct. 1911, "The Prevention of Dust in Development Drives of Mines during Drilling Operations", p. 54, discussant K. Austin.

[91] *JC[illegible]*, Feb. 1916, "Recent Investigations on Dust", p. [illegible]2, reply to discussion.

[92] Quilliam, pp. 47, 51.

[93] Thomas Johnson, p. 44.

[94] *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 237, evidence of Dr L. G.

Irvine; Cd. 7476, 1914, p. 155, qq. 23 977-23 900, evidence of H. F. Marriott; *JCHMS*, Dec. 1906, "Safety Measures in Mining", p. 237, reply to discussion; *Rand Daily Mail*, 18 July 1910, editorial.

[95] See above. chapter 2.

[96] Rosen, pp. 307, 364 423, 424, 446, 449, 457. Rosen only touches on some of the socio-economic pressures which brought about reform. This gap requires research.

[97] Rosen, pp. 157-159.

[98] Rosen, pp. 418-419, 444-450.

[99] Rosen, pp. 380-382, 398-399; Oliver, "An Address on Rand Miners' Phthisis...", p. 920.

[100] When discussing the improved health of coalworkers, Rosen, pp. 380-382, 398-399, also fails to draw this conclusion.

[101] See, for instance, Rosen, pp. 146-147.

[102] Haddock, p. 127; Kerr, p. 102; Boulton, v. 2, p. 243.

[103] Haddock, p. 181; Boulton, v. 2, p. 256. See also Hunter, pp. 1 033-1 034, who shows that by 1913 only 8 per cent of coal in Britain was cut by machines.

[104] F. T. Williams, pp. 67-78 passim. Although carbon monoxide was a serious risk to life, deaths from poisoning by this gas were relatively infrequent on the Witwatersrand. See Macaulay and Irvine, pp. 294-295. Cf. Burke and Richardson, p. 155, who incorrectly stress the high prevalence of carbon monoxide poisoning amongst the Witwatersrand miners.

[105] Cd. 2091, 1904, pp. 8, 20; *JCHMS*, 1902-1903, "Miner's [sic] Phthisis: Some Notes and Suggestions", pp. 260-262, discussants J. Flemming, W. Cullen and B. E. Tennant.

[106] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. xvi-xviii, pars. 41-57.

[107] Heymann, p. 12; Moir, p. 15.

[108] Sansom, p. 48; *Final Report of the Mining Regulations Commission*, 1910, v. 2, evidence of Dr L. G. Irvine.

[109] *JCHMS*, Aug. 1903, "Some Mine Gases: Their Toxicology and possible connection with Miners' Phthisis", pp. 22, 25, discussants A. M. Johnston and Dr L. G. Irvine; *Final Report of the Mining Regulations Commission*, 1910, v. 2, pp. 94-95, evidence of Dr J. Moir.

[110] *JCMMS*, Sept. 1906, "Safety Measures in Mining", p. 470, letter by Dr J. S. Haldane; *Mining Journal*, 28 Oct. 1905, p. 470, letter by Dr J. S. Haldane.

[111] Mavrogordato, "Contributions to the Study of Miners' Phthisis", pp. 69-76 passim.

[112] Cf. Burke and Richardson, p. 155, who incorrectly state that nitrous fumes are a cause of silicosis.

[113] International Labour Organisation, ed., *Encyclopaedia of International Health and Safety*, v. 1, p. 946.

[114] Irvine and Mavrogordato, p. 166.

[115] Mavrogordato, "Contributions to the Study of Miners' Phthisis", pp. 69-76 passim; Letcher, p. 372; Irvine and Mavrogordato, p. 168.

[116] *JCMMS*, Oct. 1906, "Safety Measures in Mining", p. 118, discussant J. W. Bradford.

[117] *JCMMS*, Feb. 1906, p. 256, "Mining".

[118] Hemp, unnumbered page; *Report of the Medical Bureau for Occupational Diseases*, 1975-1976, p. 2.

[119] Lang, pp. 341-342; Bunt, p. 437; MacConahie, p. 132.

[120] Marriott, pp. 58-65 passim.

[121] F. T. Williams, p. 112.

[122] Moss, p. 82.

[123] Van Niekerk, pp. 17-18.

[124] *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 3.

[125] BRA, HE, v. 268, file 240V, "Memorandum on Ventilation of Mines", recorded date 21 July 1910.

[126] *Rand Daily Mail*, 24 Oct. 1912, "Johnnies".

[127] TCMA, file M23 (d), memorandum by W. Gemmill, 19 June 1913, "The Miners' Phthisis Board...31 Jan. 1913".

[128] Merriman Papers, correspondence, R. Barry to JXM, 16 Feb. 1918.

[129] Kennedy, *A Tale of Two Mining Cities*, pp. 65-70. Kennedy exaggerates the initial willingness of the South African mineowners to introduce preventive measures.

[130] Kriel, p. 5; Weindling, p. 17.

[131] Quilliam, p. 48.

[132] Ziskind et al, p. 661.

[133] Webster, "Lectures on Pulmonary Pathology", p. 59; Quilliam, p. 48.

[134] Aspin, p. 56; Archibald, p. 314.

[135] *Report of the Miners' Phthisis Commission, 1902-1903*, p. xii, par. 30.

[136] TG 2, 1908, pp. 455, 475, 691, qq. 5 011, 5 322, 8 979, evidence of T. Mathews, D. Hadenfeld and F. Crean; TCMA, file M27, Miners' Phthisis Prevention Committee to Minister of Mines, 13 June 1913.

[137] *South African Mines, Commerce and Industries*, 17 March 1906, p. 4, "The Inflation of Working Costs by Inefficient Labour".

[138] Pern, p. 975; *East Rand Express*, 4 Feb. 1911, letter by "Miner".

[139] *South African Mines, Commerce and Industries*, 1 Aug. 1903, p. 449, "Leading Article".

[140] Navarro, *Medicine under Capitalism*, pp. 82-92 passim; Kriel, pp. 6-8.

[141] De Beer, p. 70.

[142] This extract from *De Re Metallica* is fully cited in Martinson, p. 5.

[143] See de Beer, p. 70, for an extreme version of this view.

[144] See Kriel, pp. 3-9, for the application of the term "justice" to medical care.

[145] De Beer, p. 70.

[146] Kriel, p. 7.

[147] Adler, p. 6, shows that many workers who run the risk of contracting dermatitis have never been apprised by management of the inherent dangers in their jobs.

[148] English, p. 30.

[149] Navarro, *Medicine under Capitalism*, p. 87; Sigerist, p. ix.

[150] Adler, p. 1; Horner et al, p. 6.

[151] Horner et al, pp. 6-20 passim; Adler, pp. 2-3.

[152] Horner et al, pp. 4, 6.

[153] I thank East Daggafontein Mines Ltd for allowing me to observe their training programmes in 1973.

[154] Matthysen. I have also had the opportunity to attend "Bunny" Matthysen's safety training seminars.

[155] Horner et al, pp. 18-19.

[156] Sigerist, p. x. See also Clutterbuck, pp. 149-150, who claims that present-day British workmen "hunger" for education on safety and health.

[157] Sigerist, p. x.

[158] Union, *Statutes*, 1916, no. 44, section 23.

[159] *MJSA*, June 1916, pp. 161-162, editorial, April 1918, pp. 161-162, editorial.

[160] The British Factories Act of 1937, for instance, instituted initial and periodic examinations for all workers in dangerous secondary-industry occupations. See Hunter, p. 178.

[161] Irvine and Mavrogordato, p. 166.

[162] AHFA, pp. 166-167; Rae, p. 37.

[163] TCMA, file M23(a), W. Watkins-Pitchford to Secretary of the TCM, 31 Jan. 1915; CAD, MNW, file MM 3629/20, W. Watkins-Pitchford to Secretary of Mines and Industries, 21 Dec. 1920. Surprisingly, Packard, pp. 200-202, does not note that the mineowners' rationalisation for perpetuating the migrant labour system was strongly linked to their need to obviate the incidence in Africans of progressive massive fibrosis, in which tuberculosis was the "complication".

[164] Irvine and Mavrogordato, p. 166.

[165] Irvine and Mavrogordato, p. 166.

[166] CAD, MNW, file MM 3629/20, W. Watkins-Pitchford to Secretary of Mines and Industries, 21 Dec. 1920; Mavrogordato, "Contributions to the Study of Miners' Phthisis", p. 44.

[167] Irvine and Mavrogordato, p. 166. See also Cartwright, *Doctors of the Mines*, pp. 40-65 passim, for an uncritical appraisal of the growth on the mines of medical services for Africans.

[168] Native Grievances Inquiry, transcript of evidence, 3 March 1914, p. 64, evidence of C. W. Williams.

[169] Mavrogordato, "Contributions to the Study of Miners' Phthisis", p. 44; CAD, MNW, file MM 3629/20,

W. Watkins-Pitchford to Secretary of Mines and Industries, 21 Dec. 1920.

[170] UG 37, 1914, p. 26, par. 186.

[171] Birdwood, pp. 5-6. I originally used this quotation in Katz, "Silicosis on the South African Gold Mines", pp. 215-216.

[172] Roberts, "Dust in Mine Air", p. 115; Beadle, pp. 31-32.

[173] Erlich et al, pp. 504-508.

[174] Horner et al, pp. 5-8; Erlich et al, pp. 504-508.

[175] AHFA, p. 170.

[176] In similar vein, Mavrogordato, "Contributions to the Study of Miners' Phthisis", p. 75, argues that the mining engineers rather than the doctors reduced the incidence of silicosis on the Witwatersrand gold mines.

[177] *JCMMS*, Oct. 1933, "W. R. Jones - Silicosis", p. 150, discussant Dr A. J. Orenstein.

[178] Kirsch, p. 1.

[179] Kirsch, pp. 1, 6; Hunter, p. 219.

[180] Kirsch, p. 2; Navarro, *Medicine under Capitalism*, p. 20; Savage, p. 8.

[181] Savage, pp. 8-12; Navarro, *Medicine under Capitalism*, pp. 67-96 passim.

[182] Hunter, pp. 219-220.

[183] Hunter, pp. 219-221; Weindling, pp. 11, 13.

[184] Hunter, pp. 219-221.

[185] Navarro, *Medicine under Capitalism*, pp. 67-167 passim; Navarro, "Work, Ideology and Science: The Case of Medicine", p. 30; Savage, p. 9. See also *Sunday Star*, 15 Nov. 1987, "Review", by Dr Trefor Jenkins.

[186] Cf. Weindling, p. 13, who argues that doctors avoid occupational medicine because "the medical ethic of self-employment resists the linking of practice with industries".

[187] *Sunday Star*, 15 Nov. 1987, "Review", by Dr Trefor Jenkins.

[188] Both Clutterbuck, p. 142, and Weindling, p. 5, note this, but do not comment on it.

[189] Hunter, p. 221; Kirsch, pp. 1-2, 9; Navarro, *Medicine under Capitalism*, pp. 93-95.

[190] Navarro, "The Underdevelopment of Health or the Health of Underdevelopment", p. 27.

[191] Hall, p. 5.

[192] Navarro, *Medicine under Capitalism*, p. 111.

[193] Navarro, *Medicine under Capitalism*, p. 111.

[194] Sigerist, p. x.

CHAPTER 4

THE HEEDLESS YEARS 1886-1899

> "But no one who had carefully watched the rise of gold mining believed that the Transvaal was a failure. It was known to be under a cloud which time would roll away, and leave not a silver, but a golden lining...Where the blustering speculator ruled and the worthless manager controlled, we now have men with scientific mining knowledge with very different assistants to carry out their ideas."---*Mining Journal*, 1893.[1]

> "After boring-machine labour in South Africa, [miners returned to England] 'only to die of phthisis', of somewhat the same description as that of the phthisis of steel grinders and stonemasons, due to the inhalation of dust, a disease widely different from the old 'miners' complaint' of Cornwall or the tuberculous phthisis, as it is generally known..."---*Cornubian*, 1902.[2]

Long before the initial introduction of mechanical rock drills in Cornwall during the 1860s,[3] generations of Cornish tin miners as well as hard rock miners in other parts of Britain and Europe had succumbed to silicosis. This disease, variously

described by a number of indefinite names, including miners' phthisis, usually manifested itself in a chronic form. This meant that the victims on the mines risked disablement and death at an average age of fifty years. Indeed, Dr William Ogle's official statistics in 1892 showed that during the 1880s they had the shortest working lives of any other occupational group in England and Wales.[4] A few health officers viewed this situation in a grave light. But most doctors seemed to regard an average of thirty years as an adequate span for the working life of a miner.

An extract from the report of the Transvaal Medical Society submitted in evidence to the Weldon Commission on miners' phthisis in 1902 supports this impression:

> Miners' phthisis is not peculiar to machine miners, for, although it is most typically and commonly seen in them, it frequently develops also in course of time amongst miners who have never used rock drills at all. *Most cases of this sort, however, have been mining for long periods, e.g., over 30 years, and it may be taken that the ordinary miner has a considerable advantage in length of life over the rock drill miner.*[5]

The extract appears to offer a straightforward comparison of the working lives of rock drillers with accelerated silicosis and "ordinary" miners with chronic silicosis. But it contains a subtle disclaimer, which has been italicised. Although the doctors noted that general miners were liable to chronic silicosis, they also made it plain that they

had no serious misgivings about the shortened careers of the victims.

During the latter part of the 19th century and the opening years of the 20th century chronic silicosis was habitual amongst miners: miners all over the world recognised that incapacitation and premature death from lung disease almost inevitably accompanied work in metal or hard rock mines.[6] They were conscious, too, that these occupational hazards were of minimal concern to the vast majority of medical professionals, members of management, and governmental authorities, who viewed these morbid factors as inescapable facts of the miners' vocation.

In short, members of the managerial and professional classes took the disease and death of miners for granted. This attitude was entirely in accord with the low regard they had for miners as a class of workers. The candid, even if brutal and insensitive, views expressed by George Albu, a prominent mining director, in cross-examining a witness before the Transvaal Industrial Commission of 1897, were typical of management. "It must be very hard on a woman," he said, "if the husband has got no further in life than a miner."[7]

This dismissive view of workers was also bound up with the health officers' *laissez faire* attitude to the working class. In assuming that workers exercised freedom of occupational choice, the health officers

concluded that miners in choosing their calling weighed the attendant risks, which included their liability to silicosis.[8] Thomas McIsaac, who had had prior mining experience in British Columbia and Canada before coming to the Witwatersrand, interpreted these views in 1903 to the Weldon Commission:

> I have never seen any special interest taken in the matter [of sickness amongst the miners]. It was recognised that the work of a miner was unhealthy, and I never saw any steps taken to make it more healthy...as a rule old men do not suffer. They [miners] do not get very old.[9]

In examining this passage we should also note that McIsaac's tone was clearly one of resentful resignation. As such it calls into question the mystique which surrounds the 19th century Cornish miners, and which historians tend to perpetuate. In doing so historians stress the innate fondness of the Cornish miners for their profession which bred in them an "unquestioning acceptance" of and an "immunity" to their harsh conditions.[10] But the evidence of McIsaac does not seem to agree with these romanticised assumptions. Further research on the perceptions of Cornish miners during this period is clearly needed.

During the 19th century miners in Cornwall - and elsewhere - usually began an informal apprenticeship as children, when they were approximately ten years of age; and they were fully fledged professionals before they were out of their teens.[11] Therefore a working career which constituted a mere thirty years foreshadowed a relatively short lifespan; and like

McIsaac, miners were obliged to face the prospect that a majority of them would not reach "old-age".[12] Consequently when overseas miners drifted to the gold fields of the Witwatersrand, after their formal launching on 8 September 1886 by Paul Kruger, President of the South African Republic,[13] as a matter of course they acknowledged - as did health officers and management - that they would have relatively short working lives.

They could not know that within thirteen years after gold mining began on the Witwatersrand hundreds of them would be dead or dying suddenly and painfully from accelerated silicosis at a relatively young age. This appeared to be a unique form of the disease and differed in many marked respects from its chronic form. In retrospect Sir Thomas Oliver, a physician from Newcastle-upon-Tyne, reported:

> My experience was gained in miners who had returned to Northumberland during and shortly after the close of the [Anglo-Boer] War. A few years previously young miners in the bloom of health had left their Northern homes for the South African goldfields, and after working there four to six years returned to Northumberland and elsewhere broken in health.[14]

The bad news soon spread through Cornwall and other British mining centres and burst upon mining circles along the Witwatersrand. These alarming disclosures came between 1900 and 1902, that is during and shortly after the Anglo-Boer War, so that Transvaal health and mining officers turned to the medical history of the gold fields during the pre-war

period. It was important for them to find out whether silicosis was an entirely new phenomenon on the Witwatersrand, as the reports seemed to indicate. They concluded that a lack of awareness of the dangers of heightend dust levels and even the existence of accelerated silicosis on the gold fields were typical of the period from 1886 to 1899.[15] This view gained additional support in 1930 from three prominent health officers, who presented a joint historical survey to the International Conference on Silicosis held in Johannesburg.[16]

This long-held view is, in fact, an unchallenged myth. Official, popular and academic historians have helped perpetuate it by either their unquestioning adoption of this conclusion, or by their uncritical acceptance and repetition of the evidence on which the health officers based their original case in 1902.[17] Dispelling this old canard therefore requires a careful analysis of the health officers' evidence.

Official medical records to assist the doctors in their investigation of the prevalence of silicosis during the period before the Anglo-Boer War hardly existed. One reason was the absence of a public officer of health. Accordingly, there were no mine statistics regarding prevalent diseases among the workforce.[18] Also, the records of mine managers were of little value. In contrast to fatal mine accidents which were noticed and recorded, deaths from disease invariably occurred beyond the boundaries of the

mining properties. Finally, because of the frequency with which workmen changed jobs on the mines, management did not notice the absence of miners who had died.[19]

There were also no reliable mortality statistics for those who died from disease in private dwellings or hospitals. The sole hospital was the Johannesburg Hospital, which was erected in 1888 as a "corrugated iron building with a canvas roof". Although by 1899 it had managed to keep pace with the needs of the mining town, it remained a relatively small institution;[20] and as late as 1906 it had only about 300 beds for white patients.[21] In the years before the outbreak of the Anglo-Boer War, as in the post-war period, it lacked the facilities to nurse, for any length of time, either those with severe illnesses or those who were chronically ill, including miners who had silicosis.[22] Also, the hospital did not keep a comprehensive register of admissions and deaths.[23] Even when the hospital, on compassionate grounds, occasionally admitted terminal cases of silicosis, it recorded the evidence only in isolated instances.[24]

The investigators' recourse to the records of doctors in private practice proved equally unrewarding. Nor were the medical practitioners' oral recall of case histories any more illuminating. Although several doctors remembered that they had attended to patients terminally ill with silicosis, they had attributed death not "to that cause, but to

ordinary chronic pneumonia" or tuberculosis.[25]

In the total absence of statistical evidence, the research team turned to the report and evidence of the Industrial Commission of 1897. As this became the linch-pin for their case,[26] a brief outline of the reasons for the commission's appointment and its terms of reference are therefore in order.

The immediate cause of the appointment of the Industrial Commission was the depression in the gold mining industry which followed the feverish stock market speculations which had reached their pinnacle in 1895 two years earlier. Kruger's government hoped that public confidence in the industry would be restored by the commission's recommendations. Also, the Republican administration anticipated that the evidence would show that the responsibility for the depression rested with the mineowners, rather than with its own allegedly incompetent policies and hostile treatment of the mining industry.[27]

The commission's terms of reference were to "report regarding all matters that may have stood in the way, or have hindered, and still may hinder the development of the mining industry".[28] The terms of reference had wide parameters and could have encompassed deficiencies in the mineworkers' daily working conditions. A detailed analysis of the evidence shows, however, that neither the witnesses nor the government commissioners, aided by advisory

representatives of the mining industry, viewed such issues as relevant to the commission's immediate task. The context of the all-pervasive depression on the Witwatersrand in the wake of the industry's slump dictated a concentration on those narrowly focused economic issues which were reliant only on the high working costs of the mines.

White mineworkers, many of whom had lost their jobs and had been hard hit in other ways by the depression, were also concerned with such issues.[29] They were anxious, too, about the possibility of additional mines closing and a further reduction in the number of available jobs. Alexander Buchan Fyffe, a member of the newly formed Rand Mine Workers' Union and the sole official spokesman for the mineworkers, in his evidence to the commission did, in fact, mention a few workmen's complaints. But his major concern, not unnaturally at this time and in this context, was with mineworkers' earnings. Indeed, the major portion of his testimony aimed to show that wages, particularly those of married men, were low relative to the high cost of living.[30] In similar vein the only other worker who presented evidence, Robert Barrow, an experienced Lancashire foreman, confined his complaints solely to the high cost of living for married men.[31]

It is on this flimsy evidence - or lack of it - that the investigating team of health officers based their case in 1902. They contended that, as neither

Barrow nor Fyffe had aired "any grievance as to the condition of health or suggestion as to disability arising from the method of mining", this constituted proof that no-one on the Transvaal gold fields was aware of the disease.[32] If they considered the evidence of Fyffe and Barrow as being the decisive factor, their conclusion was based on even shakier grounds.

There are strong grounds for believing that Barrow's evidence had been instigated and prepared by H. Eckstein and Co, the holding company for the mine of which he was foreman.[33] Therefore we can almost entirely disregard his testimony. Likewise, although Fyffe's evidence was more critical of management's policy towards the white workforce, for several reasons it can also be discounted.

First, the organisation which Fyffe represented, and which survived for only a few months, had a membership which at its peak totalled a mere 800.[34] It therefore barely represented the views of white mine employees whose numbers in December 1897 totalled 8 060. This figure includes the 293 clerical staff members and, more important, the 2 339 workers listed as miners.[35] Second, his trade union did not consist solely of miners; it also comprised artisans and entrepreneurs - Fyffe was himself an independent mining contractor - many of whom therefore had little regular or continuous underground working experience.[36] Finally, his opening statement made it

clear that his primary, if not sole, objective was "to give evidence on the working man's wages on the Rand".[37]

Local medical practitioners in 1901 and 1902 must certainly have felt reassured when they received the message that their colleagues had been unaware and ignorant of the existence of silicosis in the early days of gold mining on the Witwatersrand. Their absolution from neglect was reinforced in 1907 by one of the original members of the Transvaal Medical Society's investigating committee, Louis Godfrey Irvine, who was by then recognised as a leading expert on the disease.[38] In evidence to the Mining Regulations Commission, Irvine stated:

> Neither the employers nor the workmen, *nor, for that matter, anyone else,* [italics added] realised at that time the great danger to health and life arising from rock drill work.[39]

The 1930 medico-historical survey, already referred to, bestowed additional blessings on the doctors who had practised medicine in the Transvaal before the Anglo-Boer War. In a watered-down version of his 1907 statement Irvine, as one of the co-authors of this conference paper, stated that the period from 1886 to 1899 was "locally...one of practical ignorance of the menace of silicosis".[40]

Some Johannesburg doctors had in the course of private practice first attended to patients with symptoms of the accelerated type of silicosis in 1897,

that is ten years after the gold mining industry had begun operating.[41] As severe incapacitation and sudden and painful death amongst the Witwatersrand miners occurred on average only seven years after the onset of accelerated silicosis, the year 1897 was a significant signpost. It indicated a strong link between the malignant form of the disease and the widespread introduction, in 1892, of mechanised rock drills which exposed miners to excessive amounts of respirable silica dust. But in the triennium 1897-1899 only a handful of Johannesburg residents associated with the mining industry drew this connection;[42] and the majority of these did so with hindsight.[43]

In the light of this evidence, the health investigators' claim that there was "ignorance" of the "menace of silicosis" is ostensibly plausible. But their blanket assertion of total unawareness was, in fact, a half truth. There was indeed "ignorance" - but in only one respect. The ignorance applied to the manifestation of silicosis solely in its *accelerated* form and not in its *chronic* form.[44] The widespread prevalence of *chronic* silicosis, although it was then vaguely termed "sick miners lung", was common knowledge.[45]

It cannot be contended that there was a scarcity of medical doctors on the Witwatersrand to diagnose this condition. Within two years of the proclamation of the gold fields, Johannesburg, its chief urban

centre, had by 1888 evolved "from the position of a diggers' camp to that of one of the largest towns in South Africa;[46] and by 1893 it could boast of "stately" suburbs containing houses which cost from "£1 000 to £4 000".[47] Under such circumstances there were certainly sufficient doctors to cater for the town's inhabitants, as well as for dwellers on the East and West Rand.[48]

The strong possibility exists that the investigating officers made these sweeping claims of "total ignorance" and "total unawareness" to protect themselves and their colleagues from justifiable accusations of neglect. It can be argued further that state officials, doctors and management, by disregarding the health and welfare of miners in respect of chronic silicosis, condoned the unhealthy mining conditions which ultimately spawned the development of accelerated silicosis. Such heedlessness was all the more serious in the light of the relatively advanced state of contemporary medical knowledge about the disease. Moreover, the public accessibility of a number of official British statistical studies extended this understanding, for the scientific surveys demonstrated the excessive mortality from lung disease in hard rock miners.

The occurrence of *chronic silicosis* with its resultant premature mortality was a universal phenomenon in hard rock mining; and in all metal mining centres scant attention was paid to this

adversity for miners. On the Witwatersrand the manifestation of chronic silicosis paralleled the experiences of other similar mining districts, including Cornwall, Victoria and Western Australia.[49] During the 19th century *accelerated silicosis* was also beginning to appear in these mining regions, particularly in Cornwall. But its incidence in these centres lagged behind that on the Witwatersrand,[50] while its prevalence was less noticeable as well. The impact of accelerated silicosis in the Transvaal had important repercussions. It alerted health officers in all the hard rock mining districts of Britain and her overseas possessions to the more malignant form of the disease. Its occurence caused them to focus also on chronic silicosis, a form of the disease which, as has been noted, had until this period been almost entirely neglected.

There are several reasons for the prominence assumed by both forms of the disease in the Transvaal. The magnitude of the industry and the vast numbers of miners employed were causes of primary importance. Linked to these factors was the unique concentration of the Witwatersrand's gold deposits in "a comparatively small territory".[51] This resulted in the containment of workers in a single region. Consequently the fate of numerous afflicted workers on the Witwatersrand could be more easily accounted for than that of miners in Cornwall, Victoria and Western Australia, for example, where the tin and gold

deposits were scattered over wide terrains. If we are to explain the proliferat of silicosis in both its chronic and accelerated forms on the Witwatersrand, we need to examine the nature and modification of local working conditions. Changes in development and production techniques accompanied the expansion of gold mining from its immature beginnings to its conversion to a rich and powerful industrial enterprise. It is therefore to the early workings on the gold fields that we must now turn.

Gold was first discovered in 188- on Transvaal farmlands in outcrops, a term which must be clarified because of the relevance of outcrop mining to the occurrence of silicosis. The gold reefs, like strata which form the crust of the earth, were probably o iginally deposited in horizontal layers. But they are invariably no longer found in this position. Instead, on the Witwatersrand, these beds of rock in which the gold is impacted are inclined at an "angle of dip" to the horizon; and when they come to the surface at some point this is called the *outcrop*.[52] Such outcrops of parallel banket beds were discovered on the Witwatersrand. They run from east to west in a general straight line, termed the strike. It was soon estimated that the Witwatersrand strike extended for approximately fifty miles.[53] But during the period 1886 to 1914 the major mining activities took place in the forty-mile stretch from the farm, Randfontein, in the west, through the newly established town of

Johannesburg in approximately the centre, to the farm, Modderfontein, in the east.[54]

Gold excavation initially took place on the outcrop claims. Each claim constituted a parallelogram, the size of which was 150 Cape feet in the direction of the strike, that is from east to west, and 400 Cape feet in the direction of the dip, namely from north to south.[55] As each claim was relatively small - it was a mere 60 000 square Cape feet, or the equivalent of 64 025 square English feet - the claims were invariably combined with contiguous claims to form larger and more viable mining plots: the Village Main Reef and the Henry Nourse, for instance, constituted eleven and thirty-four such claims.[56]

Outcrop companies also often amalgamated with one another. By 1895 the Ferreira, which had previously absorbed thirty-one claims belonging to the Rand Mines, amalgamated with the Wemmer, which at that time consisted of twenty-one claims - its ten original claims plus an additional eleven subsequently taken over from the Village Reef.[57] These amalgamated outcrop properties were not necessarily long, narrow east-west strips. Indeed, while there was no standard plot size, amalgamated claims often extended from the northern outcrop itself as far south as the down-dip boundary. Consequently their north-south widths could range from 400 feet - this was the width of a single claim - to several thousand feet.[58]

Ore was extracted from the outcrop itself or below the ground in the direction of the dip, the dip being the inclination of the reef, at right angles to the strike, and running in a north-south direction.[59] The angle of the dip, namely the position at which the reef began its inclined descent, varied. Although the average dip at the surface was approximately fifty degrees, in some regions it was a mere sixteen degrees. In other places, particularly in the neighbourhood of Johannesburg in the central section, it was often as steep as eighty-seven to ninety degrees.[60] The lateral and vertical extent of the outcrops, and the angle of the dip, determined, therefore, the nature of the excavation methods adopted.

The geological composition of the surface and upper layers of the earth, namely the weathered zone, also shaped the initial digging operations. These surface layers were colloquially termed the free milling zone; and it was in this belt that the original underground excavations were located. In contrast to the "blue", hard, pyritic rock which lay approximately 100 to 200 feet below the surface, the weathered zone consisted of "red", soft, oxidised rock.[61] It was relatively easy to excavate the damp and friable surface banket. When it could not be dug up, hand-held hammer drills, known as hand drills, were used to penetrate the ore which was subsequently released "in bucketfuls" by blasting with dynamite.[62]

Apart from the ease with which it could be excavated, the banket in the free milling zone had a natural concentration of high grades of ore.[63] The grades averaged one Troy ounce, or twenty dwts, per ton; they far exceeded the average grades found in the pyritic zone below.[64] Also, the "free" gold of the weathered zone was amenable to mercury. After the banket had been broken up into smaller pieces by the stamps and then crushed by the mills, the gold was easily extracted from the residual rock by a simple process of amalgamation with the quicksilver. In contrast, pyritic ore excavated from the lower depths resisted the amalgamation process; "vast dumps of untreated tailings" running as high as fourteen dwts per ton had to be abandoned.[65] Consequently early mining was largely confined to the free milling zone. The pyritic zone was deliberately avoided, unless the gold grades were running sufficiently high to offset the loss in the tailings.

The term mining, with its scientific, technological and organisational connotations, cannot be applied to the original operations on the gold fields.[66] Ore was literally dug out of the ground or gouged out of the outcrop rock. The simple workings consisted of the following: short adits, or horizontal tunnels, driven into hilly outcrops; open-cast quarries; and shallow vertical and horizontal shafts haphazardly sunk to one or two levels in the free milling zone.[67] In 1908 M. H. Coombe, the manager of

the State Mine, vividly recalled his introduction to the Witwatersrand in 1888:

> I enquired the direction and set off to the Wemmer, that being the nearest mine on which work that could be called mining was in progress. Arriving there, I found an open cutting probably 200 ft. long on the outcrop strike and 40 to 50 ft. deep, at the bottom of which a mixed assortment of natives, Cape boys, and Europeans were at work, drilling and filling ore into buckets, which in one instance were hoisted with a whip, oxen being the motive force, and in the others with windlasses, white men being on the handles.
>
> About 100 ft. west of the western end of the open cast, a little 6 ft. x 4 ft. shaft was sunk, from which a drive was proceeding towards the cutting, all the work being done by white men. I do not remember seeing the slightest evidence of machinery on the Wemmer at this time, if I except the whip and the windlasses, and I think they were trying to keep a mill going out of the cutting. Such was the genesis of mining on the Rand.[68]

Two years later, in 1890, gold mining was still characterised by a lack of sophistication in techniques and organisation. The strike was honeycombed with shallow vertical and inclined shafts; and batteries of crushers and mills dotted the landscape like groves of trees.[69] Apart from the batteries, other power-driven equipment included only essential Cornish pumps and haulage gear for the deeper excavations. In short, there was little mechanisation. A low level of intensity characterised the scale of operations which were, in the main, located in the weathered zone.

A brief explanation of the initial mining operations on the Witwatersrand will enable us to

understand why conditions of work rendered miners liable to the risk of contracting silicosis. In so doing we must also appraise the Transvaal health officers' retrospective assessment, in 1930, that in the early workings mining conditions were healthy, a judgement which most historians have unquestioningly accepted.[70] In a sweeping statement made before the International Conference on Silicosis the Transvaal health officers claimed: "It can...be safely said that the miners were exposed to little if any injurious dust during those days."[71] They used two arguments to justify their claim: first, the prevalence of open-air quarry work; and second, the "comparative" softness and friability of the banket in the underground workings of the free milling zone.[72] Although these contentions have some plausibility, they are not sufficiently strong to support the generalisation, which must therefore be challenged.

Let us examine the first premise that open-cast quarry work does not expose workers to the risk of respirable silica. Undoubtedly this outdoor method of excavating ore has advantages for miners working in proximity to dust. For instance, it is a rather obvious proposition that after blasting heavy concentrations of dust - and fumes - cannot accumulate in the open air. It should be noted, however, that during this period, one in which no dust preventive measures were adopted, workers were exposed to free silica in a variety of other tasks. These outdoor

jobs - some of which were similar to those performed in the shallow underground workings - included hand drilling, shovelling the broken ore into buckets and bins, and tipping their contents into containers in readiness for crushing and milling procedures. In 1914 Wilfred Watkins-Pitchford, director of the newly founded South African Institute for Medical Research, exploded the myth that hand drilling "is not particularly conducive to miners' phthisis". Although at the time of writing he was referring to hand drilling in the pyritic zone, his remarks which followed are relevant to mining conditions in the free milling zone:

> This opinion certainly receives no support from the fact that stonemasons, who follow their calling in the open air, and employ only hand tools for their work, are specially liable to develop the disease. "Stonemasons' phthisis" was, indeed, definitely recognised as a disease before miners' phthisis.[73]

While hand drilling and the other equally well defined mining processes generated varying quantities of respirable silica, there was another indirect source of dust, but one which is seldom noted: dust was also frequently released, even if inadvertently, by the impacting of one rock on another during and after specific procedures.[74] Also, each battery of stamps and mills was a major source of dust. Although the stamps and mills used water for breaking and crushing the ore, the huge unenclosed machines created enormous dust clouds which perforce enveloped those workers whose jobs were to feed the machines and free

them of their residue.

There can be no doubt that such working conditions promoted the incidence of silicosis, but in a chronic rather than in an accelerated form. Yet the 1930 health officers failed to draw this conclusion. Their omission to do so is striking, because surface dust conditions during the 1920s were remarkably similar to those during the late 1880s and early 1890s. Their own experience had shown and empirical data had proved that crushing and milling operations could produce a chronic form of silicosis. This was certainly so with operators at crushing stations whose incapacitation and death had been documented since 1910.[75] These surface workers were so vulnerable to silicosis that South African legislation in 1916 made special provision to compensate them.[76] For these reasons we must reject the health officers' first proposition that open-cast mining was harmless.

Likewise, their second proposition, which stated that the dust from the weathered zone could not cause silicosis, can also be proved wrong. It is probable that the shallow underground workings in the free milling zone were reasonably well ventilated.[77] This was because air currents which were created by the numerous vertical cuttings and shafts driven from the surface to these shallow depths could diffuse dust concentrations produced in underground excavation and ore-removal jobs. But this was not the corner-stone of the health officers' proposition. Instead, they

focused only on the geological composition of the weathered zone. Implicitly they argued that mining operations in the friable free milling zone provoked only a soft, coarse, harmless dust, as opposed to the hard, fine, dangerous dust which was later produced in the pyritic zone. As there are no records or any other evidence of dust samples having been taken before the Anglo-Boer War,[78] this theory has little substantive value, and should be viewed rather as a subjective hypothesis.

Despite its attributes of softness and friability the composition of the weathered zone was hugely siliceous. Consequently the dust it produced possessed high concentrations of respirable silica and was, by definition, a hazard to health. As important, the numerous underground procedures continuously stirred up siliceous dust in close proximity to the workers; and long term exposure to this hazard would undoubtedly have induced a chronic form of silicosis. There is little satisfactory empirical evidence or established scientific data to support the health officers' contention that work in the free milling zone produced only a harmless dust, which could not cause silicosis.

In 1930 the medical profession, mining officials and the leaders of the gold mining industry were also proponents of this view.[79] In supporting it, all of them committed themselves to perpetuating the fallacy that silicosis did not exist on the gold fields

between 1886 and 1892. The endorsement by so many parties of a spurious rationale suggests a collective motive that bears scrutiny.

The world-wide indifference of health officers to chronic silicosis and to its cause, namely silica dust, has been amply demonstrated; and the prevalence of this attitude on the Witwatersrand was only logical. In contrast, health officers could not with similar casualness dismiss the occurrence of accelerated silicosis with its markedly malignant and morbid ingredients. Indeed, its initial widespread manifestation on the Witwatersrand between 1901 and 1902 certainly took doctors and mining officials by surprise. Health officers conceded their ignorance of this form of the disease. With some justice they claimed that they were unaware of the danger residing in one of the disease's intermediary producers, namely the mechanised rock drill.[80] But it was impossible for them to deny their understanding and knowledge of the aetiology of the disease. Indeed, they could instantly identify its primary cause as being silica dust.[81]

All the same, they did not take the admission to its logical conclusion. They would have been indicting their former colleagues for disregarding industrial hygiene, that aspect of preventive medicine to which they were professionally committed.[82] Both before and after 1892, the early-day health officers on the Witwatersrand, by failing to issue a single

cautionary note or warning, condoned and permitted the existence of dust levels that were conducive to the incidence of chronic silicosis. Such omissions had serious implications. By default they had extenuated mining conditions under which respirable silica had proliferated, so providing the ideal circumstances for the spawning of accelerated silicosis.

The health officers and their allies in 1930 were undoubtedly well aware of their colleagues' oversights in the past. But their need during the 1930s to promote publicly the industry's battle against the disease, and their desire to vindicate the industry's tarnished record, with respect to silicosis, prompted them to conceal the negative features of the history of the disease on the Witwatersrand. Clearly the collective motive of all the concerned parties was to exonerate the gold mining industry, including all its direct and indirect professional affiliates, from any blame initially attaching to the excessive incidence and prevalence of accelerated silicosis.

The medical practitioners and their supporters achieved their objective in two ways. First, they negated the presence of dangerous dust and the occurrence of silicosis during the period 1886 to 1892. Second, when in retrospect they were obliged to admit that harmful dust had come into existence on the mines, that is during the period 1892 to 1899, they deflected attention from chronic silicosis by stressing the liablity of the respirable silica for

provoking only accelerated silicosis. This, too, they could justify and extenuate with a measure of plausibility: they pleaded ignorance and unawareness of the adverse implications inherent in the industrialisation of the gold mines after 1892.

These rationales created a myth which literally banished chronic silicosis from the gold fields during the entire period from 1886 to 1899. The myth exonerated the failure of the early-day health officers and management to institute remedial measures for obviating the incidence of both chronic and accelerated silicosis during the entire period before the Anglo-Boer War.

There can be no doubt that in the free milling zone from 1887 to 1892 mining conditions, haphazard and unscientific as they were, were conducive to silicosis in its chronic form. But if we are to understand the incidence of accelerated silicosis from 1892 onwards, we need to examine the changes in mining techniques and organisation which caused increased and heavy dust concentrations in the confined space of the underground workings. This was the result of new developments in the industry; and it is to these that we must now turn.

The years from 1889 to 1891 were characterised by a lull in mining activities, for the industry was during this period in a precarious state.

> At Johannesburg [in 1890] some mills were going, but many were silent. The boom had

> passed and the blight had come. As the possibility and opportunity of speculation had uwindled, the crowd of irresponsibles who dubbed themselves brokers also dwindled till very few were left...At least one-third of the houses and stores were unoccupied and gloom was general. Families were leaving, offices were being closed, cheap sales of pianos and safes blocked the Market Square.[83]

On the gold fields themselves approximately seventy-three companies continued developing and producing; but as many as eighty-nine had closed down their operations.[84] The reason for the end to mining activities and the "panic" was the virtual exhaustion of the rich surface zone. At lower depths the average grade of ore was falling and, if the excavation of gold was to continue, the "dreaded" pyritic zone had next to be tackled.[85] Already the results of the few mining ventures in this zone were unpromising. Not only was the average grade of gold lower. Also, the pyritic ore - after crushing - resisted extraction. As mentioned earlier, in the banket of the weathered zone oxidation released the "free" gold particles, which then easily amalgamated with the mercury used in the simple extraction process. But a large percentage of pyritic ore was unamenable to this treatment.[86] Thus pyritic became a synonym for refractory. A mere 55 per cent of the gold from pyritic banket was recovered, and the rest had to be discarded in the tailings.[87] Also, work was more difficult and expensive in the pyritic zone;[88] and as working costs remained high, profits were further diminished.[89] These circumstances were a deterrent to fresh capital, both foreign and local, and discouraged the

development of existing enterprises and the launching of new ones.[90] The prevailing mood of pessimism was exacerbated by doubts regarding the persistence of the gold-bearing deposits.[91]

By 1892 the industry had not only recovered from the doldrums into which it had sunk: it was additionally infused with a strong and now sophisticated vigour. Two reasons were responsible for this transformation. The first was the introduction of a successful extraction process for treating pyritic ore; and the second was the establishment of proof that the gold persisted at depth.

Between 1889 and 1892 the cyanide process patented by John Stewart MacArthur and his medical partners, the brothers Dr Robert and Dr William Forrest, was developed and proved. Its successful application to the tailings resulted in a 90 per cent recovery of the total gold mined.[92] Even before the process had been modified and significantly improved, gold recovered from the tailings rose from 40 000 ounces per month in 1890 to 100 000 ounces per month in 1892;[93] and by 1893 it had become almost universal practice on the Witwatersrand to treat the tailings and slimes with the cyanide process.[94] Barring other exigencies, mining of low grade and pyritic ore could now become a profitable proposition.

Mining experience has shown that, once the surface layers of gold deposits, geologically similar to those found on the Witwatersrand, have been exhausted, there is usually little prospect for future mining at depth. This is because either the gold may suddenly disappear, a common phenomenon colloquially called "pinching out", or the grades may peter to unpayable lows.[95] But neither of these obstacles to deep level mining was present on the Witwatersrand. First, the gold did not disappear. Rather its persistence on a gently sloping plane was established at depth. Although the dip at the surface was steeply inclined at an average angle of forty-two degrees, it gradually flattened; and at a depth of 1 484 feet on the incline it was a mere twenty-nine degrees.[96] Thereafter the reefs continued their sloping descent, but at an average angle of only one degree per 100 feet.[97] Second, the gold grades did not peter out. Although the average grades were low - the ore fluctuated between high, medium and low grades - the ore deposits were more regular and uniform than in any other auriferous area in the world.[98] As a contemporary mining expert sagely observed: "Compared with the Rand conglomerates, all other gold-mines are but sporadic little flukes."[99]

These auspicious findings were made between 1889 and 1895 by the sinking of deep, diamond-drill boreholes. Boreholes were initially located on claims staked immediately south of the outcrop claims; but

High temperature air in deep underground workings is caused by the natural heat of the strata. But underground temperature conditions on the Witwatersrand were comparatively moderate: the mines were endowed with a relatively flat geothermic gradient.[106] At sea level the rock temperatures in mines rose one degree fahrenheit per sixty feet of vertical depth.[107] Because of the elevation of the highveld at 6 000 feet above sea level - and the absence of thermal springs - the gold fields of the Transvaal were "marvellously well favoured in this respect".[108]

In mines at this high altitude underground rock temperatures rose more slowly than at sea level, a one-degree increase occurring every 208 feet of vertical depth.[109] Thus an "ordinary" underground temperature at 2 000 feet on the Witwatersrand was a mere seventy degrees. This was relatively moderate compared to many overseas metal mines, and those of Cornwall in particular, where temperatures ranged from eighty to ninety-two degrees at the same depth.[110] On the Witwatersrand the temperature of the rock rose to eighty degrees fahrenheit only when underground work commenced at 3 500 feet.[111] This meant that miners on the Witwatersrand could work relatively more efficiently at the deeper levels than their counterparts elsewhere.

Yet another factor favoured deep level mining on the Witwatersrand. The inflow of water into the mines

at depth was less than in other mining centres.[112] Of course water did occur at depth. But the extensive use of Cornish pumps ensured that its presence did not hinder underground operations unduly.[113] In one notable instance, that of the Knights Deep, excessive quantities of underground water compelled the mine to stop working. The mine, however, was sold at a profit and its subsequent owners were easily able to surmount the problem.[114] Moreover, the "fortunate" accessibility of abundant and cheap coal made steam-powered operations, including pumping, relatively cheap.[115] The mineowners considered the freight charges for coal excessive. Even so, *before* the Anglo-Boer War the cost of coal, including transport, accounted for only 11 per cent of the total working costs of the mines.[116] Underground water was a relatively minor problem on the Witwatersrand gold mines.

Each of these geological conditions favoured deep level mining: the relative ease with which reefs dislocated by faults or pierced by dykes could be relocated; moderate underground temperatures; and the relative cheapness of pumping out minimal quantities of underground water. But each had disadvantageous features with respect to the health of workers. Each, too, in its way aggravated the conditions conducive to silicosis; and these are discussed later.[117] Before the Anglo-Boer War health officers had no intimation of the inimical potential of these apparent

advantages. They, like management, would have viewed the specific and unique combination of these fortuitous circumstances on the Witwatersrand as minor, but still significant, reasons for optimism in the future of deep level mining.

The deep level claims – they were called block claims in Queensland[118] – were staked immediately south of the outcrop claims. They occupied approximately 12,5 miles along the length of the strike, this being nearly 40 per cent of the total east-west length of the reef.[119] The deep level mines were classified as first row, second row and third row deeps depending on how far south they were located from the outcrops. Thus the northern boundaries of each of the three rows of deeps were on average 1 000 feet, 4 000 to 5 000 feet, and 6 000 to 7 000 feet respectively from the outcrops.[120] It is not possible to give an average working depth for each row, as differences in the angle of the dip along the Main Reef, the chief gold-bearing series mined during this period,[121] resulted in shafts intersecting the banket at enormously varied underground levels.[122]

The fortuitous concurrence, round about 1892, of the cyanide process with proof of the persistence and payability of the gold reefs at depth, came as a "relief" to those speculators who had earlier staked claims south of the outcrop boundaries.[123] Also, their faith in the deep levels was vindicated.[124] Therefore at the beginning of 1892 "scientific mining" was

introduced on the Witwatersrand.[125] It must not be thought that scientific mining was a term applied only to the deep levels, as some historians incorrectly imply.[126] Now that it was possible to extract 90 per cent of the gold from the pyritic zone, outcrop companies began systematically to exploit their claims using development and production techniques which were virtually identical to those used by the deep level mines.

"Deep level" when applied to the first row of deep level mines is a misnomer. The term has apt connotations only when used in a geographical sense to distinguish the first row of deeps from those situated on the outcrop claims.[127] In a relative sense they were invariably not deep mines: in most cases their shafts intersected the reef at under 1 000 feet in vertical depth.[128] For instance, the mean depth of the two vertical shafts on the Geldenhuis Deep, the original first level deep to come into production, in 1895, was 680 feet;[129] and the vertical shafts of the Rose Deep, the Jumpers Deep and the Crown Deep, which had started producing by 1897, were only a little deeper - at 812 feet, 1 072 feet and 1 099 feet.[130] Therefore their average vertical depths were relatively shallow. As late as 1906 the main deep level mines of the Corner House were of "moderate depth" and averaged "about 1,200 feet vertical".[131]

Mines which were deep in the literal sense of the word comprised those mines classified as second and

third row deep levels.[132] Yet the term deep when applied to mines in the second row was also relative; many of them were also comparatively shallow.[133] The important point is that mines classified as second and third level deeps were developed and became producers significantly later than the first row of deep level mines. In 1902, by the end of the Anglo-Boer War, only three of the ten companies on the second row had started developing - two of these were producing in 1905 - while the remaining seven were just beginning to break ground.[134] At the same time, that is in 1902, only two mines had been established on the third row; and both had only started shaft sinking. As late as 1905, by which time mining production had regained its pre-war levels, a mere twenty of the sixty-seven producing mines were classified as deep level companies.[135] Only the first row of deeps is therefore relevant to the period prior to the Anglo-Boer War.

With respect to depth of workings the outcrop mines were similar to those of the first level deeps. In their deeper workings the inclined shafts of the outcrop mines cut the reef at 800 to 900 vertical feet from the surface.[136] In some places outcrop mines were worked at even deeper levels. The Ferreira had by 1895, for example, reached 1 020 feet - the deepest vertical level then being worked by any other outcrop mine.[137] While the impossibility of establishing average depths needs to be recognised[138] - depths were proportional to the breadth of the property from the

outcrop to the down-dip - it can nevertheless be concluded that depth is not a criterion for distinguishing outcrop mines from the first level deeps.

Some of S. H. Frankel's calculation for dividend yields from gold during the period 1887 to 1936 have been based on the working depths of mines.[139] Therefore unless historians who make use of his figures understand the similarity in working depths of outcrop mines and first level deeps, they are in real danger of undervaluing the contributions of the outcrop mines to the total dividend yield, especially in the period 1902 to 1914.[140] It should be stressed again that in 1905, when the industry had regained its pre-war vitality, more than two-thirds of the producing mines were categorised as outcrop mines;[141] and the majority of these were significant producers right up to World War 1.[142] Therefore, if working conditions at depth were conducive to silicosis, they applied equally to both outcrop and to deep level mines.

By failing to appreciate the scientific way in which the outcrop mines were exploited after 1892, historians have fallen into a trap - that of attributing the incidence of accelerated silicosis solely to deep level mining practices.[143] But as a mining expert correctly observed in 1897: "It is recognised that a deep level is nothing more, from the mining point of view, than an outcrop with vertical

shafts."[144] Therefore differences between outcrop and deep level mines on the Witwatersrand cannot be identified by using mining techniques as the criterion.

If one takes into account that a large number of outcrop mines and first level deeps were worked together as single enterprises, the distinction between outcrop and deep level mines becomes even more blurred. Of the thirty-seven producing mines in 1894 only nine could be classified as "pure" outcrops: the remainder, excluding a handful of true first level deeps, were an amalgamation of outcrop and deep level mines.[145] Such combinations were a characteristic feature of the East Rand where first level deeps did not exist as separate enterprises. First row, deep level claims were both owned and operated by contiguous outcrop companies;[146] notable examples were the Simmer and Jack and the East Rand Proprietary Mines.[147] Likewise, the City and Suburban, close to the town of Johannesburg, was categorised as an outcrop mine, although it worked a number of first row, deep level claims too.[148]

In 1965 Blainey claimed that the Jameson Raid in 1895 was inspired by a conspiracy of deep level proprietors who formed a definitive group: it was distinct from the other defined party which comprised the outcrop owners.[149] Through his analysis of the structure of mine ownership, Kubicek later rejected Blainey's view; Kubicek convincingly showed that the

mineowners' interests in both kinds of mines overlapped.[150] By demonstrating that many individual mines in 1895 could not be rigidly categorised as outcrops or deep levels, this study, therefore, supports Kubicek's argument. It should further be noted that George Farrar, one of the principal conspirators behind the Jameson Raid, was Chairman of the East Rand Proprietary Mines, which was a mixture of outcrop and deep level mines. Consequently Farrar cannot be rigidly classified as a "deep level" proprietor.[151]

In summary we have established two important points. First, outcrop mines cannot be distinguished from deep level mines in terms of either ownership or mining techniques. Second, all the Witwatersrand mines and their owners were equally liable for the occurrence of both chronic and accelerated silicosis.

Towards the end of 1891 the mineowners systematically introduced scientific mining on the Witwatersrand gold fields. In contrast to the relatively low costs of their early haphazard mining enterprises, which had by this time exhausted the rich surface patches, the industrialisation of the mines involved high capital, development and working costs. These costs must be carefully examined because they are relevant to this study for at least two reasons. First, the industrialists' efforts to contain costs and to reduce, in particular, working costs led them to introduce underground methods which created

exceedingly high dust densities. These harmful working conditions provoked both chronic and accelerated silicosis. Second, after 1902, when the devastations of silicosis had been established, management's continued obsession with cost-cutting strategies was a major reason that effective dust controls were not implemented. Instead, the mineowners subordinated the health of underground workers, particularly of miners, to their financial priorities.

Before 1892 much gold mining capital came locally from entrepreneurs who had amassed large fortunes from the diamond mines in Kimberley. Overseas investors, particularly those from Britain, were initially dubious about the prospects of the Witwatersrand; and the "shady" dealings of the promotors sharpened their misgivings.[152] But by 1892 an optimism in the long-term future of Witwatersrand gold replaced the investors' former uncertainties. From now on most of the capital came from overseas, notably from Britain and from France and to a lesser extent from Germany.[153] Even so, until 1895 most of the profits from the mines continued to be based on windfalls, the result of speculative dealings which had nothing to do with mining *per se*.[154]

As the initial inflows of capital from overseas were inadequate, mineowners had to develop new mining strategies to strengthen and sustain investor confidence. Also, additional supplies of working

capital were needed for expensive modifications to the outcrop mines and the first level deeps;[155] and continuous supplies of development capital were crucial for the establishment of the second and third row of deep level mines. Accordingly, the mineowners employed "scientific mining men" to introduce sound methods of mining; and the mining of Witwatersrand gold became an organised industry.[156]

After 1895 profits mainly came from the performance of the mines.[157] Such profits were determined by the "pay limit".[158] Although the demand for gold was unlimited, throughout this period the gold price was fixed.[159] Consequently any increases in the cost of gold production could not be passed on to consumers. Mines had either to produce at a cost below the world price of gold or cease production, a problem which was compounded in that this era of mining expansion was characterised by a world-wide cycle of commodity price increases.[160] The "pay limit" therefore hinged on two crucial contingencies, of which contemporary mining experts were well aware: the yield of gold per ton and working costs.[161]

Had the average yields of the Witwatersrand mines been twelve dwts per ton and thus comparable, for instance, to the gold mines of Australia and of North America, the mineowners' undertakings to the shareholders to make profits would have been far less difficult.[162] But the average grade of ore on the majority of Witwatersrand mines was considerably less

than even the "moderate" predictions of optimistic statisticians who in 1895 had forecast averages of sixteen dwts per ton.[163]

If the mineowners had concentrated on mining only high grade patches of the reef and had "picked the eyes" of the mines, they would probably have generated profits relatively easily without finding it necessary to pare working costs to the bare minimum. But this would have meant abandoning vast reserves of unpayable low grade ore and facing the prospect of mines with relatively short lives.[164] Instead, by pledging themselves to mining low grade ores, the mineowners committed them to working richer grade reserves in conjunction with much poorer ones.[165] While this decision could prolong the lives of the mines, it was nevertheless speculative.[166]

The speculative features of the industry were more marked with respect to the second and third row deeps to which enormous capital costs were tied, but with only long term expectations of return on capital. The first row of deep levels on average began producing only three and a half years after shaft sinking had first begun. But the development of the second and the third level deeps took very much longer, so delaying production further.[167]

During the 1890s the mineowners tended to mine the reefs selectively: they mined relatively high grades of ore ranging from eight to eleven dwts per

ton and avoided reserves of lower grades.[168] But after the Anglo-Boer War the Randlords changed their policy. They were determined to mine at a profit reserves of only four dwts per ton.[169] This switch of policy, which coincided with the production stage of the second row deeps, had two results. First, to mine these lower grades profitably the Randlords intensified their efforts to reduce working costs. Second, the lay public, in "misinterpreting" the industry's policy of mining lower grades, believed that the grade of ore decreased with depth.[170]

Once the mineowners had committed themselves to the risks of mining low grade ores, they unduly stressed the costs of deep level mining. The mineowners' emphasis on the high development and production costs of the deep level mines - indeed, it amounted to propaganda - led laymen, as we have noted, to believe that the mines got "poorer as they increased in depth".[171] For the mining directors this was a convenient fallacy which they did not attempt to correct, unless they were directly challenged to do so.[172] It should be noted that in both the outcrop and the deep level mines the grades of gold, even at depth, ranged from high to low.[173] The mineowners' exaggeration of the higher working costs of the deep level mines was a spurious attempt to conceal the speculative nature of their undertakings.

Because of the speculative nature of gold mining, investors wanted higher returns on gold than on other

ventures. They were satisfied with average returns of 3 to 4 per cent on secure investments, but expected 9 to 10 per cent on gold.[174] The research of R. A. Lehfeldt and S. H. Frankel has shown that the average yields on capital from the Witwatersrand's low grade gold mining industry for the single period 1907-1926 was a mere 6,2 per cent.[175] Although returns on gold were higher than in other investments, gold returns did not always live up to investor expectations;[176] and after a brief boom in the market following the close of the Anglo-Boer War, many overseas investors turned their backs on Witwatersrand gold.[177] One of the reasons for this fall off of investor interest in gold was that the overall internal rate of return between 1887 and 1914, as Frankel has since calculated, was a mere 2,1 per cent.[178]

The relatively low dividend returns on gold explain why the mineowners constantly complained of the difficulties of raising fresh or additional capital, and were obsessional about the need to reduce working costs.[179] If greed is a quality which can be attributed to the mineowners it was in respect of their determination to "get every ounce of gold which is in the ground out of it".[180] It was not, as their antagonists believed, that low dividend payments were the effect of an unduly high retention of profits by the mining houses.[181]

Clearly the success of the mineowners' commitment to low grade, deep level mining depended on their

ability to produce gold at minimal costs. As the majority of items attributed to capital costs were fixed - only a few were capable of adjustment - mineowners had to make concerted efforts to reduce working costs. The intensity of their efforts to effect working cost reductions is illustrated by the following quotation. Although it was written in 1906, it expressed sentiments which applied equally in the period before the Anglo-Boer War:

> As is now well recognised, every shilling reduction in working costs brings into the sphere of practical mining hundreds of thousands of tons of low-grade ore which, without reduced costs, cannot be worked at a profit; and every shilling reduction in working costs means, if the reduction is not obtained at the cost of sound mining, a corresponding addition to dividends. Increased dividends would, no doubt, prove an effective argument with European capitalists, great and small, in favour of South African investment, and thus the money and minerals would be here for an enormous expansion in Transvaal industry - an expansion, the extent of which will be determined only by the level to which working costs can be reduced.[182]

Financial considerations prompted the establishment in the 1890s of nine major gold mining groups to which the vast majority of the mines was attached.[183] Overtrading in shares and speculation in both property and shares had caused investors to sustain huge losses during the period 1890 to 1891.[184] Such losses damaged the stock market at a time crucial for the inauguration of scientific mining in 1892. Therefore one of the aims of the group system, pioneered by Rand Mines Ltd in 1893, was to stabilise the market for future investments, particularly in the

deep level mines, and to attract outside funds especially from Europe.[185] In the long term the group system effectively cushioned low returns from its poorer producers. As average grades varied from mine to mine some were on average rich, while others were poor - the group system enabled the richer mines of the holding company to subvent the "low-grade mines", as the poorer mines were often generically termed.[186]

An additional aim of the group system was to rationalise management and administration. By providing their subsidiaries with a pool of technological and administrative resources each group was able to secure savings in capital and development costs.[187] Significantly, too, centralisation resulted in substantial reductions in working costs. As bulk buyers, the groups effected cost savings for each mine under their control.[188] Consequently both Rand Mines and Consolidated Gold Fields claimed in 1907 that their subsidiaries were collectively worked at a lower cost per ton than those of any of the other groups.[189]

The policies of the groups were frequently shaped by the consulting engineers,[190] whose influence and power is a neglected area of historical research. Also, the appointment of company consulting engineers forged an important communicative link between the mine managers and the mineowners.[191] The important role that consulting engineers played in implementing preventive measures against silicosis needs to be stressed. For instance, in 1911 Richard Barry, the

manager of the Nourse, had to obtain the approval of the consulting engineers of the Rand Mines before he was allowed to institute his sytematically designed watering system.[192] Despite its success and government commendations, neither the other mines in his group nor those of other groups adopted his system universally. The reason was its relatively high installation and maintenance costs.[193]

Likewise, in 1915 Barry encountered "personal abuse" from several consulting mining engineers, including James G. Lawn and Charles D. Leslie of Consolidated Goldfields.[194] As a member of the Miners' Phthisis Prevention Committee, Barry presented a report in which he advocated the universal adoption on the mines of the single-shift system of blasting, namely blasting only once a day, at the end of the second shift, so enabling the dust to settle. Although "75 per cent" of the committee agreed to abolish multi-shift blasting,[195] because of the considerable expense involved, the entire industry, influenced by the consulting mining engineers, did not voluntarily implement the recommendation.[196] Eventually the measure was enforced by mining regulations enacted in 1917.[197]

The prime underlying reason for the reconstitution of the Witwatersrand Chamber of Mines in 1889, after its weak start two years earlier, was also the containment of costs.[198] Although its progress was disrupted by superficial divisions

amongst the mining houses, by 1897 the Chamber had successfully obtained the whole-hearted support of the major mining companies.[199] The Chamber had no financial interest in any of the mines and its services were sustained purely by member subscriptions.[200] Similarly, the costs of additional Chamber ventures were often underwritten by its members on a *pro rata* basis.[201] The Chamber always strove to achieve uniformity of employment conditions for the workforce of all its affiliates.[202] But it could not enforce executive decisions on dissident members.[203] As Alan Jeeves has shown, the Chamber of Mines lacked the power to establish centralised control of African labour recruitment: throughout the period 1902 to 1914 the Witwatersrand Native Labour Association - the WNLA - a subsidiary organisation of the Chamber, did not achieve the monopsony it sought.[204]

Another instance of the Chamber's impotence in regard to the implementation of executive decisions was its failure to get full member compliance in introducing safeguards against silicosis. A prime example was the rejection by most of the Chamber's affiliates, on grounds of costs, of its recommendation to disallow promiscuous blasting.[205] In 1917 in-shift blasting was eventually abolished, but only as a result of state intervention and enforcement by mining regulation.[206]

The Chamber was therefore not as powerful as the public tended to believe. The government of the South African Republican, for instance, fearing that the Chamber would become an *imperium in imperio*, refused to allow it to become an incorporated organisation.[207] But the Chamber's non-interference with the managerial autonomy of the subscribing mines and its detachment on issues which might have prompted inter-mining house competitiveness,[208] promoted and sustained the popular support by its members.[209] Its greatest strength, both locally and overseas, was in the sphere of public relations.[210] The Chamber's propagandist role was, in fact, one of the most useful services it performed for the mining houses to which it was responsible. And it did an excellent job.

It is therefore all the more surprising that writers, who are taken seriously, should still misapprehend so fundamentally the Chamber's role. Belinda Bozzoli in her study, *The Political Nature of a Ruling Class: Capital and Ideology in South Africa 1890-1933*, discusses, in A. Gramsci's terms, the role of "the organic intellectuals":

> [their] task was to bridge the gap between economic interests and political and ideological realities; and to translate the abstract needs of capital into real social needs.[211]

In the Transvaal they ran the "media of capital" and "as journalists, speechmakers or writers, played an important intellectual role in guiding and leading

certain of the processes of class formation themselves".[212] Bozzoli claims further, "As the mining revolution got under way, so its organic intellectuals took on the roles of strategists and ideological vanguard.";[213] they were the "policy-makers for the emerging bourgeoisie".[214]

The facts controvert Bozzoli's argument. These people were not, in fact, the initiators of opinion. They were little more than propagandist hacks, who spoke and wrote as the Chamber wanted. Of their own independent role there was little sign. For instance, E. P. Rathbone, singled out by Bozzoli as the archetypal "organic intellectual",[215] was a faithful punter of whatever happened to be the Chamber of Mines' current line. Rathbone regularly attended the monthly meetings of both the Transvaal Institution of Engineers and the Chemical, Metallurgical and Mining Society of South Africa. Therefore he reported in the media the prevailing policy and opinions of consulting engineers and mine managers.[216] Also, he was a subscribing associate member of the Chamber.[217]

In implementing its decisions the Chamber had the support of the Association of Mine Managers, a powerful organisation whose influence has been insufficiently documented in the gold mining literature.[218] Founded in July 1893, with time this body grew in strength. By the end of the 19th century the Association consisted of representatives of all the groups and most of the mines affiliated to the

Chamber. Its close relationship with the Chamber was emphasised in 1898 by its inclusion of an additional constitutional objective: "To promote the interests of the Mining Companies represented by the Members."[219]

The Association's monthly meetings were well attended; and the executive which met more frequently, often once a week, was highly efficient. The Association did not plan the day-to-day running of the mines, which was considered to be the prerogative of individual mine managers.[220] Like the Chamber the Association's desire for general uniformity on the mines required the standardisation of many practices and procedures, a number of which were closely tied to working costs.[221]

Decisions taken by the Chamber needed the ratification of the Association of Mine Managers for their successful execution. It should also be noted that many of the schemes which have been credited to the Chamber of Mines or to organisations under its direct control, such as the WNLA, were, in fact, initiated and planned by the Association of Mine Managers.[222] For instance, in 1904 the initiative for improving the conditions under which black mineworkers were transported to the Witwatersrand came from the mine managers.[223] Similarly, in 1908 the suggestion of the Association that a convalescent home for silicotic miners be established led to the Chamber's decision to build and maintain the Springkell Sanatorium - it was opened in 1911 - on land donated by the government.[224]

More often than not the Association's undertakings, which subsequently received the Chamber's approval, were designed to cut working costs. Although, many of these cost-related plans may seem more staid than the Association's more grandiose schemes, invariably they had significant and often long-term implications. These projects, for instance, included the abolition of overtime rates for white mineworkers and the drawing up of wage schedules for black mineworkers.[225] In 1896 the mine managers' revised waged schedule, implemented in 1897, effectively reduced African workers' earnings by 33 per cent.[226] As the Association had no wish or power to interfere with the autonomy of mine managers, individual mines wer not obliged to carry out precautions devised by the Association for preventing silicosis. When mine managers neglected to introduce certain preventive measures, they often did so because their implementation would have involved their mines in additional working costs. Of course there were a number of indifferent mine managers, the so-called "laggards".[227] But there were certainly also many interested managers who were "prepared to do more in the spending of money",[228] but who were caught in the cross-fire between their governing boards and their own concern to observe both the spirit and the letter of the mining regulations.[229]

We have now established that the different levels in the hierarchical structure of management - the

mineowners and directors, the boards of the groups, including the consulting engineers, and the mine managers - were concerned with and committed to cutting working costs. These sentiments were expressed through the collective, "powerful" and "authoritative voice" of the Chamber of Mines. This body through its public relations arm shaped these views and made them palatable to the public through the press, including its annual reports and the newspapers which it controlled or made deliberate efforts to court and influence.[230]

This study will show that, despite investors' relatively long times of waiting for their return on capital from the deep levels, the capital costs for the outcrop mines were almost on a par with those of the first and perhaps the second row of deep level mines. Also, during this period the working costs of both the outcrop and the deep level mines were virtually identical. Accordingly, the following analysis will help correct the fallacy that the deep level mines, because of their higher working costs, particularly in development, were more dangerous as dust producers than the outcrop mines.[231] Both kinds of mines provoked equivalent amounts of silicosis, whether in their chronic and accelerated forms. This was because management's efficiency drives, which were linked to their cost-cutting exercises, created high silica levels on both kinds of mines. The outcrop mines generally had shorter lives than those of the

deep level mines. But until 1914, as we have shown, the outcrop mines continued to be significant gold producers.

From 1892, the year that saw the introduction of scientific mining, working costs were associated with the following processes in the cycle of gold production on the Witwatersrand: excavating and removing the conglomerate, hauling it to the surface, breaking the rock and crushing it to powder and finally extracting the gold by the amalgamation and cyanide processes. In 1897 the principal items included under working costs were white labour, black labour, explosives and coal. Coal and explosives were the two smallest items: when combined they barely constituted one-third of the total wage bill.[232]

In its attempts to reduce these expenses management focused its efforts far more on costs incurred in the underground operations of ore excavation, its removal and haulage to the surface,[233] than on the surface procedures of crushing, milling and refining the gold. Of course economies were also effected on the surface. These were achieved largely in two ways: first, by the process of sorting, introduced during the late 1890s; and second, by the innovation in 1904 of tube mills. Sorting by hand labour took place after the gyratory crushers had broken the excavated rock in pieces; and in this procedure workers separated the banket from the barren rock so that only the gold-bearing rock would be

subsequently crushed to powder by the mills.[234] After 1904 the introduction of mechanised tube mills further powdered the banket after its initial milling.[235] Consequently by 1905, according to mining directors, surface costs had been pared to the minimum.[236] But throughout the period from 1892 until 1914 underground working costs remained high; and in management's view there was greater scope and flexibility for their reduction than in surface activities.[237] Another important reason that management directed its attention to underground working costs was that they were inflated by a number of nominal capital costs.[238]

Shaft sinking was categorised as a capital cost. But after a mine had begun producing, further advancements in the one or two shafts were charged to other underground expenses and so included in working costs.[239] This occurred more frequently with outcrop mines than with deep level mines. The vertical shafts of the deep level mines were sunk into barren ground, whereas the inclined shafts of the outcrop mines closely followed the reef. This meant that in the case of outcrop mines production could invariably begin as soon as ground had been broken. Profits were reinvested and paid for the advancement of the shaft and other development work at lower levels. This did not mean that inclined shafts were cheaper in the long run than vertical shafts.[240] In fact, the reverse was true: it was cheaper to sink a vertical shaft than an inclined shaft.[241] We can therefore conclude that both

types of shaft sinking incurred short-term and long-term as well as direct and indirect financial advantages and disadvantages.[242]

Nor were deep level shafts necessarily longer and therefore more costly than the inclined shafts of the outcrop mines. Inclined shafts were frequently relatively lengthy and, in fact, often exceeded the length of the vertical shafts of the first and second row deeps. By 1897 the inclined shaft of the City and Suburban, for example, had already attained 2 000 feet and was being advanced yet further.[243]

It has been incorrectly argued that the capital costs of the deep level mines were higher than those of the outcrop mines. One reason for this contention is the incorrect premise that the outcrop mines had fewer shafts than the deep level mines.[244] In fact, the opposite was common. Nor was each deep level mine obliged to possess a minimum of *two* shafts.[245] This was certainly not true even with mines worked at depths of 4 000 feet.[246] In terms of regulations based on the precedent of coal mines in Germany, which the British administration retained in the Transvaal after the Anglo-Boer War, all mines were obliged to have not two shafts but two *outlets*.[247] Consequently, to obviate the expense of sinking more than one shaft, both outcrop and deep level mines, wherever possible, connected their shafts with those on adjacent properties.[248] Another form of capital-cost saving relating to the deep level mines and characteristic of

the Witwatersrand, was the construction of "compound" shafts. These were located on the boundaries of adjoining properties and the expenses of such joint shafts were shared by two separate companies.[249]

Shafts were considered to be permanent fixtures of the deep level mines and constituted a portion of the capital account. Unlike outcrop mines, which often began producing before their shafts had been completed, this rarely occurred with deep level mines. Consequently management concentrated on locating, planning and constructing their shafts with care to reduce the capital costs. But later modifications in the size and direction of the vertical shafts were frequent and costly.[250] Such expenses, as in the case of outcrop mines, were then charged to working costs.

Development costs, including those for drives and cross-cuts, were also nominally capital costs.[251] As with shaft sinking the policy of the mineowners was to amortise these development costs as soon as possible. Therefore after a mine had begun producing, development costs were also charged to working costs.[252] Clearly the development of a mine did not stop after it had begun producing, as several historians incorrectly state.[253] Development and production took place simultaneously on both the outcrop and the deep level mines.[254] As with the outcrop mines, it was essential for the deep level mines to reach the production stage as soon as

possible. Hence once the vertical shafts had been sunk, invariably only enough development work was completed to allow ore excavation to start. But the commitment of the mineowners to keep their mills at full capacity, obliged them to open up more and yet more production faces,[255] which demanded maximum additional development during the initial production phase.

This frenzied form of "speeding up", as we shall later see, was a major source of complaint to miners, who claimed that too little development work was done before production started. They contrasted this form of "hustle", with its accompanying high dust levels, with the more leisurely conditions which prevailed in development on metal mines elsewhere.[256] But in the context of working costs it is important to note that all these extra development costs were charged to the mine's working account. Consequently management, in attempting to reduce working costs, also had to cater for a large share of development costs, which were the direct result of the industrialists' expedient system of capital costing.[257]

The nominal capitalisation of the first and second row deeps was undoubtedly higher than that of the outcrop mines:[258] shaft sinking and initial development costs, rather than expenses for plant and equipment, account for these cost disparities. But this does not mean that the real capital costs for the same two major items were lower in the case of the

outcrop mines. It is beyond the scope of this study to make an in-depth comparison of the capital, development and working costs of the outcrop mines and the first and second row deeps. It may be added, however, that if the historical materialists wish to sustain their arguments about the complicity of certain mineowners and groups in promoting the Jameson Raid and the Anglo-Boer War, they would be better served by the rigorous use of similar quantitative data, rather than by the more impressionistic evidence which they have thus far provided.[259] For our purposes, however, these economic data clearly indicate that the mineowners' policy of amortising capital and development costs greatly increased the working costs of both these kinds of mining enterprises on the Witwatersrand.

From the start of scientific mining the problem of working costs was crucial for management. In 1897 the four principal items, as has been noted, constituted the wages for black and white mineworkers - each was a discrete item - at approximately 27 per cent and 28 per cent, the cost of dynamite at 12 per cent, and the cost of coal, including carriage charges, at 11 per cent.[260] As dynamite and freight charges were monopolies,[261] management, in frequent memorials to the Kruger government and in their evidence to the Industrial Commission of 1897, justifiably contended that the price of dynamite and coal could be reduced by approximately 50 per cent.[262]

The Volksraad assented only to the lowering of railway freight rates. But as these charges minimally reduced the price of coal, this concession did not mollify the mineowners.[263] The Republican government's consistent refusal to terminate the dynamite monopoly, even after the Industrial Commission had supported their views in recommending its abolition,[264] also reinforced the Randlords' dissatisfaction.

As management could not curtail the prices of coal and dynamite, it took steps to reduce wage costs. Barring social and economic constraints, these cost items appeared more amenable to management's control. Although in 1897 black mine employees outnumbered whites by approximately six to one, their separate wage bills were almost on a par,[265] a phenomenon which we will discuss later when we analyse the patterns of labour organisation. In 1897 the reduction of Africans'' wages by one-third in the revised schedule was successful in depressing the wages of this section of the workforce in the long term.

But cutting the wages of black mineworkers was only one blade of management's shears. The other cutting edge was improved white worker productivity. In attempting to prune its wage bill management invoked both blades. The measures were integral to each other, as Hennen Jennings, consulting engineer

for Rand Mines, explained:

> What we require from both black and white labour is greater efficiency, which, if readily obtained, renders rate of wages a secondary consideration...Of course we desire to get the unit of wage as low as is consistent with the contentment of labour.[266]

The competence with which management mobilised the efficiency of its white underground workforce, particularly miners, by the practice colloquially known as "speeding up" successfully offset the high wage bill of this segment of mineworkers. But the miners paid a heavy price for their increased productivity. The exchange for "speeding up" was the increased prevalence of chronic silicosis, which was more rapidly progressive than elsewhere, and the occurence of accelerated silicosis, an even more serious form of the disease. Contemporary health officers with typical insensitivity justified this sacrifice of miners' health by their paternalistic attitude; they claimed that miners were "ignorant" of the risks attached to mine work.[267]

Notes

[1] *Mining Journal*, 26 Aug. 1893, p. 943, "Gold Fields of the British Empire".

[2] *Cornubian*, 4 July 1902, "Mining".

[3] Cd. 2091, 1904, p. 26.

[4] *Report of the Miners' Phthisis Commission, 1902-1903*, Appendix A, Table E.

[5] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 2, q. 3, evidence of Dr F. Napier. See also Cd. 2091, 1904, p. 25, for supportive evidence.

[6] Collis, p. 10.

[7] *The Mining Industry*, 1897, p. 176, evidence of R. Barrow.

[8] Oliver, *Diseases of Occupation*, p. 292.

[9] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 108, qq. 873-874, evidence of T. McIsaac.

[10] D. B. Barton, *Essays in Cornish Mining History*, v. 1, p. 15. See also Jenkin, pp. 239-241.

[11] D. B. Barton, *Essays in Cornish Mining History*, v. 1, pp. 14-15. See also TG 2, 1908, p. 309, q. 2 889, evidence of S. S. Crowle. He indicated that boys in Queensland started work on the surface and were permitted to go underground only when they reached the age of sixteen. After 1872 boys in Cornwall were not allowed to work underground before the age of twelve. They were permitted to do a ten-hour underground shift only after reaching the age of fourteen. See D. B. Barton, *Essays in Cornish Mining History*, v. 1, p. 55.

[12] Collis, p. 2.

[13] Letcher, pp. 78-80.

[14] Oliver, *Diseases of Occupation*, p. 279.

[15] *Final Report of the Mining Regulations Commission*, v. 2, p. 240, evidence of Dr L. G. Irvine; *Transvaal Leader*, 10 and 17 Aug. 1909, both articles entitled "Capital and Labour".

[16] Irvine et al, p. 6. See also Payne et al,

p. 5.

[17] For an example of official histories, see *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 3. For an example of popular histories, see Cartwright, *Doctors of the Mines*, p. 136. For an example of academic histories, see Grey, p. 300.

[18] *GMEAR...30 June 1902*, p. 7.

[19] *GMEAR...30 June 1902*, p. 8.

[20] *TMJ*, April 1908, pp. 219-220, editorial.

[21] Praagh, p. 272.

[22] *South African Mines, Commerce and Industries*, 24 Nov. 1906, p. 251, "What shall we do with our derelict miners?"; Praagh, pp. 271-272.

[23] *MJSA*, Feb. 1919, p. 374, "Hospital Statistics".

[24] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 24, q. 132, evidence of Dr W. G. Rogers; UG 19, 1912, p. 12, par. 20.

[25] See, for instance, *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 24, 26, qq. 131, 161, evidence of Dr W. G. Rogers.

[26] This is also used as substantive evidence by the health officers who compiled *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 3; by Payne et al, p. 3; and by Grey, p. 300.

[27] J. S. Marais, pp. 186-195 passim.

[28] *The Mining Industry*, 1897, p. iv.

[29] Cammack, *Class, Politics and War: A Socio-Economic Study of the Uitlanders of the Witwatersrand, 1897-1902* , pp. 37 ff; *The Mining Industry*, 1897, pp. 15, 172-178, 299-302, evidence of G. Albu, R. Barrow and A. B. Fyffe.

[30] *The Mining Industry*, 1897, pp. 299-302, evidence of A. B. Fyffe.

[31] *The Mining Industry*, 1897, pp. 172-178, evidence of R. Barrow.

[32] Payne et al, p. 2.

[33] Jeeves, "Aftermath of Rebellion - The Randlords and Kruger's Republic after the Jameson Raid", p. 109.

[34] For details on the Rand Mine Workers' Union, see Katz, *A Trade Union Aristocracy*, p. 22; and Grobler, p. 10.

[35] *TCMAR*, 1897, p. 272, table showing "Distribution of White Employees".

[36] For biographical details on Fyffe, see Ticktin, p. 81; and *South African Mining Journal*, 22 May 1897, supplement, "The Industrial Commission".

[37] *The Mining Industry*, 1897, p. 299.

[38] CAD, MNW, file MM 1215/1910, minute, R. N. Kotze to H. W. Smythe, 23 May 1910.

[39] *Final Report of the Mining Regulations Commission, 1910*, v. 2, p. 240, evidence of Dr L. G. Irvine.

[40] Irvine et al, p. 4.

[41] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 24, 26, qq. 130-131, 163, evidence of Dr W. G. Rogers.

[42] *Report of the Miners' Phthisis Commission, 1902-1903*, p. 119, q. 1 085, evidence of J. M. Hodge.

[43] BRA, HE, v. 258, file 154M, T. J. Britten to President of the Executive of the TCM, 18 June 1906; Cope, p. 36.

[44] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 24, 110, qq. 135, 928-929, evidence of Dr W. G. Rogers and G. Blight; *GMEAR...30 June 1902*, p. 8; *South African Mines, Commerce and Industries*, 5 March 1903, pp. 1 147 -1 148, "Future of the Rock-Drill".

[45] See, for instance, BRA, HE, V. 258, file 154M, S. S. Jago to F. Oats, 13 July 1902, R. G. Nesbitt to Fo. Oats, 15 July 1902, F. Hiehens [sic] [Hichens], and A. E. Pennewan [sic] [Permewan] to F. Oats, 21 July 1902.

[46] Du-Val, p. 23.

[47] *Mining Journal*, 26 Aug. 1893, p. 943, "Gold Fields of the British Empire".

[48] *Lancet*, June 1904, pp. 1 610-1 611, "The Present State of Medical Practice in South Africa: A Warning".

[49] *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 140, evidence of Dr L. G. Irvine; *JCMMS*, Dec. 1906, "Safety Measures in Mining", p. 173, reply to discussion.

[50] *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 140, evidence of Dr L. G. Irvine; *JCMMS*, Jan. 1907, p. 230, "Miners' Phthisis on the Bendigo Field".

[51] Browne, p. 296.

[52] Kerr, p. 2.

[53] Truscott, p. 1. Of course the strike was later discovered to extend a further fifty miles and more. See, for example, Letcher, pp. 66-67.

[54] Denny, pp. 1-3.

[55] Hammond, p. vii. 100 Cape feet is equivalent roughly to 103,33 English feet.

[56] Pollak, p. 71; Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, p. 82.

[57] Pollak, p. 71; Goldmann, *South African Mines*, pp. 101-102.

[58] Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, pp. 1-484 passim.

[59] Kerr, p. 2.

[60] Truscott, pp. 11-12.

[61] Hatch and Chalmers, pp. 15-126; Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, pp. 72, 146; *The Mining Industry*, 1897, p. 203, evidence of H. Jennings.

[62] For evidence of the use of dynamite in 1888 in open quarries, see Du-Val, p. 14.

[63] Denny, p. 107.

[64] *The Mining Industry*, 1897, p. 203, evidence of H. Jennings; Gray and McLachlan, p. 376.

[65] *The Mining Industry*, 1897, p. 203, evidence of H. Jennings; Gray and McLachlan, pp. 375-376. Before 1891 the estimated value of the tailings on the Jumpers Mine was 10 dwts per ton. See Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, p. 107.

[66] *Report of the Council of the Association of Mine Managers*, 1893, "Presidential Opening Address".

[67] *Report of the Council of the Association of Mine Managers*, 1893, "Presidential Opening Address"; Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, pp. 1-289 passim.

[68] Coombe, p. 38.

[69] Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, pp. 1-271 passim.

[70] *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 3; Rosenthal, p. 344; Cartwright, *Doctors of the Mines*, p. 136; Grey. p. 300.

[71] Irvine et al, p. 5. See also Payne et al, p. 5.

[72] Irvine et al, p. 5.

[73] Watkins-Pitchford, "The Industrial Diseases of South Africa", p. 38.

[74] I thank Mr M. J. Martinson, a former senior lecturer in mining engineering at the University of the Witwatersrand, Johannesburg, and President of the Mine Ventilation Society of South Africa in 1960, for the information.

[75] BRA, HE, v. 258, file 154M, no. 18, memorandum, "Mining Regulations Committee" signed by Dr L. G. Irvine, 1910; SC 4, 1914, p. 460, qq. 459-460, evidence of Dr S. V. van Niekerk.

[76] Union, *Statutes*, 1916, no. 44, section 42(1).

[77] Payne et al p. 5.

[78] Martinson, p. 13.

[79] *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 3.

[80] Watkins-Pitchford, "The Industrial Diseases of South Africa", p. 39; *GMEAR...30 June 1902*, p. 8.

[81] *Report of the Miners' Phthisis Commission, 1902-1903*, pp. 2-3, 17-18, qq. 3, 46-49, evidence of Dr F. Napier and Dr L. G. Irvine; Irvine, pp. 221, 223.

[82] See, for instance, *Lancet*, 29 April 1905, p. 1 144, "Public Health in the Transvaal".

[83] *JCMMS*, Dec. 1908, "Reminiscences of the Early Rand", p. 204," discussant J. S. MacArthur.

[84] Goldmann, *The Financial, Statistical and General History of the Gold and other Companies of Witwatersrand, South Africa*, pp. 1-271 passim.

[85] Gray and McLachlan, p. 375.

[86] Praagh, p. 593, describes the mercury treatment.

[87] *JCMMS*, Dec. 1908, "Reminiscences of the Early

Rand", p. 204, discussant J. S. MacArthur; Gray and McLachlan, p. 375.

[88] *The Mining Industry*, 1897, p. 203, evidence of H. Jennings.

[89] *JCMMS*, Dec. 1908, "Reminiscences of the Early Rand", p. 204, discussant J. S. MacArthur.

[90] *JCMMS*, Dec. 1908, "Reminiscences of the Early Rand", p. 204, discussant J. S. MacArthur; Goldmann, *South African Mines*, v. 1, p. xviii.

[91] *JCMMS*, Oct. 1908, "Reminiscences of the Early Rand", p. 124, discussant W. A. Caldecott.

[92] Gray and McLachlan, pp. 375-383 passim; *The Mining Industry*, 1897, p. 203, evidence of H. Jennings.

[93] Gray and McLachlan, p. 375.

[94] *Report of the Council of the Association of Mine Managers*, 1893, "Presidential Opening Address".

[95] Goldmann, *South African Mines*, v. 1, p. xiv; Kerr, p. 2.

[96] *The Mining Industry*, 1897, p. 203, evidence of H. Jennings.

[97] Letcher, p. 109.

[98] Goldmann, *South African Mines*, v. 1, p. xiv; Hatch and Chalmers, p. 73.

[99] Taylor, p. 41.

[100] Hatch and Chalmers, pp. 98-100. The sinking of boreholes began as early as 1889. They were positioned close to the southern boundaries of outcrop claims and intersected the reef at shallow depths. See *South African Mines, Commerce and Industries*, 9 July 1904, p. 395, "Evolution of Rand Mining".

[101] Pr ıagh, p. 516; Letcher, pp. 109-110.

[102] Rosen, p. 156.

[103] Hatch and Chalmers, pp. 104-105.

[104] Truscott, p. 453; Goldmann, *South African Mines*, v. 1, p. v.

[105] Hatch and Chalmers, pp. 57-67 passim; Denny, pp. 4-14 passim.

[106] Letcher, pp. 373-374.

[107] *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 255, evidence of Dr L. G. Irvine.

[108] TCMA, file N8, H. F. Marriott to Secretary of the TCM, 2 July 1904. See also Phillips, p. 77.

[109] *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 255, evidence of Dr L. G. Irvine; Cd. 7478, 1914, p. 162, q. 24 160, evidence of H. F. Marriott.

[110] *JCMMS*, Dec. 1906, "Safety Measures in Mining", p. 165, reply to discussion. These temperatures were recorded by a wet bulb thermometer which measures the evaporating power of the air.

[111] *JCMMS*, Dec. 1906, "Safety Measures in Mining", p. 165, reply to discussion.

[112] Truscott, p. 271.

[113] Truscott, pp. 271, 275.

[114] Praagh, p. 572.

[115] Hatch and Chalmers, pp. 2, 251-253; Praagh, pp. 636-640.

[116] *The Mining Industry*, 1897, p. 7, evidence of J. Hay.

[117] See below, chapter 9.

[118] *Mining Journal*, 26 Aug. 1893, p. 943, "Gold Fields of the British Empire".

[119] Denny, p. 29.

[120] Denny, pp. 28-29; Truscott, p. 3, and diagram facing p. 100.

[121] Praagh, p. 592.

[122] Denny, pp. 28-29.

[123] *JCMMS*, Oct. 1908, "Reminiscences of the Early Rand", p. 124, discussant W. A. Caldecott.

[124] Hatch and Chalmers, p. 217.

[125] *Report of the Council of the Association of Mine Managers*, 1893, "Presidential Opening Address".

[126] Cammack, *Class, Politics and War: A Socio-Economic Study of the Uitlanders of the Witwatersrand, 1897-1902*, p. 9; Moroney, p. 5.

[127] Hammond, p. vii.

[128] *The Mining Industry*, 1897, p. 244, evidence of T. Leggett; Petit, p. 335; Truscott, pp. 151, 153.

[129] Praagh, p. 572; Pollak, p. 10.

[130] Pollak, p. 10.

[131] Browne, p. 291.

[132] Petit, p. 335, estimated the average depth of the third row of deeps to be between 4 500 and 5 500 vertical feet at their shallowest points.

[133] Hammond p. vii. In 1905 Petit, p. 335, estimated their average vertical depths, on the northern boundaries of the claims, to be between 1 500 feet and 3 000 feet.

[134] Denny, pp. 31, 33; Grey, p. 69.

[135] *GMEAR...30 June 1905*, p. 13.

[136] Hatch and Chalmers, p. 97.

[137] Goldmann, *South African Mines*, v. 1, p. 102. See also ibid., pp. 1-484 passim, for many other examples of shafts of outcrop mines which had vertical depths of 800 to 1 000 feet.

[138] Denny, pp. 28-29.

[139] Frankel, *Capital Investment In Africa*, p. 85, table 13.

[140] Richardson and Van-Helten, "The Development of the South African Gold-Mining Industry, 1895-1918", p. 322, have not avoided the pitfall.

[141] *GMEAR...30 June 1905*, p. 8.

[142] TG 2, 1908, p. 234, q. 2 312, evidence of G. Albu.

[143] Kennedy, *A Tale of Two Mining Cities*, p. 51; Payne et al, p. 5. By stressing that scientific mining techniques applied only to deep level mines, many historians have underrated the short-term importance of the outcrop mines. See, for instance, Richardson and Van-Helten, "The Gold Mining Industry in the Transvaal", p. 25.

[144] Pollak, p. 4.

[145] Calculations based on Hatch and Chalmers, pp. 29-35 passim, p. 86; and Goldmann, *South African Mines*, v. 1, pp. 1-484 passim.

[146] Denny, p. 32; Praagh, p. 394.

[147] Praagh, p. 572; Goldmann, *South African Mines*, v. 1, pp. 90-96. Similar mixed mines also occurred on the West Rand and on the rich cental outcrop section. See Praagh, pp. 566-573 passim.

[148] Goldmann, *South African Mines*, v. 1, pp. 37-49; Hatch and Chalmers, p. 29.

[149] Blainey, pp. 352-354.

[150] Kubicek, "The Randlords in 1895: A Reassessment", pp. 101-102.

[151] Despite his appreciation of the practicalities of mining Levy, pp. 104-108 passim, upholds Blainey's thesis; Levy views the outcrop and the deep level owners as distinctive and competitive fractions of capital. Bozzoli, *The Political Nature of a Ruling Class*, p. 35, also stresses the differences between the owners of outcrop and deep level mines.

[152] *Mining Journal*, 26 Aug. 1893, p. 943, "Gold Fields of the British Empire".

[153] Kubicek, "Finance Capital and the South African Gold Mining Industry 1886-1914", p. 393, Table 1.

[154] Jeeves, "The Administration and Control of Migratory Labour on the South African Gold Mines: Capitalism and the State in the Era of Kruger and Milner", pp. 5-6.

[155] TG 2, 1908, pp. 235-236, qq. 2 318-2 321, evidence of G. Albu.

[156] *Mining Journal*, 26 Aug. 1893, p. 943, "Gold Fields of the British Empire".

[157] Cartwright, *Gold Paved the Way*, p. 84; Kubicek, "The Randlords in 1895: A Reassessment", pp. 85-89 passim.

[158] Wilson, *Labour in the South African Gold Mines 1911-1969*, pp. 39-40.

[159] Wilson, *Labour in the South African Gold Mines 1911-1969*, p. 36.

[160] Van-Helten, pp. 531-532.

[161] Hatch and Chalmers, p. 7. See also Wilson, *Labour in the South African Gold Mines 1911-1969*, p. 40, for a clear explanation of the "pay limit".

[162] Kennedy, *A Tale of Two Mining Cities*, p. 8; TG 2, 1908, pp. 1 496-1 500, qq. 21 631-21 676, evidence of W. W. Mein.

[163] *Mining Journal*, 23 July 1893, p. 810, "Progress on the Rand Goldfields".

[164] Fraser and Jeeves, pp. 185-186, L. Phillips to F. Eckstein, 2 March 1908.

[165] TG 2, 1908, p. 254, statement of S. J. Jennings.

166 Jeeves, "The Administration and Control of Migratory Labour on the South African Gold Mines: Capitalism and the State in the Era of Kruger and Milner", pp. 6-8.

167 Denny, pp. 50, 52, 114; *East Rand Express*, 12 Aug. 1911, "Brakpan Mines, Ltd."; Praagh, p. 570.

168 Grey, p. 433; Bleloch, p. 34; Richardson, *Chinese Mine Labour in the Transvaal*, pp. 12, 18, 21.

169 Taylor, p. 125.

170 Bleloch, p. 34.

171 The historian, Blainey, p. 359, also seems to have been deceived by the mineowners' propaganda.

172 TG 2, 1908, p. 1 469, q. 21 172, evidence of F. D. P. Chaplin.

173 Praagh, pp. 566-579 passim; Fraser and Jeeves, L. Phillips to F. Eckstein, 2 March 1908.

174 *Mining Journal*, 23 Dec. 1893, p. 1 423, "Gold Mining in South Africa"; TG 2, 1908, p. 103, q. 697, evidence of L. J. Reyersbach.

175 Frankel, *Gold and International Equity Investment*, pp. 17-20 passim.

176 Cf. Grey, p. 68, who assumes that investors were satisfied with their returns.

177 Kubicek, *Economic Imperialism in Theory and Practice*, pp. 26-29.

178 Frankel, *Gold and International Equity Investment*, p. 20.

179 See, for example TG 2, 1908, pp. 93-103, evidence of L. J. Reyersbach.

180 TG 2, 1908, p. 113, q. 855, evidence of L. J. Reyersbach.

181 For a full discussion of this belief, see Katz, *A Trade Union Aristocracy*, p. 29. For supportive evidence, see also TG 2, 1908, p. 1 478, statement of E. J. Moynihan.

182 *South African Mines, Commerce and Industries*, 2 June 1906, p. 253, "Leading Article".

183 Praagh, pp. 602-603, 606.

184 Another major cyclical depression occurred in 1895-1896. See Kubicek, "The Randlords in 1895: A Reassessment", pp. 88-89.

185 Kubicek, "The Randlords in 1895: A Reassessment", p. 95.

[186] Cartwright, *Gold paved the Way*, p. 110.

[187] Jeeves, "The Administration and Control of Migratory Labour on the South African Gold Mines: Capitalism and the State in the Era of Kruger and Milner", pp. 3-4.

[188] Levy, p. 28; Letcher, p. 412.

[189] TG 2, 1908, p. 1 318, evidence of C. J. Price; Cartwright, *Gold Paved the Way*, p. 112.

[190] See for instance, Merriman Papers, correspondence, S. Evans to JXM, 18 June 1912.

[191] Bozzoli, *The Political Nature of a Ruling Class*, p. 52, fails to appreciate the important communicative channel between consulting engineers and the mineowners. Incorrectly she claims that mine managers were the sole communicative link between the mines and the mineowners.

[192] Merriman Papers, correspondence, R. Barry to JXM, 15 Dec. 1911; Jourdan, pp. 148-151.

[193] Merriman Papers, correspondence, R. Barry to JXM, 1 Aug. 1915; *JSAIE*, Oct. 1911, Jan. 1912, "The Prevention of Dust in Development Drives of Mines during Drilling Operations", pp. 57-58, 139-140, discussants J. B. Roberts and K. Austin. For details, see below, chapters 11 and 12.

[194] Merriman Papers, correspondence, R. Barry to JXM, 2 Nov. 1915.

[195] Merriman Papers, correspondence, R. Barry to JXM, 2 Nov. 1915.

[196] Merriman Papers, correspondence, R. Barry to JXM, 16 Feb. 1916.

[197] *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 269. For details, see below, chapter 12.

[198] Lang, p. 42.

[199] Jeeves, "Aftermath of Rebellion - The Randlords and Kruger's Republic after the Jameson Raid", p. 102.

[200] Lang, pp. 42-43.

[201] See, for example, TCMA, file M3(d), circular letter, 25 Jan. 1910, requesting contributions for the capital costs of the Springkell Sanatorium.

[202] Grey, p. 63.

[203] Grey, p. 185.

[204] Jeeves, *Migrant Labour in South Africa's Mining Economy*, pp. 12-15.

[205] TCMA, file M27, circular letter 24/13, 5 March 1913; Merriman Papers, correspondence, R. Barry to JXM, 20 Nov. 1915.

[206] *The Prevention of Silicosis on the Mines of the Witwatersrand*, 1937, p. 269; Merriman Papers, correspondence, R. Barry to JXM, 16 Feb. 1918. For details, see below, chapter 12.

[207] Grey, p. 57, quoting D. A. Etheredge, "The Early History of the Chamber of Mines, 1887-1897", unpublished MA dissertation, University of the Witwatersrand, Johannesburg: 1949, p. 32.

[208] Lang, p. 43.

[209] Grey, p. 52.

[210] It is beyond the scope of this study to analyse the apparent "detachment" of the Chamber. Certain historians commend the Chamber for this quality. See, for instance, Lang, pp. 85, 186, 221; Grey, p. 277; and Cartwright, *Golden Age*, p. 12. Others, particularly Katz, *A Trade Union Aristocracy*, pp. 394-397, view it as being spurious in the context of controversial social and political issues.

[211] Bozzoli, *The Political Nature of a Ruling Class*, p. 10.

[212] Bozzoli, *The Political Nature of a Ruling Class*, p. 10.

[213] Bozzoli, *The Political Nature of a Ruling Class*, p. 46.

[214] Bozzoli, *The Political Nature of a Ruling Class*, p. 10.

[215] Bozzoli, *The Political Nature of a Ruling Class*, pp. 26, 34, 35, 48, 99.

[216] See *JSAIE*, 1902-1913 passim; and *JCMMS*, 1902-1913 passim.

[217] See *ARTCM*, 1902-1914 passim.

[218] This brief survey, unless otherwise noted, is based on the Council and Monthly minutes of the Association of Mine Managers, 1901-1914.

[219] *Report of the Council of the Association of Mine Managers*, 1898, p. 4.

[220] Jeeves, "Aftermath of Rebellion - The Randlords and Kruger's Republic after the Jameson Raid", p. 108.

[221] On the need for uniformity see, for

instance, Council minutes of the AMM, 14 April 1902.

[222] See, for instance, Monthly minutes of the AMM, 15 Feb. 1904. Most writers who have investigated the gold mining industry on the Witwatersrand have neglected the influence of the Association of Mine Managers. Jeeves, "Aftermath of Rebellion - The Randlords and Kruger's Republic after the Jameson Raid, p. 107, is an exception. He hints at its importance but does not, however, explore the organisation in depth. A detailed investigation of the Association and its links with both the Chamber of Mines and the WNLA is needed.

[223] Monthly minutes of the AMM, 18 Jan. 1904.

[224] Council minutes of the AMM, 5 March 1908; Monthly minutes of the AMM, 13 Sept. 1908; *Rand Daily Mail*, 20 Nov. 1911, "Miners' Phthisis". For details, see below, chapter 12.

[225] On the abolition of overtime rates, see *Report of the Council of the Association of Mine Managers*, 1902, p. 5.

[226] Council minutes of the AMM, 26 May 1902; *Report of the Council of the Association of Mine Managers*, 1898, p. 5.

[227] *JCMMS*, Aug. 1906, "Safety Measures in Mining", p. 39, discussant J. M. Johnston.

[228] *Voice of Labour*, 24 May 1912, "At the Vaudeville".

[229] *JCMMS*, Aug. 1906, "Safety Measures in Mining", p. 39, discussant J. M. Johnston. See also Merriman Papers, correspondence, S. Evans to JXM, 18 June 1912. Bozzoli, *The Political Nature of a Ruling Class*, p. 52, overestimates the independence of the mine managers. She claims that during the 1890s they had "the virtual independence of a small businessman himself". For details, see below, chapter 12.

[230] Fraser and Jeeves, p. 118, L. Phillips to J. P. FitzPatrick, 15 Aug. 1902; CHA, WLF, Phillips letter book, no. 11, L. Phillips to F. D. P. Chaplin, 18 July 1912. Cf. Lang, p. 181, who denies that the mining magnates unduly influenced the press. The confidential letters of individual mineowners do not support Lang's view.

[231] This exaggeration is particularly noticeable in Payne et al, pp. 5-6. Also this view is implicit in the superficial survey of Irvine et al, pp. 4-6.

[232] *The Mining Industry*, 1897, p. 47, evidence of J. P. FitzPatric

[233] Kubicek, *Economic Imperialism in Theory and Practice: The Case of the South African Gold Mining Finances, 1886-1914*, p. 46.

[234] Bleloch, p. 19, n. 1.

[235] Fraagh, pp. 594-596; Letcher, p. 387.

[236] TG 2, 1908, p. 233, q. 292, evidence of G. Albu.

[237] Cartwright, *Gold Paved the Way*, p. 106.

[238] Browne, pp. 289-290.

[239] Truscott, p. 190.

[240] Cf. Blainey, p. 352.

[241] Levy, p. 11.

[242] It is beyond the scope of this work to compare in detail the financial advantages and disadvantages of both kinds of shafts. See, for instance, *Rapport van den Staats-Mijningenieur*, 1894, p. 26; and Truscott, pp. 190-193.

[243] Truscott, p. 150.

[244] Blainey, p. 354. See also Truscott, p. 153, who shows that the second row deeps had fewer but larger shafts than the first row of deep level mines and the outcrop mines.

[245] Levy, p. 11.

[246] *Report of a Commission...Mining by Single Outlet*, 1907, p. 108, evidence of H. Weldon.

[247] *Report of a Commission...Mining by Single Outlet*, 1907, pp. 6, 69, 109-110, qq. 41, 853, evidence of M. J. Francke, A. R. Sawyer and H. Weldon.

[248] *Report of a Commission...Mining by Single Outlet*, 1907, p. 108, evidence of H. Weldon.

[249] Letcher, p. 161.

[250] On the New Primrose, for example, at the sixth level at a depth of approximately 800 to 900 feet, the vertical shaft had to be changed to an inclined shaft, called a sub-inclined shaft. For examples of shaft enlargement, see Goldmann, *South African Mines*, p. 288; and Fraagh, p. 571.

[251] Cartwright, *Gold Paved the Way*, p. 12.

[252] Denny, p. 50. See also Truscott, pp. 295 ff.

[253] On the basis of the incorrect assumption that development stops once production begins, Richardson and Van-Helten, "Labour in the South African Gold Mining Industry, 1886-1914", pp. 82-83, argue that the underground white workforce decreased

in relative terms shortly before and after the Anglo-Boer War. Also, numerous official statistical sources invalidate their wrong conclusion.

[254] Truscott, p. 213.

[255] TG 2, 1908, pp. 27-28, qq. 466, 475, evidence of H. Weldon.

[256] See, for instance, Cd. 2091, 1904, p. 25; TG 2, 1908, p. 520, q. 6 100, evidence of E. Moore; *Rand Daily Mail*, 2 April 1912, letter by "X.Y.Z.".

[257] TG 2, 1908, pp. 235, 936, 1 473, qq. 2 318-2 321, 13 882-13 892, 21 230, evidence of G. Albu, G. I. Hoffman and R. G. Fricker. See also Browne, pp. 289-290.

[258] See, for instance, TG 2, 1908, p. 1 473, q. 21 230, evidence of R. G. Fricker. Fricker stressed the heavy capital costs incurred by the Simmer and Jack.

[259] See Mendelsohn, p. 140, and Marks and Trapido, pp. 58-61, who have tried to revive the debate. It should also be noted that Blainey, p. 356, provides no substantive evidence to show that deep levels mines had higher working costs than the outcrop mines. He claims that the deep level mines used far more machine drills and dynamite than the outcrop mines. Machine drills, as is later shown, were used on a large scale in the outcrop mines in both development and production.

[260] *The Mining Industry*, 1897, p. 47, evidence of E. J. Way. The estimates of J. Hay, chairman of the Chamber of Mines, and J. P. FitzPatrick, a director of H. Eckstein and Co, were similar, but their figures for the cost of black labour were lower: both give estimates of 23 per cent. See also ibid., pp. 8, 47, evidence of J. Hay and J. P. FitzPatrick. There was general consensus on management's estimates for white labour at 28 per cent. See, for instance, *Mining Journal*, 8 May 1897, p. 661, "Leading Article".

[261] J. S. Marais, pp. 27-45 passim.

[262] *The Mining Industry*, 1897, pp. 38-39, evidence of E. J. Way. See also Grey, pp. 370-374. Grey shows that by December 1903, under the administration of Milner, the price of dynamite had been halved since 1899.

[263] J. S. Marais, p. 192.

[264] J. S. Marais, p. 192.

[265] Figures derived from *TCMAR*, 1898, p. 292, tables, "Distribution of White Employees at 31st December" and "Distribution of Natives".

[266] *The Mining Industry*, 1897, p. 219, evidence

of H. Jennings.

267 *Final Report of the Mining Regulations Commission*, 1910, v. 2, p. 240, evidence of Dr L. G. Irvine.

CHAPTER 5

THE INDUSTRIAL YEARS 1892-1899

> "Undoubtedly the formation of companies with very big capitals for development of deep underground levels has affected the working of the miners. . A process of 'speeding up' has been instituted in order to keep the mills going and earn sufficient profits to pay the promised dividends. In American factories "speeding up" has increased the number of accidents, and it has probably had the same effect on the mines. There is no time to take adequate precautions. The men have to work at express speed and their health suffers. The policy operates from top to bottom."---T. Bottomley, 1904.[1]

> "It is needless to point out that this industry [the Witwatersrand gold mines] in the past has been built up by the employment of unskilled native labour in combination with skilled white labour, in the ratio of approximately nine natives to one white man."---Transvaal Chamber of Mines, 1902.[2]

In 1893 an article in the *Mining Journal* enthusiastically endorsed the widespread introduction of mechanised rock drills on the Witwatersrand mines. In commending the mineowners for their "series of achievements" the journalist singled out the

"extensive employment of power transmission". This had facilitated "the general displacement of manual labour by rock drilling", a step which he considered as important as the general adoption of the cyanide process.[3] But during the 1890s, when the proponents of mechanised drilling applauded the increased productive power of rock drills, they were apparently oblivious of the machines' capacity to harm their operators. In retrospect they stated: "The story of the drills covers a good deal of the story both of our output of rock and our output of silicosis."[4]

This chapter examines the introduction of rock drills on the Witwatersrand and traces their history and use until 1899 in both the outcrop and in the deep level mines. In so doing the study shows the enormous scale of mining on the Witwatersrand and the accelerated pace of the output of rock. The development and production methods caused heightened dust levels with an accompanying increase in the incidence and prevalence of both chronic and accelerated silicosis.

Three factors account for the revenue of Witwatersrand mines: first, the grade of ore milled; second, the price of gold; and finally, and most significantly in this context, the quantity of ore hoisted and milled.[5] The mineowners soon realised that they would have to work their mines on a very large scale to make the low grade ore pay.[6] Consequently mass production became their target. Mass production

was partly facilitated by the introduction of rock drills. But the mineowners also achieved their objective by their methods of mobilising the underground workforce. This chapter therefore analyses the labour-saving strategies and the labour-intensive practices of the mineowners, both of which contributed to the occurrence of the disease which we are studying.

Mass production on the Witwatersrand gold fields is usually associated with the period from 1902 to 1914, when most of the second row of deep level mines became fully operational. During this period the mineowners did, indeed, intensify their efforts to mass produce; they widely publicised their strategies in order to procure their capital requirements. But it is important to stress that the foundations and structures of mass production had been solidly laid and built before the Anglo-Boer War began. Unless this fact is grasped, the huge crude prevalence of silicosis, which became manifest in 1901, will remain incomprehensible. During the days of Kruger's republic the "speeding up" of mine development and ore production generated enormous dust densities which provoked the occurrence in the underground workforce, and amongst white miners in particular, of this slow-developing lung disease in both its chronic and accelerated forms.

The productive propensity of the industry was measured by its tonnage, that is the amount of ore

daily broken, hoisted and milled. It will be shown that the use of rock drills in development, in particular, enabled the Witwatersrand mines to generate their huge tonnage, which brought them world-wide acclaim. We shall first analyse the mechanisation of development on the mines, which was one of the routes management took in order to achieve mass production.

Management saw no practical need for using pneumatic, or air-driven, power drills to mine the soft friable banket in the free milling zone. But once pyritic rock had been struck, management seriously considered the advantages of rock drills.[7] Towards the end of 1889 the Jumpers Gold Mining Company pioneered their introduction on the Witwatersrand, and soon afterwards the Robinson and the Langlaagte Estates followed suit.[8] The initial rock drill units were modest in size. Each unit consisted of a small air compressor which ran one to four drills and cost approximately £3 563.[9] Once their efficacy had been proved, larger compressor plants which could handle eight to twenty drilling machines were installed.[10]

Even so, between 1889 and 1892 machine drills were imported on a very small scale, although most producing mines installed mills during this period.[11] By 1892 less than 10 per cent of approximately 140 registered companies owned rock drill plants. These companies included former producing mines, which had

been liquidated or had temporarily stopped working because of the slump, as well as mines which were current producers.[12]

Towards the end of 1892, after the systematic exploitation of the pyritic zone had begun, mechanised drills became standard equipment on developing and producing mines. By 1895 over 90 per cent of working mines owned rock drill plants;[13] some of the compressors were large enough to run forty rock drills at a time.[14] Also, the number of operational rock drills continued to grow: for the years 1896, 1897, and 1898 the number of rock drills in average annual use was 1 013, 1 351 and 1 850.[15]

It should be stressed that the extensive use of rock drills not result solely from the development, from 1892, of the first level deeps, as numerous historical accounts incorrectly suggest.[16] The prosperity of the outcrop mines was equally dependent on them.

Apart from their high capital costs, rock drills were expensive to maintain. Also, their working costs, in terms of their power supply and their consumption of explosives, were far higher than those of hand drills manned by black workers.[17] But the extra development which rock drills accomplished both offset their additional costs and promoted the profitability of the mines.

The mineowners viewed the need for the rapid development of both the outcrop and the deep level mines in the pyritic zone as being exceedingly important, as development paved the way for the excavation of the banket. Through development, that is the driving of horizontal and vertical tunnels through barren rock, the ore-bearing reefs were exposed. The places at which the underground tunnels exposed the pyritic reef were called the faces. The faces were then drilled and dynamited; and the next stage of the cycle was hauling the tons of broken ore to the mills.

Management on the Witwatersrand quickly learned that development by hand labour did not supply sufficient faces quickly enough for ore production to match the capacity of the huge mills. Therefore both before and during production rock drills were extensively deployed on both the outcrop and the deep level mines to expedite development. Their use in development ensured that more and more ore-bearing rock was laid open underground so that it could be speedily excavated and milled.

From 1892 until the outbreak of the Anglo-Boer War in 1899 the demands of the mills grew. In 1894 over two thousand stamps were continuously dropping. In 1894 the crushing of more than 2 375 000 tons of gold-bearing banket yielded approximately two million ounces of gold, which had an estimated value of

£7 000 000.[18] This single-year production was equivalent to one-third of the total output of the preceding six-year period, from 1887 to 1893.[19] As a gold producer in 1892 the Transvaal, after Australia, the United States and Russia, was ranked fourth in the world.[20] In 1894 the production from approximately fifty mines raised the Transvaal to third place in the world ranking and to first place in the following year.[21] In 1895 the Witwatersrand, as the world's top gold producer, yielded one-fifth of the globe's total annual output.[22]

The Witwatersrand did not attain its primacy as a producer solely because of it geography, namely the concentration of so many gold fields in a single region. A less obvious reason, but an equally important one, was management's institution of particular mass-production techniques which caused development and production on the Reef to be more rapid than in any other hard rock mining region.[23] By September 1899, on the eve of the Anglo-Boer War, the 6 000 working stamps[24] of seventy-eight producing mines[25] were, on average, pounding approximately 28 000 tons of ore per day.[26] Tourists to Johannesburg were astounded by the noisiness of the stamps; and one such visitor observed:

> Beyond the town are the mines from which there comes an increasing murmur, growing on nearer approach to a dull roar, as of the sea, the sound of the batteries crushing the ore.[27]

Mineworkers would have disagreed with the tranquil

connotations of the writer's simile: even at night the "incessant roar" of the mills disturbed the workers.[28] For miners, in particular, the thundering stamps epitomised the "speeding up" effected on the mines.

The daily milling in 1899 of so many thousands of tons of banket depended, as we have seen, on the development of enough faces for the excavation of gold-bearing rock. Management could not have achieved this magnitude of production, since 1892, in so short a period, without the large scale deployment of rock drills in development.

To meet the demands of the mills the pace of production was also quickened. In fact, the average tonnage excavated and hauled to the surface exceeded by 16 to 18 per cent the tonnage finally milled.[29] This was due to a proportion of the waste rock being discarded in the cost-saving surface procedure of sorting. Hand sorting took place before crushing in an attempt to provide the mills with only gold-bearing rock.[30]

Management, for many cost-related reasons, preferred to use hand labour rather than machine drills in ore excavation, or stoping.[31] But during 1894, when there was a shortage of African workers for manning the hand drills, management used rock drills to supplement hand drills in the stopes in order to maintain production levels.[32]

Irrespective of the width of the reef, stoping was always intricate, as each stope had "an individuality of its own".[33] But machine drills of the 19th century were unsuited to the complexities of the narrow reefs that predominated in the Witwatersrand mines.[34] Also, when used in narrow stopes these rock drills proved to be far more costly and hazardous than hand drills.[35] Management therefore always insisted that the substitution of rock drills for hand drills in the stopes was only a temporary expedient;[36] and it clung to this view until 1910.[37] But throughout the period before the Anglo-Boer War African hand-drill labour always fell short of the demand,[38] so obliging management to increase the numbers of machine drills in stoping. Indeed, in 1898 the State Mining Engineer of the South African Republic stated in his annual report that machine drills had become the chief means of stoping.[39]

This did not mean that hand drilling had been discontinued entirely: at least 20 per cent of stoping was still done by hand, particularly in selected narrow reefs which had a high gold content.[40] Therefore despite its additional costs and other work-related disadvantages, by 1899 "speeding up" had become the norm in production too. As in development, by 1900 production by machines was also responsible for provoking the occurence of accelerated silicosis, a feature which many medical historians tend to overlook or play down.[41]

Many popular historians are also responsible for perpetuating fallacies concerning the Witwatersrand gold mining industry. In romanticising the industry's pioneers, such historians praise their foresightedness in mechanising the industry, but fail to mention the adverse implications inherent in the mechanisation process. Likewise, many official mining-house histories are simply public relations exercises. Although such surveys purport to be based objectively on mining-house archival records, they do, in fact, invariably suppress data which are inconsistent with their writers' predetermined view of the industry's upward and smooth technical progression.[42] They gloss over or ignore entirely, as do most popular histories, one of the disfiguring historical features of the industry - the mortality of miners from silicosis.

In contrast, the autobiographical and subjective narrative of the industry by William Taylor, the diamond and gold mining pioneer, does not suffer from this defect. In 1932, in reviewing the role of rock drills in the growth of the Witwatersrand gold mining industry, Taylor, the brother of one of the early gold mining house directors, James B. Taylor,[43] explained both their significance and malignance to the industry:

> Sixty miles of the reef resounded with the constant rat-ta-tat of machine drills, and the hard conglomerate ore melted, like ice in the spring, before the weapons which science had brought into play against it.
>
> Unhappily the new methods at first exacted a ghastly price in human sufferings and death

> from the speeding-up they brought. With each rapid stroke of the machine-drill there was thrown into the air an impalpable powder, largely consisting of infinitesimal but sharp fragments of silica. This dry dust pervaded the mines, coated the walls of the drives, the trucks and the woodwork, coloured the faces and clothes of the workers, and entered their lungs. It produced a fell disease [among white miners] known by the name of Miners' Phthisis or Silicosis.[44]

Taylor's identification of the occurrence of silicosis has several important implications. In brief, his chief arguments are: first, rock drills caused silicosis; second, by implication the incidence of the disease was highest among rock drillers; third, all stoping was done by rock drills; and fourth, white miners, rather than African migrant mineworkers, contracted the illness. Taylor's review of the growth of the gold mining industry does not shirk detailing the morbid consequences of rock drill work. But his analysis of the causes of the disease and the liable intermediary instruments is based on the generalisations of contemporary commentators during the early years of the 20th century.[45] As stereotypes, Taylor's insights, therefore, have a number of inherent fallacies.

Let us examine his first premise. Taylor, like many other analysts, blames rock drills for producing silicosis. But dust, not the machines, causes the disease. We must therefore avoid the pitfall of transferring the liability for silicosis to rock drills, so incorrectly promoting machines as disease producers. If we are to understand why rock drills became dust producers we need to examine carefully the

way they were operated and the conditions in the mining zones where they were handled.

In certain respects Taylor's second observation is valid. He correctly shows that rock drillers were more vulnerable to silicosis than other underground workers. This was confirmed in 1937 by reliable statistics based on incidence data which had been compiled since the inauguration of the Miners' Phthisis Medical Bureau in 1916. Despite the industry's growing vigour, particularly after 1916, in implementing important dust precaution measures, the 1937 figures showed that 70 per cent of the current silicotic victims had been machine drillers.[46] But, as this 1937 statistic also illustrates, nearly one-third of the incidence of silicosis could not be accounted for by the use of rock drills. Additional factors must, therefore, be analysed to explain why such a relatively large proportion of the underground workforce, who had not operated rock drills, nevertheless contracted the disease.

Taylor's third argument that machine drills were exclusively used in stoping is incorrect for the period before World War 1: his simile "and the hard conglomerate ore melted, like ice in the spring" contains too much poetic licence. As we have seen, by the time of the outbreak of the Anglo-Boer War machine drills accounted for approximately 80 per cent of stoping. After the war, and particularly between 1904 and 1910, the ratio of machine-drilled faces to

hand-drilled faces dropped: during this period at least 30 per cent to 40 per cent of stoping was done by hand.[47]

Between 1902 and 1912 the number of machine drills in use did, indeed, increase in absolute terms.[48] But wherever it was practicable, management used hand drills in stoping.[49] Apart from a brief period of scarcity of African contract labourers, from 1901 until 1904, which the introduction of indentured Chinese workers alleviated, from 1904 to 1914 there was virtually no shortage of non-white mineworkers.[50] They were available in sufficient numbers to wield the less costly hand drills, which now replaced machine drills in many of the stopes. Also, after 1904 management deployed hand drills in reserves which it had developed before the Anglo-Boer War, but had not mined then, because of cost. Such reserves included both reefs which were complex and narrow and those which had exceedingly low grades.[51] Although hand drills replaced machine drills in many stopes, during the period 1905 to 1912 the number of rock drills used in both production and development was sufficient to sustain the high prevalence of accelerated silicosis.

To promote a better understanding of the weaknesses in Taylor's arguments the organisation of the workforce must be analysed. Such analysis will clarify the role of machine drills as dust producers and help explain why rock drillers engaged in development were more prone to silicosis than their

counterparts in stoping. It will also show that additional categories of underground mineworkers and other kinds of miners, including hand stopers, also contracted the disease. By unravelling these complexities we will be in a stronger position to assess objectively Taylor's last proposition. It followed the consensus of contemporary medical opinion, which asserted that "the incidence of uncomplicated silicosis is very decidedly lower in the native than in the European labour force".[52] Such a medical view seems to be incongruous. But its apparent absurdity can be better understood if we examine the origins, composition and deployment of the workforce on the Witwatersrand gold fields.

When systematic mining was inaugurated on the Witwatersrand, the deployment of labour, as contemporaries observed and as historians have since confirmed, largely followed the precedents of "deep diamond mining" at Kimberley.[53] Many Kimberley mining directors provided both the bulk of the initial finance capital and the original management for the gold mining industry. Following their successful experience at Kimberley the Randlords applied to the gold mines a labour system which had already proved effective.

This transfer of labour patterns from the one industry to the other was not an automatic decision based on the coincidence of mutual capital and ownership: engineering and economic principles were

equally important in governing the industrialists' choice. Both the underground diamond mines and the outcrop and deep level gold mines were designed according to univeral underground vein-mining practices. Therefore both at Kimberley and on the Witwatersrand the mines had similar labour requirements. Also, in both these mining centres the labour arrangements, which so closely resembled one another, were economically shaped by the composition, costs and availability of the labour resources in southern Africa.

By 1887 the original alluvial diamond diggings, established during the early 1870s, at the four Kimberley mines, had altered radically. Under the monopoly of the De Beers Consolidated Mines they were transformed into an underground mining industry with expensive mechanised equipment.[54] This was not a sudden development. After alluvial quarrying had been largely abandoned, there was a relatively lengthy transitional period: from 1875 to 1884 open deep-pit mining predominated; and later, from 1884 to 1887, these open-pit mines co-existed with a small number of underground mines.

At Kimberley one of the basic requirements for both the deep pits and the underground mines was a skilled labour force. As South Africa had only a small pool of artisans and a tiny complement of professional miners, the diamond industrialists offered high wages to attract skilled mineworkers from

overseas.[55] Trained artisans were required to install, operate and service the machinery; and the expertise of professional miners was needed for the more sophisticated development and production requirements of the new forms of mining. Most of these skilled mineworkers came from the United Kingdom. In contrast to the artisans who came from all parts of Britain, the miners emigrated largely from Cornwall and Cumberland, where tin and iron-ore mining was declining.[56]

Unlike the case of artisans, the initial demand for miners was small: they were required only as timbermen to shore up the walls of the deep pits.[57] The demand for miners increased during the early 1880s: some of the industrialists increased production in the deep pits; and from 1884 others started to construct more complex underground mines. It must be stressed that the increased demand for miners was barely influenced by the application of technology: until the late 1890s at Kimberley the mechanisation of the mining processes themselves was minimal. Apart from a handful of rock drills used for driving tunnels, both development and production in the underground diamond mines were almost entirely labour intensive.[58]

The money wages of the skilled mineworkers, which ranged from £6 to £8 per week, were substantially higher than those paid in Britain. But because of the high cost of living in Kimberley, in real terms these

wages were barely sufficient for the subsistence of married men.[59] Also, the wages of skilled white workers were markedly higher than those of unskilled African mineworkers: in the late 1880s the wages of recruited black labourers averaged between 18s. and £1 10s. per week.[60]

It could be argued plausibly that the wage disparities between white and black mineworkers at the Kimberley mines was the white mineworkers' possession of skills. But this was not consistently so, as the case of the white overseers illustrates. The overseers, most of whom were former claim holders,[61] played an important role in production at the quarries and in the open pits.[62] When the numerous diamond claims became concentrated in the hands of a few owners who pursued capitalist methods of production, the former independent diggers were from economic necessity obliged to become wage-earners.[63] As the vast majority of diggers lacked artisanal or mining skills, unskilled work on the mines was their only alternative.[64] But they were not employed as unskilled labourers. Instead, they were promoted to supervising black labourers who performed the unskilled physical work.[65]

As *employers*, the diggers had been prepared to perform unskilled physical tasks in the company of their black employees.[66] The white prospectors did not regard the concept of unskilled physical work *per se* as being distasteful. Rather, they viewed manual work

as being "degrading" only when they performed it as *wage-earners*. In this capacity they regarded their standing as having been reduced to a level which they considered suitable only for non-white people, whom they perceived as being biologically and racially inferior to themselves. Also, the unskilled wage rates, customarily paid to non-white labourers, did not enable the former diggers to conform to socio-economic standards, which social convention in South Africa held to be appropriate for whites. Such views were not peculiar to the diggers. Indeed, they were shaped by, and so conformed to, the value systems of most contemporary white South Africans.[67]

A tangled mesh of psychological and socio-economic variables accounted for the prejudice of white unskilled wage-earners towards executing unskilled physical tasks in the same workplace and in the company of unskilled non-whites.[68] While the economic competition of non-white workers was an undeniable threat to these white workers, many of whom were of low socio-economic status, it was not the sole reason for their prejudice. Displaced aggression and personality needs carried equal weight in shaping their prejudice, as did the need to conform to existing social norms.[69] Consequently former claim holders - and later Afrikaners, who had been dispossessed of their small farmholdings - in adjusting to their changed status from independent entrepreneurs to unskilled wage-earners, clung tightly to customary

white value systems which disdained the performance by whites of menial wage-earning tasks. But the pretensions of whites to superior categories of work and to standards of living higher than those of non-whites needed justification; and a simplistic version of Social Darwinism, which had become the conventional wisdom in the 19th century, provided them with a suitable rhetoric.

The idea that whites were different from and socially and economically superior to non-whites had gradually taken root in South Africa: the idea was part of the legacy of the 17th century Calvinist doctrines and of the Cape's relatively lengthy experience as a slave-owning colony. After the middle of the 19th century notions of white racial supremacy were further bolstered by the pervasiveness and influence of Social Darwinism, a socio-cultural theory of evolution, with its rider of biological determinism. Its attachment to natural laws, associated with Darwin, gave it a spurious legitimacy. This enabled its contemporary - and present-day[70] - proponents to support class stratification based on colour and to justify racial discrimination on the grounds that these inequalities were scientifically determined.[71]

Also, the resort to Social Darwinist rhetoric by the diamond mining magnates - and later by the Randlords - enabled the industrialists to justify the disparities in their employment and treatment of black

and white mineworkers. To a considerable degree they were also able to satisfy the British Colonial Office that many of the exploitative practices inherent in the cheap black labour system did not run counter to the British state's avowed principles of duty and trusteeship towards the African societies under its governance.[72] This accord between the British government and the mining industrialists is not surprising: ironically, the ostensibly commendable aims of the Colonial Office also had strong social Darwinist connotations that were used to justify the British state's imperialist policies.[73]

Management, particularly on the gold mines, had many reasons for preferring to employ cheap black labour rather than cheap white labour on the mines, and these are discussed later. But had management been explicit about these reasons, it would probably have harmed the industry's image amongst many of its professed and potential supporters, including shareholders.[74] Also, such frankness would have further intensified the hostility of the industry's antagonists, including white mineworkers. Instead, management placated public opinion by resorting to the plausible theory of Social Darwinism.[75] The remarks in 1904 of Hennen Jennings, the London-based consulting engineer of H. Eckstein and Company, illustrate the soothing impact of this theory's rhetoric on persons of white descent:

> The history of this country [the Transvaal], as well as that of others where white and

> black are thrown together, clearly proves that the white is the superior race mentally, and that the black should recognise it. In other words it is the natural attitude of the white man to feel his superiority, and relegate to the black the lower grade of manual work...
>
> Thus, to obtain the maximum population, intelligence and contentment, work among the whites must be confined to skilled departments where brain tells, and the mere muscular work apportioned to races willing to be considered inferior, and to work cheerfully for wages far below the scale required by the white populat to support their families in the condition of affairs obtaining in this country.[76]

Management on the diamond fields did not employ unskilled whites as manual labourers. Instead, it promoted them to be overseers. But these actions, which appeared to be consistent with their professed Social Darwinist views and so also with contemporary white social norms, overshadowed the industrialists' economic motives. One of the employers' major problems was to minimise, if not to eliminate, diamond thefts which led to profit losses. A the industrialists held whites, particularly former claim holders, to be innately more trustworthy than blacks,[77] a notion entirely compatible with Social Darwinist thinking, they placed unskilled white workers in charge of production in the quarries and in the open pits.[78] In this capacity each white overseer supervised approximately ten black manual labourers who dug up the blue rock with picks and shovels.[79] Ostensibly the overseers ensured that the black labourers performed their work. But this was not the overseers' main function. Rather, they acted as policemen guarding against diamonds being stolen.[80]

Even though the jobs they supervised had no skilled content whatsoever, the overseers were paid a semi-skilled wage,[81] namely approximately two-thirds of the earnings of skilled mineworkers. Clearly the high earnings of skilled workmen from overseas strengthened the established wage differences between black and white wage-earners.[82]

After management had modified its methods of production, the redeployment of unskilled whites as nominal semi-skilled overseers continued, but in different kinds of work in which racially discriminating theories again reinforced economic self-interest. By the late 1870s many open-pit mines were being worked at depths of 300 to 400 feet.[83] Therefore to speed up and cheapen production, customarily accomplished by laborious pick and shovel methods, a few industrialists introduced drilling and blasting techniques.[84] As the overseers lacked these skills, they could not supervise African hand drillers. Instead, some overseers were redeployed as supervisors of Africans engaged in shovelling and other ore-removal jobs.[85] Like their previous tasks, these new supervisory jobs also had no skilled content. Even so, this reduced body of overseers continued to be paid at semi-skilled wage rates, presumably because employers were reluctant to offend common prejudice by employing whites at non-white rates.

The significance of the overseer arrangement needs to be stressed because the supervisory system was transferred to the professional miners, but not to the artisans. This occurred first at Kimberley, with the introduction of the techniques of ore drilling and blasting in the open pits and in the underground mines, and later on the Witwatersrand, where miners were required to supervise Africans who operated hand and rock drills. But the promotion of miners to be supervisors, ironically, jeopardised their claims to exclusiveness as a skilled class of workers. Through supervising drilling the skill content of the miner's task was fragmented. As a consequence both his job classification and his skilled wage packet were threatened by the encroachment of newly qualified semi-skilled black drillers who commanded unskilled wages. Likewise, the job security of the nominal semi-skilled overseers - they, like miners who supervised hand drills, were called gangers on the gold mines - was also precarious owing to the competitive threat posed by the black workforce.

On the Witwatersrand both kinds of supervisors were caught in a similar pincer-like movement. They felt threatened by their industrialist employers, who naturally wished to reduce their wages, and on the other hand by the potential competition of lower-paid Africans.

Clearly professional miners had an identity of interests with the unskilled overseers on the diamond mines and the unskilled gangers on the gold fields, one which historians exploring worker consciousness on the South African mines have failed to identify. The precarious job security of both these categories of underground white workers is one of the reasons that skilled miners collaborated with the overseers, and not with the artisans, in forming a short-lived trade union on the diamond mines in 1884.[86] Afterwards on the gold mines, when both these categories of workers shared yet another danger, that of contracting silicosis, they once again joined forces - in the industrial-wide miners' strike of 1907.[87]

The supervisory system was crucial to the deployment of the underground labour force of the Witwatersrand gold mines. But as has been established, its origins were on the Kimberley mines. Its initiation was made possible chiefly by the composition of the workforce. Another reason that professional miners could be promoted to be supervisors hinged on the nature of mining in underground vein mines, such as those that existed in Kimberley and on the Witwatersrand. A brief description of the general design of an underground vein mine will further an understanding of the essential features of supervision. Also, once we have a knowledge of such a mine's layout, we will be able to assess later why some underground tasks and zones

had a greater liability for silicosis than others.

On a mining property one or two shafts were sunk to intersect the ore at depth. In the case of outcrop mines inclined shafts followed the lode, whereas the vertical shafts of the deep level mines were directed at striking the ore on its underside.[88] From the shafts, at levels of approximately 150 feet, horizontal tunnels, called drives, were drilled and blasted through barren rock to reach the mineral. The drives were then connected with one another at intervals of approximately 300 feet by yet another group of underground passages called winzes.

Winzes can be likened to underground shafts and, likewise, two of their functions were to prepare the ground for excavation and to provide ventilation.[89] Winzes were vertical or inclined tunnels; and the ore was frequently mined on either side of them in an upward or downward direction until the next level was reached.[90] Winzes could be dropped from the upper to the lower drives, a procedure called sinking; and when they were elevated from the lower to the upper levels they were termed "raises". By blocking out the mine in these development operations the ore was exposed at the face enabling it to be excavated.

Production of the ore-bearing rock then began. The sites where the tunnels intersected the reef were called the stopes; and during stoping, the operation used to cut steps or notches in the face, the

ore-bearing rock was released. This process was effected by boring holes with hand tools or machine drills which were then blasted.[91] But once production had begun, development did not stop. Blocking out continued to lower and lower depths to facilitate as many faces as possible being worked simultaneously.[92] After stoping, the broken ore was shovelled into trucks - as late as 1946 draught animals were also occasionally used on the Witwatersrand gold mines[93] - which conveyed it along the drives to the shafts, a procedure called tramming.[94] On reaching the shafts the ore was shovelled or tipped into containers, or bins, which were then hauled to the surface.[95] In brief, stoping and the removal of the ore to the shafts comprised the production processes.

Shaft-sinking was probably the most taxing task for a miner as it required his all-round ability and ingenuity.[96] But after this stage in mine development had been completed, the skilled miner still had to display his prowess in other development procedures and in stoping. Apart from performing unskilled physical tasks, including tramming and shovelling in the drives, winzes and stopes, he applied himself to the semi-skilled manual jobs of drilling and blasting. Also, he did mechanical semi-skilled tasks: he ran the pumps; he fitted water pipes; he laid the plates for the tramming trucks; and he timbered the shafts, drives, winzes and stopes.[97] By the time a miner had mastered all these skills he could, indeed,

be regarded as an expert or as a skilled miner; and most overseas metal miners, with sufficient experience, could lay claim to this title.

As the underground mine of De Beers at Kimberley was based in 1887 on a design used for the mining of haematite in Cumberland,[98] the following representation of iron-ore mining in Lancashire is apt. James Coward describes the laborious process through which he acquired his skills as a miner and also shows the complexities of his calling. In one respect, however, the haematite miner's job differed from those of diamond and gold miners. Because the country rock in the British haematite mines was more friable than the hard rock at Kimberley and the gold mines,[99] the iron-ore miner was obliged to do far more timbering, as Coward notes:

> I had to learn, in the first place, what they call tramming in this country. In the Old Country it is termed "bogeying"...When they see that he is quite efficient and a robust young fellow, they take him out of the bottom levels and put him in the upper workings. When he goes to the upper workings he is generally out on shovelling, and he has to fill the cars up above and tip them into the box for the trammers below. When he has no iron ore to fill, he has to get hold of a pick, the same as his partner, and he has to help to get some more iron ore loose. I want you to understand that mining in the Old Country is altogether different to what it is in this country. It is not solid rock they have to deal with. If you are not careful you would be buried in five minutes, and have the whole concern on top of you. You have to timber there every 18 inches: that is, there are side sticks, what we term in Lancashire "two forks" and a "head tree". When you have got your head tree up, you have to lag it, and when you have got sufficient ahead for another set of timbers, you must put it in. When you

> strike water in this iron ore, it comes in from all directions, not only from the top, but you must look to the sides and lag, and in many cases it will blow your bottom up by the continual rise of the water coming out of the iron ore. There is none of that here. We have perfectly solid rock...Now I would like to give a case, an instance in rising ["raising"] in the Old Country. We drive there, and perhaps we have 100, 200, or 300 feet to rise right up from the surface. We do it all there with pickwork in loose ground. We may come across some rock or clay or hard substance that we have got to blast. I want you to understand these rises are 7 feet 6 inches or 8 feet by 4 feet 6 inches. We have continually periodical rises. If the ground begins to run in on you [collapses], you have to keep your timbering close, and all the time it must be kept close until you get it timbered.[100]

Although drilling and blasting were two accomplishments of a haematite miner, he seldom had to use them in stoping, as Coward corroborated. But the hardness of both the country rock and the lode in the Cornish tin mines made drilling and blasting a daily necessity in production as well as in development. These skills were acquired during a lengthy, but informal, period of training, as the following description illustrates:

> They start their training generally on the surface, either in connection with the machinery, or if on metalliferous mines they start on the dressing floors. They go from the dressing floors to tramming and shovelling, and while they are underground they start in their spare time a little work for the miner until they are put on as helpers. Generally with hand labour a young man is put on with an old miner. He learns to use the mallet and probably does a good deal of the work with the hammer, and the old man turns the jumper, which is for double-handed work. For single-handed work we do the same thing. The young man takes the hammer and drill and he starts in the same way.[101]

Because black mineworkers almost exclusively performed hand drilling in South Africa, many contemporaries and historians have tended to regard this job as being unskilled, requiring no mental application and with average physical demands.[102] As a consequence they have, perhaps unwittingly, caused the skills required for this task to be denigrated. Alfred John Gregory, the retired Medical Officer of Health for the Cape, was a perceptive doctor who refused to accept this stereotyped version of hand drilling. In 1913 he argued:

> You must remember that the work of the underground boys, whatever may be said to the contrary, is very hard, especially hand drilling. One boy, having the knack, will do the task of boring thirty-six inches with ease, but to the average boy, it is exceedingly hard labour. I have spent some time underground, and have watched them working.[103]

Cornish miners would certainly have endorsed Gregory's contention concerning the physically taxing demands of hand drilling. But they would also have stressed its skilled requirements, which the *Cornubian* proudly noted in 1901 in its description of double-handed drilling:

> This requires long months of practice before one can become adept...One man holds the steel drill and another strikes it with a nine-pound sledge hammer, one-eighth of a turn being given between each blow, a mis-hit meaning sometimes a smashed finger or a wrist; but the miners become such adepts, despite the faint light at hand that accidents are rare.[104]

In single-handed drilling the miner directed a pointed solid steel rod, the "borer" - known on the

Witwatersrand as the "jumper" - at the face. He struck it with a heavy hammer, withdrew it a short distance to free it from the broken chips, gave it a slight twist so as to maintain the round shape of the hole, and continued these procedures until he had drilled the hole to its required depth in readiness for blasting. To remove the chips of rock the miner threw water into the hole where it formed a sludge, which he periodically removed with a wooden stick.[105] The hand driller required skilled co-ordination and physical strength to strike with the one hand "many thousands of deliberate and heavy blows" on the end of the borer, and simultaneously with the other hand to steady and twist it.[106]

Initially on the Witwatersrand, as in Cornwall, management permitted both double-handed and single-handed drilling amongst its African workforce.[107] But as management raised the productive output of its black underground labour complement, single-handed drilling became the norm on the gold fields.[108] Double-handed drilling in a single shift produced a hole sixty to sixty-six inches in depth.[109] But in single-handed drilling each black labourer was required daily to bore a hole thirty-six to forty-two inches in depth.[110]

Over and above the possession of dexterity and the physical strength required for such arduous manual work, performed in near darkness and in cramped conditions, hand drillers needed appropriate mental

skills. This knowledge, which was also informally acquired through experience, manifested itself in the expertise with which drillers placed the holes, the direction in which they bored them and in their assessment of explosive quantities.[111] Overseas miners considered this understanding so important that they would not allow a youngster of eighteen or twenty "to attempt to put a hole in unless the elder men of the party were present".[112] When blasted, misdirected holes could distort the shape of the stopes resulting in costly alterations, including retimbering: even worse, they could cause rock falls with accompanying loss of life.[113]

Nor were overseas novice miners allowed to do blasting. They were required to have at least four years' underground experience before being allowed to assist with the explosives.[114] Blasting also required the miner to apply his mental faculties to this task: ill-fired holes could cause costly damage or loss of life through weakening the hanging wall. In this process dynamite cartridges, furnished with a detonator and a fuse, were thrust to the bottom of the holes. After lighting the fuses for the many holes which were simultaneously blasted, the blasters retreated speedily to escape the explosion of the charges which shattered the rock. The acquisition of these mental skills - ones which African mine labourers also gained at an early stage[115] - was an additional prerequisite for a driller-cum-blaster to

be considered an expert.

After rock drills had been introduced in the late 1860s in Cornwall, "it was only a short step to the good combination that is miner and machine man".[116] This adage was true for metal miners all over the world.[117] By the mid-19th century, Victoria in Australia and mining centres in North America had been established for a sufficient length of time for the institution of traditional training methods.[118] As their introduction became more widespread, in these far-flung corners of the globe both local and roving miners had little difficulty in learning to operate the rock drills.[119] When rock drills were originally introduced, they were provided with water jets to cool the machines and to preserve the boring edge of the cutting tool.[120] But as management and miners considered the provision and use of water jets to be costly and time-consuming, hosing was abandoned world-wide, and water was used as wetting only for sludging.[121]

The rock drills of the 19th century were made of solid steel and were fitted on a cradle. The end of the piston, which projected beyond the end of the machine, was enlarged into a "chuck" in which a solid steel drill, or jumper, was inserted and clamped. Activated by compressed air the rock drills worked on a reciprocating principle: the piston rod of the machine drove the jumper against the face of the rock with great force and speed, rotating it between

blows. As the machines, which weighed 200 to 300 lbs, were extremely heavy, two men were required to rig them; smaller machines at a weight of 175 lbs, which were later designed for stoping, were only a trifle less cumbersome.

Two men also had to operate the smaller rock drills: one controlled the air supply and regulated the movement of the drill; and the other changed the jumpers at regular intervals. Drill-changing was a laborious and tedious operation. The blunted jumper had first to be loosened and removed before it could be replaced by its successor, which in turn had to be tightly fastened into the chuck. Despite the time taken to rig, shift and adjust the machines, and the frequent and lengthy intervals which changing the jumpers made necessary, a single machine manned by two operators could drill four holes each six feet deep in a single shift. Such holes, which were deeper and of greater diameter than hand-drilled holes, required more explosives.[122] Even so, the labour-saving capacity of the rock drill, particularly in development, outweighed its capital, maintenance and working costs.[123]

The ability to operate a rock drill fortified the skilled miner's store of accomplishments. As a hand driller, the all-round miner already possessed the requisite knowledge for positioning and directing the holes for the rock drill. Therefore he needed only a short period of training as a chuckman to equip

himself with the mechanical skills necessary to take hold of the drill handle.[124] With this additional expertise he met all the requirements necessary to qualify as a skilled miner according to management's definition of the term: "One who can do any practical work demanded from the collar of the shaft to the bottom."[125]

Practical all-round miners were not, however, required at Kimberley. Instead, the overseas miners used only one of their numerous accomplishments in a specialised capacity. When the jobs of skilled artisans are similarly fragmented, it is usually the result of advances in technology. The introduction of improved mechanical appliances occurred in numerous 19th century - and 20th century - crafts and trades. In the printing trade, for instance, the handicraft skills which the compositor laboriously acquired were not needed for operating the late 19th century mechanised linotype. A worker with a brief period of specific training could operate it. The assignment of the linotype to semi-skilled printers diluted the compositor's monopoly of skills and so rendered part of his craft redundant.[126]

At Kimberley the jobs of professional miners were fragmented, but in quite a different way. The miner's job was not fragmented, or deskilled, as the process is often less precisely referred to, by the introduction of labour-saving machines. Instead, this process occurred through management's employment of

low-paid black labourers who were, in practice, human machines. Although many of the Africans' jobs were, indeed, semi-skilled, the African mineworkers continued to be paid at unskilled wage rates, which they were prepared to accept, given their lack of alternatives.

All the overseas miners were classified as skilled mineworkers. But by segmenting underground work into distinctive categories, management on the diamond mines used only one or two of the miner's all-round skills at a time. Consequently miners almost exclusively performed discrete jobs as semi-skilled operators. As wage-earners, who worked continuously underground, they were collectively referred to as miners.[127] But their designations as developers, stopers, stope-timberers, plate layers, pump minders, and pipe fitters more specifically accorded with their tasks.[128] Like the artisans who worked at the margins of the open pits or on the surface of the underground mines, a tiny category of miners were assisted by two or three unskilled black helpers. This group of miners, known as the specialist pitmen, included timbermen, plate layers, pump minders and pipe fitters.[129] Like the skilled artisans, the specialist pitmen were not supervisors in the same sense as developers and stopers clearly were.

As supervisors, most miners were in charge of gangs of twenty to thirty black workers in shaft

sinking, driving, winzing and stoping.[130] Besides handling personally the explosives and lighting the fuses,[131] the all-round miners were called upon to use their knowledge of boring and blasting in only one other way: they demonstrated the position and direction of the holes which their black charges drilled. "Our duty is to read rocks and split them," was the sage comment in 1902 of a supervisor, or ganger, on the gold mines.[132] When miners at Kimberley were employed under contract they supervised their own ore removal.[133] But miners on day's pay were not responsible for these unskilled tasks. Tramming and shovelling were entrusted to the overseers who, as we have seen, received semi-skilled wages, which were approximately one-third less than those of the professional miners. The overseers, like the miners-cum-supervisors, were in charge of gangs of black workers of similar size.[134]

The gangs of African mineworkers constituted a large portion of the total black workforce, which consisted mainly of migrant labourers who worked for relatively short contract periods.[135] Migrant labour was not new in African societies.[136] Nor was it determined solely by the industrialisation of the diamond mines. Long before diamonds had been discovered in the 1870s at Kimberley, Africans from virtually all the societies south of the Zambezi had, as migrant workers, been offering their labour to white employers in the South African colonies and