

PULMONARY TUBERCULOSIS

IN

EAST AFRICAN NATIVE TROOPS

by

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## PREFACE

This is not a record of a planned research carried out as a wholetime occupation: it is the record of investigations made, during a tour of duty as a Medical Specialist in the Royal Army Medical Corps in East Africa Command, in an attempt to answer certain questions which forced themselves upon my notice.

The literature dealing with Tuberculosis in East African natives is scanty and with one or two exceptions pessimistic. It was the realisation that this pessimism was not entirely justified that gave the initial stimulus to these investigations.

Critical revision of the case notes has revealed two major deficiencies: the routine investigations were not always made as regularly as they should have been and in some cases the treatment given was ill-advised. The irregularity of the routine investigations was due to pressure of other more urgent work. During the period in question there were two major outbreaks of Falciparum Malaria and an epidemic of Typhoid Fever. During these outbreaks so much additional work was thrown upon the laboratory staff that routine investigations on chronic cases had to be deferred. The errors in treatment were due to ignorance on my part - an ignorance which it was the main purpose of this

investigation to rectify.

East African Natives suffering from Pulmonary Tuberculosis can obtain specialist treatment at three centres: Kibongoto Sanatorium, Tanganyika Territory; the Hospital for Chest Diseases, Mombasa, Kenya; and Mulago Hospital, Kampala, Uganda. I was able to visit each of these hospitals and it was encouraging to find that a reasonable optimism prevailed at them all.

No account of these investigations would be complete without acknowledgement of the help so freely given to me on all sides. The East African native dislikes lying in bed. The Nursing Officers of Queen Alexandra's Royal Army Nursing Corps, who were in charge of the Tuberculosis wards, were indefatigable in their efforts to enforce strict rest in bed. A succession of Medical Officers of the Royal Army Medical Corps, who acted as my House Physicians, entered enthusiastically into the investigation. Three Surgeons, Mr.C.S.Gross, F.R.C.S.E., Mr.Maule Liddell, F.R.C.S.E., and Mr.Wilfrid Barber, F.R.C.S. cooperated most willingly. Lieutenant Colonel Leslie Gordon, O.B.E., RAMC. then Assistant Director of Army Health, Headquarters, East Africa Command supplied the data upon which table 17 is based. Miss Allen, the librarian at the Medical Research Institute, Nairobi, was a mine of information about the literature available for consultation in Kenya.

Finally the photographic reproductions of the radiographs are due to the skill of Dr. D.G. Rushton.

Subject. The object of the investigations hereafter

occurs. To one and all I tender my most grateful thanks.

(1) to determine the incidence of Pulmonary Tuberculosis in East African Natives at Mockinon Road and thus to gain some idea of the Tuberculosis problem, which may face the Army Medical Services in the event of large numbers of East African Natives being called to the colours;

(2) to study the natural history of Pulmonary Tuberculosis as it occurs in East African Natives;

(3) to define the most suitable methods for the treatment of Pulmonary Tuberculosis in East African Natives.

Material. The material available for study consisted of 53 patients suffering from Pulmonary Tuberculosis of whose case notes are presented in the appendices and 1 patient suffering from Abdominal Tuberculosis, whose case notes are also included. In addition, two patients suffering from Pulmonary Tuberculosis, whose case notes are not included in the appendices, are referred to in the section dealing with the



CHAPTER I: OBJECT; MATERIAL; METHODS.

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incidence of the disease.

All the patients were serving in Military or Para-military units in East Africa Command. They came, therefore, from a highly selected population. The manner, in which this selection was made, differed slightly in the cases of the Military and the Para-military units.

In the case of the Army, recruiting parties go <sup>out</sup> on safari into the native reserves. The native chiefs, who have been forewarned, collect those young men whom they consider suitable for enlistment. These men are screened for gross disabilities by the lay members of the recruiting team. Those, who are passed, are then examined by the Medical Officer, attached to the team, whose mental attitude is succinctly expressed by the phrase "when in doubt, reject." After enlistment the recruits spend six months doing basic training at the East African Training Centre at Nakuru, Kenya. Here they are under the supervision of a Medical Officer, who is empowered to reject any recruit whom he considers unfit. Nakuru is within easy reach of Nairobi, where there is a Military Hospital, to which any doubtful cases may be referred for special investigations.

In the case of the East African Civil Constuction Force, from which the paramilitary units were formed, the recruiting procedure was basically similar. But the standards for acceptance were lower

and the recruits did not spend six months in a training depot.

Although recruiting safaris go to most parts of East Africa, the majority of the population, from which these patients came, was made up of natives from three tribes: the Kamba, the Kikuyu and its associated sept the Embu and the Meru, and the lake tribe, the Luo.

The Kamba is a Bantu tribe engaged in arable agriculture but its reserve has suffered severely from soil erosion. The reserve has two main districts: Machako's and Kitui. In Machako's district, the diet consists of millet, maize, beans, cassava, bananas, sweet potatoes, fruit and green vegetables with meat and milk as rare luxuries. By African standards the people are well to do, well fed, well housed and well dressed. In the Kitui district, cassava and beans form the staple diet though maize and bananas are imported from the Kikuyu reserve. In this district, malaria and schistosomiasis are endemic; under-nourishment is widespread; and scabies is nearly universal.

The Kikuyu tribe and its sept the Embu and the Meru are of Bantu stock. They dwell on the slopes of Mount Kenya and the north-eastern slopes of the Aberdare range and are engaged in agriculture. A Kikuyu is slender, unfit for heavy labour, suspicious, treacherous and full of guile. The diet consists of maize, beans, millet, cassava and vegetables; meat is

eaten on ceremonial occasions or in times of plenty. Malnutrition is common, including Kwashiorkor; malaria is endemic in the lowland parts of the reserve; and respiratory infections are common everywhere.

The Luo is a Nilotic tribe, engaged in agriculture in the country lying to the east of Lake Victoria. A Luo is tall, dark, strong and cheerful; he eats meat, fish and maize and drinks milk. Malaria is endemic but there is a high degree of immunity against it. Respiratory infections are also common.

Throughout their service life, all native soldiers, irrespective of their tribal habits, are given the same diet. This consists of maize, beans, potatoes, meat, vegetables, fruit, edible oil, tea, sugar and milk. An analysis of this diet is given in table 17. The men in the paramilitary units received a very similar diet but the carbohydrate content was higher and the protein and fat content lower.

Both military and paramilitary personnel are housed either in wooden huts or in bandas. A banda is a hut made of mud and wattle and thatched. In the case of the military units the beds are spaced out to give each man the requisite floor space laid down by the Army Health Authorities and mosquito nets are used. In the paramilitary units, the beds were usually closer together and the mosquito nets in a state of advanced disrepair.



To summarise, the population from which the patients came, was of mixed origin but carefully selected, well fed, well housed and well clothed. The standards of hygiene achieved in the paramilitary units were not as high as those obtaining in the Military Units.

Methods. Throughout the investigation, the methods used were those normally employed in dealing with Pulmonary Tuberculosis among Europeans.

In dealing with African patients, the history of the disease plays a much smaller part than it does in dealing with European patients. The African has only a hazy idea of time and he has a stoical and fatalistic attitude to physical ailments. In consequence it is unusual to obtain a history of more than short duration, confined to a few leading symptoms.

Discussing the diagnosis of Pulmonary Tuberculosis among East Africans in the reserves, HAYNES (27) says: "the limitations of physical examination are well recognised and need not be remarked upon here, except to point out that diagnosis must be dependent on this alone when an adequate history can seldom be obtained." He continues: "It is fatally easy, when examining the chest in a subject, who, one has reason to believe, has tuberculosis, to delude oneself either into exaggerating normal physical findings .... or actually to find non-existent



physical signs."

Each patient underwent a full clinical examination on admission to hospital. In many cases, the physical signs were minimal or non-existent and could not be elicited even after the radiograph had been examined. Pleural effusions were the exception and as a rule gave rise to the classical physical signs.

The presence of a pleural effusion was confirmed in every case by diagnostic paracentesis. Only those cases of pleural effusion in which the cells were predominantly lymphocytic (80% or over) have been included. In fact, this means that only two cases of pleural effusion have had to be excluded: pleural effusion in association with non-tuberculous pulmonary disease appears to be rare in East Africans. Guinea pig inoculation was not employed.

Repeated sputum examinations were made on all patients. As a routine, six consecutive daily sputa were sent to the laboratory every month from each patient. When the sputum is recorded as positive this means that acid fast bacilli were seen in at least two of the six consecutive specimens. This rule was adopted to eliminate as far as possible any chance of the sputa being muddled. When the sputum is recorded as negative this means that no acid fast bacilli were seen in any of six consecutive specimens. Sputum culture was not carried out and gastric lavage and laryngeal swabbing were not employed.

The Erythrocyte Sedimentation Rate was estimated in all patients. In the early stages of the investigation this was done at fortnightly intervals. But in the later stages it was done once a month as it was not considered that the information gained from fortnightly estimations justified the additional work. For the greater part of the investigation the estimations were made in the laboratory by Wintrobe's method in conformity with Army custom. But, as the results were frequently not in keeping with the other clinical data, after September 1951 all estimations were made in the wards by Westergren's method.

Tuberculin tests were employed as a diagnostic measure only in one or two cases. It was not until the investigation was well advanced that the interest-

ing problems, associated with tuberculin sensitivity in the African, were appreciated. By that time, it was considered, that insufficient data could be collected in the time remaining to make it worthwhile to investigate such problems.

Leucocyte counts proved to be of no value in diagnosis. This subject has been dealt with in detail elsewhere. JOHNSTONE (29).

Radiological examination was carried out on all patients at monthly intervals as a routine and more frequently if there was any special reason.

In Mackinnon Road the power for the X-ray plant was provided by generators run by the Royal Engineers; it fluctuated a good deal from day to day. In Nairobi, similar fluctuations were common, although the power was provided by the East African Light and Power Company. Consequently the penetration of the films was very variable and comparison was often difficult.

In selecting radiographs to illustrate the case notes every effort has been made to choose films as nearly comparable as possible. The photographs of these radiographs were taken with a Leica camera. The captions describing the radiographic appearances were made while reading the original films. The occasional discrepancies between caption and photograph are due to this fact.

Autopsies were performed on all patients who died except one; this patient died after transfer to a

hospital nearer his home. A careful naked eye search was also made for healed primary foci or other signs of tuberculosis at all autopsies performed on patients dying from causes other than tuberculosis.

years since Von Pirquet described the sensitizing reaction which bears his name, our knowledge of the prevalence of tuberculous disease and of tuberculous infection among the native races of Africa is still woefully scanty. And, owing to the inadequacy of the available data, the opinions that have been expressed on these subjects are frequently contradictory.

Excavation of the tombs of Ancient Egyptians has revealed lesions, which are considered to be tuberculous, and a tuberculous bone found in South Africa probably dates from 2000 B.C. It is therefore reasonable to conclude that in the valley of the Nile tuberculosis has existed for many centuries. This conclusion is supported by the chronic nature of the disease that is commonly seen among the Egyptian Fellahs of today.

The disease has probably also been endemic for many centuries in the communities of the North African

CHAPTER 2. THE PREVALENCE OF TUBERCULOUS INFECTION.

Although seventy years have passed since KOCH discovered the causative agent of Tuberculosis and fifty years since VON PIRQUET described the sensitivity reaction which bears his name, our knowledge of the prevalence of tuberculous disease and of tuberculous infection among the native races of Africa is still woefully scanty. And, owing to the inadequacy of the available data, the opinions, that have been expressed on these subjects, are frequently contradictory.

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Mediterranean littoral and of the East African coast as far south as Somalia, where Sir Richard Burton found it in the course of his journey to Harrar.

BURTON (9) comments on the diseases of the Somali as follows: "their maladies are few and simple ....

Consumption is a family complaint and therefore considered incurable: to use the Somali expression, they address the patient with 'Allah have mercy upon thee' not with 'Allah cure thee'." The disease has also existed in Zanzibar since the island was opened as a trading centre to Europeans.

At the southern end of the continent the situation seems to have been different. LICHTENSTEIN (36) writing in 1803 noted the absence of cough, chronic disease and syphilis amongst the Kaffirs. LIVINGSTONE (37) is almost always quoted as saying that "tuberculosis did not exist in the interior." What he really wrote was this: firstly of the inhabitants of Buchuanaland - " the diseases of the Bakwains are few. There is no consumption and no scrofula. The most prevalent diseases are pneumonia and other inflammations, as of the bowels, stomach and pleura, with rheumatism and disease of the heart." Later, after describing an epidemic which looked like pneumonia he adds: "as no inspection of the body is allowed by these people and the place of sepulture is is carefully concealed, I had to rest satisfied with conjecture." Elsewhere he says of the Makololo tribe,

which was then settled about 100 miles from the Victoria Falls - "fever is almost the only disease prevalent. There is no consumption or scrofula."

At the time when LIVINGSTONE was writing, the association between pleural effusion and pulmonary tuberculosis had not been discovered. But, in the light of modern knowledge, it is clear that immediately after denying the existence of tuberculosis among the Bakwain he brings forward strong circumstantial evidence in favour of its existence. Furthermore, it will be shown later that in the African native pulmonary tuberculosis is frequently pneumonic in character with an acute onset. In the absence of postmortem evidence, LIVINGSTONE's statements must be accepted with reserve if, indeed, they are not set aside as definitely misleading.

Another medical missionary, who was a pioneer investigator of Tuberculosis in South Africa, was MACVICAR (38). In an abstract of the thesis, which he submitted to the University of Edinburgh, he wrote as follows: "The Bantu population is affected very unequally by tuberculosis. In some districts, especially in Natal and the Native Territories in the East of Cape Colony, the people suffer much. On the other hand, there are still a few districts in the interior where tuberculous disease is almost if not quite unknown among the Bantu.

"From the evidence of Dr. LIVINGSTONE and others,

the fact is pretty well established that the Buchuana-Basuto race inhabiting the high-lying interior of South Africa was free from tuberculosis until about fifty years ago. Since then it has been introduced and is spreading among them, in some places being already very common.

"Tuberculosis has been so long known among the Kaffir-Zulu people inhabiting the coastal belt that it is hard to say if it was originally absent; but in some places it is still rare and there seems ground for the current belief that it has been introduced in modern times.

"There is a tradition in Portuguese East Africa that consumption came from the Indians and it is quite possible that this may have been the original source of the disease among the Zulus. As regards East Central Africa the evidence points to the Zanzibar Arabs as the chief introducers of tuberculosis. It is met with along the old slave routes; it is still absent from regions remote from these routes."

MACVICAR is usually quoted as considering that tuberculosis in South Africa was of recent origin. It is clear from the extensive extract of his paper, given above, that he based his view largely on the evidence of LIVINGSTONE. How much credence should be given to that evidence has already been pointed out. It now seems that, apart from his remarks on the prevalence of the disease at the time when he

was working in South Africa, MACVICAR's statements must also be treated with considerable reserve.

In 1895, GREGORY (26) could say " of all the diseases attacking the (South African) Native and (Cape) Coloured, Tuberculosis is by far the most important." And in 1896, THEALL (55) wrote "consumption, another fell disease, was almost unknown amongst the natives of South Africa until recent years."

The opinions, so far considered, have been based upon clinical observations and have not all been those of qualified doctors. When the two most important opinions - those of LIVINGSTONE and of MACVICAR -are subjected to scrutiny, it is clear that they are based on inadequate evidence and are not worthy of the authority usually ascribed to them.

After the introduction of the VON PIRQUET test, Tuberculin Sensitivity Surveys were carried out in many parts of Africa and provided objective instead of subjective evidence for the opinions based upon them.

The first tuberculin sensitivity surveys were made in West Africa at the instigation of CALMETTE. Table 1 has been compiled from the figures given by WAGON<sup>(60)</sup> in 1910; by CALMETTE (11) in 1912; by MATHIS and DURIEUX (41) in 1929; and by TOULLEC and JOLLY (56) in 1932.



TABLE I

PLACE

PLACE	1910		1912		1925		1929		1932	
	No. tested.	% Pos.	No. tested	% Pos	No. tested	% Pos.	No. tested	% Pos.	No. tested	% Pos
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	(k)	(l)
SENEGAL										
Cercle de SINE SALOUM					146	10	7	71		
LOUGA					50	12	132	35		
THIÈS					57	14	45	15		
TIVAOUANE			232	7.7	73	31	14	57		
TAMBACOUNOA					15	20	31	55		
PADOR							36	44		
MATAM							6	66		
KOLDA							142	41		
SÉDHIU			159	1.2			45	51		
ST. LOUIS			174	25.8						
BAKEL			40	25						
BIGNONA			179	23.5						
BAOL					94	21				
RUFISQUE							5	20		
Unspecified							17	23		
Totals			784	14.8	435	15	480	41		
FRENCH SUDAN										
Cercle de NIORO					91	12	133	55		
BANDIAGARA							49	30		
SAN							6	66		
KOUTIALA							35	82		
BAMAKO			173	16.7			21	38		
BOUGOUNI							64	45		
KITA							14	50		
SIKASSO							16	43		
KAYES							67	43		



PLACE

	1910		1912		1925		1929		1932	
	No. tested (a)	% Pos. (b)	No. tested (c)	% Pos. (d)	No. tested (e)	% Pos. (f)	No. tested (g)	% Pos. (h)	No. tested (K)	% Pos. (L)
SÉGOU							110	57		
BAFOULABÉ							18	44		
MOPTI							34	47		
SATADOUGOU							28	53		
TOMBOUCTOU							10	80		
GOUNDAM							48	43		
Unspecified							9	77		
<b>Totals</b>			173	16.7	91	12	662	51		
<b>HAUTE VOLTA</b>										
Cercle de	OUAVADOUGOU				366	6	70	25		
	DÉ DOUGOU				16	6	97	24		
	GAOUA						120	25		
	BOBO-DIOULASSO						23	48		
	OUAHIGOUYA						7	77		
Unspecified							6	33		
<b>Totals</b>							323	27		
<b>FRENCH GUINEA</b>										
Cercle de	MAMOU		20	5			5	40		
	KISSIDOUGOU						8	75		
	BOFFA						23	43		
	FORÉCARIAH						37	59		
	LABÉ						22	41		
	PITA						8	75		
	MACENTA						12	75		
	KANKAN						38	39		
	GUÉKÉDOU						9	64		
	BOKÉ						36	61		
	KINDIA	100	12				19	63		

PLACE.

	1910		1912		1925		1929		1932	
	No. tested (a)	% Pos. (b)	No. tested (c)	% Pos. (d)	No. tested (e)	% Pos. (f)	No. tested (g)	% Pos. (h)	No. tested (k)	% Pos. (l)
SIGUIRI							35	40		
CONAKRY					15	53				
Unspecified							15	36		
<b>Totals</b>	100	12	20	5	15	53	267	52		
<b>IVORY COAST</b>										
Cerde de	HAUT-SASSA-NORA						10	30	13	69
des	LAGUNES						24	25		
de	MAN						15	66	115	33
des	GOUROS						5	60		
du	MOYEN-CAVALLY						6	66		
du	BAOULÉ		506	7.1			62	51	29	27
de	BOUNDOUKOU								24	16
	GRANDBASSAN		196	12.7						
	GONOS								22	27
	GUIELO								24	58
	KONG								19	21
	Unspecified						19	42	39	33
<b>Totals</b>			702	8			141	46	285	34

All these workers applied the Von Pirquet test in accordance with the technique taught by Calmette. Wagon and Calmette used Koch's Old Tuberculin; but Matthis and Durieux and Toullec and Jolly used B.C.G. tuberculin. The figures quoted refer to African natives over 15 years of age. The figures of Wagon and of Calmette were obtained from surveys made in the actual towns and villages. The figures of Matthis and Durieux were obtained from a survey of recruits to the French Army carried out at Rufisque. And the figures of Toullec and Jolly were obtained from a survey of similar recruits shortly after disembarkation at Marseilles.

CALMETTE (11) comments on his figures thus: "Dans les régions tropicales, la tuberculose n'est pas sensiblement influencée par les climats. Sa fréquence est en rapport direct avec la civilisation. Elle est extrêmement rare parmi les populations indigènes de race noire dans les pays où l'Européen n'a pénétré que depuis peu d'années; mais la proportion des sujets contaminés s'accroît chez elles avec l'intensité des échanges commerciaux et de l'immigration étrangère."

MATHIS and DURIEUX (41) contrast their figures with those of Calmette and comment this: "Il semble que l'index tuberculeux dans les différentes Colonies de l'Afrique Occidentale Française a progressé avec rapidité et dans des grandes proportions."

They then proceed to give an interesting outline of the geographical and economic features of each of the Colonies from which their recruits were drawn and to demonstrate that the increase in the prevalence of tuberculous infection has occurred along the various trade routes - the rivers, the railways and the main roads.

They conclude: "Les documents que nous apportons ne sont pas tres nombreux, mais ils confirment la notion que la tuberculose est en progression en Afrique Occidentale Francaise."

This conclusion did not pass unchallenged. Six months later BOUFFARD (4), who had been Director of Medical Services in the Ivory Coast from 1924-1929, wrote that, on the basis of the small number of cases of proven tuberculosis that he had seen during that period, he considered tuberculosis was uncommon in that colony. "Deès 1925, j'ai cherché à préciser la domaine et l'importance que prenait le bacille tuberculeux dans la pathologie locale. Je n'ai pas en recours à la cutireaction, préférant le dépistage clinique des malades avec examen microscopique de l'expectoration des tousseurs chronique ---

En 4 ans, Je N'en ai rencontré que 12 Cracheurs de bacilles au dispensaire d'Abidjan où se present chaque jour plus de 300 consultants ---- Il semble bien que les cas de tuberculose ouverte devraient y être plus frequemment observées, si la maladie etait



aussi répandue que le pensent Mathis et Durieux ---  
Je persiste donc à considérer la Côte d'Ivoire comme  
un pays où le bacille tuberculeux est encore peu  
répandu. He adds that BLANCHARD had tested 200  
Sudanese recruits on arrival in Marseilles and found  
only 15% positive reactors in contrast to Mathis and  
Durieux's figure of 51%.

TOULLEC and JOLLY (56) are wary about attaching  
much significance to their figures. They write:

"Si l'on veut bien comparer nos résultats avec ceux  
de Mathis et Durieux on peut noter des écarts variant  
du simple au double suivant les cercles intéressés.  
Nous ne doutons pas que nos résultats ne puissent  
subir les mêmes critiques, mais nous nous gardons de  
leur attacher une valeur qu'ils ne peuvent avoir."

It is clear from a study of the figures in  
Table I that the utmost circumspection is needed in  
their interpretation. The figures obtained from test-  
ing recruits are open to the criticism that the  
recruits might have become infected since leaving  
their native villages. Therefore the material on  
which the surveys of Mathis and Durieux and of Toullec  
and Jolly were performed is not strictly comparable  
with the material on which the surveys of Wagon and  
Calmette were performed.

Secondly, although the technique of the Von  
Pirquet test was standardised in all the surveys, as  
has already been pointed out, the tuberculin used  
was different. Moreover with regard to the Von



Pirquet test, MALMROS and HEDVALL (39) state categorically: "The Pirquet test is by no means sensitive enough to enable us to divide a material into tuberculin positive reactors and tuberculin negative reactors, for many certainly positive tuberculin cases give a negative Pirquet reaction."

Thirdly, the number of natives tested in any one place is small and the totals vary considerably. The figures do not stand up to statistical analysis. It is inadmissible to compare Calmette's figure of 7.7% positive reactors out of 232 tested at Tivaouane with the figure of 57% positive reactors obtained by Mathis and Durieux by testing 14 recruits from the same area. Yet it is on this and similar comparisons that Mathis and Durieux base their conclusions. Again, to contrast the mean percentages of tuberculin positive reactors in the different colonies, as several recent authors have done, is equally inadmissible, as these means are derived from figures from different areas, where geographical, economic and social conditions are not comparable.

In considering Calmette's conclusions, it must be remembered that the figures incorporated in Table I form only a small proportion of the figures, which he was considering and which included several different age groups and were drawn from all the French Colonial Possessions and not only from West Africa. When his paper is read in its entirety with the mind attuned to the scientific climate of his

time, his conclusions seem justified, just as the conclusions of Dr. Livingstone seemed justified to Macvicar. It is only the modern advances in the techniques of tuberculin testing and of statistical analysis that render them suspect.

All the figures from the paper by Mathis and Durieux are included in Table 1. The hypothesis which they propound - that the increase in the incidence of tuberculous infection has occurred along the trade routes - is an extension of Calmette's conclusion and is both reasonable and attractive. It was put forward in 1867 by BUDD (7) who wrote: "Now everywhere along the African Seaboard where the blacks have come into contact and intimate relations with the whites, phthisis causes a large mortality among them. In the interior, where intercourse with the whites has been limited to casual contact with a few great travellers or other adventurous visitors, there is reason to believe that phthisis does not exist." And Macvicar has already been quoted on the spread of tuberculosis along the old slave routes. But as Mathis and Durieux themselves admit "Les Documents ne sont pas tres nombreux" and, as has been pointed out already, they will not stand up to statistical scrutiny. Their figures do not contradict their hypothesis; nor do they prove it.

The criticism, to which their conclusions were subjected by Bouffard, is a good example of the confusion which results when the prevalence of tubercu-

lous infection is compared with the prevalence of tuberculous disease without the author realising that the two incidences are not one and the same. As criticism Bouffard's paper is valueless. Its interest lies in showing the magnitude of the discrepancy that appeared to exist at that time and place between the two incidences.

Comparison of the figures of Mathis and Durieux with those of Toullec and Jolly illustrates the pitfalls waiting when attempts are made to draw conclusions from surveys made on small numbers - a fact of which Toullec and Jolly were clearly aware - "Nous nous gardons de leur attacher une valeur qu'ils ne peuvent avoir."

To sum up a critical study in the originals of the figures available from the French West African Colonies shows that by modern standards these figures are, on technical grounds, not capable of sustaining the hypothesis based upon them, namely that tuberculous infection was uncommon among the natives before the start of the 1914-1918 war; that the prevalence of tuberculous infection has increased during the ensuing decades; and that the increase has taken place mainly along the trade routes.

From the Belgian Congo, tuberculin sensitivity surveys have been reported by MOUCHET (44) in 1913; VAN DEN BRANDEN (57) in 1926; VAN HOOFF (58) in 1926; and SCHWETZ et al (52) in 1930. All these investigators employed the Von Pirquet test. Table 2 has been

compiled from their figures referring to natives over the age of 15 years.

TABLE 2.

PLACE.	1913		1926		1930	
	No tested	% Pos.	No tested	% Pos.	No tested	% Pos.
LEOPOLDSVILLE						
(a) TOWN	359	3.9	1168	50		
(b) SURROUNDING COUNTRYSIDE	188	1	564	26		
STANLEYVILLE			407	33	54	57

MOUCHET (44) concluded: "Il semble donc bien que Leopoldville constitue un foyer assez intense de tuberculose, mais un foyer localisée et d'origine relativement récente."

SCHWETZ ET AL (52) concluded: "Ni le nombre, même approximatif, des malades, ni la marche de la maladie, ne sont connus, ni ne seront reconnus avant un recensement médical complet ad hoc."

The figures obtained by MOUCHET and by VAN DEN BRANDEN are of the same order and therefore comparable. They reveal a striking increase in the prevalence of tuberculous infection in the capital of the Belgian Congo and in its surrounding countryside. But in the thirteen years, which elapsed between the two surveys, the town of Leopoldville must have grown. No mention is made of the total population in either paper. It may well be, therefore, that the increase is more apparent than real. Nevertheless these figures form a pièce of evidence which strongly



supports the hypothesis suggested by the French investigators.

In South Africa, largely due to the publicity given to the subject by the writings of Macvicar, a Tuberculosis Commission was appointed and its Report was published in 1914. With regard to the prevalence of tuberculous disease the conclusions reached were:

(1) "Tuberculosis is of comparatively recent introduction among the Bantu tribes;

(8) It is least prevalent in Zululand and the Northern Transvaal, more so in Basutoland, still more so in the Cape Native Territories and most widespread in the natives in the settled districts of the Cape Province;

(9) It is excessively prevalent among natives working in the large industrial centres and especially on the mines;

With regard to the prevalence of tuberculous infection the conclusion was: (11) "It must not be forgotten that for every case which has become established and has developed to the extent of producing marked lesions of tuberculosis, there must be others who, having been infected, are still maintaining a successful resistance to the invasion."

In 1921, MITCHELL(43) wrote of "tuberculosis, rare or unknown at the earliest days of the colony,

becoming more and more frequent during these latter days with improving communications."

In 1924, ALLAN (1) carried out a small tuberculin sensitivity survey among the natives of the TRANSKEI and CISCHEI and found the infection common and widespread. He later carried out a more extensive survey, which is incorporated in the Report on Tuberculosis in South African Natives with special reference to the disease amongst the Mine Labourers in the WITWATERSRAND (49). This report was published in 1932 and opens thus: "While reliable statistics are, of course, in the nature of things, unobtainable, it has been the almost universal experience of those qualified to judge, who have had opportunities of studying isolated communities before tribal conditions were unduly disturbed, that tuberculosis has been conspicuous by its absence both in the humans and also in their cattle."

Tuberculin sensitivity surveys were carried out at the mines on new recruits and also in the TRANSKEI and CISCHEI areas, from which a large proportion of the recruits were drawn. At the mines, it was found that out of 57,659 recruits, tested intradermally with 1/5000 or 1/10,000 Old Tuberculin, 72.5% were positive reactors. Table 3 has been compiled from the figures given by ALLAN, who carried out the survey in the Transkei and Ciskei areas; the figures refer to natives over 15 years of age.

TABLE 3.

<u>PLACE.</u>	Number tested	% Positive.
Butterworth	2195	85
Lusikisiki	233	95
Matatiele	551	56
Busutoland	590	61
Totals.	3569	77

It is clear from these figures that by 1930 tuberculous infection was already widespread amongst those natives of South Africa and of Portuguese East Africa dwelling in the parts of the country from which the Rand mines recruit their labourers. Commenting on this situation in the light of the known opinion of South African observers - that the disease is of recent origin in that country - HAYNES (27) suggests that the rate of spread may well have been accelerated by the return to the countryside of infected sputum positive labourers from the mines. This was a danger to which MACVICAR had drawn attention many years ago. CUMMINS, from his experience in other parts of the world, endorses this view.

It would be foolish to deny that there has been any increase in prevalence of tuberculous infection in South Africa during the present century. But it is difficult to believe that in a population, which is regarded as highly susceptible to the disease, the prevalence could rise in roughly fifty

years from nil to 75% without catastrophic results.

When the Canadian Indians first apparently came in contact with the tubercle bacillus, the disease assumed epidemic proportions. From 1882 to 1902, tuberculosis was responsible for 2/3s of all deaths; and it was not until 1918 that for the first time in fifty years more Indians were born than died from every cause(BUSHNELL 10). By 1928, 90% of the children were tuberculin positive reactors(FERGUSON 20) Nothing of this nature appears to have taken place among the native races of South Africa.

In contrast to the Canadian Indians is the case of the Dinkas of the Bahr-el-Ghazal Province of the Sudan. In the days of the slave trade, the Dinkas were so liable to tuberculosis that only the lowest prices were paid for them. In 1902, CUMMINS (16c) found no evidence of tuberculosis among the Dinkas, their cattle or the wild game of their country. Thirty years later after the province had again been opened to foreigners, BURROWS (8) found that 36% of natives between 10 and 25 years of age and 50% of natives over 25 years old reacted positively to 1/1500 old tuberculin given intradermally. Tuberculous infection appears to have died out when the province became isolated after the abolition of the slave trade and to have become prevalent again when the country was once more opened up. "The defined and localised pulmonary and non-pulmonary lesions and the many chronic cases with a history of five years or more" observed



by Burrows supports this theory, which accounts for the epidemiological difference from the Canadian Indians.

In South Africa, the trend of events cannot be satisfactorily explained as due to the first meeting between the tubercle bacillus and a susceptible population, as in the case of the Canadian Indians, nor to a reinfection of a population rendered resistant by the process of natural selection, as in the case of the Dinkas.

The true explanation is probably that the increase in the prevalence of tuberculous infection in South Africa has been more apparent than real. Just as the use of increasingly powerful telescopes has gradually revealed heavenly bodies hitherto unsuspected by the astronomers, so has the gradual improvement in the techniques of medical investigation revealed a prevalence of tuberculous infection formerly unsuspected.

When Livingstone came to Africa, before the discovery of the tubercle bacillus, he was on the lookout for "consumption" the wasting disease to which he was accustomed at home. Now "the obvious signs of tuberculosis are not common amongst Negroes in Africa" (KERANDEL 32) and consequently, because he was not looking in the right way, Livingstone found no consumption.

Macvicar and his contemporaries were equipped with the means of identifying the tubercle bacilli in

sputa and consequently were able to seek out cases of open tuberculous disease.

The early tuberculin surveys revealed the presence of tuberculous infection in those not suffering from open tuberculous disease. And finally the refinement of the Mantoux test revealed that the prevalence of tuberculous infection was greater than the investigations carried out by the Von Pirquet technique had suggested.

During the period between the Von Pirquet and the Mantoux eras, the apparent increase in the prevalence of tuberculous infection was accompanied by a real increase due to the greater ease in intercommunication. The figures for the Rand mines may also be a special case due to the habit of returning open cases of tuberculous disease to the native reserves.

In East Africa, the earliest work on the prevalence of tuberculous infection was done in Tanganyika. At Kilwa in 1911, PEIPER (47) tested 54 Africans over 15 years of age by the Von Pirquet technique and found 18.5% positive reactors, while 7 Indians of similar age 30% were positive reactors. On the basis of these and similar observations in younger age groups, PEIPER concluded that the disease had been introduced into the country by the Indians. In 1914, MANTEUFEL (40) tested at DAR-ES-SALAAM 388 Africans and 197 Indians over the age of 15 years and found 22.4% and 22.3% positive reactors respectively. Following PEIPER's lead, he suggested that Indian immigration

should be prohibited. In 1914, MULLER (45) tested 700 patients at the hospital in TANGA and found 33% positive reactors. A more extensive survey was made by FISCHER (21), who used the Von Pirquet technique and published his results in 1932. Between 1932 and 1938, WILCOCKS (62) carried out a survey of the whole country using the Mantoux test with 1/1000 Old Tuberculin. Table 4 has been compiled from the figures given, for natives over 15 years of age by MULLER, FISCHER and WILCOCKS.

TABLE 4.

PLACE	1914		1932		1932-38	
	No tested	% Pos.	No tested	% Pos.	No tested	% Pos.
TANGA	700	33			1009	80
DODOMA					970	48
SINGIDA					67	69
KIOMBOI					489	58
HANDENI					139	60
MOSHI					356	52
KILMANJARO (WEST)			) 56		888	66
" (EAST)					436	77
MERU					118	67
MBULU					141	45
PARE MTS					659	64
USAMBARA MTS					245	53
IRINGA					229	56
NJOMBE			67		387	60
MWANZA					493	60
USARAMO			30			
UNYAMWESI			21			
KONDE			32			
BUKOBA			42			
Totals					6627	62.5

In 1932, WILCOCKS (62B) wrote: "there is no word in Kiswahili which conveys to the native mind the same meaning as the word tuberculosis or even pulmonary tuberculosis does to the European. This may be taken as presumptive evidence that the disease has not been long known ---- The position is obscure since in the early days methods of diagnosis were poor." In 1938, in his Report on Tuberculosis in Tanganyika Territory, he concluded: "the results of the 13,313 tuberculin tests here recorded show that no part of the territory may be regarded as virgin soil."

Apart from a small survey carried out in the Nyanza province of Kenya in 1941 by CONNOLLY (14) whose results are recorded in table 5, no Tuberculin Sensitivity Surveys were carried out in Kenya or Uganda until after the 1939-1945 war.

TABLE 5.

African Recruits	Number tested.	% Pos.
BANTU: North Kavirondo	289	42
Central "	23	53
South "	97	50
Kipsigis	26	46
LUO: Central & South Kavirondo	452	52
C.M.S. Mission School		
BANTU: Aged 13-17	29	28
18-22	36	33
LUO: Aged 13-17	87	29
18-22	56	50



In 1951, a Report on Tuberculosis in Uganda was published by SANTON GILMOUR (25) Table 6 has been compiled from his figures referring to natives over 15 years of age tested intradermally with 1/10,000 Old Tuberculin.

TABLE 6.

<u>PLACE</u>	<u>Number tested</u>	<u>% Positive.</u>
BUGANDA	387	46
ANKOLE	460	49
TORO	362	49
BUNYORO	296	45
WEST NILE	262	48
ACHOLI	515	31
MBALE	343	49
BUSOGA	231	37
TOTALS	2856	44

In 1952, a Report on Tuberculosis in Kenya by HAYNES (27) was published. Table 7 has been compiled from his figures referring to natives over 18 years of age tested intradermally with 1/1000 Old Tuberculin.

TABLE 7.

<u>PLACE</u>	<u>Number tested.</u>	<u>% Positive.</u>
EMBU	1491	61
KERUGOYA	1616	73
MBERI & CIAMBERI	1338	41
MACHAKO'S	826	72
KITUI	1589	75
TEITA	775	84
TAVETA	238	82
MSAMBWENI	909	82
KISAUNI, LIKONI, CHAGAMWE	1296	78
KATIADO	244	70

TABLE 7. (contd:)

<u>PLACE</u>	<u>Number tested.</u>	<u>% Positive.</u>
KAPSABET	315	69
KERICHO	563	55
KISII	1026	63
KISUMU	823	69
MOMBASA	1863	92
NAIROBI	1814	84
TOTALS:	17298	73

It is unfortunate that Santon Gilmour elected to use 1/10,000 Old Tuberculin as this complicates the comparison of his figures with those presented by Wilcocks and Haynes. It is also unfortunate that Haynes selected slightly different age groupings from those used by most other workers, though, as he points out, statements of the ages of African natives do not have the same exactitude as those for more civilised populations. However, even allowing for these discrepancies, it is clear that these two post-war surveys give no support to the suggestion that tuberculous infection in these areas is of very recent origin.

Santon Gilmour points out that references in Aeschylus and Ptolemy to the Mountains of the Moon suggest the possibility that contact between Europeans and Central Africans may have existed 2000 years ago. He states, in contradistinction to Wilcocks, that several of the tribal languages have an old word for tuberculosis. He considers that it

is probable that tuberculosis was established in Uganda before the European invasion of the present century and that the result of this invasion has been the dissemination rather than the introduction of tuberculous infection.

Haynes considers that the widespread extent of tuberculous infection is a fact that is difficult to reconcile with the idea of a recent introduction of the disease.

In reviewing the literature on the prevalence of tuberculous infection among the native inhabitants of Africa, certain deliberate omissions have been made. On account of racial miscegenation with Europeans and Arabs, no detailed reference has been made to those papers which deal with the prevalence of tuberculous infection along the Mediterranean littoral, in Madagascar, in Zanzibar and among the Cape Coloured. The early work of ZIEMANN (65) in the Cameroons has not been quoted because it is in accord with the work done in the French West African Colonies and does not appear to have been followed up.

There is a tendency for those, who have had no personal experience of African natives, to think and write about them as if they were the same in all parts of the continent. This is erroneous. On this account, it may well be that much of the detailed information collected in this chapter is inapplicable to the subject of this thesis: Pulmonary Tuberculosis in East African Natives. But it was

necessary to survey the literature appertaining to other parts of the continent because thought on Pulmonary Tuberculosis in East Africans is still dominated by results obtained in other parts of the continent.

The "virgin soil" hypothesis rests upon the conclusions drawn from the early tuberculin surveys and upon conclusions drawn from the character of the disease in West Africans. It has been shown in the present chapter that the conclusions drawn from the early tuberculin surveys are of dubious validity; the conclusions drawn from the character of the disease will be discussed later.

How long the infection has been present in East Africa and how it has been spread are questions which can never be answered conclusively. What can be stated categorically is that throughout the three East African Territories the infection is now widespread and that this dissemination of infection has occurred without the incidence of the disease assuming epidemic proportions during the period of historical memory. There is now no virgin soil in East Africa.



CHAPTER 3. THE INCIDENCE OF TUBERCULOUS DISEASE.

The facts relating to the incidence of tuberculous disease in Africans are even scantier than those relating to the prevalence of tuberculous infection. The views of the early workers in the era prior to the introduction of tuberculin sensitivity tests have already been presented in the previous chapter and need not be repeated here.

The remaining facts fall into three distinct groups:

- (a) those relating to Africans in their natural surroundings;
- (b) those relating to Africans working under stress;
- (c) those relating to highly selected groups of Africans.

The figures relating to Africans in their natural surroundings come from the Reports by Wilcocks and by Haynes and are collected in tables 8 and 9 respectively.

TABLE 8 (After WILCOCKS)

<u>PLACE</u>	<u>Number Examined</u>	<u>Incidence of TB per 1000.</u>
SINGIDA		42
MERU		34
KILIMANJARO		30
MBULU		25
USAMBARA MTS		19
RUNGENE		16
TANGA AREA		15
MWANZA		12
PARE MTS		8

TABLE 8 (Contd:)

<u>PLACE</u>	<u>Number Examined.</u>	<u>Incidence of TB per 1000</u>
DODOMA		6
NJOMBE		4.9
HANDENI		4.8
IRINGA		3.3
KIOMBORI		2.4

TABLE 9 (After HAYNES).

<u>PLACE</u>	<u>Number Examined</u>	<u>Incidence of TB per 1000.</u>	
		<u>(a)</u> Proved.	<u>(b)</u> Proved + Suspected.
KITUI	3962	10.6	23.5
MACHAKOS	3072	5.9	14.3
TAVETA	500	4	16
KERICHO	1420	5.6	15.5
KAPSABET	575	5.2	12.2
KAJIADO	692	0	11.6
MSAMBWENI	1753	2.9	9.7
MARAGOLI	1266	4	9.5
KERUGOYA	3955	4.3	8.9
MBERI & CIAMBERI	3171	5.4	8.5
TEITA	2018	3	8.4
KISAUNI	2330	2.6	7.7
KISUMU	1683	1.2	7.7
EMBU	4279	3.5	6.1
KISII	2715	1.8	5.9

HAYNES (27) examined those natives, who returned to have their intradermal tuberculin tests read, selecting those who did not look fit. Thus of 3,962 who returned at Kitui, he actually examined 359. He writes: "It soon became apparent that it

would be necessary to divide the examination results into two groups which may be called clinical and proved tuberculosis and suspected tuberculosis. In the first group are put those cases with positive sputa or unequivocal chest signs (this virtually amounted to signs of cavitation), characteristic adenitis or bony lesions. The second group comprises cases showing suggestive adenitis (usually cervical) or with physical signs in the lungs that in all probability denoted tuberculosis but which were in themselves not felt to be sufficient evidence on which to base a firm diagnosis.

"There is no doubt that many cases of tuberculosis escaped examination altogether.

"When all the factors are taken into consideration, it is felt that an estimate based on "proved" plus "suspected" cases is likely to be nearer to the truth than one based on "proved" cases alone; even so it is more likely to be an underestimate than otherwise."

On the basis of these figures, Haynes estimated that there were about 56,000 cases of tuberculosis in Kenya in 1948.

WILCOCKS (62A) figures are for cases of tuberculosis proved by sputum examination or by radiography. His estimate for the prevalence of tuberculous disease in Tanganyika in 1938 was 11.55%, which is practically identical with that given by Haynes for Kenya ten years later.

The earliest figures relating to Africans working under stress are those given in table 10 for miners in Southern Rhodesia.

TABLE 10.

<u>Year.</u>	<u>Number Employed</u>	<u>Incidence of Tuberculosis per 100,000</u>
1912	34,494	338
1913	23,543	572
1916	37,928	161
1920	37,669	185
1921	37,605	342

In Table 11 are quoted figures for the attack rate of Pulmonary Tuberculosis among the miners on the Rand.

TABLE 11.

<u>Year.</u>	<u>Number Employed.</u>	<u>Incidence of Pulmonary Tuberculosis per 100,000.</u>
1926-27	180,461	422
1927-28	191,486	390
1928-29	193,493	314

CUMMINS (16A) gives the attack rate of tuberculous disease in the South African Labour Corps in France during the 1914-18 war as 2,907 per 100,000 per annum. BORREL (6) found the attack rate in Senegalese recruits in 1918 was 3,000 per 100,000 per annum; and in Senegalese battalions containing seasoned troops it was 3,900 per 100,000 per annum. It can be inferred from an article by RODHAIN (51) that the attack rate in LEOPOLDSVILLE in 1925 was 3,600 per 100,000 per annum.

The earliest figures relating to highly selected groups of Africans are those given by STONES(53A)



for admissions to the C.M.S.Hospital, Kampala, Uganda - He reports that from 1903-1927, 263 cases of tuberculosis were admitted out of a total of 46,756; and that the admission rate was steady at 0.560%. In 1914, MILLER (45) at Tanga Hospital, Tanganyika admitted 24 cases of Tuberculosis out of a total of 5,612, which gives an admission rate of 0.427%. In 1927, GILLAN (24) admitted 54 cases of tuberculosis to the Church of Scotland Mission Hospital at Tumutumu, Kenya out of a total of 400 which gives an admission rate of 13.5%.

These three groups of figures are not, of course, directly comparable. But each group has its own particular interest. The least interesting are those for admissions to hospital. Such figures are always affected by the personal interests of the medical staff, by the availability of hospital beds and by the fact that only the really sick seek admission. At the time when Haynes estimated that there were 56,000 cases of tuberculosis in Kenya the total hospital beds available for the treatment of all forms of disease was in the region of 2000. In consequence, cases of tuberculosis were seldom admitted.

The figures of Wilcocks and of Haynes give some idea of the magnitude of the problem which faces the Colonial Medical Service in East Africa.

The figures of Borrel and of Cummins and those from the mines in South Africa and Southern Rhodesia are of particular interest to anyone who may have to

deal with African natives under stress. But they are affected by the highly abnormal conditions under which the natives were living and working.

It would clearly be of interest if figures could be obtained for a group of natives living under good hygienic conditions and doing regular but not excessive work. Such figures are presented in the next chapter.

it of about 20% per annum due to discharged and the arrival of recruits.

During this period, 40 cases of Pulmonary Tuberculosis were admitted to the British Military Hospital at Enkhuizen Road of which 20 cases came from the military population and 20 cases came from the civilian population. 18 cases were admitted during the first twelve months and 22 cases during the second twelve months. During each annual period exactly half the cases were of military origin and half of civilian origin.

It is believed that these 40 cases were the only cases of Pulmonary Tuberculosis that occurred during the period under review. The chests of over 800 other Africans with respiratory or general symptoms were examined by radiography during the period under review and no tuberculous lesions were found.

Table II has been compiled to show the attack rate of pulmonary tuberculosis during this period.

CHAPTER 4. THE INCIDENCE OF PULMONARY TUBERCULOSIS AT MACKINNON ROAD.

From 1 May 1949 to 31 April 1951 there was in MACKINNON ROAD an African native population of 7,000. This was composed of a military population of 4,800 and a civilian population, organised on military lines of 2,200. Although the total population remained constant there was a turnover within it of about 30% per annum due to discharges and the arrival of recruits.

During this period, 40 cases of Pulmonary Tuberculosis were admitted to the British Military Hospital at Mackinnon Road of which 20 cases came from the military population and 20 cases came from the civilian population. 16 cases were admitted during the first twelve months and 24 cases during the second twelve months. During each annual period exactly half the cases were of military origin and half of civilian origin.

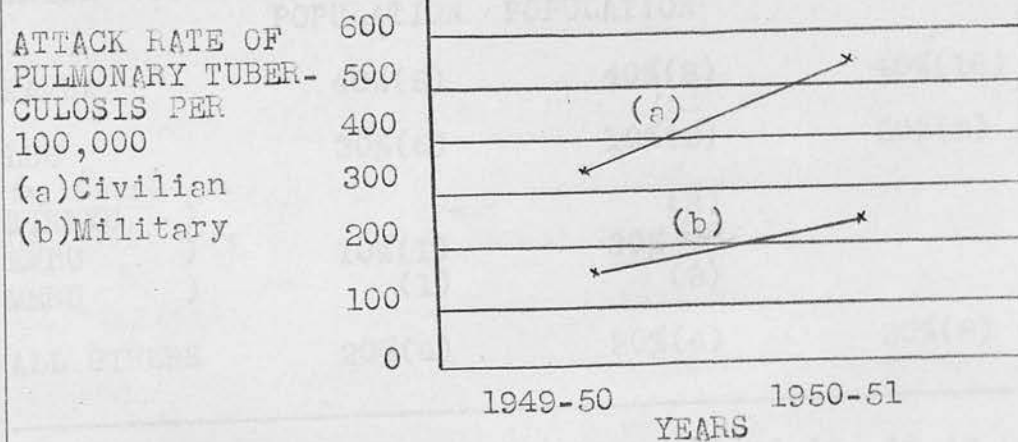
It is believed that these 40 cases were the only cases of Pulmonary Tuberculosis that occurred during the period under review. The chests of over 600 other Africans with respiratory or general symptoms were examined by radiography during the period under review and no tuberculous lesions were found.

Table 12 has been compiled to show the attack rate of pulmonary tuberculosis during this period.

TABLE 12.

YEAR.	POPULATION AT RISK			CASES OF PULMONARY TUBERCULOSIS			INCIDENCE OF PULMONARY TB PER 100,000		
	(a) Mil.	(b) Civ.	(c) Totals	(d) Mil.	(e) Civ.	(f) Total.	(g) Mil.	(h) Civ.	(j) Total.
May 49-Apr 50	4800	2200	7000	8	8	16	166	363	229
May 50-Apr 51	4800	2200	7000	12	12	24	250	545	343
Averages	4800	2200	7000	10	10	20	208	454	286

Consideration of table 12 reveals two important points: the attack rate of tuberculosis amongst the military population was less than half the attack rate amongst the civilian population during both annual periods; and the attack rate increased in both populations during the second year. This second point is illustrated graphically by figure 1, which shows that the rate of increase was greater in the civilian population.



With regard to the difference between the incidence of tuberculosis in the military and civilian populations, the possibility of a difference in tribal composition was considered as an explanation. Table 13 shows the percentage tribal compositions of the two populations.



TABLE 13.

TRIBE	MILITARY POPULATION	CIVILIAN POPULATION	AVERAGE
KAMBA	39%	41%	40%
LUO	33%	38%	35.5%
KIKUYU )	10%	19%	14.5%
EMBU )			
MERU )			
ALL OTHERS	18%	2%	10%

Table 14 shows the tribal distribution of the cases of Pulmonary Tuberculosis expressed as percentages of the total number of cases. The actual numbers of cases are given in brackets after the percentages.

TABLE 14.

TRIBE	MILITARY POPULATION	CIVILIAN POPULATION	AVERAGE
KAMBA	40%(8)	40%(8)	40%(16)
LUO	30%(6)	10%(2)	20%(8)
KIKUYU )	10%(1)	(3)	
EMBU )		30%	
MERU )		(1)	
ALL OTHERS	20%(4)	20%(4)	20%(8)

From a comparison of tables 13 and 14, it is clear that the tribal distribution of cases from the military population is what would be expected if all the tribes were equally susceptible to tuberculosis. On the other hand, this is not true of the cases drawn from the civilian population. But the number of cases is so small that it is not thought that this

discrepancy is due to anything save chance. It is concluded, therefore, that differences in tribal composition play little if any part in explaining the difference between the attack rates of tuberculosis in the two populations.

STOTT (54) in an article on Tuberculosis in Native Prisoners, was also unable to convince himself that a racial factor was an important cause of the difference in incidence between the various East African tribes.

It is considered more likely that both the difference in the attack rates and the difference in the rates of increase of the attack rates are due to the less stringent hygienic discipline, which, as was pointed out in chapter one, existed among the civilian population.

The chief interest of the figures presented in table 12 lies not in the difference between the attack rates in the two populations at risk but in the difference between these attack rates and the attack rates recorded during the 1914-18 war and also those reported from the mines in Southern Rhodesia and in South Africa. For ease of comparison all these figures are grouped together in table 15.

TABLE 15.

YEAR	MATERIAL	POPULATION AT RISK.	ATTACK RATE Per 100,000.
1912	Miners in S.Rhodesia	34,494	383
1913	"	33,543	572
1914-18	S.African Labour Corps	-	2907
1918	Senegalese Troops	-	3900

TABLE 15 (Contd:)

YEAR	MATERIAL	POPULATION AT RISK.	ATTACK RATE Per 100,000
1920	Miners in S.Rhodesia	37,669	185
1921	"	37,605	342
1926-27	Miners in S.Africa	180,461	422
1927-28	"	191,486	390
1928-29	Miners in S.Africa	193,493	314
1949-50	East African Troops	4,800	166
1950-51	"	4,800	250
1949-50	East African Civilians	2,200	363
1950-51	"	2,200	545

A study of this table reveals that the attack rates for all the populations at risk in their own continent is of the same order, whereas the attack rates for the two populations transferred by the exigencies of war to Europe is roughly ten times greater.

The explanation given by both BORREL(6) and CUMMINS (16A) for the high attack rates in the Senegalese and in the South African Labour Corps was that these troops were virgin soil. In chapter 2, reasons have been given for doubting the validity of the virgin soil hypothesis. There now seems no necessity to invoke this hypothesis to explain the great increase in the attack rates in African native troops serving outside Africa. It seems much more probable that the difference is due to the lowering of their resistance to tuberculosis by climatic conditions, to which they were unaccustomed, and

particularly by the colder climate. Even in their own country, East African natives are highly susceptible to climatic changes and the cold wet season of the long rains from March to June is commonly marked by a great increase in respiratory infections.

Commenting on his own figures, Borrel speaks of "la bonne hygiène des camps." But he makes no reference to the hygienic conditions on board the transports which brought the troops from Africa to Europe. Judging by modern troopships, it seems highly probable that the disease could have spread rapidly even during the short journey from North Africa to the South of France. In this connection, Stott, in the paper already referred to, stresses the importance of overcrowding as a cause of the epidemic of tuberculosis in Nakuru jail.

During the period under consideration, the European population of Mackinnon Road was constant at 1200; there were also about 800 Mauritian and Seychellois troops. No case of Pulmonary Tuberculosis occurred amongst the Europeans and only one or two cases amongst the Mauritians and Seychellois. There can be little doubt, therefore, that those Africans who developed tuberculosis were infected by other Africans and not by members of other races.

It is concluded that the pessimistic outlook engendered by the earlier workers on tuberculosis in African natives is no longer justified; and that in





the event of large numbers of East African natives being called to the colours the tuberculosis problem, which will face the medical services, is likely to be of a size with which they can cope without much difficulty, provided East African troops are not called upon to serve outside the tropics.

The minds of most people are too far removed from the clinical course of the disease to have been overshadowed by the descriptions of the post mortem findings. This was resulted in an attitude of extreme pessimism in all but a few clinicians.

ROBERTS (44) in 1918 stated that the four common diseases in Leopoldville were Trypanosomiasis, Malaria, Dysentery and Tuberculosis in that order. He pointed out that cases of Tuberculosis frequently presented with pneumonia or dysentery and correlated this fact with the predominance of pulmonary and intestinal lesions of post mortem. In 31 cases, in which death was due to tuberculosis in 19 cases and in which tuberculosis was an incidental finding in 8 cases, pulmonary lesions were present in 26 cases, intestinal lesions in 13 cases, miliary tuberculosis occurred in 12 cases, the serous cavities were involved in 7 cases and the liver, spleen or kidneys in 9 cases. He pointed out that in the lungs the apices were not the sites of election as in Europeans; that tuberculous foci were usually widely scattered, especially in the lower and middle zones and were usually small, though some

CHAPTER 5. THE NATURAL HISTORY OF TUBERCULOSIS:  
PREVIOUS WORK.

Most of the papers dealing with this subject are mainly concerned with the pathology of the disease in African natives as it is seen in the post mortem room. A few of the papers also refer to the clinical course of the disease. But in the minds of most people the few references to the clinical course of the disease appear to have been overshadowed by the descriptions of the post mortem findings. This has resulted in an attitude of extreme pessimism in all but a few clinicians.

MOUCHET (44) in 1913 stated that the four common diseases in Leopoldville were Trypanosomiasis, Pneumonia, Dysentery and Tuberculosis in that order. He pointed out that cases of Tuberculosis frequently presented with pneumonia or dysentery and correlated this fact with the predominance of pulmonary and intestinal lesions at post mortem. In 31 cases, in which death was due to tuberculosis in 29 cases and in which tuberculosis was an incidental finding in 2 cases, pulmonary lesions were present in 26 cases, intestinal lesions in 19 cases, miliary tuberculosis occurred in 12 cases, the serous cavities were involved in 7 cases and the liver, spleen or kidneys in 7 cases. He pointed out that in the lungs the apices were not the sites of election as in Europeans; that tuberculous foci were usually widely scattered, caseating, and tended to coalesce and form cavities, though such

cavities were seldom large. He considered that the intestinal lesions were secondary to the pulmonary disease and were similar in character to those found in Europeans. The lesions in the liver, spleen and kidney were caseating foci about the size of a cherry and those in the kidneys had not cavitated.

He writes: "Enfin, nous devons signaler que dans toutes les observations sauf une, il y avait des gros foyers ganglionnaires caséux mais non calcifiés siégeant le plus souvent dans le médiastin ou dans les hiles pulmonaires; parfois aussi dans le mésentère ou au devant de la colonne vertébrale."

His conclusions are: "L'évolution lente et progressive de la tuberculose, cas le plus fréquent chez l'européen, est beaucoup plus rare chez l'indigène du Congo.

La tuberculose se cantonnerait d'abord dans une groupe ganglionnaire et de là, a l'occasion d'une diminution de résistance quelconque, prendrait une forme offensive et se développerait.

Mais une fois lancée, l'évolution de la tuberculose apparait rapide. La rareté des adhérences pulmonaires, la dissémination sans régularité des lésions intestinales, l'abondance des bacilles dans les lésions, jointe au petit nombre de cellules géantes, plaident nettement en faveur d'une marche rapide ne donnant pas aux lésions la gradation d'âge, si fréquente chez les Européens, et fait supposer une résistance moindre de l'indigène vis-à-vis de la maladie.

La rareté des tuberculoses rénales pures et des longues tuberculoses osseuses s'ajoute aux arguments précédents pour confirmer la valeur."

This admirable paper has been overshadowed by that published in 1920 by Borrel, whose views have held sway ever since. BORREL(6) studied the disease in all its aspects as it appeared amongst Senegalese troops during the latter part of the 1914-1918 war. The most important observations in his paper are seldom mentioned. They are these: Le service de dépistage a donc sorti prématurément des bataillons 970 malades tuberculeux, dont 500 environ ont été rapatriés encore en bon état ---- les rapatriés tuberculeux étaient au début de la tuberculose, généralement non fébrile, et on peut espérer qu'une forte proportion pourra se rétablir dans la colonie d'origine!"

Earlier in his paper he had written: "Je puis dire déjà qu'un très grand nombre de ces Tirailleurs mis au repos tout de suite, à la suralimentation, à l'huile de Foie de Morue, paraissent, après deux et trois mois d'observation, définitivement sauvés: malgré tout, un certain nombre, 50% continuent l'évolution tuberculeuse mais on peut estimer à 50% les gains en vies humaines obtenus."

In other words, in half of those Senegalese suffering from tuberculosis the disease was arrested clinically. This is the most important fact established by Borrel.



From the point of view of diagnosis Borrel lays stress on the enlargement of the supra clavicular glands; the loss of the normal silky sheen of the skin; the loss of subcutaneous fat and the atonic condition of the muscles; and he points out that the signs in the chest are seldom detected until the late stages of the disease.

He divides the course of the disease into two stages: "Une période initiale, ganglionnaire, latente pendant laquelle l'état général peut n'être pas sensiblement modifié au début; il n'y a pas de fièvre, et ce n'est que peu à peu que les modifications caractéristiques de l'état général surviennent, cette période latente peut durer un, deux, trois mois;

"Une période seconde pendant laquelle apparaissent des symptômes nouveaux et tout de suite très graves: fièvre, amaigrissement, lésions généralisées, se traduisant par des lésions pulmonaires locales rapidement envahissantes: pneumonie caséuse lobaire, pneumonie lobulaire caséuse, localisation sur les séreuses, tuberculose pleurale, pleuroperitonéale, granulie initiale ou granulie survenant au cours de pneumonies caséuses ou granulie survenant après un envahissement des séreuses; cette période est très courte et, dans l'immense majorité des cas, à partir du moment où la fièvre s'allume la mort survient en quinze jours, un mois, deux mois au plus. La Tuberculose Sénégalaise prend rarement la forme chronique, locale, pulmonaire avec cavernes qui

caractérise la tuberculose de l'Européen."

Borrel carried out 500 post-mortem examinations. He found that the initial lesion was a tuberculous chancre in the upper respiratory passages from which in 90% of cases the cervical, supraclavicular, tracheo-bronchial or hilar glands became infected and grossly enlarged. In only 5% of cases was the primary lesion in the lungs.

In 30% of cases, rupture of the caseous glands had given rise to a caseous lobar pneumonia.

In 25% of cases, generalised miliary spread had resulted from the rupture of a caseous gland into the blood stream.

In 20% of cases, pneumonia and miliary tuberculosis coincided in the same lung.

In 10% of cases, the serous cavities were involved.

In 5% of cases, the lungs showed chronic cavities without enlargement of the tracheobronchial glands.

In a few cases, the route of infection had been intestinal and enlargement of the mesenteric glands with ascites had occurred in the absence of thoracic lesions.

In those cases in which miliary spread had occurred the spleen was remarkable; it was enormous and studded with tubercles the size of walnuts.

It is clear that these findings, though more detailed, are very similar to the findings of Mouchet.

Borrel was a great man and had the ability to

generalise from his findings. He wrote: "La tuberculose des troupes noires Sénégalaises est tout à fait spéciale: Elle est particulièrement grave et le Sénégalais est particulièrement sensible, à cause des conditions de sa vie antérieure à la colonie. Il représente un terrain vierge au point de vue tuberculeux, il n'a jamais été en contact pendant sa jeunesse avec le bacille tuberculeux. La tuberculose qu'il contracte par intercontagion dans la promiscuité des baraquements évolue suivant un type suraigu qui rappelle la tuberculose de l'enfant.

"Il nous paraît donc bien démontré que la sensibilité du Sénégalais à la tuberculose tient à l'absence d'immunité acquise.

"Réunis et vivant en cohabitation intime, ils sont soumis à toutes les contagions qui résultent de la promiscuité des baraquements et de la vie en commun. Les cas de tuberculose vont se multipliant avec la durée du séjour et de cette vie commune."

It is important to stress Borrel's first conclusion - that tuberculosis as it appeared in the Senegalese troops was of a special type - because the pessimistic attitude towards tuberculosis in other African races, which is now so common, has been largely engendered by applying his conclusions to all the native inhabitants of Africa.

In the South African Report published in 1932 there is little of interest about the clinical course of the disease. The importance of weighing the miners

at regular intervals in assisting early diagnosis is stressed. The most interesting contribution in this report deals with the question of hypersensitivity. It is clearly shown that the incidence of tuberculous disease was greatest in those natives who were sensitive to the highest dilutions of tuberculin; but there was no demonstrable relationship between the degree of sensitivity and the type of tuberculosis that ensued.

600 post-mortem examinations were performed - 200 cases were complicated by silicosis and in 62 cases tuberculosis was merely an incidental finding. In 338 cases of pure tuberculosis caseating lesions were found in the lungs in 172 cases (51%) of which 102 showed cavitation (30%); acute military tuberculosis was found in 53 cases (16%); in 16 cases (5%) the lesions were in the serous cavities; and in 27 cases (28%) the lesions were extra thoracic. In most cases glandular enlargement was marked but in only 58 cases were the supraclavicular glands on which Borrel laid such stress, involved. Involvement of the liver and spleen was also common.

"The military cases were of such a character as to suggest that spread had occurred from the thoracic glands though in every case the abdominal organs as well as the lungs were involved. Much of the military tuberculosis of the lungs was really a caseous acinar pneumonia rather than a military tuberculosis sensu stricto.



"Of the lung caseations, the great majority were of bronchopneumonic type, although in at least six or seven of these there were more or less extensive areas of broncho pneumonia giving rise to appearances very similar to true pneumonic phthisis. In a few, the spread was more of a direct local one in the neighbourhood of infected hilar glands.

The infection of the glands was the most characteristic part of the picture."

As in the case of Borrel's paper, the most important finding in the report is contained in a single paragraph only too easily overlooked. "It should be borne in mind that the (pathological) report only deals with .... approximately one sixth of the total number of cases of tuberculosis and/or silicosis diagnosed in this population viz those cases which die on the Rand. The other five sixths are repatriated and practically no pathological information is available regarding them. From Allan's work in the Transkei, however, it is known that although many of the repatriates die within comparatively short periods of their return home, others live for long periods and some even return to the mines and are passed for work there. It would seem to be a fairly safe assumption therefore that amongst the repatriates there are cases of a more chronic type."

Of those repatriated, 10% died within two months, 50% within one year and 60% within two years.

It is probable therefore that in a quarter of those in whom the disease develops it eventually becomes arrested.

From a comparison of the post mortem findings in the two papers, it is clear that the disease as it occurred amongst the Rand miners exhibited differences - quantitative rather than qualitative - from the disease as it occurred amongst Borrel's Senegalese. In the Rand miners, glandular enlargement appears to have been equally prominent but time appears to have been allowed for the development of extensive changes in the lungs in a greater number of cases. Acute military spread was less common but chronic haematogenous lesions were encountered more frequently. The impression gained is that there was definitely some degree of resistance to the disease even in the fatal cases amongst the Rand miners.

In view of the high percentage of tuberculin positive reactors amongst the Rand miners and the fact that tuberculous disease developed most commonly in those most sensitive to high dilutions of tuberculin, CUMMINS (49 and 16B) elaborated his theory of larval tuberculosis. According to him, the majority of the miners were already infected in their native reserves. The disease had not however been overcome but was smouldering - particularly in the lymphatic glands. Under the stress of working in the mines, the resistance of the miners to the disease diminished and further spread of the disease

occurred.

VINT (59 A & B) has published two papers, in 1929 and 1937 respectively, dealing with his findings at post mortem examinations on East African Natives of all ages. He found tuberculosis present in 192 out of 1000 cases. In 132 cases it was the cause of death; miliary disease was present in 67 cases and cavities in the lungs in 37. In the 60 cases, in which tuberculosis was an incidental finding, the lesions were active in 46 and healed in 14. He comments on the rarity of fibrous tissue formation, the small size of the cavities, the frequency of glandular enlargement and its resemblance to Hodgkins disease.

In 1947, DAVIES (19) published a paper dealing with his post mortem findings in 432 cases of Tuberculosis. The lungs were involved in 362 cases; in 70 cases the lesions were extra-pulmonary.

Of the pulmonary cases, in only 97 were the lesions confined to the lungs. In the remainder, spread had occurred to the spleen, liver, serous cavities, kidney, meninges, myocardium, bones, brain and joints in descending order of frequency.

Of the pulmonary cases, 8 showed a primary focus only; 9 showed a primary focus with associated glandular enlargement; 146 showed glandular enlargement without a macroscopic primary focus; in 5 cases there were chronic fibroid lesions.

In the pulmonary cases, the distribution of the

initial tuberculous lesions in the lungs was: pan-pulmonary 171; right apex 22; left apex 31; both apices 30; right base 11; left base 10; both bases 6; apex and base 22; whole right lung 26; whole left lung 21; collapsed lungs 12.

In the extrapulmonary cases, there was glandular involvement in most cases; the serous membranes were involved in 27 cases; the liver, spleen or kidneys in 16 cases; and the meninges in 14 cases.

Davies writes: "In Kampala, as seen at post mortem, tuberculosis is of an extremely acute type. The usual picture is that of a purulent infection without cavitation but with the formation of ragged thin walled abscesses filled with liquid semi-caseous pus. Rarely is there any attempt at limitation. The abscesses break down into the bronchi and broncho-pneumonic spread is almost invariable. Laryngeal and tracheal tuberculosis and intestinal tuberculous ulceration occur in nearly every case at autopsy. Tuberculous lobar pneumonia is also common. Miliary dissemination is common with a great number of distal tuberculous metastases.

"The anatomical evidence is clear. It shows that tuberculosis in Uganda is primitive tuberculosis, its intensity in some cases almost approaching that found by Borrel in the Senegalese. It goes to show that the vast majority of Uganda Africans never meet a tubercle bacillus throughout their lives. A very few develop a primary complex, which they heal and



even calcify, but even these are not free from the dangers of death from tuberculosis. The rest develop a massive primary complex which may be situated anywhere in the lungs. From the regional lymphatic glands dissemination takes place to any or all the other lymphatic groups which may become enlarged and caseate. From the peripheral focus or from the glands, breakdown leads to haematogenous or bronchopneumonic spread - Widespread dissemination takes place throughout the body, the whole process is very rapid and death soon takes place."

Davies was, of course, quite unjustified in concluding from his post mortem data that "the vast majority of Uganda Africans never meet a tubercle bacillus throughout their lives." How wrong he was the Tuberculin Sensitivity Survey carried out three years later by Santon Gilmour has shown.

The explanation is that both Davies and Vint's findings are heavily biased for the same reason as those recorded in the South African Report: the majority of natives with tuberculosis are sent back to die or recover in the reserves and only those too ill to travel are admitted to hospital.

In 1946, OSWALD (46) published a paper summarising his experience of Pulmonary Tuberculosis in African Native Troops during the 1939-45 war. In the section devoted to pathology he deals with the autopsy findings in 50 cases. 1 case showed military tuberculosis of the lungs, extensive tuberculous

peritonitis and gross caseous involvement of the cervical, mediastinal and abdominal lymph glands but no macroscopic evidence of tuberculosis in the liver, spleen, and kidneys. A second case showed military tuberculosis of the lungs, liver, spleen and kidneys and a large pleural effusion but only a few caseous mediastinal glands. One patient showed involvement of all the serous membranes, numerous tubercles in both lungs and caseation in the abdominal glands.

The remaining 47 cases showed acute caseating pulmonary tuberculosis which was bilateral in 46 cases. Cavitation had occurred in 46 cases and was bilateral in 26. The cavities had ragged walls and contained large quantities of pus, caseous material and debris. Extensive pleural adhesions were an almost constant finding and large pleural effusions were present in 15 cases.

Glandular enlargement of some degree in either the cervical, mediastinal or abdominal region was noted in 96% of cases and caseation was present in 66% in at least one of these three regions. Oswald considered this incidence "far in excess of that found in Europeans of similar age: it is characteristic of the disease in negroes and is similar to the findings in both the South African mine labourers and Borrel's Senegalese troops."

Tuberculous involvement was evident to the naked eye in the spleen in 20% of cases, the kidneys in 8%

and the liver in 6%. Ulceration of the ileum was found in 50% of cases.

Of the 50 cases examined post mortem, 38 came from the High Commissioned Territories of South Africa, 7 were Cape Coloured, 5 were from the three East African Territories and 1 from West Africa. It is not surprising, therefore, that the autopsy findings resembled those in the Rand miners.

In contrast to and partial confirmation of these various post mortem reports may be set the clinical and radiological findings of WILCOCKS(62A). He carried out radiological examinations in 446 natives and found calcified nodules in 206 cases. In 105 cases there were no other lesions. In 101 cases the other changes were: suspicious foci in 26 cases; pleural adhesions or fibrosis in 10 cases; and tuberculous infiltration in 65 cases. He comments: "the finding of calcified nodules in lungs containing tuberculous infiltration proves that, in these cases, first infection did not lead on immediately to gross disease."

In 250 cases with radiological evidence of tuberculous infiltration, cavitation - often multiple and without fibrosis - was seen in 111 cases; and miliary spread was seen in 52 cases. But pleural thickening was found in 91 cases; thickening of the right interlobar septum in 52 cases; and fibrosis in the lung fields in 43 cases. In short, there was evidence of some attempt at healing in 186 cases.

His findings are in agreement with those of Davies as far as the distribution of the lesions goes.

He found the right upper zone involved in 185 cases; the left upper zone in 135 cases; the right middle zone in 174 cases; the left middle zone in 170 cases; the right lower zone in 107 cases; and the left lower zone in 87 cases. A fairly common picture was infiltration in the upper part of the right lung with a fan shaped spread in the middle of the left lung. The disease was bilateral in 171 cases; involved the right lung only in 54 cases; and involved the left lung only in 25 cases. There was definite or suspicious glandular enlargement in about 100 cases.

OSWALD (46) based his clinical findings on a study of 416 patients. He found that the cases fell into four easily distinguishable groups: parenchymal, glandular, serous and miliary.

There were only 3 cases of miliary tuberculosis. Two of these cases died and have already been referred to in detail. The third case had miliary lesions in both lung fields with little constitutional upset and was evacuated to South Africa.

The serous group contained 78 cases. All cases had a pleural effusion but in 14 cases there was polyserositis. The clinical course of those patients with unilateral pleural effusion only was similar to that seen in young adult Europeans. But as soon as more than one serous cavity became involved the prognosis became considerably less favourable.

The glandular group comprised 53 cases in which the most prominent feature in the chest,



radiologically, was the presence of enlarged mediastinal glands. The degree of enlargement varied from "prominent hilar shadows" to large masses of glands occupying a considerable part of the chest. The enlargement was bilateral in 27 of the cases. The clinical course depended upon the type and extent of the parenchymal lesions. When the disease appeared to be limited to the mediastinal glands there was usually little evidence of toxæmia. Oswald was of opinion that "the prognosis depends almost entirely upon whether the disease can be contained within the mediastinal glands. If it can, there is every reason to anticipate a favourable outcome, but once the lungs have become seriously affected there is little hope of arresting the progress of the disease."

The parenchymal group was made up of 282 cases (68% of the total) and was subdivided into acute; subacute, and chronic.

There were 212 cases in the acute parenchymal subgroup. The lesions were predominantly exudative and spread rapidly with little respect for interlobar boundaries; cavitation and bronchogenic spread were common. In the most acute cases after a hectic course with prostration, high fever and emaciation, death occurred in one to three months.

In those patients who survived for more than three months, cavitation was marked. Prior to death or repatriation, cavities were seen in 180 cases.

25% of these patients were too ill for repatria-

tion and therefore remained in hospital until they died. Of the remainder, Oswald stated "it is most unlikely that even half of them would live for a year from the time of evacuation."

The subacute parenchymal subgroup totalled 49 cases. The initial lesion was in one or both upper zones in all but 2 cases. Cavitation had occurred in 21 cases. Oswald considered "the ultimate prognosis was comparable with that in a similar group of Europeans."

The chronic parenchymal subgroup contained 21 patients with scattered infiltration in one or both upper zones. No patient showed radiological evidence of cavitation and the majority had normal sedimentation rates.

Table 16 shows the incidence of the four clinical types of disease in the four groups of Africans comprising Oswald's material.

TABLE 16

TYPE OF DISEASE	CAPE COLOURED	HIGH COMMISSIONED TERRITORIES	EAST AFRICANS	WEST AFRICANS
PARENCHYMAL:				
(a) ACUTE	54%	60%	43%	22%
(b) SUBACUTE	25%	8%	11%	6%
(c) CHRONIC	9%	6%	1%	6%
GLANDULAR	1%	9%	19%	38%
SEROUS	10%	15%	27%	28%
MILIARY	1%	2%	-	-

This table illustrates well the fact that tuberculosis does not develop in the same manner

in Africans from different parts of the continent. In the South African natives glandular and serous tuberculosis forms less than 25% of the cases; in the West African natives these types account for 66% of the cases; the East African natives appear to come midway between the natives of South and West Africa in their response to tuberculous disease.

A similar incidence of glandular involvement was reported from Nigeria by JONES (30) in 1949. In 126 autopsies tuberculosis was the cause of death in 36 cases and an incidental finding in 19 cases. Gross glandular involvement was present in 29 of the 55 cases with tuberculous disease.

Of the 36 cases in which tuberculosis was the cause of death, 4 cases showed military tuberculosis of the lungs, spleen, liver and kidneys; 1 case showed tuberculous pericarditis; 8 cases showed tuberculous bronchopneumonia. All these cases had widespread glandular enlargement. The remaining 23 cases had large cavities in one or both upper lobes with bronchogenic spread through the rest of the lungs. These cases showed no glandular enlargement.

Jones was of the opinion that "the violence of the disease in the African must be explained on the basis of his inborn constitutional peculiarities."

The papers, which have been reviewed in the present chapter, were mostly written by men whose chief interests lay in aspects of tuberculosis other than the clinical course of the disease -

namely, the tuberculin sensitivity incidence or the post mortem findings. An attempt to construct a composite picture of the natural history of the disease from these findings may be misleading. It is, nevertheless, worthwhile to do so in order to contrast the composite picture with the actual clinical findings to be detailed in later chapters.

In the first place, then, there is good evidence from the Tuberculin Sensitivity Surveys which have been carried out that most African natives, becoming infected in their natural surroundings, are able to arrest the disease during the primary stage. In a small number the disease is not arrested and after remaining indolent for a varying length of time it becomes florid or widely disseminated and death rapidly ensues. In the majority, the initial infection is followed by the development of a very marked sensitivity to the tubercle bacillus and to tuberculo-protein so that, when reinfection occurs or a larval focus breaks down, the ensuing disease is acute and the local destruction of tissue is so great that the likelihood of arrest of the disease is small. In a few, the initial infection is not followed by this very marked sensitivity, so that, when reinfection occurs or a larval focus breaks down, the powers of resistance are unimpeded by excessive destruction of tissue and chronic fibroid phthisis ensues. There remain those who are insensitive to intradermal tuberculin tests. Some of these are true virgin soil



and will react to a massive infection in the manner described by Borrel. The remainder are those who have been previously infected but have lost their sensitivity to the tubercle bacillus and its breakdown products. These latter usually retain their resistance to the tubercle bacillus and therefore when reinfected they will develop chronic lesions.

According to the angle from which the problem is viewed so will opinions about the natural history of the disease vary. Viewed from the post mortem rooms of general hospitals, which only admit such cases of tuberculosis as they cannot get rid of, the existence of thousands of natives with arrested disease is unsuspected. Viewed from the angle of the Tuberculin Sensitivity Surveyor the hypersensitivity, so frequently found, bodes ill for such individuals as develop the disease. To the historian delving into a literature dominated by the autopsy findings the outlook is also gloomy. But the practising physician, as will be shown in the succeeding chapters, is entitled to temper this outlook with restrained optimism.

CHAPTER 6. THE NATURAL HISTORY OF TUBERCULOSIS:  
CLASSIFICATION OF CASES.

The conclusions, which are to be detailed in the next four chapters, are based upon the study of fifty four patients admitted to British Military Hospitals in East Africa between May 1949 and December 1951. Thirty four of the patients were soldiers and twenty were civilians serving in a paramilitary unit. The material was therefore highly selected. This point has already been referred to in detail in the first chapter.

The classification of these cases has been no easy task. The classification, that is now put forward, is the result of several revisions and is tendered in no dogmatic manner, for it may well be that several of the cases are still incorrectly classified.

The cases are grouped according to the stage of the natural history of the disease which they appear to illustrate. These stages are: (a) the primary stage; (b) the serous stage; (c) the intermediate stage; (d) the reinfection stage.

The criteria adopted for allotting cases to the primary stage have been the presence either in the radiograph or at autopsy of a primary focus and of enlarged lymphatic glands.

The criterion adopted for allotting cases to the serous stage has been the presence of an effusion in one or more of the serous cavities. Radiographic evidence of an effusion was confirmed in all cases

by paracentesis.

Cases in which there was radiological or post mortem evidence of the concurrent existence of glandular enlargement and lesions usually regarded as typical of reinfection tuberculosis have been allotted to the intermediate stage.

Cases with lesions usually regarded as typical of reinfection tuberculosis and without any sign of glandular enlargement have been placed in the reinfection stage. The term reinfection is used throughout without any implications whatever as regards the exogenous or the endogenous origin of the reinfection.

There are already many different classifications of tuberculosis. There seemed little point in adding to the confusion by introducing yet another one. And the classification adopted is one in common use, modified by the introduction of the intermediate stage so as to be applicable to Africans.

A great deal of ink has been spilt in the past in attempts to draw a clear distinction between primary and reinfection tuberculosis. But the decisive words on this matter were written in 1936 by PINNER (48): "It is essentially a matter of definition where to draw the line between primary tuberculosis and the reinfection type of tuberculosis. There is, in reality, no sharp dividing line."

This point is well illustrated by the cases placed in the intermediate stage. There are three

possible explanations for the concurrent existence of typical reinfection lesions and enlarged glands in the same individual. It may be claimed that the lesions are not due to reinfection but are examples of progressive primary lesions. The progressive primary focus was described in detail by AUERBACH (3). He reported 17 cases which all occurred in children between the ages of 9 months and 9 years. 1 child was white and 16 were American negroes; 15 of the children were under 3 years of age.

PAGEL (31) states that primary tuberculosis in adults obeys the laws of the primary complex as seen in children and gives twelve examples of progressive primary disease in adults.

Secondly it may be claimed that the lesions are true reinfection lesions and that the glandular enlargement is part of the process of reinfection. Such a claim would cut right across the classical concepts of the process of tuberculous reinfection. It would almost constitute iconoclasm for its own sake.

Thirdly it may be claimed that in East Africans the resolution of the tuberculous process in the enlarged glands is a very gradual process and that in these intermediate cases this process is still occurring at the time when the reinfection lesions develop. This explanation seems the most satisfactory of the three. It involves no real break with the classical teaching and it underlines the wisdom of Pinner's dictum.



The cases grouped together in the serous stage are mostly primary infections which have become complicated by pleural effusions. But a few of the cases appear to have underlying reinfection lesions. It is for this reason that the term serous stage has been used in place of post-primary stage.

A comparison of the radiographs of some of the cases with florid disease in the group of reinfection cases with other cases in the groups of primary and intermediate cases shows that it may be impossible to decide in which group a case should be placed, by studying the radiological evidence alone. In several cases post mortem evidence required the transfer of a case from one group to another.

The question may therefore well be asked: does the differentiation of tuberculous disease in East African natives into primary and reinfection types have any practical importance? Oswald, for example, made no attempt to do so and classified his cases as glandular, serous and parenchymal.

The desire to bring order out of chaos appears to be an inherent characteristic of man. In studying a collection of case notes, some form of classification is necessary. To be of value the groups in a classification of any given disease should have a definite relation to pathology, to prognosis and to treatment. These points will be dealt with in detail in the ensuing chapters. Here it must suffice to say that the classification outlined above appears to fulfil its proper function.

CHAPTER 7. THE NATURAL HISTORY OF TUBERCULOSIS: THE PRIMARY STAGE.

Seven cases illustrate the primary stage of Tuberculosis in the East African Native.

One of these patients - Private MAKARANGA (Appendix A, Case 1) - suffered from the type of disease described by Borrel. He was admitted to hospital on account of a hydrocele; was found to have falciparum malaria and urinary schistosomiasis; developed an intra-abdominal mass and later a swelling in his left supraclavicular fossa; and died two months after admission. At autopsy, a primary tuberculous lesion was found in the ileum; the mesenteric, para-aortic, mediastinal and supraclavicular glands were all enlarged and many of them were caseous. A small tuberculous focus was found extending from the meninges into the left occipital lobe of the brain, but there was no evidence of generalised spread.

This case exemplifies the progress of primary tuberculosis in a patient, who either had no resistance or whose resistance was markedly lowered by other diseases. The post mortem appearances were astounding and provided ample confirmation of the brilliance of the descriptions given by Borrel.

One of the most interesting patients in the whole series was Private RUGONGO (Appendix A, Case 2). At the time of his admission to hospital early in January 1950, there was a small focus visible only with difficulty as the periphery of the right mid

zone. In the course of three weeks this focus enlarged and became wedge shaped and a lateral radiograph showed that it lay in the posterior and apical segments of the right upper lobe. During this period the right hilar glands also became enlarged.

During the next six weeks this wedge shaped opacity gradually extended towards the hilum but retained its shape. This change in the radiological appearances was interpreted as being due to the gradual centripetal spread of the disease along the segmental bronchi.

Shortly after this there was a marked extension of the area of consolidation in the right lung and by the middle of March bronchogenic spread had occurred into the left lung.

While these changes were occurring the patients general condition was slowly deteriorating and by the middle of May it was clear that, unless more active steps were taken he would in all probability die. Accordingly a right phrenic crush was performed and reinforced with a pneumoperitoneum. Both the radiological appearances and his general condition remained static after this until mid July when very slowly improvement began. By December, the patient was looking extremely well and his sputum was negative.

At this time the radiograph showed that the lesions in the left lung were still present and that a large opacity remained in the right lung field.

Streptomycin had by this time become available in small quantities. This patient was given 1 G daily for 60 days. The first result of this therapy was the reappearance of tubercle bacilli in his sputum. This was interpreted as being the result of widening of the lumina of the segmental bronchi due to healing of the mucosal lesions.

By the time the course of Streptomycin was completed, the patient had gained 10 lbs in weight, his E.S.R. had fallen to normal and clearing had started in the radiological appearances. By May 1951 his sputum had again become negative and clearing of the lesions was still progressing.

The pneumoperitoneum was maintained until the middle of July and was then allowed to become absorbed. The patient was discharged to the reserves in September when it was considered that the disease had been arrested. He was seen again in February 1952, through the courtesy of Dr. Bisley, the Medical Officer at Meru; at that time he looked very well, he had gained 10 lbs in weight and his E.S.R. was normal.

This case illustrates the progress of the disease in a native whose initially low resistance was built up gradually over a period of many months. It also shows one way in which the fairly common picture of caseous pneumonia may develop. In March 1950 there was a very marked change for the worse in the radiological appearances. It is impossible to be dogmatic about the interpretation of this change. But it is



interesting to speculate whether it marks the onset of local hypersensitivity. There was no evidence of the development of generalised hypersensitivity until May 1950 when the ESR became raised. There was also no evidence of the development of resistance to the disease until July 1950. If this interpretation is correct, this case also provides a beautiful illustration of Rich's view that while hypersensitivity and acquired resistance normally develop *pari passu* they are separate pathological reactions. In this case, the development of hypersensitivity appears to have outstripped the development of acquired resistance.

Civilian KILOBA (Appendix A, Case 3) was admitted to hospital in February 1951 with a large opacity in the right lower zone and associated enlargement of the right hilar glands. After three months in bed, there had been no improvement in the radiological appearances and his ESR was still raised. A right phrenic crush was therefore performed and reinforced by a pneumoperitoneum.

From that time he made slow but steady improvement. But the lung field was still not clear in November. He was then given a course of thiacetazone and this was followed by a rapid improvement in the radiological appearances. The pneumoperitoneum was abandoned early in December and he was discharged to the reserves after Christmas, at which time his disease was considered to be arrested.

This case illustrates the progress of the disease in a patient with sufficient resistance to contain but not to overcome the disease.

Private SIDINYA (Appendix A, Case 4) was admitted to hospital in July 50 with multiple opacities in the left lung field and enlarged left hilar glands. He made steady progress and was discharged to the reserves in February 51, at which time it was considered that his disease was arrested.

Civilian MUSYOKA (Appendix A, Case 5) was admitted to hospital in October 49 with opacities scattered throughout the right mid zone and enlarged hilar glands on the right side. He made steady progress and was discharged to the reserves in March 50 at his own request, although the disease was considered still to be active.

Private DIDIGO (Appendix A, Case 6) was admitted to hospital in July 51 with a large opacity extending from the left hilum outwards into all zones of the left lung field and with associated enlargement of the left hilar glands. He was discharged to the reserves in March 52 when it was considered that his disease was arrested.

These three cases illustrate the process of recovery which occurs in patients with an adequate degree of resistance. In hospital with rest in bed and an adequate diet this takes about seven months. It is probable that in the reserves on a less satisfactory diet the process may take rather longer.

Corporal NGUNDI (Appendix A, Case 7) was first seen at hospital as an outpatient in March 51, when he complained of having been out of sorts since January 51. His radiograph revealed prominent hilar shadows but was considered to be within normal limits and his ESR was normal. He reported for a follow up examination in June 51. His radiograph showed some increase in the size of the left hilar shadow and some indefinite mottling in the left lung field but his ESR was still normal. He returned for a further follow up in August 51 and his radiograph now showed definite opacities in the left mid and lower zones and his ESR was 9 mm/1 hr. He was therefore admitted to hospital where he remained until December 51. He was then discharged to the reserves because his wife had become ill and there was nobody else to look after his children. At that time his lung fields were completely clear but the hilar glands were still slightly enlarged.

The chief interest of this case lies in the fact that this patient had not been feeling well for six months before even minimal changes were recognisable on radiological examination.

From a study of these seven cases the following conclusions may be drawn. Firstly, when natural resistance is absent or depressed on account of other diseases, tuberculous infection rapidly spreads throughout the lymphatic glands and at a later stage becomes generalised by haematogenous spread. The

result is death.

Secondly, if natural resistance is low and acquired resistance is slow to develop, the development of hypersensitivity may result in florid pulmonary lesions. In the absence of treatment, death ensues.

Thirdly, if the forces of resistance and the forces of the disease are evenly balanced an indolent lesion develops which remains unchanged in appearance over many months.

Fourthly, if resistance is high, recovery occurs within seven to twelve months.

Fifthly, the primary focus may be in any part of the lung fields. In these six cases it was in the right upper zone in one case; in the right mid zone in one case; in the right lower zone in one case; in the left mid and lower zones in one case; and in all zones of the left lung field in two cases. The primary focus is more often multiple than single and this suggests that the original infecting dose must be massive.

Sixthly, although the E.S.R. is usually raised, a normal E.S.R. does not exclude the existence of active primary disease.

Finally, the disease probably exists in a sub-clinical form for some months before it is recognised. This is due partly to the indifference with which the native regards the early symptoms and partly to the difficulty of deciding when the hilar glands have become enlarged.



CHAPTER 8. THE NATURAL HISTORY OF TUBERCULOSIS:  
THE SEROUS STAGE.

Sixteen cases illustrate the serous stage of the disease. These cases are arranged in three subgroups:

(a) cases in which there is radiological or post mortem evidence of underlying primary disease;

(b) cases in which there is no unequivocal evidence of underlying disease;

(c) cases in which there is radiological evidence of underlying reinfection disease.

Private MNICHABE (Appendix A, Case 11) was admitted to hospital in November 50 with a massive pleural effusion in the right side of his chest. A fortnight after admission he developed ascites and oedema of the feet and ankles. Six weeks later he developed a pericardial effusion. He died eleven weeks after admission to hospital.

At autopsy, the left lung and the pleural cavity were normal. The right lung was collapsed, adherent to the right diaphragmatic cupola and contained multiple small cavities in the lower lobe. The pleural layers between the lobe and the diaphragm were thickened and adherent and contained a number of small multilocular cysts. There was a large pleural effusion on the right side. Large masses of caseous mediastinal glands were present. The pericardium was bound to the diaphragm by tuberculous granulation tissue. Both the parietal and cardiac layers were thickened with tuberculous granulation tissue and the cavity

was largely obliterated. The liver was rigidly adherent to the diaphragm over the base and right lateral surface. The cut surface and peritoneal covering were heavily seeded with small hard tuberculous nodules. The cut surface of the spleen exhibited diffuse tuberculous nodules. All groups of abdominal glands were enlarged and caseous. The entire peritoneal cavity was heavily seeded with small tuberculous nodules and there was a great deal of free fluid.

Strictly speaking this patient should not have been included in this series, since he was a native of Northern Rhodesia and not of East Africa. But the post mortem findings made such an excellent illustration of the type of disease described in South Africa, that it was deemed advisable to include this case by way of contrast with the other cases.

This case also illustrates the rapid downhill progress of the disease in a patient with a high degree of hypersensitivity and a low degree of resistance.

Two patients (Appendix A, Cases 12 and 13) had enlarged hilar glands and scattered parenchymal lesions in the left lung fields which became visible as the effusions were absorbed.

Nine patients (Appendix A; Cases 14,15,16,17, 18,19,20,21 and 22) showed no unequivocal radiological evidence of underlying disease. In some of these cases the hilar shadows appeared, and were recorded as being, unduly prominent. But such statements are

so subjective and are altered so much from time to time when the same radiographs are reviewed that it seems best to group all these cases together.

In the absence of any radiological evidence to the contrary, it is assumed that these cases illustrate the post primary stage of the disease in patients with a high degree of hypersensitivity and a definite degree of resistance.

In four patients, pleural effusion was a complication not of primary disease but of reinfection disease (Appendix A Cases 23, 24, 25 and 26).

Lance Corporal ODERO (Appendix A, Case 23) was admitted to hospital in April 51. By the end of May 51, his effusion had almost completely cleared. In June 51, his father died and he was allowed compassionate leave. On his return it was found that his effusion had reaccumulated. He was eventually discharged to the reserves in January 52.

Private WAMBUA (Appendix A, Case 25) was admitted to hospital with a left sided pleural effusion which developed two months after he had been treated for what was regarded as broncho-pneumonia in his left lower lobe. The radiograph revealed the presence of adhesions on the right side, which had displaced the cardiac shadow anteriorly and to the right. These adhesions were later proved by the discovery of some old radiographs to be at least two years old. The interpretation of this case is difficult. It is possible that the old adhesions had resulted from a previous pyogenic pneumonia. Against

this interpretation are two facts: firstly, pleural effusions in association with pyogenic pneumonia are extremely rare in East African natives - at a rough estimate, they occur in not more than 0.5% of cases; secondly, the patient could not recollect having had any previous illnesses. The alternative explanation, which is thought to be the correct one, is that the original tuberculous lesion was in the right lung and was followed by a pleural effusion on that side and that the so-called bronchopneumonia in the left lower lobe was in reality a manifestation of reinfection disease.

Civilian KATHUKU (Appendix A, Case 26) had chronic bilateral apical tuberculosis - a form of the disease which is seldom seen in East African natives. There was also radiological evidence of a previous pleural effusion on the opposite side of the chest. His effusion cleared slowly and he was left with a large area of thickened pleura.

In these last two patients it is interesting to note how the natural history of the disease repeats itself on both sides of the chest - parenchymal lesion leads to pleural effusion and pleural effusion to thickened pleura.

In the sixteen cases under discussion, absorption of the pleural fluid occurred in anything from five to twenty two weeks and there was residual pleural thickening of varying extent in six cases. (Appendix A, Cases 18, 21, 22, 23, 25 and 26). This compares



favourably with the series treated by WOOD (64), who recorded gross residual pleural thickening in 70%.

The conclusions which may be drawn from a study of these cases are as follows:

Firstly, pleural effusion is the commonest complication of primary disease in East Africans. In this series there is a total of twenty two cases of primary disease of which twelve developed pleural effusion. Secondly, the progress of the serous stage in East Africans appears to differ little from that in Europeans. Thirdly, as Oswald points out, the prognosis is poor when more than one serous cavity is involved. But this is probably because polyserositis only occurs in patients with a low degree of resistance and not because the involvement of more than one serous cavity has any causal relation to the prognosis. Fourthly, pleural effusion is not an uncommon complication of reinfection disease. In this series pleural effusion complicated four out of thirty three cases of reinfection disease. In two of those cases there appeared to be a fixed tendency towards pleural involvement. This suggests that in these two cases the balance between the degrees of hypersensitivity and resistance had remained unaltered over a long period.

CHAPTER 9. THE NATURAL HISTORY OF TUBERCULOSIS:  
THE INTERMEDIATE STAGE.

Three cases illustrate the intermediate stage: two cases were recognised as so doing during life; the other case found to do so at post mortem examination.

Lance Corporal F'OBUYA (Appendix A, Case 8) was admitted to hospital in July 50 in the primary stage of his disease with an opacity in the left lower zone and enlargement of the left hilar glands. He made a good recovery from the primary infection but four months after admission a small opacity developed in the left infraclavicular region and rapidly cavitated. A left artificial pneumothorax was induced and the cavity closed. Unfortunately fluid developed in the artificial pneumothorax and was very slow in clearing and adhesions prevented the re-expansion of the lung. The patient refused to undergo a thoracoplasty. Nevertheless at the time of his discharge to the reserves in January 52 his ESR was normal, his sputum negative and he was gaining weight. He was seen two months later by the Medical Officer of his district who reported that he looked healthy and that his ESR was normal.

A study of the radiographs and of the sputum reports in this case show quite clearly that at the time when the reinfection lesions in the left infraclavicular region developed neither the parenchymal nor the glandular elements of the primary stage had

completely resolved.

Lance Corporal CHACHA (Appendix A, Case 9) was admitted to the Station Hospital, Mauritius in May 50 on account of a urinary infection. In June 50 a routine radiograph revealed tuberculous infiltration in the right mid zone and infraclavicular region. He was evacuated to East Africa in December 50, by which time there was tuberculous infiltration of the whole of the right lung; a small opacity in the left mid zone; and no evidence of enlargement of the hilar glands.

A right phrenic crush reinforced by a pneumoperitoneum failed to control the spread of the disease. In July 51 a right thoracoplasty was performed. This was followed by the development of intrapleural fluid after the second stage of the thoracoplasty. The patient died suddenly as the result of the intrapleural fluid rupturing through the wall of a cavity in the right lower lobe.

At autopsy a small healed subpleural focus was found in the left lung; the tracheobronchial glands were enlarged and contained caseous foci; the right lung showed widespread tuberculous infiltration and two large cavities.

This patient was not under observation during the primary stage of his disease. There was never any radiological evidence of glandular enlargement. But the post mortem evidence makes it quite clear

that in his case also the reinfection lesions developed before the complete resolution of the glandular enlargement of the primary stage had occurred.

Private MUNYITHIA (Appendix A, Case 10) was admitted to hospital in October 50. He stated that he had been unwell and had had a troublesome cough since January 50. A radiograph taken on the day of admission revealed numerous small opacities scattered throughout the whole of the left lung field; similar opacities and a cavity in the right lower zone; and enlargement of the right hilar glands.

His condition deteriorated steadily and he died 3½ months later.

At autopsy widespread tuberculous bronchopneumonic lesions were found in both lungs. The hilar, para-aortic and mesenteric glands were enlarged and contained caseous foci. There were three small tuberculous ulcers in the ileum.

This patient did not come under observation during the primary stage of his disease and no obvious primary focus was found at autopsy. But the radiological appearances at the time of his admission to hospital suggest that the primary focus had been in the right lower zone.

The radiographs revealed the presence of a large cavity in the right lower zone which had at one time a fluid level in it. No mention of cavitation is made in the report in the post mortem examination. This is not an error: similar discrepancies occur in other cases. The explanation is that these are



tension cavities due to tracheobronchial tuberculosis and that after death these cavities become deflated. It may be remembered that both MOUCHET (44) and VINT (59B) comment on the small size of the cavities seen in their post mortem material. Probably the explanation is the same.

Describing the progressive primary focus AUERBACH (3) says it "reveals a large irregularly demarcated area of caseation with no definite capsule. Evacuation of the liquefied caseous mass results in an irregular shaggy excavation. The remaining pulmonary parenchyma gives evidence of haematogenous, lymphatic and bronchogenic spread. The lymph nodes are extensively enlarged, completely caseous and frequently have undergone central liquefaction."

From the radiological appearances of the case under discussion, it might be argued that this case was one of a progressive primary focus. The post-mortem findings are similar to the description given by Auerback except for the absence of the irregular shaggy excavation.

On the other hand the appearances at post mortem could well have resulted from the disease progressing in the same way as the other two cases. All roads lead to Rome and there is no reason why two roads should not lead to similar post mortem appearances.

What does seem to be certain is that these three cases illustrate the progress of the disease from the primary to the intermediate stage in patients with very different degrees of resistance. Lance

Corporal F'OBUYA was slowly developing sufficient resistance to overcome his primary infection and his reinfection lesion was small and localised. It is interesting to contrast his case with that of Private RUGONGO (Appendix A, Case 2) as the time relations between the development of hypersensitivity and the development of resistance appear to be reversed in the two cases.

Lance Corporal CHACHA appears never to have developed sufficient resistance to do more than fight a losing battle with his disease. Nevertheless had surgical measures been adopted at an earlier stage the issue might have been very different.

Private MUNYITHIA had very little resistance left at the time of his admission to hospital. But he had been ill without reporting sick for nine months prior to his admission. It may well have been that initially he had at least as high a degree of resistance as Lance Corporal CHACHA.

The conclusions that may be drawn from a study of these three cases are as follows. Firstly, reinfection disease may develop before the resolution of the primary stage is complete. Secondly, there may well be more than one pathological mechanism by which this state of affairs is brought about. Thirdly, the absence of radiological evidence of glandular enlargement does not necessarily mean that the disease has reached the reinfection stage, although in practice such an assumption must usually be made.

Finally, the radiological appearances and the progress of the disease are largely determined by the interaction of the degree of hypersensitivity and the degree of resistance.

Tuberculosis that may be found in East African Africa

The case of CIVILIAN (Appendix A, Case 1) forms a convenient bridge between this group, and the cases discussed in the previous chapter.

Civilian was admitted to hospital in July 1940 with a solitary cavity in the left infra-clavicular region with small nodules scattered round it. It proved impossible to close the cavity by means of a left phrenic nerve retractor and a pneumothorax; and a left artificial pneumothorax was closed temporarily. The patient refused to undergo a thoroplasty. He was therefore discharged to the reserves in October 1941.

This case is comparable to that of LANCE (Appendix B, Case 1) and illustrates the course of tuberculous disease in a patient with a high degree of resistance and a low degree of hypersensitivity.

Under (Appendix B, Case 2) was admitted to hospital in October 1941 with a large cavity in the right upper zone which contained a central cavity and a small area of consolidation above in the left mid zone. A right artificial pneumothorax was induced but the presence of adhesions prevented closure of the cavity. The patient refused

CHAPTER 10. THE NATURAL HISTORY OF TUBERCULOSIS:  
THE REINFECTION STAGE.

Twenty eight cases illustrate the various manifestations of the reinfection stage of Pulmonary Tuberculosis that may be found in East African Natives.

The case of Civilian MWASIA (Appendix B, Case 27) forms a convenient bridge between this group, and the cases discussed in the previous chapter.

Civilian MWASIA was admitted to hospital in July 50 with a solitary cavity in the left infra-clavicular region with small opacities scattered round it. It proved impossible to close the cavity by means of a left phrenic crush reinforced by a pneumoperitoneum; and a left artificial pneumothorax only closed it temporarily. The patient refused to undergo a thoracoplasty. He was therefore discharged to the reserves in October 51.

This case is comparable to that of Lance Corporal F'GBUYA (Appendix A, Case 8) and illustrates the course of reinfection disease in a patient with a high degree of resistance and a low degree of hypersensitivity.

Gunner MUSYOKI (Appendix B, Case 28) was admitted to hospital in October 51 with a large opacity in the right upper zone which contained a central cavity and a small area of bronchogenic spread in the left mid zone. A right artificial pneumothorax was induced but the presence of adhesions prevented closure of the cavity. The patient refused to



undergo adhesion section. He was, therefore, discharged to the reserves in February 52. By that time the lesion in the left lung had cleared.

This case illustrates the fact that an East African native with a high degree of resistance is capable of dealing with a bronchogenic spread if it is not very extensive.

Private ODWOR (Appendix B, Case 29) was admitted to hospital in January 51 with a large cavity in the apex of the left lower lobe and several patches of tuberculous bronchopneumonia in the left mid and lower zones. A pneumoperitoneum was induced but had no effect on the cavity. The patient refused any further treatment and was therefore discharged to the reserves in May 51. Before he left hospital the disease had spread into the left upper zone. Throughout his stay in hospital this patient was thoroughly unco-operative and ill disciplined.

Private PAULO (Appendix B, Case 30) was admitted to hospital in August 50, with a large opacity in the left lower lobe with a cavity in the centre of it. One month after admission a left phrenic crush was performed and reinforced with a pneumoperitoneum. He made steady progress from then until March 51 when he felt so well that he became intolerant of discipline, refused to stay in bed and refused to have his pneumoperitoneum refilled regularly. As a result, the cavity increased again in size and his general condition deteriorated. He finally became

quite unmanageable and was discharged to the reserves in August 51.

These two cases illustrate the downhill course of the disease in unco-operative patients who probably had sufficient resistance to control the disease with the aid of treatment.

Private OBWAKE (Appendix B, Case 31) was admitted to hospital in February 50 with a thin walled cavity in the right midzone and opacities in the right upper zone, right lower zone and left mid zone. Attempts to close the cavity by a right phrenic crush reinforced with a pneumoperitoneum and later by a right artificial pneumothorax were unsuccessful. The patient refused to undergo a thoracoplasty and was discharged to the reserves in May 51. After fifteen months in hospital, the opacity in the right upper zone and the cavity in the right mid zone were still present and the right lower and left mid zones were clear.

This case illustrates the ability of the East African native with a fairly high degree of resistance to control and resolve the spread of the disease, even though the sputum remains persistently positive.

A similar ability is illustrated by the next case. Civilian KIEMA (Appendix B, Case 32) was admitted to hospital in July 50 with a cavity in the right mid zone and large opacities in both right and left mid and lower zones. A pneumoperitoneum failed to close the cavity. The patient refused to allow a right phrenic crush to be performed. When he was dis-

charged to the reserves in May 51 the cavity was still present but there had been considerable clearing of the opacities in both lung fields. This patient was one of the few patients to have haemoptysis which is not a common complication of the disease in East African natives.

Civilian MWATHE (Appendix B, Case 33) was admitted to hospital in August 50 with an opacity in the right upper zone, a thin walled cavity in the right mid zone and scattered exudative lesions in the right lower zone. In September further spread occurred to the left lung. In October a right artificial pneumothorax was induced and successfully maintained until January 51, when fluid developed and the right lung collapsed completely. The fluid persisted in spite of repeated paracentesis until September when the patient was left with a large area of pleural thickening. He was discharged to the reserves in October 51.

This case illustrates how well an East African native can stand up to the disease even in the presence of such disastrous complications of therapy, which will be discussed in detail in a later chapter.

In discussing the post mortem appearances in the case of Private MUNYITHIA (Appendix A, Case 10) it was pointed out that in East African natives cavitation was often due to tracheobronchial disease. The radiological appearances and the behaviour of the cavities under collapse therapy in the seven cases so far dealt with in the present chapter lend further support to this conclusion.

In the previous seven cases, only single cavities have been visible without the use of tomography. The next seven cases illustrate the course of the disease in patients with multiple cavitation.

Private ABONDIO (Appendix B, Case 34) was admitted to hospital in October 51 with multiple cavities in the left lung and a small opacity in the right lung. He refused to undergo a left thoracoplasty and was discharged to the reserves in February 52. Although there had been little change in his condition during his stay in hospital, it was considered that a disastrous spread might occur at any time and accordingly his disability was assessed at 100%.

Private MUCHIRI (Appendix B, Case 35) was admitted to hospital in September 50 with a cavity in the right upper zone surrounded by exudative lesions and with several opacities in the left mid zone. Adhesions prevented the induction of a satisfactory pneumothorax. In December 50 a further cavity appeared in the left mid zone. Pneumothorax was again prevented by adhesions. This patient was discharged to the reserves in May 51 when his disability was also assessed at 100%.

Trooper KIKWAI (Appendix B, Case 36) was admitted to hospital in October 51 with a large cavity in each upper zone and widespread infiltration giving an appearance of reticulation in both lung fields. Adhesions prevented a left artificial pneumothorax from being successful. The patient refused to allow adhesion section. He was discharged to the reserves



in January 52, when his disability was assessed at 100%.

Corporal NGERU (Appendix B, Case 37) was admitted to hospital in October 49 with widespread infiltration in all zones of both lung fields. With rest in bed the exudative lesions improved but cavitation developed in both upper zones. As it was clear that little could be done for him, he was discharged to the reserves in March 50, when his disability was assessed at 100%.

Civilian MURASHUI (Appendix B, Case 38) was admitted to hospital in December 50 with widespread infiltration in both lungs and multiple cavities in the left lung. After six weeks, during which time further cavitation developed he absconded from hospital.

Civilian ANDIKA (Appendix B, Case 39) was admitted to hospital in December 49, with infiltration and cavitation in both upper and both mid zones. In February 50 further spread of the disease occurred in the left lower zone. A left phrenic crush was therefore performed and reinforced by a pneumoperitoneum. But in spite of these manoeuvres, there was little change in the patient's condition until he absconded from hospital in September 50.

Private CHULI (Appendix B, Case 40) was admitted to hospital in September 49 with a cavity at the left apex and a confluent bronchopneumonia throughout the left lung. With rest in bed and an adequate diet, his condition remained stable until January 50 although

further cavitation occurred in the left upper zone. In January 50, he developed Falciparum Malaria which responded well to treatment with mepacrine. But this additional burden had been too much for him and his condition steadily deteriorated until in mid February he appeared to be at death's door. A left phrenic crush was performed on 21 Feb 50 and reinforced in mid April with a pneumoperitoneum. These measures had a salutary effect on his general condition. But in June 50, he developed a tuberculous laryngitis and his general condition again started to deteriorate. By October 50, further cavitation had occurred and continued unchecked to the end of the year. In January 51 a left thoracoplasty was performed. This was followed by the development of disease in the right upper zone which was controlled with Streptomycin. From the conclusion of his operation, he made steady progress and gained over a stone in weight. He was discharged to the reserves in September 51. His sputum was still positive and his disability was assessed at 100%.

Of these seven cases just recorded, five were discharged to the reserves with their disability assessed at 100% and two absconded from hospital; had they not absconded their disability would also have been assessed at 100%. In six of the cases the disease was bilateral at the time of admission and was not amenable to treatment except by chemotherapy, which was not available. These six patients remained in hospital for periods varying from three

to nine months with a mean of six months. At the end of that period they were all able to walk out of hospital to travel back to their native reserves. Therefore although the ultimate outlook is gloomy, it is clear that they all possessed some effective degree of resistance to their disease. Their prospects seem to be entirely dependent upon the chance of a widespread bronchogenic spill or upon the onset of intercurrent disease which would lower their general resistance. The seventh case demonstrates the progress of the disease when widespread bronchogenic spread occurs. Presumably he had had the cavity at the left apex for some time prior to admission to hospital; and presumably the areas of confluent bronchopneumonia were the result of bronchogenic spread and led to his reporting sick. From then, in spite of his indomitable spirit, his chances of recovery, had he not been in hospital, would have been slight. As it turned out, with the assistance of treatment he was able to develop sufficient resistance to the disease to enable him to return from the threshold of death and travel back to live at least for a time in his native reserve. His case exemplifies the fact that however depressing the general condition and the radiological appearances of a patient may be, if he can be tided over the acute stage, resistance to the disease may develop when it is least expected.

Private KASOLO (Appendix B, Case 41) was admitted to hospital in January 51 with an extensive area of

consolidation in the left upper and mid zones and early cavitation in the left apical and infraclavicular areas. With rest in bed and a pneumoperitoneum the consolidation cleared revealing two large cavities. In May the first stage of a thoracoplasty was performed. Unfortunately, owing to an error in surgical technique, the patient died shortly after leaving the theatre. At post mortem examination, a large cavity filled with fluid pus was found in the left upper lobe and a small patch of consolidation in the anterior segment of the right upper lobe. There was no evidence of glandular enlargement or of extrapulmonary spread.

The radiological appearances in this case were not unlike those in the case of Private CHULI (Appendix B, Case 40) but the progress made by Private KASOLO prior to operation suggested that he possessed a much higher degree of resistance. By equating the radiological and post mortem appearances in this and the next five cases (Appendix B, Cases 42, 43, 44, 45 and 46) it has been possible to allocate other cases to the reinfection group with a fair degree of certainty.

Civilian M'MBAROKI (Appendix B, Case 42) was admitted to hospital in August 50 during the height of an epidemic of Typhoid Fever. He was at first thought to be suffering from that disease until a radiograph revealed widespread bilateral tuberculous disease with multiple cavitation. He remained in hospital with little change in his condition for a



year and died suddenly following a severe haemoptysis. Unfortunately no post mortem examination was performed.

This case is of some importance as it illustrates how long an East African native may sustain life even though he has advanced tuberculosis. Clearly such a patient must have a considerable degree of resistance.

Private MWANZI (Appendix B, Case 43) was admitted to hospital in April 50 with infiltration in the right upper and mid zones, cavitation in the left upper zone and collapse in the left mid and lower zones. He remained remarkably well until November 50 although his radiographs showed a steady worsening of the disease. But from that time, he went rapidly downhill and died on 31 Dec. 50. The post mortem appearances were remarkable as both lungs had been converted into large sacs filled with fluid pus. There was no evidence of glandular enlargement or extrapulmonary spread.

This case also illustrates the remarkable ability to sustain life, in the presence of almost complete destruction of the lungs.

Civilian ADUKE (Appendix B, Case 44) was admitted to hospital in September 50, with advanced bilateral disease. His condition gradually deteriorated and he died in February 51. At autopsy both lungs were bound to the thoracic wall with dense adhesions. Only one cavity was found in the right upper lobe - though numerous cavities had been seen in both lungs in the radiographs. Both lungs were riddled with small tubercles. There were three small

laryngeal ulcers but no other evidence of extrapulmonary spread. There was no glandular enlargement.

Civilian OMENDO (Appendix B, Case 45) was admitted to hospital in April 51 with bilateral infiltration and cavitation. His condition deteriorated steadily and he died in August 51. At post mortem examination there were pleural adhesions at both apices. The right lung contained three cavities and the left lung contained three cavities. Both lungs showed numerous areas of tuberculous bronchopneumonia. There was no evidence of glandular enlargement or of extrapulmonary spread.

These two cases illustrate the rapid downhill progress, so much stressed by other authors.

Private NDUNDA (Appendix B, Case 46) was admitted to hospital in August 51 with widespread infiltration and cavitation in both lungs. His condition deteriorated rapidly and he died ten days later.

The chief interest of this case lies in the photograph of the patient taken on his admission, which illustrates how well an East African native with advanced tuberculosis may look. Such an appearance is by no means uncommon and is the reason for the scepticism, with which statements about the absence of tuberculosis in Africans by authors, who have not had radiological facilities, must be greeted.

Private MWENDO (Appendix B, Case 47) was first seen in November 50 when a radiograph showed several small opacities at the left apex. Presumably these

were thought to be inactive as he was not seen again until his admission to hospital in February 51, when the radiograph showed that spread had occurred into the right upper zone, where cavitation had started. The radiological appearances deteriorated slowly until a right phrenic crush was performed in May 51, after which there was considerable improvement. A thoracoplasty was planned but the patient refused operation and was therefore discharged to the reserves in Nov. 51. This case illustrates how a florid acute lesion may suddenly develop in a patient with apparently high resistance and how such disease may be partially controlled.

Private KABIRA (Appendix B, Case 48) was admitted to hospital in June 50 when his radiograph showed a large cavity in the right upper zone and scattered areas of infiltration in the right mid zone and in all zones of the left lung. During the next four months his condition remained stationary. A thoracoplasty was then performed but it was technically inadequate and the large cavity remained unclosed. In Mar 51, spread of the disease occurred into the right lower zone. This was controlled by chemotherapy. The patient was ultimately discharged to the reserves in Nov. 51.

This case is a more chronic example of the type of lesion illustrated by the previous case.

Civilian WANYANGA (Appendix B, Case 49) was admitted to hospital in Feb 51, when his radiograph

showed widespread infiltration in all zones of the left lung. Early in March, he had a severe haemoptysis. After this had been controlled, a left phrenic crush was performed and reinforced with a pneumoperitoneum. This was followed by some improvement. But in the latter half of July, he had a succession of severe haemoptyses. In an attempt to control these, a left artificial pneumothorax was induced. This manoeuvre was successful and had an excellent effect upon his general condition in spite of the presence of adhesions. The pneumothorax was maintained until Oct 51, when the patient absconded from hospital.

The chief interest of this case lies in the fact that his general condition improved steadily after the phrenic crush although the radiological appearances deteriorated. It seems likely that the radiological appearances were largely due to aspirated blood rather than to fresh disease.

Private SYONDO (Appendix B, Case 50) was admitted to hospital in Apr 51 when his radiograph showed an opacity in the left infraclavicular region with streaking towards the hilum but no glandular enlargement. Spread of the disease occurred during the next three weeks and a left artificial pneumothorax was induced and maintained until mid July, when the patient absconded from hospital. He returned on 18 Sep 51, stating that during the intervening period he had been under the care of his witch doctor and that he had now returned solely in order to obtain his discharge from the army. His radiograph



showed marked improvement. He refused all further treatment and was discharged to the reserves in November 51.

Sapper PAULO (Appendix B, Case 51) was admitted to hospital in May 51, when his radiograph showed several opacities in the right upper and mid zones. After two months rest in bed a right artificial pneumothorax was induced and maintained until his discharge in Mar. 52. It was hoped that his pneumothorax would be maintained at MULAGO Hospital, Kampala to which he was referred.

Private KIEMA (Appendix B, Case 52) was admitted to hospital in Nov. 50, when his radiograph showed several opacities of varying size and density in his left left lung field. He was discharged six months later without any radiological change having occurred.

Private NGII (Appendix B, Case 53) was seen as an outpatient prior to his discharge from the army after seven years service. His radiograph showed chronic fibrous tuberculosis with displacement of the trachea, heart and mediastinum to the affected side.

Civilian SINGA (Appendix B, Case 54) was admitted to hospital in Feb. 50 and discharged three months later. His radiograph showed diffuse infiltration in the upper and mid zones on both sides and a cavity in the right upper zone. No change occurred during his stay in hospital.

These last five cases illustrate the chronic forms which the disease may assume in patients with

high resistance and without hypersensitivity.

One of the questions which the South African investigators sought to answer was: "In the case of positive tuberculin reactors, is there any quantitative relation between the degree of the reaction and the liability to tuberculosis or to the type of an aftercoming tuberculosis?" They concluded that "there is definite evidence of a quantitative relation between the degree of reaction and the liability to infection but no definite relation between the degree of reaction and the type of aftercoming tuberculosis ---- The common type of tuberculosis is the generalised or metastatic type and its occurrence seems to be independent of the degree of tuberculo-allergy present."

At the time when these words were written, resistance and allergy in tuberculosis were thought to be two aspects of one process. Thanks to the work of Rich, resistance and hypersensitivity may now be regarded as two separate processes which develop independently of each other and which wax and wane with varying speeds in different people in different environments.

This concept gives the key to the correct interpretation of radiological appearances and materially contributes to the assessment of the individual case. In discussing the cases considered in this and the three previous chapters this concept has been used. It is considered that the type of disease from which an individual suffers and the course which the disease follows is largely determined by the inter-

play of these two processes - resistance and hypersensitivity. If resistance is high and hypersensitivity minimal, the disease will either be arrested or of a chronic nature. If resistance is low and hypersensitivity is marked, either death will ensue rapidly or extensive and irreparable damage will be done to the tissues. Between these two extremes all forms and degrees of disease may be placed. In these last few chapters an attempt has been made to arrange the cases according to the interplay of these two processes.

It is also considered that in the past a grave error has been made in stressing the absence or low degree of resistance in the natives of Africa. Resistance in varying but often high degree is usually present. The florid type of disease with its acute course is due to the high degree of hypersensitivity which is present rather than to absent or low resistance.

This distinction is not an entirely academic one. If the type of disease were determined by lack of resistance, it should respond favourably to general measures directed to improving resistance. It will be shown in a later chapter that this does not occur. In the treatment of Pulmonary Tuberculosis the question is always: can this patient be sustained until hypersensitivity wanes?

The importance of the degree of hypersensitivity in determining the nature of the disease in the individual must remain only a working hypothesis

until it has been proved firstly that lung and skin sensitivity to tuberculo protein run pari passu; and secondly that measures directed to controlling hypersensitivity result in controlling the course of the disease. The attempts by the older workers to influence the course of the disease by injections of tuberculin were either ineffective or disastrous. Nevertheless a controlled experiment with tuberculin plus chemotherapy might be worth serious consideration.

collapse therapy is indicated since it definitely prevents spread of the disease and reduces tuberculin.

In 1934, CUYLER, ROBERTS and CIPER (17) were of the opinion that the affected lung should be put to rest as soon as the diagnosis is made. "Delayed treatment is especially disastrous in American negroes as have been several cases where the patient died within a few months of diagnosis. It is our policy to employ ambulatory chemotherapy as a preferred form of treatment in our negro patients and in white patients in whom possible as possible."

In 1936, WASH and RICH (18) analyzed 100 cases of pulmonary tuberculosis occurring in patients living under ideal conditions and concluded that these conditions had affected the progress of the disease once it had become established. Little is to be learned from this study.

On the other hand, in 1939, KATZMAN, KERRY and WATKINS (19) stated that "American negroes are much more susceptible to tuberculosis in all its stages and death at least as much and possibly more often."



CHAPTER 11. THE TREATMENT OF PULMONARY TUBERCULOSIS:  
PREVIOUS WORK.

There are very few papers dealing with the treatment of Pulmonary Tuberculosis in Africans. Papers dealing with the treatment of American Negroes have therefore been included in this survey of previous work.

In 1933, CHADWICK, MARKOE and THOMAS (12) came to the conclusion that in American Negroes prompt collapse therapy is indicated since it definitely prevents spread of the disease and reduces toxæmia.

In 1934, CUTLER, RODGERS and CIPPES (17) were of the opinion that the affected lung should be put to rest as soon as the diagnosis is made. "Delayed treatment is especially disastrous in (American) negroes: we have seen minimal cases become far advanced within a few months ending in death. We now apply ambulatory pneumothorax as a preferred form of treatment in our negro patients and institute collapse in them as early as possible."

In 1935, WALSH and MASON (61) analysed 200 cases of pulmonary tuberculosis occurring in patients living under ideal conditions and concluded that "these conditions had affected the progress of the disease, once it had become established, little if any at all."

On the other hand, in 1938, KETTELKAMP, MURPHY and TRUMPE (33) stated that "American negroes are amenable to sanatorium treatment in all its phases and derive at least as much and possibly more bene-

fit from it than do the whites."

In 1937, FISHER (22) wrote of American negroes: "Generally speaking, nothing short of early, complete collapse of the diseased lung has proved sufficient in the negro."

In a detailed paper on collapse therapy in American negroes, CULLEN and HOFFMAN (15) in 1942, wrote: "When collapse therapy is indicated and performed, the results are nearly as good as in the white race. Delays are dangerous and halfway measures are not to be employed. A prolonged period of rest to observe the progress of a lesion so seldom accomplishes any good that it is seldom worth employing. Collapse therapy offers a greater chance for success in the Negro than any other procedure and should be employed as widely as possible."

"Pneumothorax without pneumolysis is an inadequate measure.

"Phrenic nerve surgery seems to carry the same degree of unpredictability and disappointment as in the white.

"Thoracoplasty led to sputum conversion in 60% of cases.

"There is only one difference between the White and the Negro when surgery is considered that we wish to emphasise. The Negro is sicker and usually has more extensive disease. His prognosis is admittedly poorer. There is all the more reason, therefore, when considering collapse therapy in the Negro, to

widen the scope of the indications for performing these measures."

But in a paper published eighteen months later HOFFMAN(28) concludes: "Were the opportunity again presented to decide on the use of pneumothorax, it would be withheld in a rather large number of cases. Many of the massive exudative lesions were the ones that were followed by spontaneous pneumothorax, massive fluid formation and contralateral spread. It can hardly be denied that some patients give every evidence that their tuberculosis will follow an uninterrupted, progressive course and that any collapse therapy used will be unavailing. These cases can be recognised with reasonable certainty and no collapse measures should be employed."

In 1945, ANDERSON and WINN (2) reported the results of pneumoperitoneum and diaphragmatic paralysis in 110 American Negroes and concluded "that there is appreciably no significant variation in response to this type of therapy in the white and coloured races."

In 1946, OSWALD (46) wrote: "In this series, 14 artificial pneumothoraces had been induced at other hospitals prior to admission: none was induced in our hospital. Acute bronchogenic spreads or pleural effusions or both ensued in nearly every case. These cases clearly indicate the futility of collapse therapy for acute or advanced lesions. It may be that really "early" cases might benefit from a pneumothorax

which could be properly maintained for two to three years. These, however, are very few and far between and their chances of controlled refills over a long period in their home countries are remote.

No case presented lesions suitable for thoracoplasty or other major surgical measures of collapse therapy."

In 1946, DAVIES (18) the medical superintendent of the Sanatorium at Kibongoto, Tanganyika Territory published the results of treatment with pneumothorax in 1384 cases over a period of 12 years. Of 600 cases with severe open pulmonary tuberculosis 293 were still alive and 307 were dead. Of those still living 197 had been under treatment for over one year; 102 for over five years; and 21 for over ten years. Of 784 cases with less severe disease, 466 cases were still alive and 318 were dead. Of those still living 358 had been under treatment for over one year; 152 for over five years; and 27 for over ten years. DAVIES concluded: "The average expectation of life for an African with pulmonary tuberculosis is not more than 18 months without special treatment."

In 1949, KIRBY, SIMPSON and CREGER (35) recorded sustained improvement in 4 out of 15 American Negro adults with tuberculous pneumonia treated with streptomycin. Properly timed adequate collapse therapy appeared to be an essential adjuvant to streptomycin administration in bringing about permanent improvement. The early postoperative course had been



encouraging in two patients in whom pneumonectomies were performed.

The literature may be summarised as follows: the untreated case is unlikely to live longer than eighteen months; therefore the delays in instituting treatment which are permissible in dealing with Europeans are dangerous in dealing with Africans; in properly selected cases, there is little difference in the response to treatment between white and negro races; but the poor prognosis for the untreated necessitates the widening of the indications for collapse treatment and this may frequently bring such disasters in its train that the more experienced physician will doubt whether in many cases such treatment is worth while.

CHAPTER 12. THE TREATMENT OF PULMONARY TUBERCULOSIS: METHODS AND RESULTS.

As was pointed out in the introductory chapter, the definition of the most suitable methods of treatment of pulmonary tuberculosis in East African Natives was only one of three objects in this investigation. Of these three objects the most important was to study the natural history of the disease as it occurs in East African Natives. In the early stages of the investigation, therefore, treatment was essentially conservative in order to interfere as little as possible with the attainment of the second object. In the later stages of the investigation, when the natural history of the untreated disease had become apparent, treatment became progressively more radical and more rapidly applied.

In this chapter, the various forms of treatment employed and the results obtained are discussed.

It can be seen from the case sheets that all patients were treated initially by strict bed rest. Strict bed rest was indeed the ideal that was aimed at. But the East African Native has a rooted objection to remaining in bed unless he is feeling extremely ill. At Mackinnon Road, the Nursing Staff were, by dint of unwearied exertion, remarkably successful in keeping the patients in bed. But at Nairobi, where the wards faced onto a main road, it was practically impossible to prevent the patients from leaving their beds and from sitting by the fence, through which

they could watch the passing traffic and gossip with the passers-by.

Of the 7 cases with primary disease, only three patients were treated by bed rest alone. Private SIDINYA (Appendix A, Case 4) was so treated for 7½ months and at the end of that period it was considered that his disease had been arrested. Civilian MUSYOKA (Appendix A, Case 5) was similarly treated for 5 months. He was then discharged at his own request. Although there had been considerable improvement in the primary focus, the hilar glands were still enlarged and it was considered that the disease was still active. Pte MAKARANGA (Appendix A, Case 1) died.

In the remaining four patients, who were treated initially by rest in bed, the disease progressed unfavourably and additional forms of therapy were employed.

It is, therefore, concluded that rest in bed alone is not adequate to allow arrest of primary disease in the majority of East African patients.

Of 12 cases of primary disease complicated by pleural effusion and treated by bed rest alone, 1 patient died, 8 patients were discharged with the disease regarded as quiescent and 3 patients were discharged at their own request when the disease was still active. But in 4 out of the 11 cases, discharged from hospital, extensive areas of thickened pleura had developed. It is therefore considered

that rest alone is not an entirely satisfactory method of treating pleural effusions.

All three patients in the intermediate stage of the disease deteriorated on bed rest alone. It is therefore concluded that this is an inadequate form of treatment for such cases.

Of 11 cases of reinfection tuberculosis treated by bed rest only, 4 patients died, 1 patient deteriorated, 5 patients were unchanged and 1 patient improved. In 6 of the 7 patients, who were eventually discharged from hospital, the radiological appearances suggested that it was only a matter of time before a large scale bronchogenic spread would occur and death ensue. It is therefore considered that rest in bed alone is an inadequate method of treating such cases.

But that rest in bed does play an important part in the management of the disease is clearly illustrated by the case of Private PAULO (Appendix B, Case 30). This patient made no progress when initially treated by rest in bed alone. But, following a phrenic crush and pneumoperitoneum, he improved steadily until he was feeling so well that he refused to stay in bed. As a result the cavity, which appeared to be closing, reopened, and his general condition deteriorated again.

These cases confirm the statement of CUTLER, ROGERS and CIPPES (17) that "delayed treatment is especially disastrous;" and the statement of CULLEN and HOFFMAN (15) that "a prolonged period of rest



so seldom accomplishes any good that it is seldom worth employing."

All the patients were given a European diet throughout their stay in hospital. For administrative reasons, it was much simpler to prescribe a European diet than a high protein diet and a glance at table 17 will show that the European diet was in fact high in protein and fulfilled the optimum dietetic requirements laid down for the treatment of cases of tuberculosis.

TABLE 17

NUTRIMENT	NORMAL	NORMAL	OPTIMUM
	AFRICAN MIL: DIET.	EUROPEAN HOSP. DIET.	DIET FOR T.B.
PROTEIN	101.8G.	119G.	100-120 G.
FAT	116.6G.	136.1G.	
CARBOHYDRATE	438.2G.	453.2G.	
TOTAL CALORIES.	3,222.	3,516	3,500.
CALCIUM	1848 mgms	1375 mgms.	900 mgms
IRON	33.6 mgms	24.3 mgms	13 mgms
VITAMIN A	2769 I.U.	6898 I.U.	5000 I.U.
THIAMIN	969 I.U.	736 I.U.	600 I.U.
RIBOFLAVIN	1.81 mgms.	2.91mgms	3 mgms.
NICOTINIC ACID	21.1 mgms.	19.6 mgms	20 mgms.
ASCORBIC ACID	81 mgms.	141. mgms	75 mgms.

Dietetic experiments are fraught with difficulty so it was not considered to be worth while to attempt to provide controls. It is therefore impossible to draw any conclusions about the effect of diet.

But when Dr.H.N.DAVIES, Medical Superintendent of Kibongoto Sanatorium, Tanganyika Territory, heard details of the diet employed, he was most envious and opined that it played a great part in obtaining good results.

Collapse therapy was attempted in the treatment of 28 patients, of whom 8 were suffering from primary disease and 20 from reinfection tuberculosis. The forms of collapse therapy employed were phrenic crush, pneumoperitoneum, artificial pneumothorax and thoracoplasty. In the early stages of the investigation, phrenic crush and pneumoperitoneum were the forms most frequently employed; in the later stages of the investigation artificial pneumothorax and thoracoplasty were more frequently employed.

Phrenic crush was employed in the treatment of 10 patients of whom 2 were suffering from primary disease, 1 was in the intermediate stage and 7 were suffering from reinfection tuberculosis. In all cases the phrenic crush was reinforced by a pneumoperitoneum. In the two patients with primary disease, these measures were followed by marked clinical and radiological improvement. (Appendix A, Cases 2 and 3). In the patient who was in the intermediate stage, cavitation had already started and these measures were unsuccessful in closing the cavity. Of the 7 patients with reinfection disease, temporary improvement in the exudative lesions occurred in three patients (Appendix B, Cases 30, 40 and 47). No improvement resulted in the other four patients

(Appendix B, Cases 27, 31, 39 and 49). In none of these 10 patients was the disease entirely controlled by phrenic crush and pneumoperitoneum alone. It is concluded that in East Africans phrenic crush and pneumoperitoneum should only be employed for the purpose of allowing exudative lesions to settle down and that they should not be used in attempting to bring about cavity closure.

Pneumoperitoneum without a phrenic crush was employed in the treatment of 5 patients. In 2 patients (Appendix A, Case 10 and Appendix B, Case 45) it was induced as a forlorn hope. In 2 cases, (Appendix B, Cases 29 and 32) the intention was to perform phrenic crushes as well but the patients refused operation. In 1 case (Appendix B, Case 41) it was employed to prepare the patient for a thoracoplasty. In none of these cases did any marked improvement occur. It is considered that the induction of a pneumoperitoneum alone has no part to play in the treatment of pulmonary tuberculosis in East African Natives.

Attempts to induce an artificial pneumothorax were made in 16 patients of whom 2 were suffering from primary disease, 1 was in the serous stage, 2 were in the intermediate stage and 11 had reinfection disease.

In the 2 patients with primary disease (Appendix A, Cases 6 and 7) shallow artificial pneumothoraces were induced and maintained for  $2\frac{1}{2}$  and  $3\frac{1}{2}$  months respectively. They were abandoned as soon as the

parenchymal disease appeared to have cleared. The rapid improvement that followed this method of treatment was most impressive. Unfortunately no further cases of primary disease were admitted, so it was impossible to investigate this method of treatment more fully.

In the 1 patient in the serious stage of the disease, (Appendix A, Case 23) a pleural effusion had complicated underlying reinfection disease. An air replacement was performed with the idea of minimising the formation of pleural adhesions. But as preexisting adhesions were found the pneumothorax was abandoned as soon as the pleural space had become dry.

An artificial pneumothorax was induced and lost in one patient (Appendix A, Case 9) before he reached East Africa. In the other patient in the intermediate stage of the disease (Appendix A, Case 8) an artificial pneumothorax was induced and was successful in closing a small cavity. Unfortunately fluid developed in the pneumothorax after two months and two months later a spontaneous pneumothorax occurred. The fluid was absorbed but the spontaneous pneumothorax was still present after five months. The patient refused to undergo a thoracoplasty. In spite of these complications the patient was extremely fit at the time of his discharge.

Artificial pneumothoraces were induced in 11 patients with reinfection disease. In four patients, (appendix B, Cases 34, 35, 36 and 52) extensive ad-



hesions were encountered and the pneumothoraces were abandoned. In three cases, a reasonable pneumothorax was induced but adhesions prevented cavity closure and the pneumothorax was ultimately abandoned (Appendix B, Cases 28,31 and 49).

In the case of Civilian MWASIA (Appendix B, Case 27) an artificial pneumothorax was successful in closing a cavity. Unfortunately the pneumothorax was inadequately maintained and the cavity reopened. Thereafter, in spite of increasing the size of the pneumothorax, the cavity remained open.

In the case of Civilian MWATHE (Appendix B, Case 33) a successful pneumothorax was followed after two months by the development of complete collapse of the lung and the appearance of fluid in the pleural cavity.

Private SYONDO (Appendix B, Case 50) absconded from hospital after a successful pneumothorax had been maintained for two months. But during that period marked improvement had occurred.

The only really successful artificial pneumothorax induced in those with reinfection disease was that induced in Sapper PAULO (Appendix A, Case 51). Adhesions were present but they did not prevent good selective collapse. It was maintained for eight months and on his discharge he was referred to a hospital near his home where it was hoped that the pneumothorax would be maintained.

It is clear, from a study of these eleven cases, that the presence of adhesions is common in patients

with reinfection disease. In some cases, these adhesions were doubtless present when the patients were admitted to hospital. But it is possible that in other patients they may have formed while time was spent waiting for the exudative lesions to cool off. For this reason, in the later stages of the investigation artificial pneumothorax was induced earlier without untoward complications. On the other hand, the presence of adhesions may well have prevented disastrous complications in other cases by ensuring that the pneumothoraces were abandoned. For it was not until post mortem examinations had revealed the important part played by tracheobronchial disease in pulmonary tuberculosis in East African natives that the interpretation of the radiographs was placed on a sound footing. Had this point been appreciated earlier the disastrous complications which developed in the case of Civilian MWATHE (Appendix B, Case 33) might well have been avoided. In retrospect, therefore, the words of HOFFMAN seem particularly apt: "Were the opportunity again presented to decide on the use of pneumothorax, it would be withheld in a large number of cases."

Three patients with reinfection disease and one patient in the intermediate stage were treated by thoracoplasty. The first patient so treated was Private KABIRA (Appendix B, Case 48). The operation was decided upon as a last resort in spite of the presence of bilateral disease. The first stage of

the operation was performed in October 1950. The second stage was delayed until January 1951, owing to difficulty in obtaining the services of a sufficiently skilled anaesthetist. In consequence, the surgeon encountered some difficulty with adhesions during the second operation. After the completion of the second stage, the patient made very little headway until he had received streptomycin and PAS for 60 days. Following this treatment, he improved greatly and was eventually discharged to the reserves in November 1951, but sputum conversion was not achieved.

The second patient in whom a thoracoplasty was performed was Private CHULI (Appendix B, Case 40). A bronchogenic spill into the other lung followed the operation but this was controlled by streptomycin. No PAS was available at this time. Sputum conversion was not achieved, but the patient's general condition was greatly improved and it was possible to discharge him to the reserves seven months after his operation.

The third patient on whom thoracoplasty was attempted was Private Kasolo (Appendix B, Case 41). Owing to an unfortunate error in surgical technique he died before leaving the theatre after the completion of the first stage.

The fourth patient in whom thoracoplasty was performed was Lance Corporal CHACHA (Appendix A, Case 9). He was doing very well after the second stage until he developed intrapleural fluid. He

died suddenly early one morning as the result of this intrapleural fluid rupturing into a cavity and drowning him.

As a result of these two deaths, it was impossible to persuade any other patient to undergo operation.

An examination of the postoperative radiographs of these patients shows that the surgeons had not taken away enough of those ribs which they removed. This is a point which further experience would probably have rectified.

Although the results from thoracoplasty were not outstandingly successful, it is considered that this operation should be performed more frequently and at an earlier stage in the disease.

During this investigation, antibiotics and chemotherapeutic drugs were only used on a very limited scale, as the quantity of these preparations available was very small.

Streptomycin was given alone to one case and streptomycin and PAS were given in combination to five cases (Appendix A, Case 2 and 9; Appendix B, Cases 40, 41, 48 and 51). The indications for the employment of these preparations were (a) to cover thoracoplasty operations in four patients; (b) tuberculous laryngitis in one patient; and (c) suspected bronchial disease in one patient. Except in the case of one thoracoplasty patient who died in the theatre and another who died a fortnight after



the second stage the results were excellent.

One patient was treated with thiacetazone. The exhibition of this drug was followed by the final resolution of his primary complex, which had previously been delayed for many weeks (Appendix A, Case 3). This drug has been used successfully in a number of cases by Dr. W. S. HAYNES at Mombasa. He has found that in East African natives toxic reactions are rare and that, when they do occur, they clear up rapidly if the drug is withdrawn.

The advent of these new preparations is bound to revolutionise the treatment of pulmonary tuberculosis in East African natives and to render out of date the conclusions which may be drawn from the study of these cases.

These conclusions are as follows:

- (a) rest in bed and dietetic measures are by themselves inadequate forms of treatment;
- (b) phrenic crush and pneumoperitoneum are only palliative measures;
- (c) a shallow artificial pneumothorax appears to be a most useful form of treatment in patients with primary disease but this line of treatment needs exploration;
- (d) cases of reinfection tuberculosis, suitable for treatment by artificial pneumothorax, are uncommon;
- (e) major surgery is, therefore, required in most cases of reinfection disease.

TABLE 18.

Treatment	Stage of Disease	Result				Total.
		Im- proved	No Change	Worse	Died	
REST	PRIMARY	2	-	4	1	7
AND	SEROUS	15	-	-	1	16
DIET	INTERMEDIATE	-	-	3	-	3
ONLY	REINFECTION	1	5	18	4	28
PNEUMO -	INTERMEDIATE	-	-	-	1	1
PERITONEUM.	REINFECTION	1	2	-	1	4
PHRENIC	PRIMARY	2	-	-	-	2
CRUSH AND	INTERMEDIATE	-	1	-	-	1
PNEUMOPERI- TONEUM	REINFECTION	3	4	-	-	7
THORACOPLASTY	INTERMEDIATE	-	-	-	1	1
	REINFECTION	2	-	-	1	3
STREPTOMYCIN	PRIMARY	1	-	-	-	1
AND PAS.	INTERMEDIATE	-	-	-	1	1
	REINFECTION	3	-	-	1	4
THIACETAZONE	PRIMARY	1	-	-	-	1

In countries, where the facilities for treating Pulmonary Tuberculosis are incomplete, the physician finds himself recurrently upon the horns of the dilemma: if he refrains from treating a patient, the man will certainly die; if he widens the indications for such therapeutic procedures as are at his command, serious complications may ensue. In East Africa, the majority of physicians echo the Somali phrase recorded by Burton - Allah have mercy upon thee - and refrain from treatment. But here and there courageous physicians choose the other alternative.

In this investigation each method of treatment was explored in turn and the indications for its use extended as far as reasonably possible. The results, which are summarised in table 18, are definitely encouraging.

In 1933, HARRIS (19) quoted from the Annual Medical Report for Uganda written in 1931 by FLANNERY (20): "Tuberculous tuberculosis is still one of the most serious of diseases met with in Uganda. I have not seen it since I came to this country. I cannot claim to have observed a single case."

In 1940, H. J. DAVIES (18) stated that in his experience the average expectation of life for a tuberculous native with tuberculous tuberculosis was not more than eighteen months without treatment, but life could be considerably prolonged by treatment with isoniazid.

In 1947, J. A. DAVIES (19) concluded from his study of post mortem material in Uganda that "a very high percentage of natives develop a primary complex which they do not even realize, but even those who are not free from the danger of death from tuberculosis. The post mortem study of a massive primary complex... demonstrated... termination occurs... and death soon takes place."

In 1951, HARRIS (20) stated in the Annual Medical Report for Uganda: "The mortality of natives who have tuberculosis... they are... they are..."

CHAPTER 13. PROGNOSIS.

In 1930, KLEINE (34), who had been working in the interior of Tanganyika, came to the conclusion that in East African Natives Pulmonary Tuberculosis runs a relatively benign course and may be overcome if the natives are well fed and kept quietly at home in the reserves.

In 1933, STONES (53 B) quoted from the Annual Medical Report for Uganda written in 1931 by FLEMING (23): "Pulmonary Tuberculosis is still one of the most hopeless of diseases met with in natives. I have had 28 cases through the ward this year. I cannot claim to have arrested a single case.

In 1946, H.N.DAVIES (18) stated that in his experience the average expectation of life for a Tanganyikan Native with Pulmonary Tuberculosis was not more than eighteen months without special treatment. But life could be considerably prolonged by treatment with collapse therapy.

In 1947, J.N.P.DAVIES (19) concluded from his study of post mortem material in Uganda that "a very few natives develop a primary complex which they heal and even calcify, but even these are not free from the danger of death from tuberculosis. The rest develop a massive primary complex ---- widespread dissemination occurs --- and death soon takes place.

In 1951, SANTON GILMOUR (25) wrote of the natives of Uganda: "The majority of cases were young adult males. They are sick when they seek treatment ---



are acutely toxic and go rapidly downhill. In contrast, there are known cases whose lesions have become quiescent ---- and who have since led normal lives. Some films show a typical healed primary complex. Idiopathic pleurisy occurs without evidence of subsequent lung involvement and cases have been followed after recovery into active working life.

In 1951, CLARK (13) reviewed 300 cases of Pulmonary Tuberculosis seen at Fort Hall, Kenya and concluded: "I have been much struck by the steady, inevitable and rather rapid decline in the majority of my cases - even in the cases which I have every reason to believe were early cases when I first saw them. There are exceptions but they are very few." But earlier in his paper, he had made this important statement: "If X-rays were available, it would almost certainly mean that more early cases were diagnosed and the prognosis would appear better than it does with the means of diagnosis at our disposal."

This remark is amply confirmed by the study of table 19 which shows the end results obtained in the present series.

TABLE 19.

Stage of Disease	Results			
	Improved	No change	Worse	Died
PRIMARY	6	-	-	1
SEROUS	15	-	-	1
INTERMEDIATE	1	-	-	2
REINFECTION	7	8	7	6
TOTALS	29	8	7	10

It is, of course, impossible to make any dogmatic statements about prognosis without following these cases up for many years. An attempt was made to follow up all the cases discharged from hospital. But despite the assistance afforded by the administrative officers of the Colonial Medical Service only four were traced by the District Medical Officers.

Private RUNGONGO (Appendix A, Case 2) was in excellent health five months after discharge from hospital.

Lance Corporal F'OBUYA (Appendix A, Case 8) was in excellent health two months after discharge from hospital.

Private KINDA (Appendix A, Case 15) was in excellent health twenty one months after discharge from hospital.

Private ONGERI (Appendix A, Case 22) had deteriorated and had a high sedimentation rate two years after leaving hospital.

There seems to be little doubt that the remarks of BOGEN (5) in 1931 about American Negroes are also true of East African natives: "At one time it was believed that once tuberculosis in a coloured person was diagnosed the prognosis was hopeless. It is now known that prognosis depends upon the stage at which the disease is discovered."

CHAPTER 14.      SUMMARY AND CONCLUSIONS.

The literature dealing with the incidence of tuberculous infection among the native inhabitants of those parts of Africa, where miscegenation has not occurred, has been reviewed. It is suggested that the techniques employed by the older investigators were inadequate and that their results were consequently incorrect. It is also suggested that many of the available figures would not stand up to statistical analysis and that the figures in the different papers are not truly comparable one with another. In consequence too much stress should not be placed on any theories erected on the basis of these papers. And in particular what is said to have been the case many years ago in different parts of Africa is certainly not true of East Africa at the present time. Tuberculous infection is now widespread throughout all three East African Territories and this spread of infection has occurred without the outbreak of an epidemic of tuberculous disease.

The literature dealing with the incidence of tuberculous disease in the natives of Africa has been reviewed. The attack rate of tuberculous disease in a population of selected East African natives living under good conditions and subjected to the stress of peace time soldiering has been recorded. It is concluded that in the event of large numbers of East African natives being called to the colours the incidence of tuberculous disease should not

provide a problem that cannot be easily dealt with by the Army Medical Services.

The literature dealing with the natural history and pathology of tuberculosis in the natives of Africa has been reviewed. The natural history and pathology of the disease as it now occurs amongst adult male East African natives serving in military or paramilitary units in East Africa has been described. It is concluded that such natives now have a definite degree of resistance to the tubercle bacillus and that the acute nature of the disease is conditioned chiefly by their high degree of hypersensitivity.

The literature dealing with the treatment of pulmonary tuberculosis in African natives has been reviewed. This literature is very scanty and recourse has been made to the literature on the treatment of pulmonary tuberculosis in American negroes. The methods of treatment employed in dealing with fifty four cases of pulmonary tuberculosis occurring in adult male East African natives are described. The main conclusions reached are that rest in bed alone is seldom sufficient; that collapse therapy is disappointing; that major surgery is required in most cases of reinfection disease. The rôle of antibiotics and chemotherapeutic drugs has been briefly referred to: it is considered that they will revolutionise treatment in East Africa as they have done elsewhere.

The literature dealing with the prognosis of tuberculosis in East African natives has been



reviewed. The results obtained in fifty four cases have been recorded. It is concluded that prognosis is determined by the stage at which the disease is discovered and that the pessimistic outlook so prevalent among doctors now working in East Africa has been engendered by lack of diagnostic facilities.

The outlook of most workers on Tuberculosis in Africa seems to be coloured too darkly by reference to the past - reference too frequently resting at hearsay and not pursued in the library. The present position is indeed grave but this is due to economic circumstances and not to any inherent peculiarity of the disease.

"Say not the struggle naught availeth,  
The labour and the wounds are vain,  
The enemy faints not, nor faileth,  
And as things have been they remain."

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