LUNG CANCER IN A MINING DISTRICT IN RHODESIA*

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In 1957 I showed that there was an unusually high incidence of cancer of the lung among African mineworkers in the Gwanda district of Southern Rhodesia.[1] It was found over the next 6 - 7 years (January 1957 - November 1963) that this level was being maintained and, in fact, exceeded: that is, a firm diagnosis of cancer of the lung was made in 37 cases, compared with the previous 22 cases in 8½ years.

Relative to the general population, the incidence in 1957 was worked out as being 18/100,000 adult males, and 56·8/100,000 in the smaller population of male mineworkers. To calculate in a similar way the incidence for the 1957 - 1963 period, the Gwanda census figures for 1962 have been used, comprising the data of 61,910 African persons, of whom 15,670 were males born before 1946. This gives an estimate of 33·8/100,000 cases of lung cancer for male adults in the general population. In the case of the male mining population, using figures provided by the Office of the Government Mining Engineer (which total 17,509 man-years for employees in the Gwanda goldmines over the 1957 - 1963 period) the incidence is 205·6/100,000. Both these figures are very appreciably greater than those for the 1948 - 1956 period.

Two factors unconnected with the actual aetiology of the tumours certainly contributed to the difference: a realization that cancer of the lung was not uncommon in the African; and the growth of specialist chest services at Mpilo Central Hospital, Bulawayo, so that cases suspected of having the disease could be sent to be investigated by a specialist team. Of the 37 cases in the second series, 15 were so referred, whereas with the first series the absence of any special facilities in the only large hospital to which patients could be sent resulted in a final diagnosis being made only at postmortem in 20 out of the 22 patients.

It is proposed to re-examine conclusions reached in 1957 about the causes of lung cancer in Gwanda in the light of fresh evidence presented by these 37 new patients.

PARTICULARS OF PATIENTS

These are set out in Table I, together with a summary of the diagnostic criteria. Histological confirmation of the diagnosis, in the majority of instances from postmortem specimens, was obtained in 26 patients. In the remaining 11, diagnosis was by bronchoscopy, thoracotomy, biopsy of secondary growths, or postmortem examination, but histology was not done or the results were mislaid.

There is a further group of 6 patients in whom a diagnosis of cancer of the lung was made by X-ray and tomography. Although in some of these bronchial carcinoma seemed highly probable, they are not included in the series; their particulars are nevertheless appended to the table.

Of the 37 cases, only one was a non-miner. All the others had worked on mines: 32 were so employed at the time of coming to hospital, while 3 of the remaining 4 had other jobs (herdsman, case 8; sweeper at meat factory, case 24; sweeper at cement factory, case 32), and one, case 25, was destitute.

Evidence of Arsenicalism

In 1957 I came to the conclusion that the main factor causing lung cancer in Gwanda was most probably arsenic, which occurs in large quantities in the form of arsenopyrite or mispickel in the Gwanda goldmines west of the 29th meridian.[1] However, skin changes of arsenicalism had not been noted in the patients of the series (although observed and described in a later patient), and a map showing where 10 of the lung cancer patients had worked did not indicate longer periods of employment in arsenic-rich mines—in fact, 5 gave histories of having worked only in mines to the east of Gwanda, normally regarded as arsenic free.

In compiling the present series of lung cancer cases the appearance of the skin of the palms and soles was particularly noted, and careful mining histories were taken.

Thirteen of the 37 patients had hyperkeratoses, and in 6 of them a rash of the trunk having the features of the 'raindrop' pigmentation of chronic arsenicalism was also observed.

The palmar hyperkeratoses could be roughly divided into 'mild', with scanty and scattered small wart-like papular projections, often taking the form of little hard, dry, hyperkeratotic horns; 'moderate', with keratotic papules more numerous, but not coalescent; and 'marked', with papules numerous and on a thickened plaque-like base. The photographs (Figs. 1 - 3) illustrate this reasonably well, with Fig. 1 showing a mild scattering of keratoses, Fig. 2 (case 4) moderate scattering, and Fig. 3 (case 9) marked changes.

As well as being present in 13 cases with carcinoma of the lung, palmar hyperkeratoses were seen in 27 patients suffering from a variety of other illnesses, admitted to Gwanda Hospital during the 1957 - 1963 period. All these patients were miners or had a mining history; and in fact, although looked for in all adult males admitted to the hospital, no palmar hyperkeratoses were at any time found in any patient without such a history.

THE MINING ENVIRONMENT

Fig. 4 is a map of the Gwanda gold-belt, showing the situation of the mines at which the lung cancer patients and the miners with hyperkeratoses had worked. Circles refer to lung cancer patients without hyperkeratoses, squares to those with cancer and hyperkeratoses, and triangles to patients with hyperkeratoses only. A completely blacked-in symbol represents a period worked at a mine of more than 6 years, a half-blacked symbol represents a period of 3 - 6 years, and an unblacked symbol represents a period of 1 - 3 years. These periods were chosen to fit in with suggested development periods for arsenical hyperkeratoses (2½ years according to Fierz, quoted by Schultz[2]) and industrial carcinoma of the lung (6 - 27 years in 6 of Pirchau and Sikl's 9 cases from Schneeburg[3]). It is assumed, rather empirically, that a period of 1 - 3 years in a suitable environment is sufficient to initiate the

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TABLE I. PARTICULARS OF LUNG CANCER PATIENTS

No.	Age	Mining history (× = significant period underground in arsenical mine)	Cig. smokers (No. = amount smoked/day)	Non-cig. smokers	Hyperkeratoses	Method of diagnosis	Site of growth	Histology	Additional diseases	Comment
1	46	×				Postmortem	R main bronchus	Widespread Ca. of bronchial origin		
2	46	×	Home-made			Postmortem	L upper lobe	Anaplastic epidermoid Ca. showing much necrosis		
3	65	×	Bought (4) & home-made			Postmortem	R main bronchus	Epidermoid Ca.		
4	45	Outside mines incl. 2 yrs topaz mine at Gatooma		×	× (mod.)	Postmortem	L upper lobe	Epidermoid Ca.		
5	60	×	Bought (4)			Biopsy through bronchoscope	L middle bronchus	Anaplastic epidermoid Ca.	Stage II silicosis	
6	45	×				Postmortem		Very fibrous epidermoid Ca. of bronchial origin	TB with silicosis	
7	40	×		×		Postmortem	R upper lobe	Epidermoid bronchogenic Ca.		
8	65	×	Bought (4)			Postmortem	R upper lobe	Very anaplastic bronchogenic Ca.	TB with silicosis	
9	40	Surface on arsenical mine (Sabiwa) 20 yrs	Bought (16)		× (marked)	Postmortem	R main bronchus	Small oat-cell Ca.		
10	56	Surface arsenical mine 4 yrs	Bought			Postmortem	L upper lobe	Epidermoid Ca. of bronchus		
11	56	×	Home-made			Postmortem	L upper lobe	Primary bronchogenic Ca. with marked anaplastic change in some areas		
12	?	Non-arsenical mines	Bought (2)			Postmortem	R upper lobe. ?2 primary foci	Anaplastic adenocarcinoma of bronchus		
13	58	×	Bought (8)			Postmortem	R upper lobe bronchus	Anaplastic epidermoid bronchogenic Ca.	Stage I silicosis	
14	50	×				Postmortem	R upper lobe bronchus	Anaplastic or 'oat-cell' Ca.		
15	50			×	× (mod.)	Postmortem	R lower lobe bronchus	Epidermoid bronchogenic Ca.		
16	55	×	Bought (4)		× (mod.)	Postmortem	R lower lobe bronchus	Oat-cell Ca.		
17	58	Outside mines, unknown ore composition		×	× (mod.)	Postmortem	R mid. lobe bronchus	Oat-cell Ca.		
18	50	Mining history mislaid				Postmortem	R lower lobe bronchus	Primary anaplastic bronchial Ca.	Silicosis II	
19	65	Mining history mislaid		×	× (mod.)	Postmortem	R lower lobe	Epidermoid Ca.		
20	56	×	Bought (1–5)		× (mild)	Postmortem	R lower lobe bronchus	Anaplastic epidermoid Ca.		
21	34	×			× (mild)	Postmortem	R lower lobe	Infiltration with small round cells prob. spheroidal-cell Ca.		
22	?	×				Postmortem	L upper lobe	Oat-cell Ca.		
23	36	Outside mines with unknown ore composition	Bought (2–3) & home-made		× (mild)	Postmortem	?	Anaplastic epidermoid Ca.		
24	45	×		×	× (mod.)	Bronchoscopy & biopsy	R lower lobe	Anaplastic Ca.		
25	46	Mining history incomplete				Scalene gland biopsy	L bronchus	Sec. deposits of bronchial Ca.		
26	60	×				Diagnostic panel at Mpilo Hospital	L hilum			Cobalt-60 therapy given with improvement
27	45	Mining history incomplete				Postmortem	L lower lobe	Anaplastic Ca. epidermoid type	TB with silicosis	
28	54	×		×		Biopsy sec. in gland in neck. Malig. cells in pleural fluid	L hilum	Epidermoid Ca.		
29	44	×			× (mod.)	Bronchoscopy	L main bronchus			Mass 3 in. diam. fixed to chest wall in 1 pect. fold
30	55	Mining history mislaid	Bought (8)			Postmortem	R lower post. bronchus			Metastases on surface of heart and in R lobe thyroid
31	56	×	Home-made			Bronchoscopy	L lung			Specialist report: 'Ca. lung confirmed by bronchoscopy'
32	39	×	Bought (8)			Bronchoscopy	R upper lobe			Bronchoscopy showed Ca. protruding from R upper lobe bronchus
33	50	×	Bought & home-made			Postmortem	R upper lobe		TB with silicosis	Growth infiltrated bronchi and SVC
34	50	×				X-ray biopsy sec. growth	R lower lobe			Mass on R scapula: adenocarcinoma probably secondary
35	40	×	Bought (8)		× (mild to mod.)	Thoracotomy	R upper lobe			Thoracotomy: inoperable R upper lobe Ca. invading SVC

TABLE I (CONT'D

No.	Age	Mining history (× = significant period underground in arsenical mine)	Cig. smokers (No. = amount smoked/day)	Non-cig. smokers	Hyperkeratoses	Method of diagnosis	Site of growth	Histology	Additional diseases	Comment
36	39	Non-miner	Bought (24)			Diagnostic panel at Mpilo Hospital	R upper lobe			R upper lobe Ca. with SVC obstr.; 5 mths later supraclavic masses, secs. in skull and thorac. parieties. Died —no postmortem
37	55	Unreliable history			× (mod.)	Scalene gland biopsy	L bronchus		TB with silicosis	Specialist report: biopsy revealed sec. deposit
38	47	Not signif.		×		X-ray and tomography	R hilum			
39	55	×				X-ray	R lung near hilum			Sent for bronchoscopy but no record can be traced
40	54	Outside mine 1 yr, hammer underground		×	× (marked)	Bronchogram	R middle lobe			
41	55	×	Bought (3–4)			X-ray and tomograph	L upper lobe			Large mass in L upper lobe with complete obstr. upper 1 bronchus
42	40	Non-miner	Bought (4)			X-ray and tomograph	R lower lobe			Large Ca. R lower lobe with enlarged hilar glands and complete obstr. of bronchi. SVC obstr.
43	55	×		×		X-ray and tomograph	L upper lobe			Large mass constricting main L upper lobe bronchus. Bronchoscopy refused

process leading to eventual hyperkeratoses or cancer of the lung. This is in fact upheld by Amor (quoted by A. T. Doig in Merewether's book)[4] who found that a period of one year was sufficient exposure time for the eventual development of lung cancer in workers extracting copper and nickel from Canadian ores.

Fig. 1. Hand of patient showing palmar keratoses of 'mild' degree.

What is noticeable in the map is the fairly even distribution of triangles (symbols for time worked by hyperkeratotic patients) over the whole gold-belt area, in contrast to squares and circles (lung cancer patients) which preponderate in the area west of Gwanda village, which is also the region of highest arsenical content of ore.

More detailed information about the possible point of contact between arsenic and the patient is set out in Tables II and III. In analysing the data in these tables,

TABLE II. CANCER OF THE LUNG WITH PALMAR HYPERKERATOSES

Type of work	Case No.	Years worked	Total years mining	Region: Gwanda West	Region: Gwanda East	Region: Outside mines	Cross-ref.
A: Hammer or jackhammer work underground	4	2	12			×	
	15	28	28	×			
	17	8	32½			× (S.A.)	C
	20	4	23	×			B
	35	6	19	×			B
	37	3	?		×		
B: Surface work	9	20	20	×			
	16	8	26	×			C
	20	19	23	×			A
	35	12	19	×			A
C: Underground in other jobs (lasher, pump-man, bell-man, timberman, etc.	16	18	26			×	B
	17	26	32½			×	A
	21	10	12	×			
	23	10	10			×	
	24	16				×	
	29	6	18	×			

only mines in the highly arsenical western region of the Gwanda gold-belt are considered significant, or those in the eastern region or mines outside the Gwanda gold-belt which are known to contain arsenopyrite in the gold ore. Also—although a period of one year worked on an arsenical mine is taken as being long enough to allow potentially toxic absorption to take place—owing to the long latent period of industrial lung cancer, no work done within a period of 6 years before the development of symptoms is considered significant.

Taking these considerations into account, out of the 13 lung cancer patients with hyperkeratoses 4 (cases 4, 15, 20 and 35) had worked with a jackhammer or hammer underground in arsenical mines, and were exposed at the rock face to the maximum amount of dust in the mining environment. Two of these (cases 20 and 35) had also

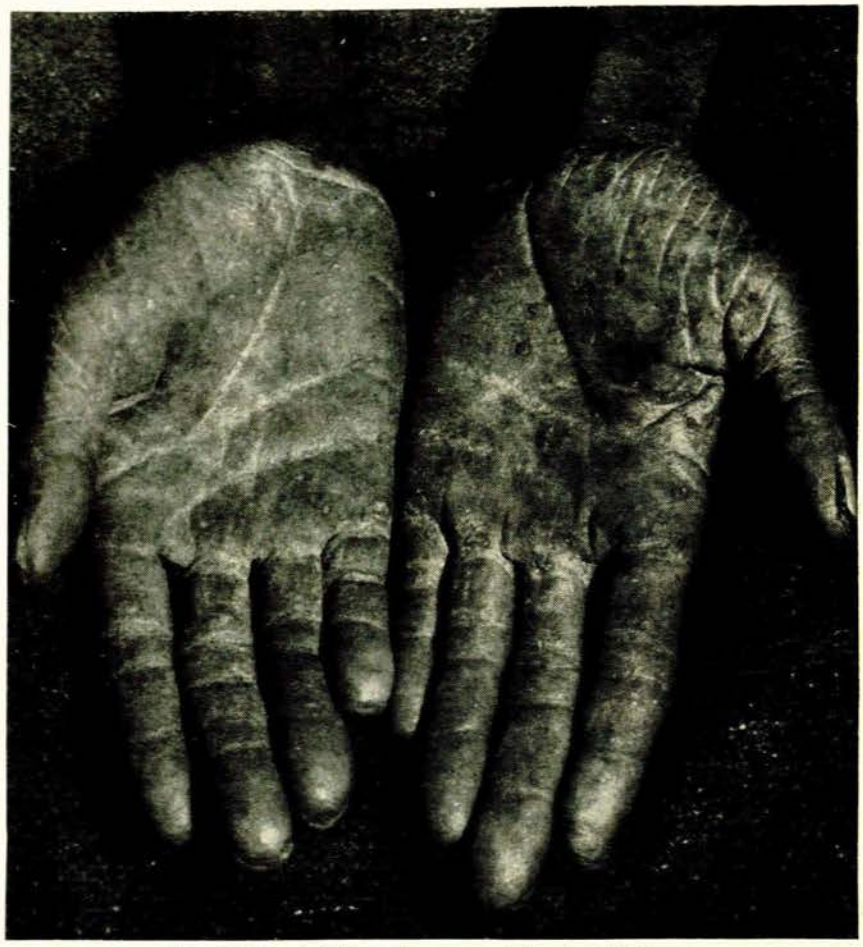

Fig. 2. Case 4. Palmar keratoses of 'moderate' degree.

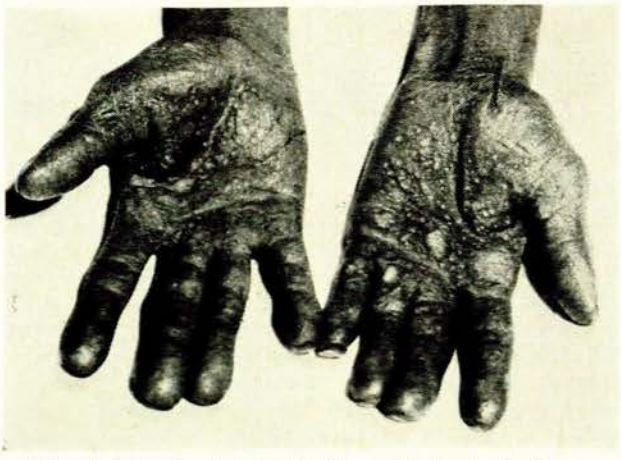

Fig. 3. Case 9. Palmar keratoses of 'marked' degree.

worked for long periods on the surface in the dust-laden atmosphere of the crushing mill, i.e. as mill 'feeders'. A fifth patient (case 16) had worked as lasher underground for 18 years in western-area mines, as well as on the surface at the roasting plant of another western mine, the Vubachikwe, where he himself noted there was 'much dust'. Two more patients (cases 21 and 29) had been 'lashers' (workers using shovels) underground in western-area mines and another (case 24) a lasher underground in the Fred mine, Filabusi, and for 10 years in a mine near Fred, where he says his hyperkeratoses appeared. Of the remainder in this group, Nos. 17 and 23 had worked on mines outside the Gwanda gold-belt, with unknown ore composition, for 32 and 10 years respectively. The mining history of case 19 was mislaid, while doubt was thrown on the history of only 3 years' jackhammer work at the Geelong mine before admission in case 37 by virtue of the fact that he was found to have silicosis with tuberculosis, confirmed by the Silicosis Bureau.

Thus, of 9 cases out of the 13 with hyperkeratoses with mining histories capable of being analysed, 8 had worked underground in mines with arsenical ores, 4 of them at the site of maximum dust pollution, i.e. as jackhammer or hammer workers at the rock face. The time of appearance of hyperkeratoses had been noted by 2 patients; in one it was while doing jackhammer work in a western mine, in the other while 'lashing' in an arsenical mine in the Filabusi district. Only one patient is, so to speak, 'odd man out'—No. 9, whose entire mining experience (20 years)

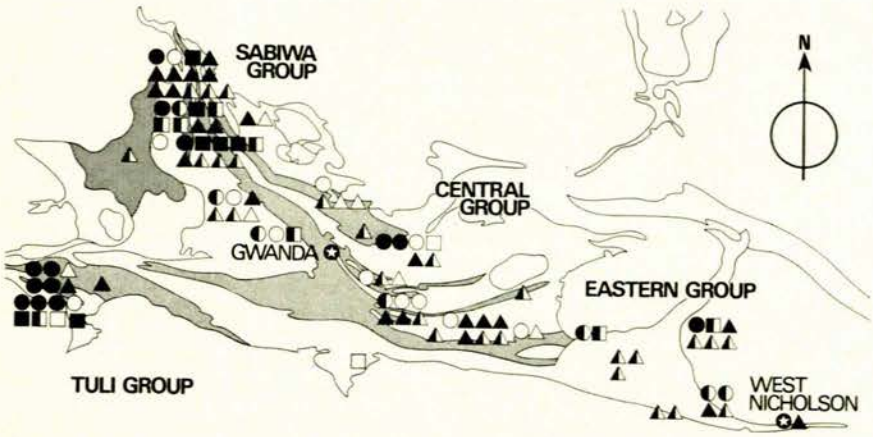

Fig. 4. Map of the Gwanda gold-belt. The area outlined is the region of gold-bearing rock. Shaded portion = quartzschist; unshaded portion = greenstone group (greenstone and greenstone schist). Symbols represent time worked. Circles = lung cancer patients without palmar hyperkeratoses; squares = lung cancer patients with hyperkeratoses; triangles = patients with palmar hyperkeratoses but without lung cancer.

TABLE III. CANCER OF THE LUNG WITHOUT PALMAR HYPERKERATOSES

Type of work	*Case No.*	*Years worked*	*Total years mining*	*Region*			*Cross-ref.*
				Gwanda West	*Gwanda East*	*Outside mines*	
A: Hammer or jackhammer work underground	1	1	21	×			B
	2	5	10	×	×		B, C
	5	30	34	×	×	×	C
	6	12	25		×	×	C
	10	10	14		×		B
	13	12	17		×		C
	26	5	17			×	B, C
	31	2	32	×			
		2	32			×	B, C
	33	9	28			×	B, C
B: Surface work	1	20	21	×			A
	2	2	10		×		A, C
	3	13	30			×	C
	10	4	14	×			A
	11	4	8	×			C
	12	11	11			×	
	22	4	15	×			C
	26	12	17	×			A, C
	31	8	32	×			A, C
	32	3	4		×		C
	33	3	28			×	A, C
	34	13	23	×			C
C: Underground in other jobs (lasher, pump-man, bell-man, timberman, etc.)	2	3	10			×	A, B
	5	4	34			×	A
	6	12	25			×	A
	3	17	30	×			B
	7	8	13	×			
		5	13			×	
	8	2	2		×		
	11	4	8	×			B
	13	5	17		×		A
	14	12	13½	×			
	22	11	15	×			B
	26	1	17	×			A, C
	31	2	32	×			A, B
		17	32			×	A, B
	32	1	4			×	B
	34	1	23	×			B
		9	23			×	B

had been on the surface of Sabiwa, a western mine, pushing a wheelbarrow, presumably a relatively non-dusty job, yet he developed 'marked' hyperkeratoses of the palms.

Examining now the 19 lung cancer patients without hyperkeratoses whose detailed mining histories are known (Table III), 7 (cases 1, 2, 5, 6, 13, 31 and 33) had worked significant periods with a jackhammer or hammer underground in arsenical ore mines. A further 4 had worked underground in such mines in other capacities —pushing coco-pans and lashing (case 7), attending to fuses (case 8), lasher underground (case 14), attending to pumps (case 28)—and there were two who had also done hammer work (cases 5 and 6). Five patients had done surface work, but in addition had worked underground: 4 as lashers (cases 11, 22, 26 and 34) and one (case 3) as cage driver in a reputedly very dusty atmosphere. Patient No. 32 had not worked underground in Gwanda, but had been lasher underground for a year at the Fred, a mine at Filabusi containing minor arsenopyrite.

Only two of this group had not been exposed to a dusty atmosphere in arsenic-containing mines: No. 12, who had done 11 years on the surface as mill-feeder in mines outside Gwanda (Inyati and Kezi) believed not to be arsenical; and No. 10, whose 10 years underground with a hammer were at Big Ben, not listed as arsenical, but who had worked 4 years on the surface in a western arsenical mine (Susannah).

DISCUSSION

The two groups analysed above are roughly similar. In both the great majority of patients had worked underground in arsenical mines, and in each group nearly half the patients were employed at the rock face. This is a significant fact, since in any mine the men actually employed as jackhammer or hammer men are outnumbered by those engaged in a variety of other jobs, both underground and on the surface. It suggests that there is a direct relation between dust concentration and the development of carcinoma of the lung, and that the arsenic—and there is enough evidence so far to make arsenic the most probable carcinogen—is absorbed by way of the respiratory tract.

Regarding the occurrence of keratoses in roughly only one-third of the miners, perusal of the available literature reveals the interesting fact that, whereas in the reports of lung cancer developing after ingestion of arsenic in medicines or contaminated water the majority of patients have pathological changes in the skin (e.g. all 6 patients recorded by Robson and Jelliffe had keratoses, and 4 of

them intra-epidermal epitheliomata[5]), industrial exposure to arsenic does not appear necessarily to follow this pattern. For example, no case of epithelioma of skin was seen in 21 cases suffering from nasal and lung cancers following arsenic poisoning during extraction of copper and nickel from Canadian ores.[4] In the case of the Schneeburg miners, Neubauer[6] thought arsenic as a cause of cancer of the lung unlikely because of the absence of characteristic skin changes (although Currie found that palmar hyperkeratoses did occur among men washing the cobalt ores). Nevertheless, in spite of the absence of such changes, Rochstroh's demonstration[7] that lung cancer is equally as prevalent among the workers at the smelting plant at Aue, Saxony, where the nickel, cobalt and arsenide ores from Schneeburg are processed, as in the Schneeburg miners themselves has revived the arsenical theory. Radioactivity at the Aue smelter is minimal, and the common factor between smelter and mine is not radioactivity, but arsenic and nickel. It appears that inhaled arsenic may affect primarily the lungs, although the reason why in some instances there is an associated increase of nasal cancers (as in the processing of Canadian ores) and in others (as in Schneeburg and Aue, and, in all probability, Gwanda) no such increase, is unknown.

Although on the available data the pulmonary route seems the obvious one, the alternative possibility of arsenic absorption by the mouth cannot be disregarded, in view of the inadequate washing facilities that obtain at many of the smaller mines, and also since contamination of surface water was shown as long ago as 1954 to occur when water entering the mill at Blanket A mine (western region) was found to contain 270 parts of arsenious oxide (As_2O_3) per million, and 'slimes' up to 430 p.p.m. Contact with such surface sources of arsenic could explain 2 out of the 3 cases in the series that do no fit into the expected pattern: Nos. 9 and 10 had both worked only on the surface in highly arsenical mines, in what must have been relatively non-dusty jobs, i.e. pushing a wheelbarrow, and sweeping in the compound, respectively. Patient No. 9, aged 40 years, had marked hyperkeratoses of the palms, and his lung cancer was therefore suspected of being arsenical in origin, although he was also a heavy smoker for an African labourer (8 cigarettes/day) and had been smoking since the age of 15 years. Patient No. 10 was also a cigarette smoker (amount not recorded) and in view of his age (56 years) and the absence of any proof of arsenic absorption such as hyperkeratoses, his smoking could have been the main cause of his lung tumour.

Cigarette smoking is the one generally accepted aetiological factor in lung cancer. Ochsner[8] is sceptical of all other causes, and quotes Auerbach as saying that he has never seen a non-smoker with primary epidermoid cancer of the lung. Ochsner himself states: 'After considerable experience with bronchogenic neoplasm, we are convinced that a history of smoking is the most important diagnostic criterion in carcinoma of the lung, and that a lesion of the lung that might be a neoplasm in a non-smoker is almost without exception either adenocarcinoma or is not malignant'.[8]

In the 1957 series of lung cancer cases from Gwanda, all 11 patients who were questioned on the matter admitted that they smoked (10 smoked cigarettes, one a pipe). In the present series of 37 lung cancer cases, smoking histories were taken from 25 patients: of these, 6 were found to be non-smokers, and one of the remainder smoked a pipe only. In one of these patients (case 17) the mining history had been mislaid; in another (case 19) it was incomplete. Both, however, had marked palmar hyperkeratoses, indicating previous absorption of arsenic. Of the 5 with known mining histories (cases 4, 7, 15, 24 and 28) all had worked underground in arsenical mines, and 3 showed marked palmar hyperkeratoses.

Conversely, all those who had not worked underground in arsenical mines (cases 9, 10, 12 and 36) were cigarette smokers. Patient No. 36, who smoked the greatest number of cigarettes (24/day), was the one non-miner in the series.

Conclusions, therefore, can be drawn from this small series, to favour both the arsenical and cigarette-smoking aetiology of lung cancer; but in fact, if the proportion of patients with complete mining histories who worked underground in arsenical mines (25 out of 29) is compared with the proportion of patients who smoked cigarettes (19 out of 25), there is a closer correlation in this series between working underground in arsenical mines and lung cancer than between cigarette smoking and lung cancer (86·2% compared with 74·1%).

Twelve patients (cases 2, 3, 5, 8, 11, 13, 16, 20, 31, 32, 33 and 35) worked underground in arsenic mines and smoked cigarettes; and it is suggested that this pattern, i.e. 2 causative factors acting in association, is probably responsible for most of the lung cancers among miners in Southern Matabeleland.

SUMMARY

Thirty-seven male patients with carcinoma of the lung were admitted to Gwanda district hospital during the period January 1957 - November 1963. Thirty-six of these men were miners or had worked in mines in the past; and they represent an incidence of lung cancer among the male mining population of Gwanda district of 205·6/100,000.

An analysis of the available mining histories reveals that the majority of these patients (25 out of 28) had worked underground in dusty conditions in goldmines with highly arsenical ore bodies.

Six patients were non-smokers, but had worked underground in arsenical mines; while all 4 patients who had not worked underground in arsenical mines smoked cigarettes. The majority of the cases, however, were cigarette smokers and had also been exposed to arsenical dust underground for significant periods.

The conclusion drawn is that, while either exposure to arsenical dust or cigarette smoking can by itself initiate cancer of the lung, it is most probably both factors acting together that is responsible for the high incidence of this disease in Southern Matabeleland.

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