MALNUTRITION IN THE SOUTHERN RHODESIAN BANTU

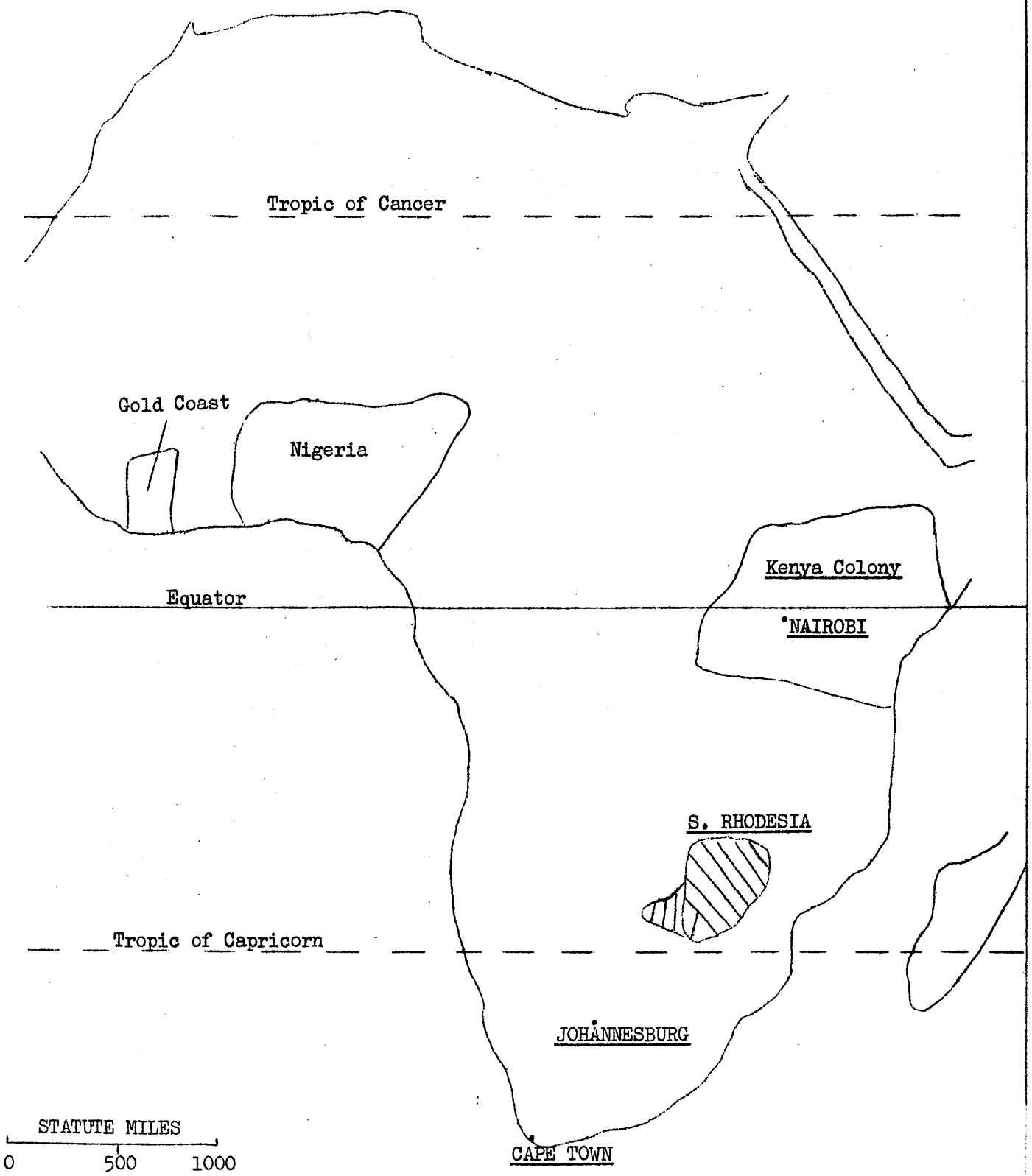
by

Charles R. Forrest, M.B. Ch.B.,
Southern Rhodesian Medical Service

Submitted as a thesis for the degree of Doctor of Medicine of the University of Glasgow

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

AFRICA


Tropic of Cancer
Gold Coast
Nigeria
Kenya Colony
Equator
NAIROBI
S. RHODESIA
Tropic of Capricorn
JOHANNESBURG
CAPE TOWN
STATUTE MILES
0
500
1000

I N D E X

-----oOo-----

INTRODUCTION

The objects of this investigation were to discover the importance, extent and type of malnutrition among the natives of Southern Rhodesia. I have made an attempt to present a complete and balanced picture. The opportunity was taken to study certain aspects in some detail - the skin manifestations and scurvy. I have tried to stress negative findings and indicate possible lines of research for specialised workers. Where a great deal of work has already been done, I have limited myself to discussing the question in relation to its local importance. I wish to emphasise that although my observations and conclusions have been drawn from Southern Rhodesia, malnutrition is common to a vast area of South and Central Africa. This contribution should be regarded as a small link in the growing understanding of a problem, which affects millions of human beings spread over the greater part of the African Continent.

SOUTHERN RHODESIA AND ITS INHABITANTS

Southern Rhodesia is a compact area of the central African plateau lying between 15°30' and 22°30'. The climate varies from mediterranean to subtropical depending on the altitude, which ranges from three to five thousand feet. The chief sources of wealth are gold mining and agriculture, especially tobacco farming. Since the second world war there has been a great increase in industry. European occupation, inspired by Cecil Rhodes, commenced at the end of the last century. Rhodes visualised the colony as a permanent home for white people; with this end in view the country was divided into areas of European and Native settlement. Emigration has been encouraged, and the European population has reached 130,000. (1950 census). Since the introduction in 1890 of British administration, the African population has more than doubled; in 1949 it was estimated at 1,898,000. (Public Health Report, 1950) The original Bantu were sparse upon the land and practised a semi-nomadic agriculture, which allowed the soil time to recover. With increased cultivation, soil erosion has become a serious problem, particularly in the native reserves. The world-wide problem of more mouths and fewer

crops exists in Rhodesia, which in recent years has been converted from a food-exporting to a food-importing country.

The Bantu race is generally supposed to have originated in the area of the Great Lakes. Successive waves of invaders swept south, eventually reaching the present Cape Province in the eighteenth century. In 1837 a marauding army of the powerful Zulu nation suffered a serious reverse. Rather than risk the penalties of defeat, these Impis, instead of returning to Natal, retraced the steps of their ancestors northwards. These displaced Zulus founded the Matabele Empire which extended over the area now known as Southern Rhodesia. The Mashona tribes which are thought to be descendants of an earlier Bantu influx, were suffered to exist in a state of subjugation. The primitive native was an agriculturalist, cattle breeder, and hunter. His numbers were controlled by tribal warfare and disease. Land and game were plentiful. The arrival of the white man changed everything. The European came as a settler; as the conqueror, he naturally claimed the best resources for his own. In Rhodesia the population is divided by the colour bar into dominant white and subservient black sections. The European has not only monopolised the professions, commerce and all the skilled trades, but has

debarred the African from the lucrative pursuits of mining and tobacco growing. Economically the Bantu constitute a cheap labour force; local custom and sometimes legal sanctions perpetuate this status. No apology is offered for this excursion into local politics. Nutrition has an intimate association with social problems. It is impossible to be unbiased towards racial questions. A believer in the present status quo in Central Africa would justifiably point out the material benefits conferred on the native. The Bantu has had the opportunity of many centuries to develop his country. He has produced no civilisation, no culture, not even a written language. Fifty years ago he had not advanced beyond a tribal system of society.

Although without a direct bearing on my subject, an understanding of the African patient is essential for the appreciation of this thesis. The title is perhaps misleading as a large number of the natives concerned originate from the surrounding territories of Northern Rhodesia, Nyasaland and Portugese East Africa. They come to Southern Rhodesia for work and often settle down permanently in the colony. Physically they are indistinguishable, the only variations being in language and tribal custom. They all show the characteristics of the Bantu race, viz., dark skin,

squat nose, thick lips and curly hair, this last being the most constant feature. The primitive African attributed illness to supernatural agencies. This implicit belief in witchcraft is dying out, but still lingers on even in outwardly sophisticated natives. The modern African is abandoning the medical creed of his ancestors and turning to the panacea of injections. This faith in the needle is conscientiously exploited by many practitioners, European and African, qualified and otherwise. The important factor is the syringe, the native is not concerned with the substance injected. In many cases he seeks the advice of the government doctor as a last resort. Hospitalisation is still unpopular, natives frequently request discharge in the condition a European would expect admission. There is, however, a growing confidence in modern medicine (apart from injection practice). More and more Africans are presenting themselves at the State hospitals.

All the parasitic infections are widespread; the native has aptly been described as a 'walking zoo'. Different surveys have revealed varying percentages of schistosomiasis, depending on the method of investigation. Gelfand (1950) in a comprehensive and careful study showed

that 82 per cent of adults are infected. In the same investigation, it was demonstrated that over 30 per cent of Africans suffer from ankylosiomiasis. The vast majority of natives suffer from chronic malaria. To quote Dr. Gelfand (1948):-

> "Malaria is the most common disease of Africa, and for practical purposes we may look upon the African as almost certain to be infected".

The same authority has rightly stressed the importance of multiple pathology. The finding of a single parasitic infection must not be taken as the cause of a patient's symptoms. It is not uncommon to encounter natives harbouring malaria, bilharzia and hook-worm, yet enjoying what the African considers excellent health. The native in many cases is able to achieve a symbiosis with his numerous parasites. The social and economic position of the Bantu is reflected in their medical conditions. Scabies, impetigo, tuberculosis, trachoma, leprosy, and all the venereal maladies are routine diseases; in other words the results of poverty, ignorance and dirt.

Method

Since joining the Southern Rhodesian Medical Service, I have been employed as a general duty officer in all parts of the colony. My results have been taken from observations made in every area of the country. When I began on this survey I was under the impression that I was breaking virgin soil. However, I gradually realised that much of the work I undertook or contemplated, had already been covered. Medicine in Rhodesia suffers from a lack of co-ordination. Papers either are not published or appear in an obscure often non-medical publication. My colleagues have been exceedingly helpful in allowing me to use their knowledge and experience, with the result that the number of personal communications is unusually large. As most of my authorities are resident in Africa, I have taken the liberty of departing from the usual convention by giving the designation of my informants in the references.

The approach to the subject has been entirely clinical. My conclusions have been drawn from a survey of 500 Africans. The original intention was to divide the subjects into indigenous and alien natives. I soon realised that as many of the foreigners either become permanent residents or spend the greater part of their active life in the colony, this distinction was unnecessary and unpractical. The

presence of malnutrition among alien natives is evidence that results obtained in Rhodesia are of some application to adjacent territories. I was at some pains to:-

(a) Examine a fair cross section;

(b) Include representatives from the more privileged classes.

Malnutrition was assumed to be present when a subject showed one or more of the following signs:-

(1) Pellagra

(2) Scurvy

(3) Follicular seborrhoea, cheilosis, angular stomatitis, cheilosis. (These manifestations have been classed as ariboflavinosis).

(4) Phrynoderma.

Hyperkeratosis of the skin and enlargement of the parotid glands have been recorded, but have not been included in calculating the total percentage suffering from malnutrition. To anticipate a possible criticism, I realise the lesions described as ariboflavinosis are not specific to a deficiency in that vitamin. In an exaggerated form, however, they make up the characteristic features of that syndrome. With many physical signs the pathological merges imperceptibly into the normal; this seems particularly true in nutrition. I had considerable difficulty in deciding on the minor deviations. I made it a rule

only to accept definite changes, and to ignore all doubtful cases.

In the appropriate sections, the individual lesions have been discussed in some detail. Before any sign was recorded as present, certain conditions had to be satisfied. The diagnosis of pellagra was based on the classical dermatitis. This rash is quite distinct from the rough cracked desquamating skin seen in many Africans, which I have called hyperkeratosis. Unless the gums were hypertrophic, discoloured and showing at least one bud, scurvy was not recognised. Follicular seborrhoea was only marked positive when several typical comedones were visible. I found difficulty with cheilosis, angular stomatitis and glossitis; only well developed examples were accepted. A very large percentage of natives when examined in a good light show small areas of permanent gooseflesh, just above and below the olecranon process. A few follicles in that site was not taken as phrynoderma, definite raised projections had to be visible on the extensor surface of a limb. The criteria applied were intentionally severe. The percentage of malnutrition thus obtained is certainly not an overestimate.

I have seen two experienced physicians examine a patient's mouth and disagree as to whether the gums show scorbutic changes. All nutritional surveys suffer from

this human element. Each observer consciously and unconsciously adopts different criteria. The nomenclature is also unstandardised. This is particularly true of the cutaneous manifestations. Pellagroil skin, cracked skin, giraffe or reptilian skin and crazy pavement dermatitis are all current terms, applied to similar if not identical conditions. In an attempt not to increase the confusion, I have classed all abnormal skin under 'hyperkeratosis'.

GENERAL DISCUSSION

Nowhere is the adage that medicine is not an exact science better illustrated than in nutrition. A great deal of work has been done in this field. Unfortunately, many investigators fail to agree and sometimes flatly contradict each other. Popper (1944) claimed that normal kidneys contain no vitamin A but that in renal disease its presence could be demonstrated. An opposite conclusion was reached by Laurie et al (1941) who considered that it is the healthy kidney that stores the vitamin whereas it disappears from the diseased organ. The importance of the vitamins has been questioned by such competent authorities as Meiklejohn (1949) and the Gillmans (1951). Two carefully controlled experiments on human subjects have shown the daily requirements of vitamins A and C are almost certainly substantially less than previously recommended. (Medical Research Council, 1948, 1949) These results cast doubts on many earlier investigations. It must be admitted that the medical profession has been fascinated by these substances to the exclusion of the other essentials of the diet. Just before the last war the annual world output of original papers on vitamins was over three thousand (Bicknell and

Prescott, 1946). The more mundane proteins, salts, etc., have not received the same attention. Many nutritional mysteries still require solution, including the relative importance and interdependance of the different food factors. The vitamin plays a part, probably a vital one, but it is only a member of a complex physiological orchestra. This thesis, however, deals mainly with vitamins, only because the effects are easily recognisable clinically. Equally valuable research could be carried out on problems such as the protein consumption. The frequency of minor vitamin deficiency conditions should be regarded as a manifestation of the general inadequacy of the diet in all components.

The Gillman's work on malnutrition is challenging and original. The concept of a dearth of one substance producing a particular syndrome is no longer accepted. Instead deficiency disease is explained in terms of "disturbed physiological relationships". This explanation is possibly nearer the truth in pellagra than the nicotinic acid theory. In the writer's opinion, the Gillman brothers deserve careful study and should be accepted, but with reservations. Their statement that a vitamin deficiency state cannot be induced in a healthy subject is incorrect,

as illustrated by the Sheffield experiment which curiously enough they did not know about. However, this mistake does not detract from the value of their researches into African malnutrition, and is only quoted to show that their sweeping theorisation is sometimes at fault.

Malnutrition is of importance in most branches of African medicine. Maladies which are extremely rare in British practice are common in Rhodesia. Thus pellagrins are frequently seen in the mental wards, (Higgs, 1951, personal communication); Bitot's spots and night-blindness are often encountered at the eye clinic. (Sparrow, 1951, personal communication). In addition to the accepted nutritional disorders several conditions have in part at least a dietary aetiology such as hepatic cirrhosis and carcinoma. In a series of a thousand consecutive post mortems carried out at Salisbury Native Hospital, ninety-six showed cirrhotic change and there were eight cases of primary carcinoma of the liver. Other disorders, where the nutritional basis is less well defined, are often found in Native Hospitals; these include tropical ulcer and complex anaemias.

Trowell (1950) in Uganda and the Gillmans in Johannesburg, working along similar but not identical lines have reached the same basic conclusion:-

"From infancy the African is handicapped by his food".

The Gillmans would go further and say from conception. Conditions vary in the two areas. Trowell's deductions are drawn mainly from Kwashiokor and its after effects. The Gillmans although giving considerable attention to this disease, have concentrated on pellagra which strangely enough is uncommon in the Northern Territories. My own contribution from Rhodesia which lies between these two centres, confirms the findings of these two authorities. The majority of natives examined showed signs of past or present malnutrition. These cutaneous lesions can be taken as evidence of internal damage (Gillmans); therefore it can be said:-

"The bodies of most Rhodesian Bantu bear the scars of malnutrition".

In other words, the native receives permanent injury from his diet. This does not mean that the subject is incapable of strenuous work, or that he does not enjoy what he considers good health. The effect of this chronic mass malnutrition is reflected in the low social, intellectual and physical achievements of the race.

THE AFRICAN DIET

In terms of the Constitution and the Land Apportionment Act, Southern Rhodesia is divided into several land categories. Of these the European area occupies about half the total area of the Colony and contains about 37 per cent of the population. The native areas occupy only about 34 per cent of the Colony's total area but contain about 63 per cent of the population. These native areas are set aside purely for native occupation. However, approximately half the adult male population is permanently absent in employment in the European area and outside the colony.

The native is in a stage of rapid transition from peasant agriculturist to industrial worker. This change from village to city and often slum life has forced the African to alter his tribal eating habits, which like the dialects vary considerably even within the boundaries of Rhodesia. The basis of the village diet is stiff maize porridge (Sodza); no meal is complete without it, in fact the term may be used as a synonym for food. It is of interest and perhaps importance that maize is not indigenous to Rhodesia. It appears to have been introduced from Natal by the Matabele invader in the middle of the last century.

Owing to the ease of cultivation and high yield, it quickly displaced the small sorghum grains* which had been the staple crops. It is known that maize was in general use by 1912 (Baker-Jones 1952, personal communication). Complementary to Sodza is the relish (Usavi). There is no European equivalent of Usavi which is probably best regarded as a thick sauce. However, it is much more than a flavouring agent and is the usual method of serving vegetables. Usavi is the highest expression of Bantu culinary art and the ingredients vary from rats to sweet potatoes. Meat and milk are consumed in varying quantities, the former often only being available for special occasions. Additional nourishment is obtained from such unlikely sources as flying ants, caterpillars, grass hoppers and field mice. A valuable and popular article of food is the ground nut. Kaffir beer is drunk by the majority of adult males. This beverage has not received much attention from other investigators, but taken regularly in moderation it could be a valuable addition to the diet.✓ Unfortunately the native tends to imbibe

* Local names being rupoko, munga and mapfundi.

✓ The nutritional values of the common African foods are given in the appendix.

large quantities at irregular intervals. Dairy produce plays little part in the unsophisticated African's menu. He is unfamiliar with the making of butter or cheese, eggs are the subject of various taboos and are not consumed in any quantity. Bulk is the main consideration in native eating, no meal is satisfactory unless the stomach is distended. Like the Indian peasant the African has two meals a day at about 11 a.m. and dusk; by preference he starts the day fasting. Because the Bantu have survived for centuries it is generally assumed that this traditional diet is satisfactory. While this was possibly true for the placid life of the kraal (village) the regular eight or more hours of manual work demanded by European supervision throw a much heavier burden on the metabolism.

The daily average calorific intake of the rural native has been estimated at 2,360. This figure was arrived at by a detailed investigation of all the food stuffs known to be available in the reserves. The writer has discussed this point with the Chief Nutrition Officer who considers the estimate to be reasonably accurate, and would not allow more than 30 calories per diem from unaccounted sources such as rodents, etc. Deductions made from direct observation put the daily intake below two thousand. Similar calculations have been made regarding protein.

The average daily intake has been estimated at 71.8 gms. (mainly from maize); the weekly consumption of meat and fish in the native reserves is given as 4½ ozs. and 0.3 ozs. respectively. In comparison with many native peoples, the Rhodesian Bantu are fortunate. The calories available are higher than in Columbia, Mauritius, China, Japan, Indo-China, Pakistan, Burma, Ceylon and India. The protein consumption is higher than all the Eastern countries and some of the American republics (Baker Jones, 1952).

The urban native either receives food from his employer or a money allowance in lieu. Rations are issued uncooked, communal feeding is practised by a few firms but is not popular. Tea, sugar, jam, bread and mineral waters are often purchased as additions to the basic diet, seldom meat, milk or vegetables. A social investigation by a prominent welfare worker (Ibbotsen, 1942) on over twenty-five thousand natives employed in urban areas, revealed the fact that many Europeans consider 2 lbs. of meat and 14 lbs. of mealie meal an adequate weekly ration. This survey was done ten years ago and there has undoubtedly been some improvement. However, this statement still holds good for a large number of employers. The same worker also observed that unmarried men are often too lazy or too tired to do

much cooking and exist mainly on bread and tea.

The generalisations of the preceding paragraphs have been quoted from official documents.* To check these, and to obtain more personal and detailed information, a hundred Africans (mainly hospital patients) were questioned about their food (see appendix). All nutritional diseases were excluded, and as all classes of natives attend Government Hospitals, it is felt that a reasonable cross section of the population was investigated. This approach is of value as the differences between the diet of the rationed, urban, self supporting and rural native are illustrated. The following facts were established. Mealie meal is the universal staple, only one subject did not eat this cereal. Meat is generally eaten in varying amounts. The anti-scorbutics received special attention, consumption varies from practically nil to a regular intake of fruit or vegetables. The ascorbic acid content of at least seventeen of the diets was extremely low. The criteria applied were not strict, any ration containing more than one orange per

* See (1) Baker-Jones; (2) Annual Reports of Nutrition Council; (3) Report of Social Security Officer.

month was not counted as unsatisfactory. Therefore the only recognisable sources of vitamin C for nearly 20 per cent of the population are meat and Kaffir beer. Dried beans are a common food stuff, but ground nuts which have been in short supply for several years are no longer a regular article of diet for the town native. Milk is drunk in varying amounts; the town dweller regards it as an addition to tea while for some village natives milk ranks only second to mealie meal. The use of milk is not universal, prejudice exists among certain tribes who regard it as suitable only for infants and calves. Most natives eat eggs, but at such long intervals that they cannot be regarded as an important food stuff. It is of interest that several men claimed never to have even tasted an egg. As has been previously mentioned, the egg is subject to various tribal taboos. Some communities believe they are harmful to pregnant women, others that eggs will cause convulsions in children.

The Government of Southern Rhodesia has at least in theory recognised the importance of nutrition. A nutrition council has been set up, reports have been presented and recommendations made, but always to the interested few. The Mines and Minerals Act lays down a minimum ration. This

scale is only obligatory for mine employers, and is not enforced anyway. To my certain knowledge these and other industrial regulations are generally ignored. In the appendix the rations scales of various employers (including the Government) are recorded. The management of Mine A was enlightened and progressive, its labour could be described as well fed and cared for. However, a comparison with the official rations revealed a discrepancy in meat, ground nuts and vegetables.

Government Rations (weekly)		Mine A Rations
Maize Meal	10½ lbs.	14 lbs.
Ground nuts	1 lb.	½ lb.
Dried beans	2 lbs.	2½ lbs.
Salt	3½ ozs.	6 ozs.
Sugar	-	6 ozs.
Vegetables	2 lbs.	1 lb.
Meat	2½ lbs.	2 lbs.
Tobacco	-	2½ ozs.

Mine A gave rations for dependants, this practice is by no means universal and there is no legal compulsion to do so.

The attitude of employers varies from paternal solicitude to cynical indifference. Few Europeans even

from enlightened self interest realise the importance of nutrition or have any understanding of the subject. Farmers who fully appreciate that a horse will only give its best work when adequately nourished, fail to apply this fact to their labourers. One hears statements that "the nigger can live on the smell of an oily rag" coupled with tirades on their inefficiency. Even when adequate supplies are available the native employee often receives miserable food. I have seen scurvy in farm workers, and been told the "Baas (master) gives us meat when a beast dies". On a large ranch where the cost of a cow is negligible, the cattle hands were given meat once a month, (the firm in question produces a world famous brand of canned beef). Many employers, particularly farmers, in addition to rations allow the native to cultivate a piece of land for his own use. This practice is by no means universal and of course only possible in country districts. Frequently the African does not take advantage of this garden which can be of great benefit to a family. With careful husbandry, it is sometimes possible for a farm labourer to both feed his family and still have a surplus for sale.

The native is usually described as conservative.

Although suspicious of any European innovation (however well intentioned), he is readily capable of acquiring new tastes and habits. In 1950 Coca-Cola was almost unknown in Rhodesia, through vigorous advertising this mineral water has now (1953) penetrated into the heart of the reserves and is consumed in large quantities by the native population. Tea, some years ago, was the subject of a similar campaign. Harmless as these commodities are in themselves, they are expensive items to an impoverished population. There is a large trade of protein food stuffs from the Native to the European areas, at least a percentage of the profits must be exchanged for these luxuries. While criticising the European for his ignorance and indifference, the difficulties which face a reformer should be appreciated. Many schemes fail through lack of co-operation. African labour is migratory, and it is too much to expect an employer to feed up a native who will leave him in a few months or weeks. There is an unfortunate tendency for the employer to evade his responsibility by giving money instead of food. This is also popular with the African, who is free to spend his augmented wages as he pleases.

It has been assumed for the sake of brevity that one ration is consumed by one native, in the case of married men

this is not true. Many families subsist almost entirely on the head of the family's allowance. On a monthly income of often less than £3,* there is little cash left for extra food after meeting essential expenses such as fuel, clothing, taxes, etc. Many men have to live on a fraction of their rations. It is impossible to estimate the portions of food eaten by members of a household; however, it should be realised, that in many instances the quantities theoretically alloted to one worker have to be divided among several mouths.

Expressing the calorific value of mealie meal† in terms of white bread, the weekly rations of the less fortunate Bantu workers are equal to twenty pounds of bread and two pounds of meat (including bone). This diet is deficient in all the essential components, and certainly not conducive to full health and efficiency, especially for a labourer. The calorific value (official figures) of the Government rations is approximately 2,500 calories per day. It contains 114 to 140 grams of protein and 57 to 78 grams of fat, only about 21 grams in each case being first class. The

† Mealie Meal (96 per cent extraction) = 1,633 calories per lb.
White bread = 1,136 calories per lb.

* The minimum legal wage for a native employee is £2:2:0 per month (with rations and quarters provided). However, many Africans earn much larger sums.

calcium content is in the region of 0.4 grams, phosphorus from 1.6 to 2.2 grams and iron from 9.7 to 12.3 mgms. The vitamin content is given as A 966 units, Vitamin B1 543 units (1.6 mgms.), Riboflavin 1.5 mgms., Nicotinic 30 mgms., and Vitamin C as 28 mgms. The chief faults of this ration are the insufficient quantities of Vitamin A and calcium; in other respects it could be described as just adequate. Unfortunately, all the available evidence shows that the majority of natives do not obtain anything like this allowance, and therefore the average African's diet must be deficient.

EXAMINATION OF FIVE HUNDRED AFRICANS

Five hundred Africans were examined for signs of malnutrition. The results of this investigation may be conveniently followed by discussion on the deficiency diseases encountered in Southern Rhodesia. The examinations were conducted in various parts of the Colony and included hospital patients, convicts, employees of industrial concerns, police and native troops. Skin changes, lesions of the buccal cavity, and enlargement of the parotids were recorded. The liver was palpated as a routine in two hundred and fifty cases. No correlation between the extent of the deficiency signs and hepatic enlargement could be demonstrated. Moreover as the liver was palpable in over ten per cent, it was decided to accept minor degrees of hepatomegaly (up to two fingers in the nipple line) as a normal finding in the African. In an attempt to obtain a fair cross section, as many different classes of native as possible were examined. When using hospital patients all nutritional syndromes and chronic wasting diseases such as pulmonary tuberculosis were excluded. To avoid any criticism that only the less fortunate categories had been investigated, a hundred police were included in the survey. These men may be considered as belonging to the privileged

classes. Out of the five hundred Africans examined, there were five cases of pellagra and one of scurvy, all occurring in convicts, phrynoderma was present in 37 per cent. Lesions usually described as ariboflavinosis were found in 44 per cent. 65 per cent of all the subjects showed one or more of the clinical signs of deficiency. Two changes which were recorded but not assessed as evidence of malnutrition were hyperkeratosis (50 per cent) and enlargement of the parotid glands (5 per cent). These phenomena will be referred to more fully in a later paragraph. The figures for the police and army were scurvy and pellagra nil, phrynoderma 30 per cent and ariboflavinosis 40 per cent. The physique and general well being of these men was good and the changes were definitely milder than seen in the general population. However, these findings prove the point that nutritional deficiencies occur in all sections of the native population.

TABLE I

Results of clinical examination of 500 normal Africans

Section	Scurvy	Pellagra	Phryno-derma	Aribo-flavin-osis.	En-larged Paro-tids	Hyper-kera-tosis	Percentage showing definite signs of malnutrition
	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent
General Popu-lation	.2	1	37	44	5	50	65
Police and Army			30	40			

Until my own examinations were almost completed, I was under the impression that no survey of the Southern Rhodesian native had been undertaken. In 1942 500 patients in the medical wards of Salisbury native hospital were examined for signs of malnutrition. The findings were not published in any medical journal, and were obtained for the instruction of a National Health Commission. I have corresponded with the officials concerned, and no copy of the paper is now extant. (Martin, Baker-Jones and Gelfand, personal communications 1952).

The following Summary is taken from the report of the Social Security Officer (1944):-

"Of 500 native cases admitted to medical wards in Salisbury in the first half of 1942, 58.6 per cent were suffering from malnutrition. With indigenous natives the proportion was 45.9 per cent compared with 67.5 per cent in the case of aliens. About 3 natives in every 4 were suffering either from malnutrition or a disease such as bilharzia or hook-worm. It is considered by the Public Health Department that this sample reflects very fairly the incidence of malnutrition among natives in general. The natives were admitted to hospital suffering from pneumonia, tuberculosis, severe malaria and other complaints. There was hardly any instance of an admission for malnutrition, bilharzia or even hook-worm. The tests imposed were so severe that the incidence of malnutrition shown is considered a conservative one".

This work could be criticised on the grounds that the patients of a medical ward are not a fair cross section of the population. Individuals weakened by malnutrition are generally accepted as being susceptible to infection. However, even sustaining this objection, the Salisbury investigation indicates a high incidence of malnutrition.

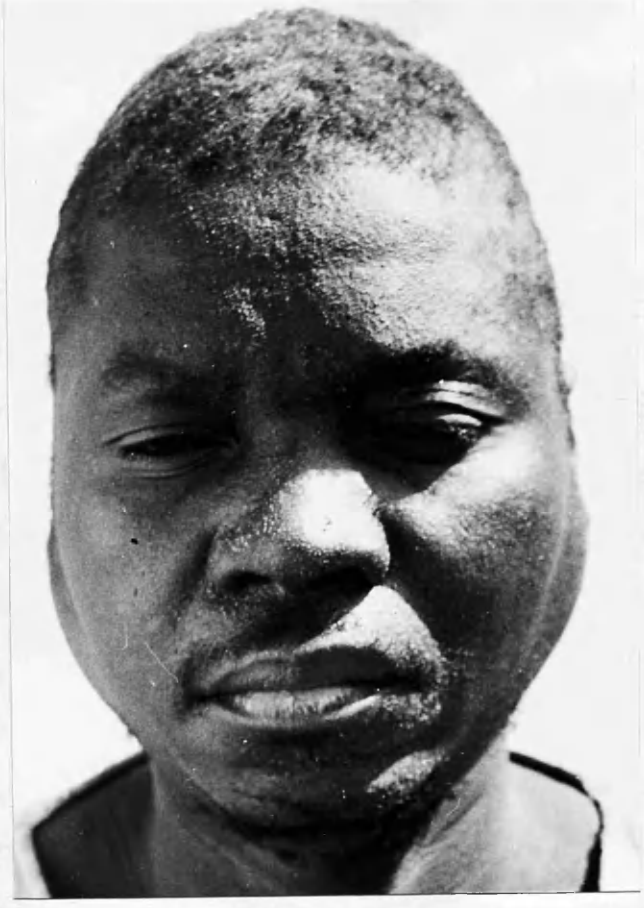
Trowell (1948) examined five hundred African Railway workers in Nairobi. As the clinical investigation was conducted along similar lines, a comparison can be made with my own findings. Phrynoderma was detected in 38 per cent. The conjunctivae was abnormal in 30 per cent and 3 per cent showed Bitot's spots. Riboflavin deficiency was found in 4 per cent; as Trowell only noted the mouth changes and did not include follicular seborrhoea, these figures do not represent a serious difference (about 3 per cent of the Rhodesian natives had these lesions. Hyperkeratosis which he called crackled skin was present in 42 per cent. The parotid glands were judged to be enlarged in 18 per cent. 2 per cent had an enlargement of the liver of over two fingers in the mid-clavicular line. In an attempt to assess the results of three and a half years of war time rationing, Sydenstricker (1944) examined 5,000 British civilians from groups which might be expected to show adverse effects. "There were three elderly people with typical Bitot's spots but without other evidence of deficiency disease. Many women and children had mild folliculosis but no other signs or symptoms of Vitamin A deficiency. One mild case of pellagra was seen, and eight women had characteristic signs of riboflavin deficiency. Thirty-five other individuals had

corneal vascularisation of the type which may be due to ariboflavinosis, but without other signs of malnutrition. No scurvy was seen. The differences between European and African are too great to require comment.

Fifty per cent of my subjects showed abnormalities of the skin which have been classed as Hyperkeratosis. Normal African skin is soft and supple in texture with a glossy sheen. Slight reticulation may be found over the shins particularly in those who wear shorts and are constantly exposed to minor trauma. Trowell describes hyperkeratosis as an increase of the reticulation with cracking and superficial desquamation along the natural lines of the skin. In the established case definite superficial cracking occurs between irregularly shaped glazed areas of desquamating epidermis. When advanced the appearance resembles the dried skin of a snake. Some observers use the term crazy pavement for this condition. Trowell, however, reserves that description for the enamel paint appearance seen in severe Kwashiokor. Gillman's criteria are similar but include various changes in pigmentation; he applies the term crazy pavement to the adult. In computing my percentage in addition the changes described above I included a state where the reticulation was minimal but where there was a widespread roughness of the skin with an extensive

desquamation. Enough has been said to show the confusion which has arisen. The nomenclature is chaotic; Hyperkeratosis, pellagroid skin, crackled or giraffe skin and crazy pavement dermatitis have all been used, apparently with the same meaning but unfortunately this is not certain. Platt (1945), Trowell, Gilman and Gelfand (personal communications) agree that at least sometimes hyperkeratosis is a manifestation of malnutrition. The lesions are stated to respond slowly to a full diet but have not been ascribed to any particular component. In my experience when it is found in association with pellagra, the hyperkeratosis persists after the pellagrous rash has cleared up. I have given nicotinic acid to several cases (not associated with pellagra) but have been unable to detect any improvement.

I have given 5 per cent as the incidence of parotid gland enlargement. With many physical signs the pathological fades imperceptibly into the normal. Every clinician adopts different criteria and with minor abnormalities there is often disagreement. This is particularly true of parotid swelling. Trowell describes a method of palpating the gland. I never became proficient in his technique and have only taken notice of cases where there was a visible increase. In the Kenya Survey 18 per cent had parotid swelling with marked enlargement in 6 per cent. Considering



Figs. 1 and 2 Examples of enlarged parotids. The upper photograph would be classed as moderate, and the lower as advanced enlargement of the parotid glands.

the different standards, the discrepancy is probably slight. The Gillmans state that all the salivary glands are involved in the pathology of malnutrition, and they demonstrate definite histological changes.

It must be emphasised that the Bantu are far from starvation. On the average, the natives are of slight physique and stature; a reasonable proportion are, however, well built and muscular. Scurvy and pellagra may appear in plump, well-covered individuals; conversely, vitamin deficiencies are stated not to occur in starving people (Gillmans) In the preceding section the African diet has been discussed in the usual terms of calories, fat, protein, carbohydrate, mineral salts and vitamins. These criteria are relatively crude and do not tell the whole story. Maize, for instance, has a known effect on the Vitamin B complex metabolism which in certain circumstances can be toxic. Further investigation will probably reveal other important factors. The Rhodesian diet, judged by the usual standards, may be superior to those of most tropical countries. However, the final test is how do the Bantu fare on their diet. The answer is that over 60 per cent show tangible evidence of its insufficiency.

The machine is not devoid of fuel nor often even short of it. However, owing to the poor quality of the fuel, minor defects frequently appear in the engine, and occasionally a complete breakdown.

COMMON NUTRITIONAL DISEASES

For the sake of clarity, the deficiency diseases are here ascribed to a single vitamin. It is fully realised that nutritional syndromes are highly complex, and seldom if ever is the deficiency of one single factor alone responsible.

Vitamin A

There is some disagreement among authorities as to the minimum daily requirements. The National Nutrition Council of S. Africa advises 3-400 international units of the vitamin. The Food and Nutrition Board of the United States recommends 5,000 I.U. (assuming two-thirds is obtained from Carotene). The Medical Research Council concluded from their researches that 1,300 I.U. appeared to be the minimum protective dose and recommended 2,500 I.U. of vitamin A (or 7,500 I.U. of carotene) for healthy young adults. They caution that this value should not be relied on as an infallible standard for assessing the adequacy of the diet in a nutritional survey. As the rural African includes milk and green vegetables in his meals his intake is probably reasonable. However, with many employed and urbanised natives the position is unsatisfactory. The white mealie meal almost universally consumed in preference

to the yellow variety contains little or no vitamin A or carotene. In the case of a native existing entirely on meal and meat the total weekly consumption must be under 1,000 I.U. (beef is stated to contain 230 I.U. per lb.) Janisch (1941) put the average daily consumption of the Johannesburg native at 607 I.U. per diem. The Mines and Minerals Act Ration (when supplied in toto) is estimated to contain 966 I.U. per diem, (mainly as carotene). The proof of the inadequate consumption of vitamin A is the frequent occurrence of the clinical states of deficiency. Only the eye and the skin will be considered.

The ophthalmologists at the three centres which maintain eye clinics (Bulawayo, Salisbury and Umtali) agree that vitamin A deficiency is not uncommon. Dr. Sparrow of Salisbury has been particularly helpful. Night-blindness, Xerophthalmia and Bitots spots are the usual manifestations, in other words the conditions caused by prolonged mild depletion. Keratomalacia however is rare. Vitamin A deficiencies are by far the most common in African ophthalmology. The eye changes described as part of other malnutrition syndromes are seldom encountered.

Phrynoderma is extremely common in Southern Rhodesia and therefore merits special attention. The condition has been called a variety of names including toad skin, shark skin,

follicular hyperkeratosis, keratosis pilaris, icthysosis, follicularis, lichen pilaris, lichen spinulosus, etc. The following account is mainly taken from Bicknell and Prescott. The skin changes are preceded by dryness of the skin. The eruption first appears on the limbs and front and lateral aspects of thighs and the extensor aspect of the forearm in the region of the elbow. In an advanced case the whole of the body surface is involved with the exception of the face and head. In a well established case the follicles are often very prominent on the buttocks. The lesion consists of dry, horny, round or oval sharply defined papules, varying in diameter from that of a pin head to as much as quarter of an inch. The size of the papules increases with the duration of the deficiency, in the early stages being more easily felt than seen, while later the skin looks from a distance as if many split lentils had been stuck upon it. Often broken or coiled up unerupted hairs are found either projecting through the papule or imprisoned beneath. Itching, at least in Africa, is not a symptom. Microscopical examination of the skin shows that the papules are composed of masses of keratinised cells which have been shed from the piloslbaceous follicles, becoming compressed in their centres into horny amorphous plugs. These block

the hair follicles and the sebaceous glands which tend to atrophy. There is also hyperkeratinisation of the epidermis, especially round the papules, and a thickening of the stratum corneum and sometimes an increase in the pigment cells. A mild lymphocytic infiltration near the base of the follicle is also seen. The sweat glands are not markedly changed, but do not appear to be secreting, and they are often plugged with keratineus material.

Wiltshire in 1919 published a description of phrynoderma from Serbia. He recognised it as an early sign of scurvy although not pathognomonic of that disease. The condition was later described independantly by Nicholls (India 1933), Lowenthal (East Africa 1933) and Frazier and Hu (China 1931). Cases were diagnosed in England by Goodwin (1934) and Pemberton (1940), also at about this time in the U.S.A. and Malaya. All these writers observed the association of vitamin A deficiency, and the beneficial results of therapy with this substance. More recently the condition has been recognised in West Africa where riboflavin has been incriminated. (Gelfand,1948) Credit for the original description probably belongs to Lind (1772) who stated that in some cases of scurvy the skin became anserine.

Fig. 3 Severe phrynoderma (Michael, Case No.9) The follicles were unusually large and numerous.

Fig. 4 The same patient after fifteen weeks treatment with ascorbic acid. Skin now normal.

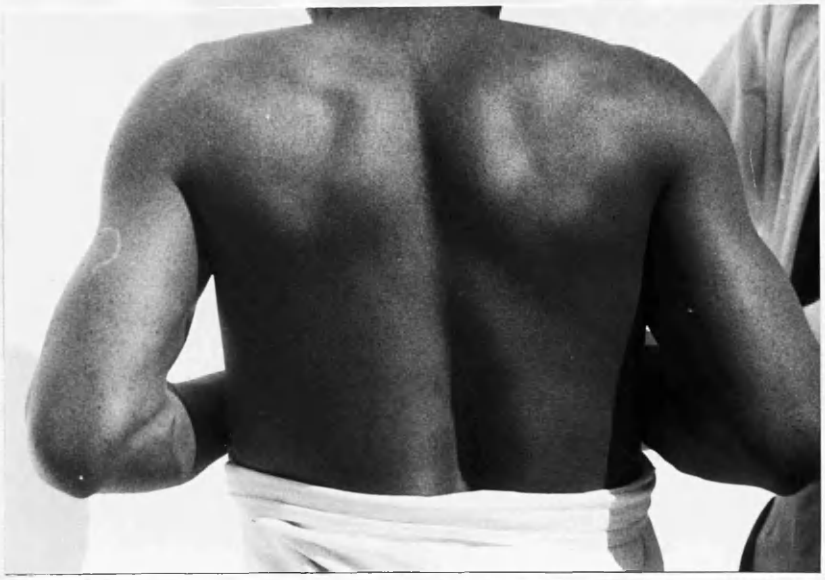
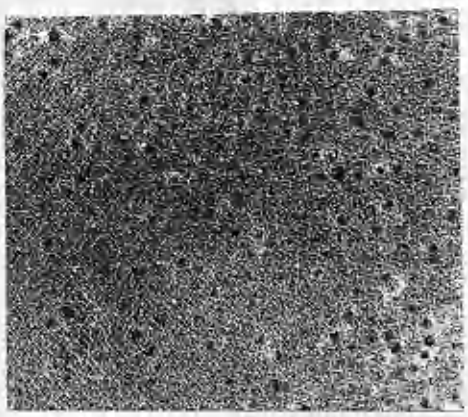
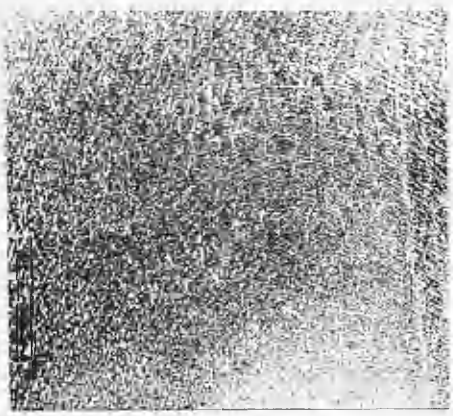


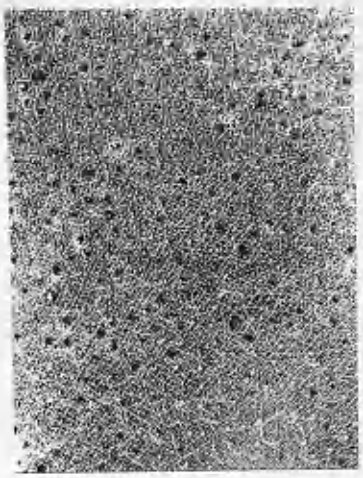
Fig. 5 Michael's back (Case No. 9) before treatment. Numerous large follicles present.

Fig. 6 After twelve weeks on ascorbic acid; the hyperpigmented patches, which replace the follicle in the process of healing, are visible. (See text)

Phrynoderma has been studied in three recent investigations viz., the Medical Research Council's experiment on Vitamin A deficiency, the Sheffield experiment on Vitamin C and the Minnesota experiment on starvation (Keys 1948). The first investigators remark, "In several of the subjects follicular hyperkeratosis was present at the start and varied during the experiment, but the variations have no relation to the vitamin A intake". In the Sheffield experiment all the ten volunteers developed the eruption. The statement is made, "Deficiency in vitamin C is only one of the causes of follicular hyperkeratosis". Keys observes, "A substantial proportion of persons subsisting on a European type of famine diet exhibit rough gooseflesh like areas of skin. The condition resembles the follicular hyperkeratosis sometimes associated with vitamin A deficiency. The causation is puzzling because its incidence is highly variable and does not seem to be clearly related to the state of vitamin A nutrition". The dietary referred to by Keys, although deficient in many aspects, has an adequate vitamin content. Gillman was unable to repeat the work of Lowenthal. He regards the condition as a non-specific manifestation of malnutrition. These skin lesions appear in states of malnutrition. There







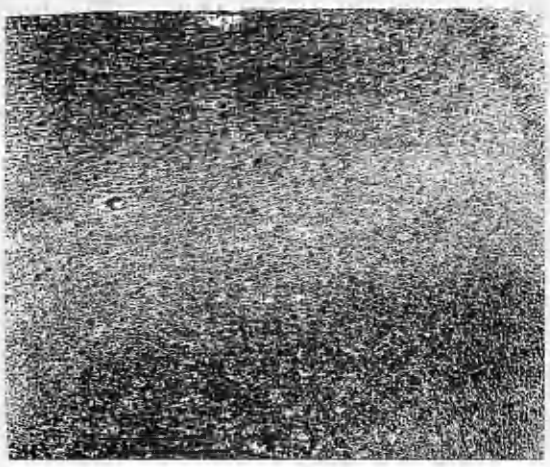


Fig. 7 A photograph of Michael's (Case No. 9) right upper arm showing advanced phrynoderma. Skin impressions were taken by Stannus's method (1944). The prints on the left were obtained before treatment; on the right, after nine weeks on vitamin C. The Stannus method is ingenious and does provide a permanent visual record. It is, however, somewhat laborious and does not always record minor degrees of phrynoderma. The prints should be viewed through a reading glass giving about five magnifications.

is definite evidence of the importance of ascorbic acid. The connection with vitamin A lacks experimental proof. An objection raised to the Medical Research Council's findings, is that observations are drawn from a previously adequately fed subject who is suddenly totally deprived. It is conceivable that a chronic but not complete lack of a food factor over many years in a generally malnourished subject may produce different results. It is difficult to disregard the observations of several independent workers on the importance of vitamin A. The aetiology of phrynoderma is obviously complicated and mysterious. The position will remain so until the details of skin metabolism and pathology are elucidated.

In Wiltshire's 3,000 cases, 87 per cent showed phrynoderma. All ten of the Sheffield volunteers developed the skin changes which with one exception became haemorrhagic. Wiltshire very definitely states that the follicles in the Serbian troops were not haemorrhagic. Thirteen of my nineteen scurvies had phrynoderma which is a higher percentage than found in the survey (37 per cent). As bruising and petechiae are not usually visible in a black skin, I am unable to say whether haemorrhage was a feature. There was no obvious correlation between the severity of the scurvy and the skin condition. Five of these patients had a

Fig. 8 Moderate degree of phrynoderma seen on the leg of a scurvy patient. (Scurvy Case No. 3) Compared with Michael's arm (Fig. 3), the papules are small and scattered.

Fig. 9 Phrynoderma occurring on the buttocks. This site is usually only involved in fairly advanced cases.

marked degree of the eruption. However, I have seen more advanced examples in the apparently healthy African. Observations made on five of the scurvy patients (three of them in the marked category) were confusing. One patient showed no response after five weeks and was discharged. Three cases showed a slight improvement after six or more weeks on large doses of vitamin C. This improvement consisted of the follicles becoming less prominent, the process of healing as seen in the really favourable case was absent. The one exception (Case number 11 in the series) showed a rapid improvement about the fourth week, and was almost cured six days later. Unlike the other scurvy patients his follicles disappeared leaving areas of hyperpigmentation which were larger than the original papule. The texture of the skin changed from rough and dry to smooth and glossy. Some investigations were carried out on subjects who had no other signs or symptoms of either an A or C deficiency. Michael was a case of unresolved pneumonia transferred from an outlying clinic. In addition to advanced phrynoderma (Fig. 3) he had enlarged parotids and well marked follicular seborrhoea of the nose and naso-labial folds. He was put on 300 mgms. of ascorbic acid daily. After three weeks the comedones

had almost disappeared; traces of these lesions, however, lingered until the eleventh week. The phrynoderma persisted unchanged until the sixth week when the papules started receding. After an initial spurt, the improvement continued slowly, and he was not finally clear of follicles until he had received fifteen weeks of treatment. Long before his skin was clear the consolidation of the right lung had resolved. Asina lost three fingers of the right hand in a factory accident. Fairly advanced phrynoderma was present, also a moderate degree of follicular seborrhoea. When he demanded discharge after eight weeks of therapy, the skin was unchanged, although by the twenty-eighth day the comedones had disappeared. Two convicts with mild follicular hyperkeratosis were given 120,000 I.U. of pure vitamin A (Prepalin) for eleven and six weeks respectively. No definite change was seen in either. Chiringa was admitted to hospital with fractures of the right metatarsal bones. He was the most advanced case of phrynoderma seen to date. He displayed the appearance of split lentils stuck on the skin. He was put on the same dose of Prepalin. In three weeks a definite improvement was visible, in six the papules had gone and the skin was of normal texture. The patches of hyperpigmentation faded slowly and could be discerned after fourteen weeks of treatment.

Fig. 10 Picture of Chiringa's legs (Case No. 8) untreated. Although this photograph was taken from a distance, the enlarged papules are clearly visible. The general appearance was suggestive of 'split lentils stuck on the skin'.

Fig. 11 The same patient after six weeks on vitamin A. The papules have been replaced by patches of hyperpigmentation.

TABLE II

Response of Phrynoderma to Vitamin Therapy

	Disease	Phrynoderma	Treatment	Summary of Investigations on Phrynoderma: Response to Treatment
1.	Scurvy Case No.5	Moderate	Vit.C 300 mgm. daily	No response after five weeks
2.	Scurvy Case No.8	Marked	Vit.C 1,000 mgm. daily	Only slight improvement after six weeks
3.	Scurvy Case No.11	Marked	Vit.C 1,000 mgm. daily	Rapid improvement occurred four weeks after start of therapy. Almost cured in five weeks.
4.	Scurvy Case No.15	Moderate	Vit.C 1,000 mgm. daily	Slight improvement after eleven weeks.
5.	Scurvy Case No.16	Marked	Vit.C 1,000 mgm. daily	Slight improvement after five weeks, thereafter remained static for the two weeks before his final discharge.
6.	Prisoner Leonard	Mild	Vit.A 120,000 I.U.daily	No improvement after eleven weeks.
7.	Prisoner Pedzai	Mild	Vit.A 120,000 I.U.daily	No improvement after six weeks.
8.	Chiringa Fractured Metatarsals	Very marked	Vit.A 120,000 I.U.daily	Definite improvement in under three weeks. After six weeks follicles had disappeared and skin of normal

TABLE II (continued)

Disease	Phrynoderma	Treatment	Summary of investigations on Phrynoderma: Response to Treatment
8. Chiringa (continued)			texture. Hyperpigmented patches still visible after fourteen weeks treatment.
9. Michael Unresolved pneumonia	Very marked	Vit.C 300 mgm. daily	Definite improvement seen at sixth week. Clear of follicles after fifteen weeks of treatment. Comedones improved before the phrynoderma.
10. Asina Laceration of hand	Marked	Vit.C 300 mgm. daily	No improvement after eight weeks. Comedones had disappeared after four weeks.

The hope that a satisfactory explanation for the condition in the Rhodesian Bantu could be demonstrated, was not realised. However, it has been shown that vitamins A and C are curative in some cases, and of no benefit in others. Apart from all other considerations, the writer would like to suggest the appearance of phrynoderma is partly

dependent on a susceptibility of the individual's skin.

Thiamine

Very little rice is grown in Southern Rhodesia; however, Beri-Beri does occur. (Gelfand: Morley-Smith 1950 personal communicati on). The w riter has had some experience of this disease in Malaya. No definite example has been seen in five years, in the few doubtful cases where vitamin B1 has been exhibited the response has not been convincing. It can be stated with confidence that thiamine deficiency is not a serious problem in the Colony.

Nicotinic Acid and Riboflavin

It became clear shortly after starting practice in Africa, that no standards for pellagra exist even in Rhodesia. The incidence of the disease from different centres reflects the criteria adopted by the individual physician. In this discourse the diagnosis of pellagra has been reserved for patients showing the classical symmetrical rash, pellagra sine pellagra has not been recognised. Despite the well known observations of Stannus (1911) on the prisoners of Zomba Jail, Nyasaland, it must be accepted that there is an intimate association between maize and pellagra. The exact aetiology of the

Figs. 12 and 12A — A case of pellagra showing a Casal's necklace, and the typical bilaterally symmetrical rash. This patient was also suffering from follicular seborrhoea, cheilosis and angular stomatitis.

malady is still not clear. It is not proposed to touch this vexed question. While not pretending to understand all their theories, I have made repeated reference to the work of the Gillman brothers. The basic idea is simple, "The majority of Johannesburg natives suffer from chronic malnutrition". "Pellagra is one of the several manifestations of an acute episode in this process of malnutrition". The pattern of malnutrition is common to Rhodesia and Johannesburg, also the diet is essentially the same. Conclusions reached in Johannesburg hold good for Rhodesia. A similar state of affairs apparently exists in Kenya. Pellagra, however, is a rare disease in the Northern territories. As Professors Trowell and Gillman have conferred on this question, the different incidence is not a matter of diagnosis. No satisfactory explanation for the scarcity of pellagra in Kenya has been advanced. It is a curious fact that pellagra is uncommon in the equatorial regions although malnutrition is rife. I see no reason to disagree with the Gillman conception. The limited investigations undertaken in Rhodesia confirm their findings.

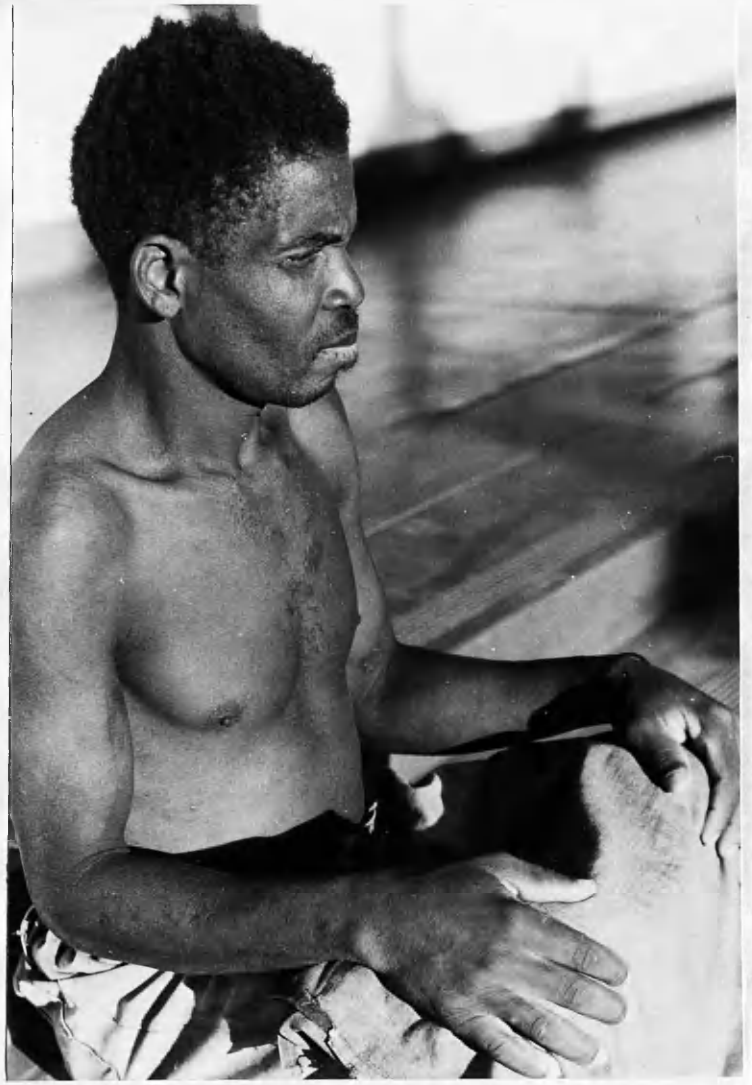
Pellagra is fairly common in Southern Rhodesia. Although more cases of scurvy are seen in hospital practice, probably a higher percentage of natives suffer from

pellagra which is often too mild to make them seek treatment. The patient shown in Fig. 13 came to hospital complaining of a large hydrocele. He agreed that he felt weak, but was obviously satisfied with his general health. The descriptions in standard text books have been taken mainly from American sources. Pellagra was formerly the scourge of the South, where it presented in a very severe, often epidemic, form with a mortality up to 60 per cent. The clinical picture in Rhodesia is rather different. Several senior members of the Public Health Department have assisted in preparing the following account of the local variations of the disease. (Personal communications from Morris, Montgomery and Gelfand). Fulminating fatal cases have been encountered, but the disease is generally mild and chronic. The classical triad of diarrhoea, dermatis and dementia is rare. The mouth lesions are not a prominent feature of the syndrome. Alcoholism is of little or no importance in the aetiology. The future of any disease is difficult to predict. According to the Gillmans, there is a possibility the relatively benign type seen in Africa at present will change into a virulent disease with serious consequences to the community.

There is another syndrome at least as common as pellagra.

These patients invariably complain of weakness. They have a characteristic miserable expression. Lethargy is marked and unlike the majority of sick Africans, they prefer to rest in bed. Wasting is often present but not always, and some cases could be described as podgy. The most striking feature is numerous yellowish-white plugs of inspissated sebum projecting from the orifices of the sebaceous follicles.[1] These filiform excrescences are seen particularly on the bridge of the nose and the naso-labial folds. In an advanced case the whole face is involved. There is usually some degree of angular stomatitis and cheilosis. The tongue may show discolouration. Phrynoderma is often marked. The condition is usually referred to as malnutrition, or acute malnutrition. These lesions are essentially an exaggeration of the signs found in so-called normal Africans. The picture closely resembles, or is identical with ariboflavinosis. In Rhodesia the itching dermatitis of the scrotum is extremely rare. Even on direct questioning I have never obtained a history of pruritis, nor have I seen an eczema of the perineum. The ocular changes are not very marked. Trachoma is prevalent in the Colony and there is local disagreement over the causation of corneal vascularisation.

[1] The rash is here termed follicular seborrhoea, and the individual diseased follicle as a comedone.



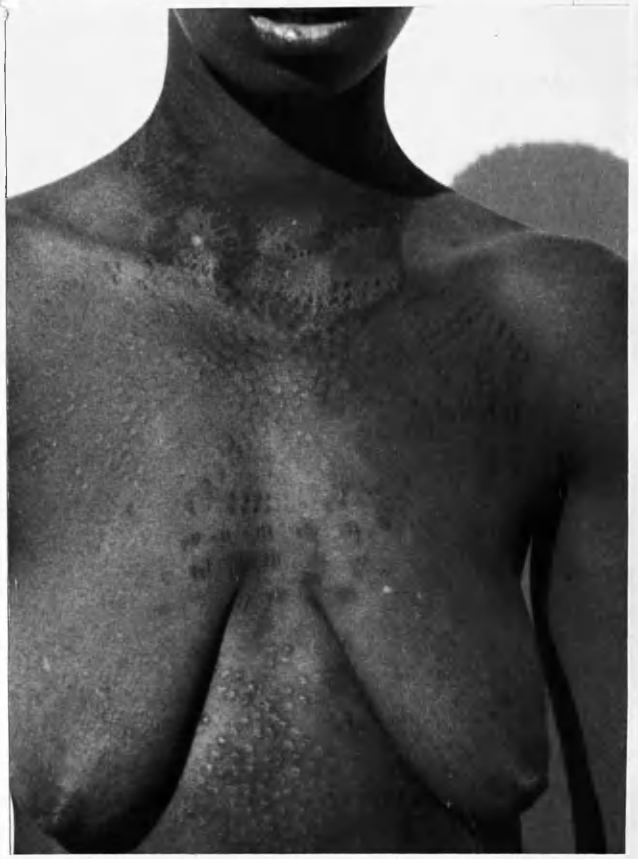
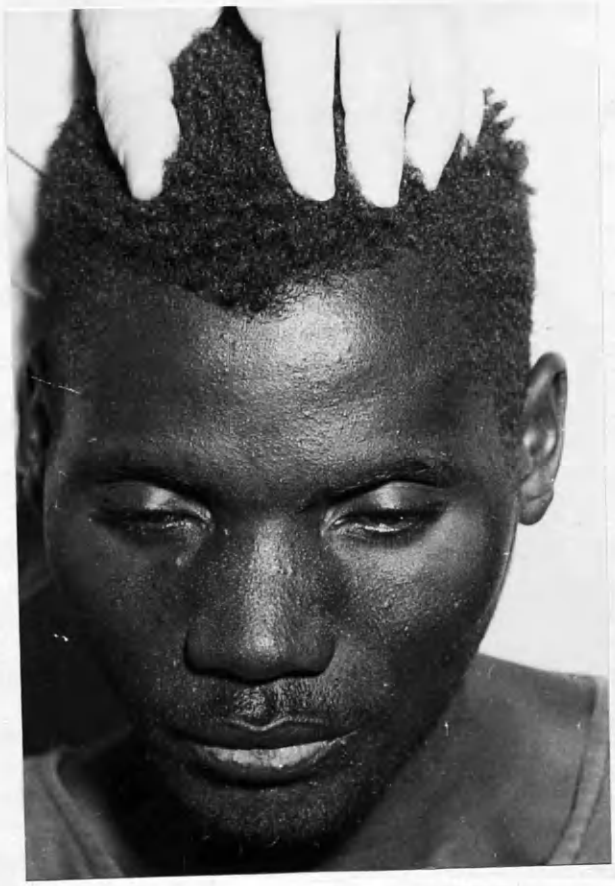


Fig. 13 A mild ambulant case of pellagra. This patient came to the out-patient department complaining of a large hydrocoele. (See text)

Fig. 14 Casal's necklace in a woman. The rash is fading under treatment.

I do not feel myself competent to make any statement. Varying degrees of follicular seborrhoea, stomatitis and cheilosis are often seen in pellagrins; it is an old observation that these lesions become more prominent when a pellagrous rash is responding to treatment. Intermediate forms showing the features of both diseases occur. To summarise the position:- The minor signs of deficiency present on the majority of Africans merge into the pattern of acute malnutrition, which has an intimate association with pellagra. It is reasonable to postulate the metabolism of many Africans is deranged. If this derangement becomes a definite breakdown, one of these syndromes will appear. From a practical point of view, these disorders are more than specific deficiencies of riboflavin and nicotinic acid. They are the manifestations of a chronic state of malnutrition.

In the examination of 500 Africans, ariboflavinosis has been estimated at 40 per cent. Follicular seborrhoea was by far the most common manifestation. The mouth changes were present in a clear cut form in just over 3 per cent. Although follicular seborrhoea is usually described as a sign of vitamin B2 deficiency, it is not pathognomonic. Although it could be argued that this abnormality should be



Figs. 15 and 16 Two examples of acute malnutrition. Both photographs show the characteristic miserable expression. Follicular seborrhoea is present to a marked extent in both patients. The man in Fig. 15 was suffering from an angular stomatitis which was more advanced than the picture suggests. (This subject has also been shown as an example of parotid swelling, Fig. 2). Fig. 16 shows some cheilosis. A lens will show the individual lesions more distinctly.

described as a non-specific sign of malnutrition, for reasons already stated, I have adhered to the usual classification.

While investigating the effects of ascorbic acid on two patients with phrynoderma, it was noticed the follicular seborrhoea disappeared. (Pg. 40) The response was striking enough to be definitely attributed to the vitamin C. This observation, as far as I can make out, is new. Clinically, follicular seborrhoea is not particularly common in scurvy; only three of my cases had it to any extent. The Gillmans state that histologically the comedone closely resembles the hyperkeratotic follicle of phrynoderma. The mechanism of nutritional signs is not yet fully understood. A possible explanation may be advanced:- Although a particular manifestation is generally associated with a certain deficiency, some at least of the signs are not specific, and may appear in a variety of circumstances. Further investigation may show the aetiology of follicular seborrhoea to be as complex as that of phrynoderma.

Kwashiokor

By far the most serious nutritional problem in Southern Rhodesia is Kwashiokor. I regret I can contribute nothing really new to the understanding of this disease.

Figs. 17 and 17A

An advanced case of kwashiorkor. This photograph shows several of the salient clinical features:-
(a) The fine hair; (b) general irritability; (c) oedema; (d) the pigmented, peeling skin which has been called the crazy pavement, (Trowell), or the cracked enamel paint appearance, (Gillmans).

The objects of this section are to stress the importance of Kwashiokor, and also to focus attention on certain findings which appear to have at least a local bearing on the subject. The manifestations of adult malnutrition are eminently treatable (at least as far as the acute phase is concerned). The reverse is true for Kwashiokor; the disease carries a high mortality. During one year at Umtali Native Hospital (120 beds), 23 deaths from this condition were recorded. In the week 1st to 7th January, 1952, four children died in the hospital, and post mortems were performed on two cases brought in by the police for medico legal investigations, (personal experience). In Salisbury the disease accounted for 52 out of 2,000 autopsies. (Gelfand) The syndrome has a very wide distribution in South, Central, East and West Africa. The history of kwashiokor illustrates many of the difficulties of tropical medicine, where workers are isolated and without access to medical literature. Proctor (1926) is credited with the original English description. Although working in the same part of Kenya, Gillan (1934) redescribed the disease as a new clinical entity. Williams (1933) claimed to have discovered a new disease in West Africa. The position was consolidated by Trowell (1940)

who showed that the disease was found in many tropical countries and not confined to Africa as originally thought. In recent years kwashiokor has been extensively investigated. Some workers claim the syndrome is the infantile manifestation of pellagra. An attractive case can be advanced to support this theory (The Gillmans); unfortunately, kwashiokor is common where pellagra is unknown. (Trowell, Williams). In my opinion, this controversy is of academic rather than practical importance. Davies (1948) as the result of a pathological study stated the primary failure occurs in the pancreas. Kwashiorkor was the subject of a conference[1] held in Gambia during November, 1952. (Annual Report of Nutrition Council, 1952) The disease was shown to have a world wide distribution. It has been described under no less than 42 different names since the beginning of the century. The name of kwashiorkor has now been universally adopted in preference to the numerous synonyms. It was established that kwashiorkor is always associated with a high calorific and low protein intake. The condition is quite distinct from marasmus. Although there is probably some other factor besides protein starvation involved in the aetiology, kwashiorkor is prevented by a diet containing mixed proteins, particularly of animal and legume origin.

1 Footnote: C.C.T.A. Nutrition Conference & Joint W.H.O./F.A.O. Nutrition Committee.

Bantu infant feeding varies considerably. It is unusual for a native mother to be short of milk. However, when this happens, gruel is forced on the babe. Lactation is often prolonged and sometimes continues up to two years. Supplementary feeds of thin porridge are introduced any time after the fourth month. In a favourable case, by the time a child is weaned, it is able to partake of adult fare. In a proportion of cases, when breast milk is no longer available, they are fed almost entirely on maize. In other words, the unfortunate child has to meet the demands of early life on a diet which is little superior to bread and water. The beneficial properties of milk (other than human) are not generally appreciated by Africans. In the early months, the African baby usually thrives, and compares not unfavourably with his European counterpart. It is perhaps significant that kwashiorkor does not attack under a year, the maximum incidence being between two and three years. The healthy African youngster is a pleasant little being. He is contented and extremely cheerful. His skin is smooth and dark, and his hair grows in wiry black pepper corns. A definite percentage exhibit the

Data has been obtained from colleagues, officials of the Native Affairs Department, nursing staff and by direct question of African mothers.

opposite characteristics. They are fretful and unhappy. Their skin is pale and the hair is soft with a reddish brown tinge. Both Trowell (1950) and Gillman are emphatic that many African children pass through a period of kwashiorkor, they apparently recover but the upset leaves permanent pathological changes in their tissues. "The seeds of cirrhosis are sown in childhood".

To summarise briefly, kwashiorkor is not only Rhodesia's most serious nutritional problem, but a dangerous factor in African infantile mortality. The modern view of the aetiology blames a high calorific diet with a low protein content; the food given to many native children fulfils these conditions. There is strong evidence that a large proportion of African infants go through a stage of kwashiorkor which has a permanent and detrimental effect on their development.

Vitamin C

Vegetables, wild fruits, green leaves and milk are included in the fare of the village native. Except in drought years, when he is forced to exist on purchased mealie meal, the rural African probably obtains an adequate quantity of ascorbic acid. When living away from the land

the position is less satisfactory. Special attention was paid to antiscorbutics in the diet survey (See appendix). Eighteen out of the hundred diets were classed as poor in vitamin C. The criteria applied were not strict. Any ration containing more than one orange a month was excluded. When applicable, the subject was asked when he had last consumed vegetables, fruit or milk. Several men apparently had not partaken of these foods for about a year. In the case of the four alleged teetotallers, the only fresh food available was meat. Dried fish was substituted for meat in one ration which contained no recognisable source of antiscorbutics. Ten subjects in addition to meat indulged in milk in tea and Kaffir beer; four other rations also included one orange per month. Mealie meal contains no vitamin C; the content of uncooked fresh beef is estimated at 9 mgms. per lb. (South African food tables). The weekly consumption of many natives cannot exceed 27 mgms. of ascorbic acid, (allowing a meat ration of 3 lbs. which is unusually generous). A limited investigation undertaken in Bulawayo of the urinary ascorbic acid showed a marked degree of unsaturation, even in the more privileged classes such as medical orderlies (Morris, 1952, personal communication) The dose of vitamin C required to prevent scurvy is very low,

and probably varies from individual to individual. The Medical Research Council proved that 10 mgms. daily will protect from clinical scurvy, and suggest the minimum protective dose may even be somewhat lower. However, to ensure a margin of safety, an intake of 30 mgms. per day is recommended. The final test of the antiscorbutic properties of any community's diet is the incidence of scurvy. Although a larger percentage of Bantu show the signs of pellagra and ariboflavinosis, scurvy is the deficiency which drives the Rhodesian native to seek treatment. As scurvy is the only notifiable avitaminosis, no official figures are available for comparison, but I have no hesitation in stating that, in my experience, scurvy is the most common deficiency disease seen among adults in hospital practice. Over a period of nine months, as a general duty officer in native hospitals, I treated over twenty cases of scurvy compared with less than a dozen examples of the other malnutrition syndromes. By the term deficiency disease is meant a breakdown in health; the asymptomless manifestations such as phrynoderma are not included. Because of the importance of scurvy, I have devoted a special section to the disease.

SCURVY

A long and fascinating paper could be written on the history of scurvy. Hippocrates has been credited with recognising the condition in soldiers. Since ancient times the disease has been of economic and military importance. Epidemics with serious consequences have occurred in such different enterprises as the Crusades and the colonisation of Canada. Scientific knowledge dates from the publication of Lind's (1772) classical treatise. At this time the diagnosis was fashionable and applied to any and every ailment. Lind deprecated the abuse of the term, and defined the disease as we know it today. Although the clinical picture appears to have changed, his description can still be read with profit. Unlike the modern writers, he clearly differentiated the early from the late symptoms, and the common from the rare. The reader is taken through the stages of the disease from the first sign of pallor to the various mechanisms of death. Some of his statements are surprising: he mentions a psychological factor, and comments on the beneficial effects of exercise in the early stages. Lind produced his treatise mainly to demonstrate the prophylactic and

therapeutic value of antiscorbutics. However, he was convinced other factors besides a faulty diet had a part in the aetiology. He was puzzled why scurvy should appear in a crew after a few weeks on sea food; while at other times sailors could live on identical rations and remain healthy for at least three years. Lind decided climate was very important, cold and wet being particularly harmful. Bad weather caused great discomfort among the common seamen, even their bedding was constantly wet. These hardships must have put a great strain on the metabolism. I see no reason to dispute Lind's observation. I have encountered similar anomalies in Southern Rhodesia, but am unable to advance such a satisfactory explanation. Some natives develop scurvy on a diet containing antiscorbutics, while others live for years on a ration devoid of all fresh food. Only a small percentage of potential scorbutics ever develop any definite signs. Enquiry along the lines of infection or increased work has not been productive. A possible but not entirely convincing answer is individual idiosyncrasy.

Scurvy was the first nutritional disease to be recognised in Southern Rhodesia (Baker-Jones, 1952).

In the early years of the colony, it caused considerable havoc. In 1908, one thousand and six hundred cases, with over two hundred deaths, were reported in the mines. In the same year 13·8 per cent of deaths (from all causes) were attributed to scurvy. In 1910, a committee enquiring into the prevalence and prevention of pneumonia and scurvy, considered the incidence of the latter disease to be underestimated. (Report of Social Security Officer, 1944) Although still a common illness, the position has improved considerably since the unenlightened pioneer days, when African labour was fed exclusively on mealie meal.

The chief objects of this investigation were:-

i. Make an estimate of incidence of scurvy in Southern Rhodesia.

ii. Elucidate the clinical picture of African scurvy and ascertain any difference from the classical European disease.

iii. Demonstrate the association of vitamin P.

iv. Establish the importance (or otherwise) of avitaminosis C in the aetiology of tropical myositis.

Although it is not possible to quote the incidence in accurate terms of percentage per native population, I hope to give some idea of the prevalence and seriousness of the condition. During my service in this Colony, I have

diagnosed several cases which I class as incidental scurvies, because either they came to hospital with complaints not directly associated with avitaminosis C or were found in the course of mass examination of convicts or native employees.

The following are typical examples:-

Incidental Scurvies

	Date	Place	Age and Sex	Concurrent disease
	October 1948	Gatooma	Old woman	Septic hand
	October 1948	Gatooma	Adult male	Colles fracture
	December 1949	Marandellas	Adult male	Tendon sheath infection
	April 1950	Umvuma	Female adult	Secondary syphilis
*	May 1950	Umvuma	Female adult	Malaria
	May 1950	Antelope	Female adult	Routine maternity
	May 1950	Antelope	Adult Male	Pulp infection
	June 1950	Gwanda	Adult male	Iritis

* This case was seen in a village during a malarial survey.

Incidental Scurvies (Continued)

Date	Place	Age and Sex	Concurrent diseases
June 1950	Belingwe	Adult male	Carcinoma of lung
June 1950	Belingwe	Adult male (Convict)	Routine inspection
July 1950	Nyamandhlovu	Adult male	Inspection of saw mill

Apart from the gums, and vague aches and pains, these patients did not show any of the usual signs or symptoms of scurvy. My attention was first drawn to the frequency of these mild ascorbutic states by Case Number 1, whose affected gums I discovered when giving her a general anaesthetic. These patients were seen all over the Colony at every season of the year.

Bulawayo Native Hospital has 320 beds and serves a population of over 80,000. In the twelve months ending December, 1949, seventy cases of scurvy were admitted. In the official Health Report for the same year, it is stated that 173 scurvies were treated as in-patients in Government Hospitals, (with two deaths). The native population is estimated at 1,898,000. These figures are, however, no

index of the real incidence of the disease. Mild cases are treated as out-patients, while many remain undiagnosed and are cured or relieved during the harvest months. Localised outbreaks have been known to occur in the more arid and unfertile districts of the country. Dr. Gavin Wright (1951), a colleague in the Government service, has sent me details of cases occurring among sawmill workers.

TABLE III

Details of Scurvy in Sawmill Workers (Wright)

Cases of Scurvy		Sawmill No. 1	Number of Employees 730	
Year	Number of Cases	First case of Outbreak Reporting	Last case of Outbreak Reporting	Odd Cases
1948	13	25.9.48	16.11.48	-
1949	18	23.9.49	5.12.49	12.5.49
1950	32	7.8.50	15.12.50	2.2.50 11.3.50 10.5.50
1951	1			
Cases of Scurvy		**Sawmill No. 2**	**Number of Employees - 1948 - 195, 1949 - 238 1950 - 173, 1951 - 163**	
1948	15	14.10.48	24.11.48	2.1.48 6.3.48 31.3.48

TABLE III (Continued)

Year	Number of cases	First case of Outbreak Reporting	Last case of Outbreak Reporting	Odd Cases
1949	21	3.9.49	26.10.49	31.1.49 31.3.49 20.7.47
1950	13	29.9.50	4.12.50	6.6.50 9.6.50 28.7.50
1951	-			

These figures supply valuable information, showing that approximately 9 per cent of a labour force can be affected in a single year, the seasonal incidence is well illustrated. In 1951 green vegetables were included in the rations with beneficial results. In Sawmill No. 1, out of a total of 64 cases, 25 were severely crippled and the remainder had a variable but definite disability. Dr. Wright also comments upon the scarcity of the condition in the African female.

Scurvy is apparently only found in certain areas of Central and Southern Africa. Trowell in Uganda and the Gillmans in Johannesburg have both informed me that the

disease is scarcely, if ever, seen in their Bantu patients. However, the disease is not uncommon in Capetown. (Bronte-Stewart, 1951)

In the Northern Territories, sweet potatoes and plantains are common articles of food. However, there seems to be little dietary variation between Rhodesia and South Africa. Physicians in Johannesburg are puzzled by the dearth of avitaminosis C. This problem can only be solved by a first hand investigation of the different areas, and is outside the scope of this thesis.

There are several widely accepted ideas about scurvy among Rhodesian practitioners:-

1. Scurvy does not occur in kraal natives, i.e. when living in their natural state.
2. Women are immune.
3. There is a seasonal incidence related to the harvest.
4. Infantile scurvy is unknown.

In the main, these generalisations are true, but exceptions do occur. I have on one occasion seen scurvy in a village woman. Males are certainly more frequently and seriously affected than females, who seldom develop more than a subclinical manifestation, e.g., four of the incidental scurvies were females (36 per cent), but only

one of the series studied in hospital was a woman (5 per cent). Cases of avitaminosis C are seen at all times of the year, but the maximum incidence is between November and February. Nine of my nineteen cases were admitted during December and January. The green mealie (corn on the cob) ripens in February and March; this is a favourite with natives who often eat them raw. At this season various wild fruits and berries also become available. Barlow's disease must be very rare in the African. Dr. Gelfand (personal communication) tells me he has seen cases; Dr. Harrington (personal communication) has never come across an example of this disorder. Although on the look-out for the condition, I have never seen an infant patient suggestive of the disease.

To illustrate the African variations of avitaminosis C, I have collected 19 cases. These have been recorded in a standard form. The history was divided into presenting complaints, and those elucidated by questioning. Each patient was also asked about his diet. In the physical examination, emphasis was laid on the presence of other deficiency states. The African can often give a graphic description of a symptom when actually present, e.g., "There is a red hot snake in my bladder", but is vague

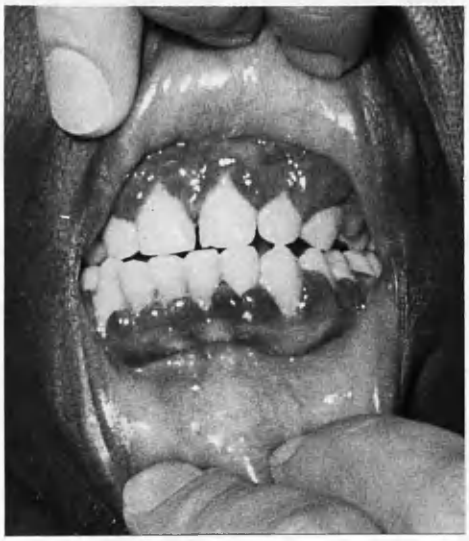


Fig. 18 Scurvy gums showing a moderate degree of hypertrophy and several typical buds. (Case No. 1)

Fig. 19 A more advanced example of scurvy gums. (Case No. 3)

Fig. 20 This photograph shows the lower incisor teeth partially buried by a generalised hypertrophy of scorbutic gums. (Case No. 10)

as to past discomforts. The native has little sense of or interest in time; the majority have no idea of their age. The duration of illness as stated is often inaccurate and tends to be an underestimate.

To avoid fallacies I only accepted definite cases for the series. My criterion was the condition of the gums which had to be discoloured, hypertrophic and showing scurvy buds (nodes). The only exception to this rule was Case Number 8, whose gums, although grossly abnormal, were not typical and remained unaltered after large doses of ascorbutic acid by mouth and parenterally. After some hesitation, it was decided to include this patient in the series as a doubtful case, because of the interest as a diagnostic problem.

The standard of dental hygiene in the Bantu is low; I had to discard several cases as a certain differentiation could not be made from oral sepsis. The historical writers describe gross changes in the mouth such as teeth being completely obscured by the gingival hypertrophy. (Hess, 1920). I have never seen pathology of that nature, even in patients bedridden by the disease. In fact, the mouth signs are often completely overshadowed by the lesions of the locomotor system. Out of 19 cases, 3 had not

Fig. 21 This photograph shows the abnormal gums of Case No. 8. Considerable hypertrophy was present, but no change was observed after six weeks treatment with large doses of vitamin C. Lind mentions permanent gingival changes in chronic scurvy; this patient was possibly an example. (See text).

Fig. 22 Scurvy gums in an advanced stage (By courtesy of Dr. Gelfand).

noticed the unhealthy state of the gums; in 3 it was a presenting symptom, the remainder, on questioning, stated they had experienced buccal changes. In one case (Number 5) I was able to confirm the old clinical observation that the disease does not affect edentulous areas.

The impression gained from descriptions of scurvy in standard text books led me to believe the clinical picture in the African was very different from the European. However, after reading MacMillan and Inglis's (1944) study of fifty-three cases, it is clear that no basic difference exists. By far the most common complaint is painful limbs. This is always mentioned among a medley of symptoms, but even in specialised works on vitamins, it is not stressed as the usual presenting symptom. In the unselected patients recorded, only two did not present with painful limbs; one of these was a multiple deficiency (Case Number 2), the other (Number 6) was suffering from an ulcer and when questioned did admit to the usual leg pains. MacMillan and Inglis state

> "A cardinal symptom was painful limbs, the discomfort first appearing in the legs".

Out of their fifty-three cases it was absent only in two.

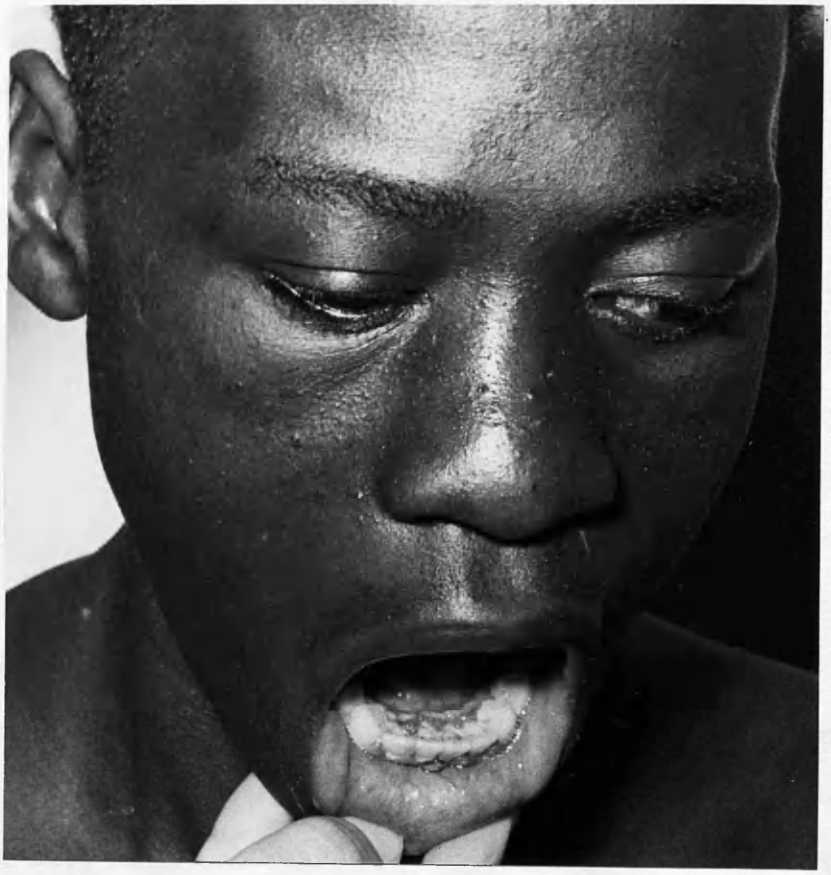


Fig. 23 A not very successful attempt to demonstrate scurvy buds on the inside aspects of lower incisor teeth. This patient (Case No. 14) also shows a mild degree of follicular seborrhoea.

Thirteen of their patients also had deep haemorrhages in the arms, a complication I have never seen in the African. In the excellent investigation made by the Medical Research Council, the limbs were not affected: As other symptoms of the clinical disease did not appear, it was postulated that classical scurvy is a mixed deficiency. While in no way denying this contention, it is felt that the different circumstances of the volunteers and the Bantu patient should be made clear. In the pure avitaminosis C of the Sheffield experiment, the subjects were not doing strenuous work. Nearly all my cases were labourers in some form or other. In two patients where the involvement of the leg was slight in comparison to the mouth, their occupations were of a lighter nature. Case Number 9, who was a tailor's assistant, and Case Number 12, who made a living helping a carpenter.

The condition of the affected legs showed great variation from gross brawny swellings to complete normality. The amount of disability was not proportional to the extent of palpable induration. In three cases the limbs were normal (apart from localised tenderness) and one of these was crippled (Case Number 1). The induration generally involved the lower aspect of the thigh and upper third of

Fig. 24 Scorbutic swelling affecting the knee and surrounding tissues. The day after this photograph was taken, 40 cc's of blood were aspirated from the joint.
(Case No. 16)

the calf muscles, the popliteal fossa being a favourite site. I could detect no correlation between the extent of signs in the mouth and the legs. Under treatment the majority of ascorbutic swellings disappeared, but in a few cases after an initial response some swelling persisted as is illustrated in Cases 4 and 16.

The classical sign of haemarthrosis was encountered twice, (Cases 5 and 16), in each instance the knee joint being affected.

Fourteen patients were X-rayed; antero-posterior and lateral views were taken at the site of maximum discomfort. The plates were examined by Dr. Hedderman (Radiologist, Bulawayo General Hospital). No pathology could be detected in 13; in one case there was a doubtful elevation of the periosteum of the lower end of the tibia. It can be definitely stated that the striking changes of infantile scurvy are not seen in the adult. Radiology is of no positive value in the diagnosis of avitaminosis C.

Oedema was never observed, although a careful examination was always made. This sign receives considerable attention in the older descriptions. In the ship scurvy described by Lind, oedema was a common and important manifestation. Two other symptoms monotonously

recorded in the text books, haematuria and night blindness, received special attention. None of my patients admitted to any nocturnal visual disturbance. This was unexpected, as night blindness is common in the African, (Sparrow, 1952, personal communication) and it would have been reasonable to expect a multi C and A deficiency to occur. No history of haematuria could be obtained, with the exception of Case Number 1, who had passed blood in childhood. MacMillan and Inglis comment that "frank haematuria was not encountered in their patients".

The role of vitamin C in erythropoiesis has been the subject of much study and difference of opinion. Ascorbic acid is accepted by physiologists and haematologists as being essential for the maturation of the erythrocyte. Whitby and Britton (1946) state:-

> "Vitamin C has some specific action in erythropoiesis and its absence results in dyshaemopoieses".

McDowall (1950) mentioned vitamin C as being of considerable importance in the formation of the erythrocyte. Israels (1943) from a study of scurvy patients concluded, "Ascorbic acid deficiency causes a depression of erythropoiesis rather than failure of maturation at any stage". Among clinicians, conflicting views exist. Shafer in a balanced review of the problem discusses the two schools of

thought. It is evident that, although anaemia is often present in scurvy, it is not a constant finding. Certain workers (Vilter et al, 1945) postulate that the anaemia is dependent on an additional deficiency. "They consider that the concept of a multiple deficiency state with many factors besides avitaminosis C adversely affecting the bone marrow, would offer a satisfactory explanation of the many conflicting reports on the subject of scorbutic anaemia". The most recent contribution to the problem, viz., the Medical Research Council's experiment, does not connect vitamin C with erythropoiesis. No alteration was observed in the blood counts of the volunteers. However, the exact cause of scorbutic anaemia is still unexplained, likewise the fact that it is usually corrected by ascorbic acid.

The findings in the patients under discussion were extremely variable. Accepting 80 per cent (Sahli) as the lower limit of Bantu normal*, with 50 per cent and below as severe anaemia, the eighteen cases where the haemoglobin was estimated may be classified.

* See Appendix

TABLE IV

Haemoglobins of Scurvy Patients

No Anaemia	Slight and Moderate Anaemia	Severe Anaemia
(Hb above 80 per cent)	(Hb between 80 per cent and 50 per cent)	(Hb 50 per cent or less)
Case No. 2	Case No. 1	Case No. 4
Case No. 3	Case No. 5	Case No. 16
Case No. 8	Case No. 6	Case No. 17
Case No. 10	Case No. 7	
Case No. 12	Case No. 9	
Case No. 13	Case No. 11	
Case No. 19	Case No. 14	
	Case No. 15	
Total 7	Total 8	Total 3

There was no obvious relation between the anaemia and the severity of the other symptoms. A patient may be completely crippled with little or no reduction of the haemoglobin, viz., Cases 1 and 10. With vitamin C, normal levels were reached, often very quickly, e.g., Case 16, who rose from 36 per cent to 85 per cent in less than five weeks. One

exception was patient Number 6, whose haemoglobin fell during treatment; possibly he illustrates the Gillman contention:-

> "In certain states of disordered metabolism, the exhibition of the deficient factor will exert the opposite of a curative effect".

MacMillan and Inglis's observation is similar, although the European patient is perhaps more liable to develop an anaemia.

The scorbutic leucocyte has received scant attention compared to the erythrocyte; however, from the literature available it is clear that the deviations are inconsistent and relatively unimportant. MacMillan and Inglis stated that the white count varied between 2,800 and 12,000 per c.mm. in their patients. In the cases reported by Israels, and Jennings and Glazebrook (1938), no abnormality was found in the total or differential leucocyte counts. Cranden's white count fell from 5,000 to 3,500 per c.mm. and was promptly restored by the exhibition of vitamin C.

> "No significant change was observed in the Sheffield volunteers in the course of their depletion".

My observation showed a leucopaenia with a decrease in granular cells occurring in a minority of cases. Even when the total white count was normal, there tended to be a relative neutropaenia.

There is a dearth of references to the behaviour of the central nervous system in scurvy. Bicknell and Prescott remark

"Exaggeration of patellar and biceps reflexes have been described".

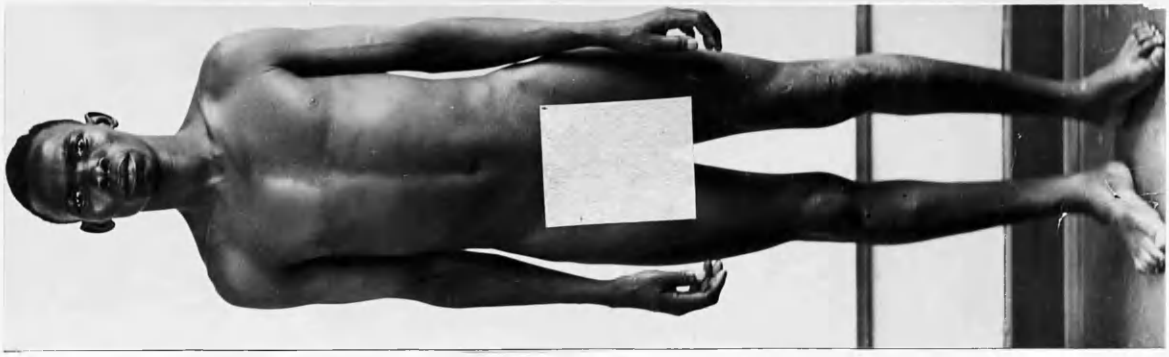
Hess mentions rare cases where haemorrhage has occurred into the cord. Two of my cases (Numbers 13 and 19) showed minor neurological disturbances; the changes were those of a lower motor neurone lesion and occurred in the affected limb. The signs disappeared with therapy and were presumably the result of some scorbutic lesion in or near the peripheral nerves; the clinical picture did not resemble beri-beri. Although of little practical importance, these nervous manifestations are felt to be worth recording.

Cardiac dilatation is generally accepted as a common feature of scurvy. However, I was unable to confirm this; none of my patients had any clinical enlargement of the heart. In the one fatal case, the heart was found to be within normal limits. However, the sudden and unexpected death of this patient is in accordance with the classical descriptions (Lind, Lord Anson, 1748). Two of the Sheffield volunteers developed cardiac emergencies. It is reasonable to postulate that in avitaminosis C the

action of the heart can be deranged, moreover in my opinion any sign of cardiac involvement calls for vigorous treatment. There is an understandable tendency to regard scurvy as a mild medical curiosity. While this is generally true, the fatal potentialities of the disease should not be forgotten. The old physicians maintained a different attitude to this "virulent scourge".

In the epidemics of scurvy occurring among the crews of 18th century warships, the disease was probably complicated by infection. It is reported that pneumonia and dysentery appeared in different out-breaks. In this series no disorder of bowel function was observed. The respiratory system also escaped involvement, the one important exception was Case Number 11 who presented as a sick pneumonia. The details of this patient are discussed in his case history.

It is emphasised by all the authorities that deficiency states are always multiple. While not presuming to contest this axiom, with the African it is sometimes difficult to demonstrate clinically. Scurvy is not limited to the emaciated native and is seen in the apparently well fed. The photographs of five patients originally selected for another purpose show the general nutrition which can be accepted as average for the local Bantu. The observation that scurvy can occur without wasting is not new. Wiltshire



Figs. 25 and 26 These five patients comprised all the scurvy cases in Bulawayo African Hospital on a certain day in January, 1951. None of these men showed any wasting; their physique is at least average for the Rhodesian native.

commented that some of his cases were "among the finest physical specimens he had seen". On several occasions I have encountered scurvy in well covered natives of good physique, e.g., Case Number 18. However, three (16 per cent) of my patients had signs of a B complex deficiency (Cases 2, 5 and 11); this is certainly a much higher incidence than found among the general African public. Minor degrees of comedones, hyperkeratoses and phrynoderma were not counted as deficiency states. The frequency of scurvy in employed natives can be taken as an index of the unsatisfactory rations supplied by many employers. It can also be assumed that these diets are deficient in more factors than ascorbic acid.

By his publications, and in conversation, Professor Gillman has made me realise the importance of controlled conditions in nutritional experiments. He has also emphasised the uncritical attitude of many observers. Rest and adequate diet are important factors in medicine and are often ignored in vitamin investigations. Short of solitary confinement, I felt it was hopeless to exclude my scurvy patients from the ordinary hospital meals which usually contain a reasonable quantity of anti-scorbutics As an alternative, I instituted an observation

period. The majority improved without specific treatment, and two mild cases achieved clinical cure (Numbers 18 and 19). In assessing the efficacy of any treatment, this response to rest and the hospital rations must be considered. Severe scurvy remained static until given vitamin C, e.g., patient Number 1. Several patients actually deteriorated during the observation period, viz., Cases Numbers 10, 14 and 17. The following conclusions may be drawn:-

1. The moderate amount of vitamin C contained in the hospital ration combined with a decrease in physical activity was sufficient to cause a disappearance of symptoms in mild cases (the Sheffield volunteers were cured with 10 mgms. of ascorbic acid daily).

2. However, in the well established case exhibition of the specific vitamin is required, and in very severe cases it is not justifiable to withhold treatment for experimental purposes.

Originally I employed a dosage of 100 mgms. of ascorbic acid T.D.S., latterly this was increased to 300 mgms., plus 100 mgms. intramuscularly daily. The larger dose was employed in an attempt to ease the bed shortage by an accelerated cure, and also to see if any latent nutritional defects could be provoked into becoming detectable. This latter objective was not realised, as no new deficiency pattern developed during treatment. The

increased dosage appeared to produce a more rapid cure, but in the absence of controls no dogmatic statement can be made. With the exception of the doubtful case (Number 8), the response to treatment was very satisfactory. The milder examples were fit for discharge in less than two weeks. In severer cases, the course was more protracted and sometimes a complete cure was not achieved, as some abnormality of limb or gait persisted after several weeks in hospital (seven in the case of patient Number 16). Within twenty-four to seventy-two hours of receiving treatment, the pain became less; this subjective improvement was not usually accompanied by any change in the signs. The mouth cleared up before the limbs. The time required for the gums to heal varied from three to sixteen days, a rough average being nine from the start of therapy. To correct any other subclinical deficiencies, multivitamin capsules were prescribed either at the end of the stay in hospital or to be taken after discharge.

Vitamin P has been incriminated in the aetiology of scurvy. However, Scarborough, as a result of his investigations, decided,

> "There is no evidence that vitamin P plays any part in human scurvy".

Although the African diet must be extremely poor in vitamin

P, my observations confirm Scarborough's findings. Four patients were treated with vitamin P. Permidine (Glaxo) was given in daily doses of 1.5 gm.; this is above the amount usually recommended as adequate. Two patients did improve; however, these were mild cases which, in my opinion, would have responded like the controls and I am not satisfied the hesperidin had any specific effect. One patient deteriorated from the ambulant to the crippled. The last case (Number 17) died.

Although originally attracted to the theory that tropical myositis was a manifestation of avitaminosis C complicated by infection (Gelfand, 1948), I have been unable to establish a definite connection. This condition is common in Rhodesia. The disease is almost entirely confined to males, and occurs most frequently in the young adult. The general condition of the patient is often excellent, and the onset is generally sudden. There is severe pain in the affected muscles which are indurated and exquisitely tender. Some cases resolve, but a large number go on to suppuration and require surgical drainage. An interesting practical point is the difficulty in deciding on the presence or absence of pus, which often lying deep to muscle, may never fluctuate, and actually feel carcinoma hard. The clinical picture is similar

but not identical with classical scurvy. The explanation of bacterial invasion of a scorbutic haemorrhage appears simple and probable. Against this pathology the following facts should be considered:-

1. The other symptoms of scurvy are usually absent. Only on one occasion have I seen definite scurvy gums associated with a myositic abscess.

2. There is not the same seasonal incidence and the disease is seen in sites which are seldom affected by avitaminosis C, viz., the upper limb and the rectus abdominis muscle.

3. Tropical myositis occurs in territories and among classes of Africans where scurvy is unknown. (Personal communications from Trowell and Meiklejohn)

4. None of my scurvies produced pus, and certain authorities (Hess) state that scorbutic lesions do not suppurate.

It is a reasonable supposition that with the occurrence of definite scurvy a subclinical state of avitaminosis C must be widespread. All the authorities agree that scorbutic gums only appear when the deficiency is well established. The diagnosis of a subclinical state is difficult. Various manifestations have been described, but none of these are pathognomonic. Phrynoderma, anaemia, increased capiliary fragility can occur in a wide range of conditions; this is especially true of the African. The writer suggests a new symptom. Natives frequently

complain of pain in the legs. Several sufferers were investigated. Clinically, no abnormality could be detected. The Wassermann reaction, routine urine analysis and haemoglobin percentage gave no information. The B complex was not beneficial, but the discomfort was quickly relieved by ascorbic acid. Admittedly this statement lacks scientific verification. Vitamin C therapy, however, has been found most useful for these unsatisfactory cases who, in the past, were sometimes suspected of malingering. The proper investigation of these subclinical cases requires the laboratory. An interesting study could be made of the blood chemistry of the Bantu, whether suffering from frank avitaminosis C or in apparent health. The estimations of ascorbic acid in urine, plasma and leucocytes all have limitations and require skilled interpretation. The certain availability of clinical examples of scurvy and the deficient diet eaten by many natives, suggest Southern Rhodesia as a productive field for the bio-chemist.

Vitamin D

There is complete agreement between radiologists and physicians that rickets and osteomalacia do not occur in Central and East Africa.

SUMMARY

1. From evidence already available, it is clear that malnutrition is rife in two widely separated centres of Central and South Africa (Kenya and Johannesburg). It is postulated that malnutrition, with local clinical variations, affects the majority of the indigenous inhabitants from the equator to the Transvaal. Until further surveys are made in the intervening districts, a dogmatic statement about the whole area cannot be made. It is suggested that malnutrition is a cause of the indifferent attainments of the Bantu race.

2. The food of the Rhodesian native has been examined in some detail. It is clear, both from Government documents and direct observation, that the diet eaten by many natives is unsatisfactory. Even the official mining rations are inadequate. Moreover, a large percentage of natives exist on allowances inferior to this scale.

3. Sixty-five per cent of Rhodesian natives show clinical signs of malnutrition. It is considered that this estimate is conservative.

4. The vitamin A content of many African diets falls below any of the recommended minimum prophylactic doses. Diseases usually attributed to vitamin A deficiency are common in Southern Rhodesia:-

   (a) Bitot's spots, night-blindness and xerophthalmia are frequently seen at the three ophthalmological clinics maintained by the Government Medical Service.

   (b) Phrynoderma is found in 37 per cent of normal natives. This condition has been observed in association with avitaminosis A, scurvy and chronic starvation. A limited investigation was carried out on the therapeutic effects of vitamin A and ascorbic acid. It was shown that these substances are curative in some cases and of no benefit in others. The aetiology of phrynoderma is complex, individual idiosyncrasy is probably a factor in its development.

5. Thiamine deficiency is uncommon and unimportant in Southern Rhodesia.

6. Pellagra and ariboflavinosis are considered together. The characteristics of these diseases are

present in a minimal form in a large percentage of normal Africans. The theme of this discussion is that these syndromes are not to be regarded as specific deficiencies of certain vitamins, but manifestations of an acute episode in the process of chronic malnutrition.

7. Kwashiorkor is not only Rhodesia's most serious nutritional problem, it is a common cause of death in African children. A great many infants pass through a subclinical phase of this disease; the apparent recovery is not complete, and permanent damage is done to the tissues. The modern aetiology incriminates a high calorific diet containing inadequate proteins. The African methods of infant feeding are particularly suitable for the development of Kwashiorkor.

8. The only sources of vitamin C for many employed natives are meat and kaffir beer. The fact that so many Africans maintain health on these low intakes is practical proof that the minimum protective dose of ascorbic acid required to prevent scurvy is very small. Certain aspects of scurvy were investigated:-

   i. Scurvy is a common and important disease in Southern Rhodesia.

   ii. There is no real difference in the clinical picture of the European and African disease.

iii. Vitamin P is of no therapeutic value in the treatment of scurvy.

iv. The association of avitaminosis C with tropical myositis was not established.

9. For practical purposes, vitamin D deficiencies do not occur in Southern Rhodesia.

CONCLUSIONS

It is difficult to present a balanced picture of any problem. Malnutrition in Rhodesia is not expressed in dramatic terms of famine and starvation. It is an unexciting story of an insidious process wreaking its effects on the bodies of the population. Although the majority are affected, it is only occasionally that the deficiencies develop into the definite syndromes recognised by the clinician as ariboflavinosis, pellagra or scurvy. In the African there is no sharp distinction between health and disease: most live in a twilight state.

Infantile mortality in Africa has always been enormous. It is easy to be complacent towards kwashiorkor. Apart from the deaths in the acute phase, it must be realised that the organs of many young children already bear the stamp of malnutrition.

No attempt can be made to forecast with certainty the health of any community. Certain factors, however, can be considered. The population has doubled in fifty years of European occupation; the increase shows no sign of abating. Industrialisation is bringing more and more natives into the towns and under the spell of

the cheap carbohydrate. Soil erosion is a serious problem in the already over-crowded reserves. The purchasing power of native wages is decreasing in the face of rapidly increasing prices. It is not too much to say that wise planning is required to safeguard the nutrition of the African population.

It would be unfair to give the impression that the Government of Southern Rhodesia has entirely ignored its responsibility towards the native. Vigorous schemes of food production and soil conservation have been undertaken. The medical service, despite greatly increased costs, has been extended. There are, however, several obvious improvements to be recommended. An adequate ration must be enforced for all employed natives. The system of vegetable gardens should be encouraged. Education of both the European and African is very necessary. Missionaries could assist in this campaign and should also be invited to co-operate with the Department of Native Agriculture. The children require special attention; their needs are best catered for by infant welfare centres and health visitors. Birth control has been seriously advocated; there is, however, hesitation in introducing the African to a practice so alien to his traditional way

of life. It seems desirable to substitute, at least partially, some other food for maize. Even assuming wholehearted co-operation, there are technical difficulties in the introduction of European crops like potatoes, wheat and oats; even a return to the small sorghum grains would not be easy. All these measures are immediate practical steps which, however, do not go to the root cause of the problem. The only fundamental solution is a radical improvement in the African standard of living.

APPENDIX.

Kaffir Beer, Mealie meal and ground nuts are of importance in Bantu diet but unknown in the European, the nutritional content of these substances is recorded for the convenience of the reader. No estimations have been done on local products, the values quoted are taken from South African Food Tables (Fox and Goldberg, 1944)

(1) Mealie meal (96% extraction) per 1 lb contains:-

Water	=	55 gm
Crude Protein	=	43.0 gm
Carbohydrate	=	323 gm
Fibre	=	9.5 gm
Calories	=	1,633 gm
Calcium	=	91 mgm
Iron	=	17.3 mgm
Phosperous	=	922 mgm
Vitamin A	=	nil
Theamin	=	1.14 mgm
Riboflavin	=	0.54 mgm
Nicotinic acid	=	5.4 mgm
Ascorbic acid	=	nil

(2) Kaffir Beer per pint contains:-

Vitamin A	=	nil
Theamin	=	0.12 mgm
Riboflavin	=	0.18 mgm
Nicotinic acid	=	1.5 mgm
Ascorbic acid	=	4.8 mgm

(3) Ground Nuts (Shelled) per 1 lb contains:-

Crude Protein	=	125 gm
Fat	=	220 gm
Carbohydrate	=	63.6 gm
Fibre	=	11.4 gm
Calories	=	2,737 gm
Calcium	=	277 mgm
Iron	=	9.1 gm
Phosperous	=	1,657 mgm
Vitamin A	=	Trace
Theamin	=	2.27 mgm
Riboflavin	=	2.27 mgm
Nicotinic acid	=	68.1 mgm
Ascorbic acid	=	nil

AFRICAN RATION SCALES.

The ration scales of various government and commercial enterprises are shown in the following page. It can be stated with experience that the native does not always receive his full allowance. Items such as ground nuts and vegetables are subject to periodic shortages. The depredations of contractors, cooks and storekeepers can be considerable. A personal experience of the native catering may serve as a useful illustration. The meat supplied to a certain government hospital was found regularly to contain more than the 25% of bone stipulated in the contract. It was also found that the native cooks stole at least a third of all the meat issued to the cookhouse. When these two abuses were corrected, the amount available for the patients was doubled.

RATIONS SCALES PER ADULT MALE (WEEKLY)

Commodity	Govt. Mine Rations	Mine A	Police Recruits	African Soldiers	Convicts	Tobacco Farm	Civil Servants	Hospital Patients
Mealie Meal	10½ lbs	14 lbs	10½ lbs	10 lbs	14 lbs	14 lbs	10½ lbs	7 lbs
Ground Nuts	1 lb	½ lb	2 lb	1 lb 12oz	-	-	2 lb	2 lb
Dried Beans	2 lbs	2½ lbs	Nut beans or rice 2 lbs	Nuts beans or rice 2 lbs	14 oz	1½ lbs	1 lb	1 lb
Salt	3½ oz	6 oz	3½ oz	3½ oz	5 oz	-	4 oz	4 oz
Sugar	-	6 oz	-	7 oz	-	-	-	-
Vegetables	2 lbs	1 lb	2 lbs	2 lbs	2 lb 10 ozs.	-	2 lbs	2 lbs
Meat[x]	2½ lbs	2 lbs	7 lbs	3 lbs	1 lb 10 ozs.	1 lb	2½ lbs	2½ lbs
Fat			1¾ lbs		5 ozs		may partially replace ground-nuts	as per Civil Servant
Dependants		approx. ½ rations for wife				7 lbs for wife		
Other Items		Tobacco 2½ oz		Coffee or Tea 1¾ oz	Soup 7 pints Good conduct prisoners extra meat, tea or coffee	Gardens available	Sweet Potatoes 7 lbs.	Sweet Potatoes 4 lbs

[x] The weight of meat is inclusive of bone, up to 25% is officially allowed in Government Contracts.

AFRICAN DIET HISTORIES

A hundred Africans were questioned about their food, the majority were hospital patients. All nutritional conditions were excluded and preference was given to traumatic cases. With a few exceptions the assistance of an interpreter was required. The enquiry was standardised as much as possible, each subject being questioned about the following points.

(a) The source of his food viz. Rations from employer or buys own food.

(b) The basic elements of the diet, invariably meat and mealie meal, sometimes beans and ground nuts.

(c) Fruit and vegetables in the rations supplied.

(d) Milk consumption.

(e) Antiscorbutics available from sources other than the employer's rations. Where the intake appeared to be low, the patient was asked when he last partook of vegetables, fruit, or milk.

(f) Kaffir beer.

(g) 60 men were questioned about their egg consumption.

Where possible literal answers have been preserved. Amounts when volunteered have been recorded. There was no means of checking these and quantative statements should be accepted with caution. Seventeen of the diets examined were judged to be deficient in vitamin C. viz. did not contain more than one orange or its equivalent per month.

(1) Disease: Jaundice. Employment: Kitchen Boy

(a) Rations supplied by employer.
(b) Consists of mealie meal, meat, bread, and tea.
(c) No fruit, vegetables or milk included.
(d) Supplied with sufficient milk for tea.
(e) Never buys vegetables, fruit or milk. He cannot remember when he last ate any of these articles of diet.
(f) Denies drinking beer.

(2) Disease: Septic Foot. Employment: Builder's Assistant.

(a) Buys own food.
(b) Consists of mealie meal, meat, occasionally tea and sugar.
(c) Does not buy fruit.
(d) Cannot remember when he last ate antiscorbutic food.
(e) Denies drinking beer.

(3) Disease: Septic Hand, and Syphilis. Employment: Labourer:

(a) Rations supplied by employer.
(b) Consists of Mealie meal, meat and beans (dried).
(c) No vegetables or milk supplied.
(d) No milk in rations but buys one cup daily for tea.
(e) Never buys vegetables or fruit and has not eaten same for the last 4 months.
(f) Drinks beer once a week.

(4) Disease: Hernia. Employment: Miner.

(a) Rations supplied by employer.
(b) Consists of:- Mealie meal, meat and beans.
(c) Cabbage once a week, 1 orange per week, no milk.
(d) Does not buy any Antiscorbutic food.
(e) Drinks beer twice a week.

(5) Disease: Strangulated Hernia. Employment: Labourer.

(a) Mealie meal from employer. Given 3/- per week ration money. He buys meat sometimes bread, tea, and cigarettes.
(b) Never buys vegetables.
(c) Occasionally buys bananas or oranges.
(d) Occasionally buys milk for tea.
(e) Had bananas last week.
(f) Drinks beer once a week.

(6) Disease: Cartilage Injury. Employment: Miner.

(a) Rations supplied by employer.
(b) Consists of:- Mealie meal and meat.
(c) Spinach twice weekly or a cabbage in lieu.
(d) No fruit of milk.
(e) Sometimes buys condensed milk.
(f) Denies taking beer.

(7) Disease: Fractured Femur. Employment: Electrician's Assistant.

(a) Rations supplied buy employer.

(b) Consists of mealie meal, meat, and beans (dried).

(c) Cabbage once a week.

(d) No fruit or milk supplied in rations.

(e) Never buys any antiscorbutic food.

(f) Drinks beer once a week.

(8) Disease: Injury to hand. Employment: Textile Worker.

(a) Buys own food.

(b) Consists of mealie meal, meat and bread.

(c) No vegetables, fruit or milk supplied.

(d) Does not buy any antiscorbutic food. He has not eaten any vegetables or fruit for about one year. Had some milk with tea 10 days ago.

(e) Drinks beer once a week.

(9) Disease: Assualt Case. Employment: Store keeper.

(a) Rations supplied by employer.

(b) Consists of mealie meal, meat, and beans.

(c) No vegetables or milk supplied.

(d) Very occasionally buys fruit.

(e) Never buys milk.

(f) Denies drinking beer.

(10) Disease: Head Injury. Unemployed: Living in his Kraal.

(a) Procures his own food.

(b) Consists of:- Mealie meal, and milk, which he drinks every day as sour milk.

(c) Eats wild berries.

(d) No vegetables.

(e) Occasionally kills a goat.

(f) Drinks beer about 8 times a month.

(11) Disease: Assault Case. Employment: Farm Labourer.

(a) Rations supplied by employer.

(b) Consists of:- Mealie meal, Meat and sugar.

(c) Spinach once a week.

(d) No fruit or milk.

(e) Sometimes buys condensed milk - approximately once a week.

(f) Drinks beer sometimes 3 times a month.

(12) Disease: Car Accident. Employment: Butcher Boy.

(a) Rations supplied by employer.

(b) Consists of:- Mealie meal 40 lbs a month. 1 lb meat daily.

(c) No vegetables, fruit or milk.

(d) Buys butter and milk about 3 times a week and condensed milk for tea. Last ate vegetables about six months ago.

(e) Drinks beer twice a week.

(13) Disease: Mine Accident. Employment: Miner.

(a) Rations supplied by employer.

(b) Consists of:- Mealie meal, meat and dried beans.

(c) Occasionally spinach in rations.

(d) No fruit or milk.

(e) Occasionally buys milk. Last ate green food 1 month ago.

(14) Disease: Burns of Face. Employment: Living in his Kraal.

(a) Grows own food.

(b) Mealie meal and milk every day, drunk as sour milk.
Occasionally meat, viz. once a month.

(c) Green pumpkin leaves occasionally, and wild berries.

(d) Does not take beer.

(15) Disease: Hernia. Employment: Carpenter.

(a) Rations supplied by employer.

(b) Consists of:- Mealie meal and meat daily.

(c) Spinach daily.

(d) No fruit.

(e) No milk from employer, but obtains some from his own cattle.

(f) Buys beer generally every day.

(16) Disease: Orchitis Employment: Cement Worker

(a) Buys own food.

(b) Consists of:- Mealie meal and meat.

(c) Obtains wild vegetables about once every second week.
No fruit of milk. Does not buy any fruit or milk.

(d) Drinks beer once a week.

(17) Disease: Tumour of Kidney. Employment: Farm Labourer.

(a) Rations supplied by employer.

(b) Consists of:- Mealie meal - 14 lbs a week. Also meat.

(c) Pumpkins and potatoes ad lib.

(d) No fruit or milk.

(e) Drinks beer twice a week.

(18) Disease: Hydrocele. Employment: Labourer - Filling grain bags.

(a) Rations supplied by employer.

(b) Consists of:- Mealie meal and meat.

(c) Cabbage once a week. Carrots once a week.

(d) No fruit or milk.

(e) Occasionally buys condensed milk.

(f) Drinks beer once a week.

(19) Disease: Synovitis. Employment: Carpenter.

(a) Buys own food.

(b) Consists of mealie meal and meat.

(c) Obtains wild vegetables, which he buys as an alternative to meat, exact frequency not obtainable.

(d) No fruit.

(e) Obtains some milk about once a week.

(f) Does not drink beer.

(20) Disease: Burns of legs. Employment: Steam Laundery.

(a) Buys own food.

(b) Consists of:- Mealie meal and meat.

(c) No vegetables fruit or milk.

(d) Last ate Antiscorbutic Foods about four months ago.

(21) Disease: Fractured Tibia. Employment in R.A.R.

(a) Rations received from Army.

(b) Consists of:- Mealie meal, meat and tea daily. Rice once a week.

(c) Cabbage once a week.

(d) Oranges every Friday.

(e) Given milk with tea.

(f) Drinks beer once a week.

(22) Disease: Laceration of Hand. Employment: Coal Miner.

(a) Rations received from employer.

(b) Consists of:- Mealie meal, meat, monkey nuts, beans and sugar.

(c) Cabbage once a week.

(d) Oranges once a week.

(e) Sometimes drinks sour milk.

(f) Beer issued with rations. Claims not to indulge.

(23) Disease: Laceration of hand. Employment: Labourer.

(a) Rations received from employer.

(b) Mealie meal, meat and beans.

(c) No vegetables, fruit or milk.

(d) Buys wild vegetables about 3 times a week.

(e) Does not drink beer.

(24) Disease: Dislocation Right Ankle. Employment: Mission Teacher.

(a) Buys own food.

(b) Buys mealie meal, meat sugar and tea.

(c) Buys cabbage.

(d) Buys oranges once or twice weekly.

(e) Buys milk for tea.

(f) Buys beer once or twice a week.

(25) Disease: Bilateral Synovitis of knees. Employment: In business.

(a) Buys own food.

(b) Buys Mealie meal and meat.

(c) Buys cabbage, onions and tomatoes about once a week.

(d) Buys oranges and bananas twice a month.

(e) Does not drink beer.

(26) Disease: Fractured Tibia. Employment: Labourer on Roads.

(a) Rations received from employer.

(b) Mealie meal, meat, and monkey nuts. Dried beans.

(c) No fruit, vegetables or milk.

(d) Does not buy Antiscorbutic food. Does not remember when last he consumed vegetables, including wild vegetables, fruit or milk.

(e) Drinks beer twice a week.

(27) Disease: Fractured Tibia. Employment: Garage Boy.

(a) Rations from employer.

(b) Dried beans, mealie meal, and monkey nuts.

(c) Occasionally given green mealies and Pumpkins. Last issue approximately four months ago.

(d) No milk in rations.

(e) Does not buy vegetables or fruit, occasionally obtains wild vegetables.

(f) Drinks a large amount of beer on Saturdays.

(28) Disease: Prostatectomy. Employment: Lorry Driver.

(a) Rations from employer.

(b) Consists of:- Mealie meal, sugar and meat.

(c) No milk, fruit or vegetables from employer.

(d) Never buys any milk, vegetables or fruit.

(e) Occasionally eats wild vegetables.

(f) Drinks beer twice a week.

(29) Disease: Injury to Knee Joint. Employment: Mechanic.

(a) Rations from employer.

(b) Consists of:- Mealie meal, meat, dried beans and sugar.

(c) One whole cabbage per week, carrots once a week. No fruit or milk.

(d) Buys condensed milk for tea about once a week.

(e) Does not procure wild vegetables.

(f) Does not drink beer.

(30) Disease: Car Accident. Employment: Carpenter.

(a) Buys own food.

(b) Consists of:- Mealie meal, tea, sugar and meat.

(c) Buys condensed milk about once a week, cabbage three times a week.

(d) Sometimes eats wild vegetables.

(e) Drinks beer daily.

(31) Disease: Laceration of Hand. Employment: Car Mechanic.

(a) Buys own food.

(b) Consists of:- Mealie meal, meat and sugar.

(c) Buys 8 - 10 tomatoes a week, condensed milk once a week.

(d) Buys wild vegetables sometimes.

(f) Denies drinking beer.

(32) Disease: Car Accident. Employment: Tailor.

(a) Buys own food.

(b) Consists of:- Mealie meal, meat and sugar.

(c) Buys cabbage twice a week, milk daily. No fruit.

(d) No wild vegetables.

(e) Drinks beer daily.

(33) Disease: Knee Injury. Employment: Bricklayer.

(a) Rations from employer.

(b) Consists of:- Mealie meal, meat and dried beans.

(c) No milk, vegetables or fruit given in rations.

(d) Buys fresh milk about five times a month, but not vegetables or fruit.

(e) Occasionally eats wild vegetables, has not eaten same for the last month.

(f) Drinks beer when he has the money.

(34) Disease: Laceration of Leg. Employment: nil. Lives in Kraal.

(a) Obtains food from his own farm.

(b) Consists of:- Mealie meal, meat, milk. (Drunk both fresh and sour).

(c) Cabbage occasionally, and eats wild vegetables more often.

(e) Drinks lots of beer at week-ends.

(35) Disease: Sigmoid Colon Tumour. Employment: Live in Kraal.

(a) Obtains food from own farm.

(b) Consists of mealie meal, meat, milk, (fresh and sour).

(c) Cabbage once a week.

(d) Eats wild vegetables quite frequently.

(e) Denies drinking beer.

(36) Disease: Laceration of hand. Employment: Painter.

(a) Buys own food.

(b) Consists of mealie meal and meat.

(c) Buys cabbage twice a week, milk for tea, no fruit.

(d) Does not eat wild vegetables.

(e) Drinks beer twice a week.

(37) Disease: Burns of leg. Employment: Glue Factory.

(a) Buys own food.

(b) Consists of mealie meal, and meat.

(c) Occasionally buys milk for tea, no vegetables, buys three oranges weekly.

(d) Drinks beer daily.

(38) Disease: Appendicitis. Employment: Works in Brewery.

(a) Buys own food.

(b) Consists of mealie meal and meat.

(c) Buys milk for tea, rarely buys vegetables or fruit.

(d) Eats no wild vegetables.

(e) Denies drinking beer.

(39) Disease: Laceration of Hand. Employment: Works in Brush Factory.

(a) Rations from employer.

(b) Consists of mealie meal and meat.

(c) No milk, or vegetables given in rations.

(d) Buys milk for tea.

(e) Eats wild vegetables daily.

(f) Denies drinking beer.

(40) Disease: Enteritis. Employment: Teacher.

(a) Buys own food.

(b) Mealie meal, meat (once a week) occasionally beans and monkey nuts.

(c) Eats wild vegetables once a week.

Does not buy milk or fruit.

(e) Eats an egg about once a month.

(g) Does not drink beer.

(41) Disease: Alcohdic (acute). Employment: Labourer in Cement Factory.

(a) Rations partly from employers, viz. cooked midday meal.

(b) Buys own evening meal, lunch consists of mealie meal, meat and vegetables (Cabbage, peas or potatoes).

Buys:- Mealie meal, cabbage, cauliflower, meat, sugar and tea, occasionally buys own fruit.

(c) Does not eat eggs.

(d) Drinks Beer.

(42) Disease: Accident Case. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, monkey nuts, meat, once a week.

(c) Vegetables once a week viz. one cabbage occasionally and sour milk.

(d) Does not buy vegetables or fruit. Eats eggs when available.

(e) Does not drink beer.

(43) Disease: Hernia. Employment: Farm Labourer:

(a) Rations from employer.

(b) Meat once a week, dried beans and mealie meal.

(c) No vegetables or milk in rations. No regular source of vegetables.

(d) Last ate vegetables six months ago.

(e) Drinks beer at week-ends.

(44) Disease: Carcinoma of Bladder. Employment: Farm Labourer.

(a) Rations from employer.

(b) Meat once a week, (piece as big as fist), mealie meal, dried beans.

(c) No vegetables or milk in rations.

(d) Buys oranges once a week.

(e) Has not eaten vegetables for one year. Does not eat eggs.

(f) Drinks beer at week-ends.

(45) Disease: Peritonitis: Employment: Farm Labourer

(a) Rations from employer.

(b) Consists of mealie meal, meat once a month approx 4 lbs, dried beans.

(c) No vegetables, milk or fruit in rations. Eats about two eggs monthly.

(d) Does not have a regular source of vegetables, milk or fruit.

(e) Cannot remember when he last ate antiscorbutic food.

(46) Disease: Fractured Femur. Employment: Quarry Worker.

(a) Rations from employer.

(b) Mealie meal, meat twice a week, dried fish once a week, and beans.

(c) Cabbage, sweet potatoes and oranges every week.

(d) Beer twice a week.

(e) Does not eat eggs.

(47) Disease: Septic Toe. Employment: Garage Hand.

(a) Buys own food.

(b) Mealie meal, meat cabbage and milk.

(c) Does not drink beer.

(g) Had eggs last in January.

(48) Disease: Unresolved Preumonia. Employment: Cook Boy

(a) Rations from employer.

(b) Meat, mealie meal, sugar, tea, and enough milk for tea.

(c) No vegetables or fruit from employer.

(d) Vegetables from own garden which he eats every day.

(e) Drinks beer.

(f) Claims he ate an egg for the first time in his life a week ago.

(49) Disease: Sinusitis. Employment: Kraal Native.

(a) Food from his own farm.

(b) Mealie meal, kaffir corn, munga, beans and meat about once a week.

(c) Eats wild vegetables.

(e) Beer about twice a week.

(f) Eats eggs about twice a week.

(50) Disease: Cirrhosis of Liver. Employment: Kraal Native.

(a) Food from own farm.

(b) Mealie meal, kaffir corn, munga, and beans, eats meat once a week.

(c) Eats wild vegetables every day.

(d) Drinks beer three times a week.

(e) Eats eggs nearly every day.

(51) Disease: Malaria. Employment: Hotel waiter

(a) Rations from employer.

(b) Meat, mealie meal, sugar, tea,

(c) Cabbage and potatoes every day.

(d) Denies drinking beer.

(e) Has not eaten an egg for one year.

(52) Disease: Myositis. Employment: Washing Boy.

(a) Rations from employer.

(b) Mealie meal, meat and chicken.

(c) Buys vegetables two or three times a week.

(d) drinks beer at week-ends.

(e) Eggs from employer nearly every day.

(53) Disease: Chronic Malaria. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, meat twice a week, dried beans, no monkey nuts.

(c) No vegetables, milk, or fruit in rations.

(d) No private source of vegetables, milk or fruit.

(e) Has not eaten antiscorbutic food for about eight months.

(f) Denies drinking beer.

(g) Has not eaten eggs for about eight months.

(54) Disease: Poliomyelitis. Employment: Laundry Boy.

(a) Rations from employer.

(b) Mealie meal, and beans, no meat or monkey nuts in Rations.

(c) No vegetables in rations.

(d) Buys wild vegetables twice a week.

(e) Denies drinking beer.

(f) Does not eat eggs.

(55) Disease: Myositis. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, meat once a week, dried fish once a week.

(c) No vegetables or milk in rations.

(d) Does not have a regular source of fruit, vegetables or milk.

(c) Claims he has not eaten any antiscorbutic food for four years.

(f) Drinks beer at week-ends.

(g) Has never eaten eggs.

(56) Disease: Osteitis. Employment: Store Boy.

(a) Buys own food.

(b) Mealie meal, meat, wild vegetables, and cabbage.

(c) Buys milk for tea.

(d) Does not drink beer.

(e) Eats eggs about three times a week.

(57) Disease: Accident. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, meat once a week, dried beans.

(c) No vegetables in rations, oranges once a month.

(d) No outside source of antiscorbutic foods.

(e) Does not drink beer.

(f) Eats about three eggs a month.

(58) Disease: Fractured Femur. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, meat, about once a month, dried beans once a week.

(c) Oranges once a week.

(d) No antiscorbutic food.

(e) Supplied beer once a week.

(f) Ate eggs about a year ago.

(59) Disease: Fractured Femur. Employment: Farm Labourer

(a) Rations from employer.

(b) Mealie meal, dried beans once a week, meat once a month (about 5 lbs).

(c) No fruit, milk or vegetables in ration.

(d) No private source of antiscorbutic food.

(e) Last eat vegetables about $1\frac{1}{2}$ years ago.

(f) Does not take beer because of religious convictions.

(g) Last ate eggs six years ago.

(60) Disease: Fractured Tibia and Fibula. Employment: Railway Worker.

(a) Rations from employer.

(b) Mealie meal, meat once a week, dried beans once a week, sugar once a week.

(c) Vegetables viz., cabbage, spinach, and potatoes, no milk in rations.

(d) Buys cabbage occasionally.

(e) -

(f) Does not drink beer.

(g) Eats eggs about once a week.

(61) Disease: Cyst of knee. Employment: Kraal Native.

(a) Grows own food.

(b) Consists of rapoko, no mealie meal, meat once a month and dried beans.

(c) Vegetables from farm.

(d) -

(e) -

(f) Drinks beer weekly.

(g) Eats eggs very occasionally, viz., once in six months.

(62) Disease: Fractured Tibia. Employment: Garden Boy.

(a) Buys own food.

(b) Mealie meal and meat.

(c) Occasionally gets presents of cabbage and sweet potatoes also milk.

(f) Denies drinking beer.

(g) Last ate eggs about two years ago.

(63) Disease: Retention of Urine. Employment: Kraal Native.

(a) Grows own food.

(b) Mealie meal, rapoko, munga, monkey nuts, dried beans, meat once a week.

(c) Grows cabbage, drinks milk every day.

(f) Denies drinking beer.

(g) Eggs about two or three times a month.

(64) Disease: Malaria. Employment: Quarry Worker.

(a) Rations from employer.

(b) Mealie meal, meat daily, tea and bread.

(c) Cabbage every day, milk once a week.

(d) Does not buy any antiscorbutics.

(e) -

(f) Drinks beer at week-ends.

(g) Last ate an egg three years ago.

(65) Disease: Head Injury. Employment: Pipe Worker.

(a) Buys own food.

(b) Mealie meal, meat, bread, tea.

(c) Buys cabbage and beans every week.

(d) -

(e) -

(f) Denies drinking beer.

(g) Eggs occasionally viz., once in two months.

(66) Disease: Abscess Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, meat 1 lb. a month.

(c) Milk every day.

(d) No antiscorbutics from other sources.

(e) -

(f) Beer at week-ends.

(g) Has never eaten an egg.

(67) Disease: Pneumonia. Employment: Cook Boy.

(a) Rations from employer.

(b) Mealie meal, meat daily, tea and bread.

(c) Cabbage, potatoes, milk in tea every day.

(d) -

(e) -

(f) Denies drinking beer.

(g) Eggs twice a week.

N.B. This patient says he receives liberal food supply because of his occupation. The ordinary labourers are not so well off.

(68) Disease: Laceration of Face. Employment: Road Labourer.

(a) Rations from employer.

(b) Mealie meal, meat once a week (2 lbs.) dried beans.

(c) One cabbage per week and peas.

(d) Occasionally buys milk.

(e) -

(f) Denies drinking beer.

(g) Last ate an egg three months ago.

(69) Disease: Conjunctivitis. Employment: Mill Boy.

(a) Rations from employer.

(b) Mealie meal, meat once a week.

(c) Oranges once a week, cabbage and potatoes weekly.

(d) Buys milk for tea.

(e) -

(f) Denies drinking beer.

(g) Eats eggs about once a month.

(70) Disease: Enteritis. Employment: Truck Driver.

(a) Buys own food.

(b) Mealie meal, meat twice a week.

(c) Buys vegetables nearly every day, viz., cabbage and lettuce. Milk for tea, buys oranges once a week.

(d) -

(e) -

(f) Denies drinking beer.

(g) Ate eggs two months ago.

(71) Disease: Fractured Femur. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, meat once a month, lasts for two weeks, dried beans once a week, does not drink milk.

(c) Has a plot and grows cabbage and onions.

(d) Nil.

(e) -

(f) Denies drinking beer.

(f) Last ate eggs 9 months ago.

(72) Disease: Employment: Jute Worker.

(a) Food from employer.

(b) Mealie meal, meat 2 lbs. per week. Dried fish once a week.

(c) Carrots, cabbage, tomatoes, potatoes every week.

(d) Buys milk for tea.

(e) -

(f) Does not drink beer.

(g) Ate an egg about a year ago.

(73) Disease: Fractured Femur. Employment: Lorry Driver.

(a) Buys own food.

(b) Mealie meal, meat (every second day), bread and tea.

(c) Buys cabbage and potatoes, and milk for tea.

(d) Nil.

(e) -

(f) Denies drinking beer.

(g) Last ate an egg 3 months ago.

(74) Disease: Typhoid. Employment: Driver.

(a) Grows own food.

(b) Mealie meal, munga, meat twice a week.

(c) Grows cabbage, onions and carrots.

(d) -

(e) -

(f) Occasionally drinks beer.

(g) Eggs every day.

(75) Disease: Typhoid. Employment: Bakery Boy.

(a) Partly from employer, with money allowance (5/- per month).

(b) 12 lbs. mealie meal a week, buys meat once a week, milk about three times per week.

(c) Buys cabbage every day.

(d) -

(e) -

(f) Drinks beer at week-ends.

(g) Last ate eggs about 1½ years ago.

(76) Disease: Hernia. Employment: Herd Boy.

(a) Rations from employer.

(b) Mealie meal and approximately 1 lb. meat weekly.

(c) Spinach, cabbage or potatoes daily. No milk.

(d) -

(e) -

(f) - Drinks beer at week-ends.

(g) Last ate eggs about four months ago.

(77) Disease: Pneumonia. Employment: Carpenter.

(a) Rations from employer.

(b) Mealie meal, and meat daily.

(c) Cabbage and spinach daily, 2 oranges per week, no milk.

(d) -

(e) -

(f) Does not drink beer.

(g) Eats eggs about twice a month.

(78) Disease: Fractured Pelvis. Employment: Cobbler.

(a) Rations from employer.

(b) Mealie meal, meat every second day.

(c) Cabbage and wild vegetables, buys milk for tea.

(d) -

(e) -

(f) Beer at week-ends.

(g) Last had an egg about five months ago.

(79) Disease: P.U.O. Employment: Labourer.

(a) Buys own food.

(b) Buys mealie meal, meat three times a week, beans once a week, dried fish once a week.

(c) Buys milk (to drink alone) three times a week, vegetables every second day (white loaf and cabbage).

(d) -

(e) -

(f) Beer occasionally.

(g) Last ate eggs about two years ago.

(80) Disease: Accident Case. Employment: Farm Labourer.

(a) Rations from Employer.

(b) Mealie meal, beans, 3 dried fish a week (no meat in rations).

(c) No vegetables, milk or fruit in rations.

(d) Has no other source of antiscorbutics.

(e) Has not eaten any vegetables, fruit or milk for three years.

(f) Beer on Saturdays.

(g) Eggs twice or three times a week (from own fowls).

(81) Disease: Virus Pneumonia. Employment: Jute Mill Worker.

(a) Rations from Employer.

(b) Mealie meal, meat twice a week, dried fish once a week.

(c) Cabbage and potatoes twice a week, does not drink milk.

(d) -

(e) -

(f) Teetotaller.

(g) Last had eggs four months ago.

(82) Disease: Septic Laceration of leg. Employment: Labourer.

(a) Buys own food.

(b) Mealie meal, meat about three times a week.

(c) Tomatoes, cabbage, potatoes, spinach. Does not buy milk, oranges once a week.

(d) -

(e) -

(f) Beer at week-ends.

(g) Last ate eggs about seven months ago.

(83) Disease: Compound Fracture. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, dried beans, dried fish once a week.

(c) No vegetables, fruit or milk in rations.

(d) No other source of Antiscorbutic foods.

(e) Last ate green foods about four years ago.

(f) Teetotaller. (Muslin).

(g) Last ate eggs about a month ago.

(84) Disease: T.B. Knee. Employment: Kitchen Boy.

(a) Rations from employer.

(b) Mealie meal, meat once a week. Monkey nuts once a week. Dried fish weekly.

(c) Cabbage once a week, milk every day.

(d) Buys oranges and bananas.

(e) -

(f) Does not drink beer.

(g) Eggs about once a week.

(85) Disease: Head Injury. Employment: Goal Guard.

(a) Buys own food.

(b) Mealie meal, meat daily, monkey nuts about once a month.

(c) Does not buy vegetables, buys milk for tea and oranges once a month.

(d) -

(e) -

(f) Does not drink beer.

(g) Had an egg about five years ago.

(86) Disease: Hernia. Employment: Shop Assistant.

(a) Buys own food.

(b) Mealie meal, meat daily, dried beans once a week, monkey nuts about once a month.

(c) Buys cabbage and spinach once every second day.

(d) -

(e) -

(f) Teetotaller.

(g) Eggs occasionally (about once in two months).

(87) Disease: Compound Fractured Tibia. Employment: Lorry Driver.

(a) Rations from Employer.

(b) Mealie meal and meat twice a week.

(c) Cabbage, spinach and fresh beans every week.

(d) -

(e) -

(f) Drinks beer at week-ends.

(g) Eggs about once in every six weeks.

(88) Disease: Hydrocele. Employment: Police Boy.

(a) Rations from employer.

(b) Mealie meal, meat approximately two pounds per week, monkey nuts and dried beans and cooking oil.

(c) Three cabbages per week, five oranges per week.

(d) Buys condensed milk for tea.

(e) -

(f) Drinks beer at week-ends.

(g) Last ate eggs about a year ago.

(89) Disease: Colles Fracture. Employment: Lavatory Attendant.

(a) Rations from employer.

(b) Mealie meal, meat twice a week, dried fish once a week. Sugar once a week.

(c) Twelve potatoes once a week, half a cabbage and some spinach every week, does not drink milk.

(d) -

(e) -

(f) Does not drink beer.

(g) Last ate eggs about two months ago.

(90) Disease: Pneumonia. Employment: Farm Labourer.

(a) Rations from employer.

(b) Mealie meal, dried beans, monkey nuts, meat once a month (enough for one day).

(d) No private source of antiscorbutics.

(e) Has not eaten any antiscorbutics for six months.

(f) Drinks beer every Sunday.

(g) Last ate eggs about a year ago.

(91) Disease: Myositis. Employment: Builder.

(a) Buys own food.

(b) Mealie meal, and meat.

(c) Buys cabbage, spinach, dried beans and oranges once a week.

(d) -

(e) -

(f) Does not drink beer.

(g) Has not eaten eggs for the last three years.

(92) Disease: Septic Foot. Employment: Kraal Native.

(a) Grows own food.

(b) Mealie meal, meat about once a week, monkey nuts.

(c) Eats cabbage, spinach, potatoes and dried beans, buys oranges once a week.

(d) No milk.

(e) -

(f) Denies drinking beer.

(g) Cannot remember eating eggs.

(93) Disease: Umbilical Hernia. Employment: Kraal Native.

(a) Grow own food.

(b) Mealie meal, rapoko, meat once a week and dried beans.

(c) Grows cabbage, spinach, does not drink milk.

(d) -

(e) -

(f) Does not drink beer.

(g) Last ate an egg about two years ago.

(94) Disease: Pneumonia. Employment: Road Labourer.

(a) Rations from employer.

(b) Mealie meal, meat once a week (approximately three pounds). Dried beans, monkey nuts.

(c) Given fifteen oranges per month.

(d) -

(e) -

(f) Beer at week-ends.

(g) Last ate eggs about one year ago.

(95) Disease: Planter Warts. Employment: Farm Labourer

(a) Rations from employer.

(b) Mealie meal, meat once a week, dried beans.

(c) Cabbage and tomatoes. No milk available.

(d) -

(e) -

(f) Beer at week-ends.

(g) Eats eggs once every six weeks.

(96) Disease: Asthma. Employment: City Cleaner.

(a) Rations from Municipality.

(b) Consist of mealie meal, meat once a week, dried fish once a week, dried beans.

(c) One cabbage per week. No milk or fruit in rations.

(d) -

(e) -

(f) Does not drink beer.

(g) Last ate eggs about a year ago.

(♀7) Disease: Pleurisy with effusion. Employment: Garage Attendant

(a) Buys own food.

(b) Mealie meal, meat three times a week. Occasionally dried beans.

(c) Eats two cabbages a week, an orange once a week, buys milk for tea.

(d) -

(e) -

(f) Does not drink beer.

(g) About two eggs a month.

(♀8) Disease: Stricture. Employment: Post Office Messenger.

(a) Buys own food.

(b) Mealie meal, meat daily, no dried beans or monkey nuts.

(c) Buys cabbage and milk for tea.

(d) -

(e) -

(f) Does not drink beer.

(g) Last ate eggs ten years ago.

(99) Disease: Employment: Kraal Native.

(a) Grows own food.

(b) Mealie meal, meat about once a week. Monkey nuts and dried beans.

(c) Eats pumpkin, oranges and bananas nearly every day. Sour milk daily.

(d) -

(e) -

(f) Beer at week-ends.

(g) Eggs twice a week.

(100) Disease: Hernia. Employment: Farm Labourer.

(a) Rations from employer.

(b) Consist of Mealie meal, meat once a week (about 2 lbs), once a month given A ration of dried beans or ground nuts.

(c) Two cabbages per week.

(d) -

(e) -

(f) Drinks beer at week-ends.

(g) Eats eggs once a month.

AFRICAN HAEMOGLOBIN ESTIMATION (MALE).

Place.	No.	Injury (if any).	Haemoglobin.	
Que Que	1	Compound fracture Toe.	91%	Sahli
"	2	Fracture Radius & Ulna	94%	"
"	3	Fracture Pelvis	89%	"
"	4	Hydrocele	82%	"
"	5	Fracture of tibia	114%	"
"	6	Fracture of Femur	101%	"
"	7	Multiple lacerations	75%	"
"	8	Laceration of ankle	33%	"
"	9	Laceration of Elbow	97%	"
"	10	Skin graft (old burns)	116%	"
"	11	Medical Orderly	100%	"
"	12	Laceration of Knee	75%	"
"	13	Acute Conjunctivitis	88%	"
"	14	Medical Orderly	92%	"
"	15	Laceration of fingers.	94%	"
Gwanda	16	Compound fracture Rt Femur	86%	"
"	17	Skin graft.	95%	"
"	18	Laceration of fingers Lt	85%	"
"	19	Laceration of feet	85%	"
"	20	Laceration of fingers Rt	91%	"
"	21	Laceration of fingers Rt	98%	"
"	22	Conjunctivitis	93%	"
"	23	Planter Warts	89%	"
"	24	Native Orderly	85%	"
"	25	Native Orderly	89%	"
"	26	Ward boy	89%	"
"	27	Native Orderly	84%	"
"	28	Native Orderly	86%	"
"	29	Sprained wrist	89%	"
"	30	Traumatic Conjunctivitis	89%	"
"	31	Ward boy	80%	"
Bulawayo	32	Native Orderly	84%	"
"	33	Native Orderly	85%	"
"	34	" "	80%	"
"	35	" "	86%	"
"	36	" "	88%	"
"	37	" "	79%	"
"	38	" "	77%	"
"	39	" "	81%	"
"	40	" "	80%	"
"	41	" "	82%	"
"	42	" "	80%	"
"	43	" "	81%	"
"	44	" "	81%	"

Place	No.	Injury (if any)	Haemoglobin.
Umtali	45	Native orderly	87% Sahli
"	46	" "	86% "
"	47	" "	79% "
"	48	" "	80% "
"	49	" "	90% "
"	50	" "	88% "

The estimations were performed by myself in daylight using the same Sahli haemoglobinmeter. (100% = 14 gms after 5 minutes). The subjects were selected from traumatic surgical patients, eye cases and the African nursing staff. Any - one claiming ill health was excused. The average reading was 87% As five normal Africans had haemoglobins of under 80%, it was felt reasonable to accept this figure as the lower limit of normal.

CASE NUMBER 1

Station	Que Que
Date of Admission	5th July, 1950.
Name	Beaton
Age	Elderly Male
Employment	Mining

History

(a) Presenting symptoms and duration

1. Headache.
2. Pain in shoulder blade.
3. Pains in both legs and knee.

Duration vague, ? one week.

(b) Questioning

1. Mouth symptoms - gums have been bleeding and teeth loose. Patient vague as to duration.
2. Night blindness.
3. Haematuria - occurred in childhood.

Diet History

Has been in present employment for one year. Buys his own food which is mealie meal, sugar, bread and meat. He does not buy vegetables and has not eaten any for one year.

Examination

General Nutrition state - very emaciated, calf muscles measure 10" at widest point.

Mental State - lethargic and depressed.

Gums - typical scurvy gums (several buds present)

Skin/

Skin

1. Hyperkeratosis - present to marked degree on arms and legs.
2. Pellagra - nil.
3. Phyrnoderma - nil.

Mucus Membranes - moderately well injected.

Clubbing - nil.

Oedema - Nil.

Jaundice - nil.

Dyspnoea - nil.

Other vitamin deficiency signs

1. Face - nil.
2. Eyes - nil.
3. Lips - nil.
4. Tongue - nil.
5. Scrotum - nil.

C.V.S. - no abnormality.

Lungs - no abnormality.

Abdomen - no abnormality.

C.N.S. - no abnormality,

Locomotor System (including gait)

Pains located in right knee and left calf. Left calf appears normal. No swelling or inflammation. Tenderness on moderate pressure. Right knee is within normal limits. No heat, swelling or limitation of movement. Patient can stagger a few steps.

Investigations

Blood	20.7.50	Hb 77%, R.B.C. 3.3 M per cmm. Cells normocytic.
	8.8.50	Hb 98%, R.B.C. 4.1 M per cmm. Stated to/

to have M.T. parasites on admission (quinine given for several days).

Urine — On admission was stated to contain red blood cells and S. Haematobium. I could not confirm this finding either by microscope or mirocidioscope.

Treatment

19.7.50 Intramuscular vitamin C 100 mgms. daily.

26.7.50 Tabs, vitamin C 100 mgms. T.D.S. (Intramuscular therapy discontinued).

31.7.50 Nicotinic acid 50 mgms. T.D.S. and Betalin 1 cc. daily.

Progress Notes

26.7.50 Gums show marked improvement but there is little change in general condition. Shows no inclination to get out of bed and still has great difficulty in walking.

31.7.50 In the last two days a great improvement has been noticeable. States he has no pain either walking or at rest. Unrequested, demonstrated his ability to walk up the ward. Mentally is more alert and has lost his former miserable expression. Gums are now normal.

8.8.50 Patient absconded.

Date of Discharge

See above.

Condition on Discharge

Condition greatly improved but still very thin.

Comments and Conclusions

A severe case of scurvy who was bedridden by the disease. I first examined him on the 17th July, 1950. For twelve days he had been receiving treatment for malaria and bilharzia without improvement, which occurred immediately he was given ascorbic acid. This case illustrates the importance of routine mouth examination in African medicine.

The anaemia responded to Vitamin C. alone It is interesting to compare the slight drop in his haemoglobin with the crippling involvement of his locomotor system.

CASE NUMBER 2

Station	Que Que
Date of Admission	15th August, 1950.
Name	John
Age	Elderly male
Employment	Underground miner

History

(a) Presenting symptoms and duration

1. Cough for 2 months.
2. Haemoptysis, last occurred about two months ago.

(b) Questioning

1. Mouth symptoms - mouth painful, teeth feel loose, gums bleed.
2. Night blindness - nil.
3. Tongue has been cracking for last 3 months.
4. Pain in limbs - nil.
5. Haematuria - nil.

Diet History

Fed on dried beans, monkey nuts and meat (twice a week), no fresh vegetables in ration. He has not eaten any fresh food since last green mealie season, (about five months ago).

Examination

General Nutritional state - moderate. Not emaciated.

Mental State - lethargic.

Gums - several scurvy buds which bleed on moderate pressure. However, there is the general retraction of gums and the typical appearance of scurvy lacking.

Skin/

Skin

1. Hyperkeratosis - marked especially hands and feet.
2. Pellagra - nil.
3. Phyrnoderma - marked.

Mucus Membranes - well injected

Clubbing - slight

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil

Other vitamin deficiency signs

1. Face - nil.
2. Eyes - no vascularisation of cornea.
3. Lips - angular stomatitis, lips rough but no actual cheilosis. Raw patches on mucous membranes of mouth which bleed readily.
4. Tongue - raw at tip with atrophied patches.
5. Scrotum - normal no pruritis.
6. Marked follicular seborrhoea of nose, cheeks and chin.

B.P. - 140/80

C.V.S.- no abnormality.

Lungs - no abnormality.

Abdomen - no abnormality.

C.N.S. - no abnormality

Locomotor System (including gait) - no abnormality.

Investigations

Blood On admission (and subsequently) Hb 98%. R.B.C. 4.9 M per cmm. W.B.C. 5,200 cmm.

Treatment/

Treatment

16.8.50 Mist. Pot. Chlor. mouth wash.
Yeast tabs. 2 T.D.S.
Nicotinic acid tabs. 2 T.D.S.
Intramuscular vitamin C 1 amp. (100 mgm.) daily.

24.8.50 Tabs. Riboflavin, 2 mgm. T.D.S.
Cod liver oil ½ oz. T.D.S.

Progress Notes

22.8.50 Gums show definite improvement. Buds now disappearing. Angular stonatitis and follicular seborrhoea disappearing. No chest symptoms but still complains of weakness. Skin unchanged.

27.8.50 Buds no longer visible, but gums still bleed readily. There has been a retraction of gums since start of treatment. No change in skin condition. Patient looks more alert. States he feels stronger. No chest symptoms.

31.8.50 Follicular seborrhoea still present but less marked.

2.9.50 The actual mucous membranes of gums appears normal but the disappearance of the general hypertrophy has made the retraction more conspicuous.

14.9.50 Transferred to larger centre for more accurate X-Ray of chest.

23.9.50 Returned to Que Que. The X-Ray of chest showed no abnormality. Follicular seborrhoea has entirely disappeared. Skin unchanged.

25.9.50 Discharged with recommendation to be employed only on the surface and to be checked up at regular intervals.

Conclusion

This case is classified as a mixed Riboflavin and ascorbic acid deficiency. The hyperkeratosis persisted and was uninfluenced by therapy with the vitamin B complex. It is interesting to note that although this patient had signs highly suggestive of aribоflavinosis he did not complain of and denied any itching of his scrotum.

CASE NUMBER 3

Station	Que Que
Date of Admission	26th August, 1950
Name	Grey
Age	Elderly Male
Employment	Mining

History

(a) Presenting symptoms and duration

1. Penile sores for 3 months.
2. Pains in legs of 5 months duration. (Pain present at rest).

(b) Questioning

1. Mouth symptoms - gums have been bleeding, duration vague. Teeth not loose.
2. Night blindness - nil.
3. Haematuria - nil.
4. Pains in limbs - vide supra. Patient does not connect the painful limbs with V.D.

Diet History

Has been employed by same European for several years. Rations consist of dried beans, mealie meal, once a week given cabbage and lettuce.

Examination

General Nutritional State - thin, not emaciated.

Mental State - normal.

Gums - well marked scurvy gums. Bleed on slight pressure. Numerous buds.

Skin -

1. Hyperkeratosis - slight.

2/

2. Pellagra - nil.

3. Phyrnoderma - moderate.

Mucous Membranes - well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Eyes - nil.

3. Lips - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no abnormality, sounds pure. No enlargement.

B.P.- 108/75.

Lungs - no abnormality.

Abdomen - no abnormality. Liver and spleen not palpable.

C.N.S. - no abnormality. Reflexes normal. Sensation and co-ordination normal.

Locomotor System (including gait). Pain situated in right calf. There is a fullness of the affected area. No heat, induration or oedeme. No limitation of movement. Walks with right knee and ankle stiff, i.e. definite limp.

Remarks.

Penis - multiple soft sores. Prepuce indurated hypertrophic and secondarily infected.

Investigations

Blood on admission (and subsequently) -

Hb 80%, R.B.C. 4.1, W.B.C. 5,900

13.9.50/

13.9.50 Hb 88%, R.B.C. 4.6 M per c. mm.
W.B.C. 4,800 per c. mm.

28.8.50 W.R. Negative.

Urine - N.A.D.

Treatment

25.8.50 1. Penicillin 100,000 units soluble stat. and Procain Penicillin 1 cc. daily.

2. Sulphatriad tabs. 4 stat. 2 tabs. 2 x 4 hourly.

28.8.50 Vitamin C100 mgms. t.d.s.

Progress Notes (N.B. Date of disappearance of specific lesion)

28.8.50 Scurvy condition unchanged.

31.8.50 Gums now quite normal, pain in legs still present.

3.9.50 Pain in legs still present but much better. Limp less marked.

6.9.50 Pain has disappeared from legs. Gait now normal and is able to run.

8.9.50 Circumcision performed under spinal anaesthesia. No excessive haemorrhage observed at operation.

Date of Discharge

13th September, 1950. Discharged to V.D. section.

Condition

Gums normal. Gait normal and free of pain. Circumcision healing satisfactorily.

Comments and Conclusions

The presence of a multiple pathology is not uncommon in African practice, venereal disease is often an incidental finding. His diet included fresh vegetables but not meat. However deficient the ration may have been in protein and vitamins, he had been able to exist for several years. There was no anaemia, i.e., Hb 80% on admission. It is interesting that it rose to 88% on therapy. This patient responded unusually quickly, the gums being normal on the third day of treatment.

CASE NUMBER 4

Station	Que Que
Date of Admission	6th September, 1950.
Name	Mary
Age	Elderly female
Employment	Housewife

History

(a) Presenting symptoms and duration

Swollen, painful right leg of one month duration.

(b) Questioning

1. Mouth symptoms - bleeding for 1 month.
2. Night blindness - nil.
3. Haematuria - nil.

Diet History

Described as normal African food, viz., mealie meal, meat and sometimes vegetables. She buys spinach and cabbage, but is unable to say how often.

Examination

General Nutritional State - normal.

Mental State - dull and stupid but excessively cheerful.

Gums - spongy, purple and bleed profusely on pressure. One typical bud present.

Skin -

1. Hyperkeratosis - slight.
2. Pellagra - nil.
3. Phyrnoderma - nil.
4. Dyspnoea - nil.

Mucous/

Mucous/

Mucous Membrane - pale

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Eyes - nil.

3. Lips - nil.

4. Tongue - smooth, absence of normal papillae. Not sore.

C.V.S. - not enlarged, sounds pure.

Lungs - within normal limits.

Abdomen - liver enlarged (3 fingers), spleen enlarged (4 fingers). No ascites.

C.N.S. - within normal limits. No evidence of peripheral neuritis.

Locomotor System (Including gait) - cellulitis of right leg, (limb swollen, hot and tender from just below knee to dorsum of foot). Left ankle hot, swollen and tender.

Remarks

The inflmmatory condition of right leg is clinically a cellulitis and lacks the relatively localised induration of a myositis.

Investigations

Blood on admission (and subsequently) -

Hb 44%, R.B.C. 2.2 M per cmm., W.B.C. 2,400, P.M.Is 28% Lymph 64%, Ecs. 8%.

14.10.50 Hb 87%, R.B.C. 4.5 M per cmm. W.R. Negative.

Stool - hookworm ova present.

Treatment/

Treatment

6.9.50 Sulphatriad Tabs. 6 and Tabs. 2.4 hourly.

7.9.50 Vitamin C 100 mgm. T.D.S.

9.9.50 Penicillin 100,000 units soluble and 1 cc. procaine daily.

13.9.50 Stop penicillin and sulphatriad.

18.9.50 Hookwork treatment given.

Progress Notes

9.9.50 Gums definitely improved.

11.9.50 Leg improved. Less swollen and tense.

12.9.50 Gums much improved. Hardly bleed on firm pressure.

18.9.50 Leg much improved. Swelling less, only slight tenderness. Gums normal. Left ankle is now normal.

23.9.50 Is now complaining of a painful area about the size of half-crown at junction of distal third and proximal two-thirds of dorsum, left foot. It is sharply defined by tenderness. Skin distal to area is discoloured and coooler than rest of foot. Sensation over discoloured areas normal. Toes normal and not involved. Neither Dorsalis pedis nor anterior tibial arteries palpable.
Condition of right calf much improved. Still swollen, (right calf 13½" at broadest part and left calf 11½"). No longer hot, no oedema. Tenderness limited to small area of about 2" long and ½" broad, this area is situated at junction of upper and middle thirds of posterior aspect of calf. The muscles of the whole calf have an unusual wooden feel.

29.9.50 Patient is now able to walk, no change in physical signs.

5.10.50 Walks more easily but still limps. Measurements are unchanged. Right calf only very slightly tender on pressure. The skin over discoloured area has desquamated revealing normal skin. Pain is less and only felt when walking.

N.B./

N.B. - the mentality of this patient makes accurate recording of her symptoms difficult. This hypomaniacal state is very common in old African women.

16.10.50 Measurements and condition of leg unchanged. Limp barely perceptible. The skin over dorsum of left foot is quite normal. Patient is restive and requests permission to go home. Her wish is being granted as there is an acute bed shortage and she may abscond. On questioning she states that for some time before the right leg became painful, she had felt weak. She now claims robust health.

Date of Discharge

16th October, 1950.

She has been instructed to report in two weeks, but the likelihood of seeing her again is slight.

Condition on Discharge

See note of 16th October, 1950.

Comments

A definite scurvy occurring in a female. The leg on admission was infected, presumably a cellulitis superimposed upon an intra-muscular haemorrhage which has failed to absorb completely. Hence the persistence of the swelling after a dramatic initial improvement. Although her statements have been recorded they must be accepted with caution. Haemoglobin and red cells showed the usual improvement on vitamin C therapy., It is assumed that the enlargement of the abdominal viscera was either malarial or bilharzial (or both).

The importance of examining the mouth is well illustrated. The patient was first seen on a very busy afternoon and admitted with a diagnosis of cellulitis right leg. The associated scorbutic condition was not suspected until her gums were examined the following morning. I was tempted to do a biopsy, but it was not felt justifiable to submit the old woman to the discomfort of operation.

CASE NUMBER 5

Station	Que Que
Date of Admission	21st September, 1950.
Name	Kuleti
Age	Old Man
Employment	Mining

History

(a) Presenting symptoms and duration

Pain in right thigh and knee of one month's duration, (Pain present at rest).

(b) Questioning

1. Mouth symptoms - nil.
2. Night blindness - nil.
3. Haematuria - nil.

Diet History

In present employment 6 years. Provided with rations which consist of meat, mealie meal and dried beans. No green vegetables supplied. Has not eaten any green vegetables for about a year.

Examination

General Nutritional State - moderately thin but not emaciated.

Mental State - normal intelligence.

Gums - patient edentulous except for 4 lower molars and premolars in right lower jaw. Between teeth, gums are slightly hypertrophic with one definite bud. In toothless areas gums normal.

Skin -

1. Hyperkeratosis - moderate.
2. Pellagra - Typical Cassal's necklace with pellagrous rash of left forearm.

3/

3. Phyrnoderma - moderate.

Mucous Membranes - moderately well injected.

Clubbing - slight.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Eyes - nil.

3. Lips - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - Heart sounds poor quality, no enlargement and no murmurs.

B.P. - 135/70.

Lungs - move equally, no dullness, occasional rales at both bases.

Abdomen - nil abnormal. Liver and spleen not palpable.

C.N.S. - pupils react normally. Reflexes normal. Sensation and co-ordination normal.

Locomotor System (including gait) - the right thigh is swollen especially in the lower third. The muscles of the right thigh are hard and tender. Right knee - hot, tender on gentle pressure. Patella tap present. This patient is unable to walk and propels himself on his buttocks.

Investigations

Blood on Admission (and subsequently) -

Hb 67%, R.B.C. 3.3 M per cmm., W.B.C. 4,500.

5.10.50 Hb 77%, R.B.C. 3.8 M per cmm.

16.10.50 Hb 91%, R.B.C. 4.0 M per cmm.
Film - cells normocytic.

Treatment

22.9.50 Betalin 1 cc. intramuscular daily.

26.9.50 Betalin stopped. Vitamin C 100 mgms. T.D.S.

12.10.50 Nicotinic Acid 100 mgms T.D.S. Cod Liver Oil ½ oz. T.D.S.

Progress Notes (N.B. Date of disappearance of specific lesion)

26.9.50 No improvement on Betalin. Still unable to walk.

28.9.50 Pains improving, discomfort mainly in knee. Gums still unchanged.

30.9.50 Gums show definite improvement, bud disappearing. Legs less swollen and only slightly tender to touch. Knee still swollen but less tender. Patient is now able to walk.

2.10.50 About 40 cc. of blood withdrawn from right knee.

3.10.50 Scurvy bud has disappeared. Walking much better.

5.10.50 No tenderness in muscles of leg even on firm pressure. Swelling greatly reduced. Measurement of thigh taken 2" above patella - right 13½", left 13". Effusion has recurred into knee joint which however is only slightly tender.

12.10.50 Effusion now subsiding. Patient is able to walk at a smart trot. Pellagra unchanged.

16.10.50 Pellagra disappearing.

23.10.50 Pellagra has disappeared. No other B complex deficiency has become manifest. The hyperkeratosis and phyrnoderma remain unchanged. There is still a slight synovitis of right knee.

25.10.50 Still no change in hyperkeratosis or phyrnoderma. Slight synovitis persists, but there is no pain or limitation of movement. No swelling of thighs (measured 2" above patella - left 13", right 13"). He is now able to run.

Date/

Date of Discharge

26th October, 1950.

Condition on Discharge

See note of 25.10.50.

Conclusions and Comments

A severe case of scurvy. The importance of a careful examination of the gums is well illustrated, the diagnosis in this case was made from the condition of the mucous membranes between his four remaining teeth. The old clinical observation of edentulous gums being unaffected by the disease was confirmed. The exhibition of betalin had no effect on this patient, however, the very dramatic effect of ascorbic acid, in the writer's opinion, fully justifies a diagnosis of scurvy. The presence of a haemarthrosis of the knee joint may be taken as additional evidence. Although the true pellagra responded quickly to therapy (nicotinic acid started on 12.10.50 and skin rash disappeared on 23.10.50), the hyperkeratosis and phyrnoderma were unaffected by this treatment.

The usual response of the blood to Vitamin C was observed, the high colour index persisted but no abnormality of the red cells was detected in the blood films.

CASE NUMBER 6

Station — Que Que

Date of Admission — 29th September, 1950

Name — Mangochi

Age — Adult

Employment — Gold Mining.

History

(a) Presenting symptoms and duration

Ulcer right leg of six months duration (originally struck by a stone).

(b) Questioning

1. Mouth symptoms - gums bleeding for 1 year and teeth loose for two weeks.
2. Night blindness - nil.
3. Haematuria - nil.
4. Pains in limbs - has had pains in the muscles of both calves for one year.

Diet History

Has been employed two years at present mine. Rations from employer which consist of meat, mealie meal and monkey nuts. He can only remember two issues of green vegetables in two years, never buys green vegetables for himself.

Examination

General Nutritional State - emaciated.

Mental State - normal.

Gums - typical scurvy gums, bleed on slight pressure.

Skin -

1. Hyperkeratosis - yes (moderate).
2. Pellagra - nil.

3/

3. Phyrnoderma - yes (moderate).

Mucous Membranes - moderately well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no cardiac enlargement. Systolic murmur at all areas.

B.P. 118/70.

Lungs - no abnormality.

Abdomen - spleen palpable (2 fingers).

C.N.S.- pupils react normally. Reflexes all present and brisk. Sensation and co-ordination within normal limits.

Locomotor System (including gait) - slight tenderness of both calves. Right leg lacks about 15 degrees of full extension, left leg about 50 degrees. Walks with marked limp, both knees held stiff. Superficial ulcer lower third medial aspect right leg, about $3\frac{1}{2}$" and $2\frac{1}{2}$" at broadest part.

Investigations

Blood on admission (and subsequently) -

Hb 73%, R.B.C. 4.4M., W.B.C. 7,400 per c/mm., R.B.C. normocytic.

24.10.50. Hb 63%, R.B.C. 4.4 M.

30.10.50 Hb 71%, R.B.C. 5.1 M.
W.R. Negative.

Treatment/

Treatment

3.10.50 Vit. C 100 mgm. T.D.S.

31.10.50 Cod Liver oil ½ oz. T.D.S.
Yeast Tabs. 2 T.D.S.

Progress Notes (N.B. date of disappearance of specific lesion)

5.10.50 Scurvy Gums still present but only bleed slightly on vigorous pressure. Muscle tenderness diminishing.

11.10.50 Gums show improvement but not yet normal. No muscle tenderness. Ulcer healing. Limp still very pronounced.

16.10.50 Gums show further improvement, but some hypertrophy still present. Little change in gait.

23.10.50 No definite change in gums. Walks without a limp and can produce a stumbling trot when requested to run, the ulcer is healing well.

28.10.50 Bleeding tendency of gums is greatly reduced, but spongy appearance still present. Ulcer healing well.

Comments and Conclusions

Unfortunately transfer deprived the writer of the satisfaction of seeing this patient cured. Comment can be made on these points, viz.,

The complication of an ulcer with scurvy raises interesting speculation, whether avitaminosis C could be associated with the etiology of the ulcer. Although it is reasonable to suppose the tissue of scorbutic patients would be especially liable to break down it has not been borne out by this series. The patient's lesion was not typical of any particular kind of ulcer. Until the Negative Wasserman was received, I regarded the condition as probably specific. Reviewing this case at a later date, I realised the Wasserman should have been repeated. I refrained from the local custom of giving arsenic (whatever the etiology), because of the interference with metabolism produced by heavy metals (Hughes

Therapy did not produce the usual increase in the haemoglobin percentage. The drop which occurred is difficult to understand, and is too great to be explained by experimental error, as the estimations were made on the same apparatus by by the one observer (myself). The splenomegaly is not a very unusual/

unusual clinical finding in healthy Africans, and is generally ascribed to Chronic Malaria. It is of interest that the earlier writers described splenic enlargement as part of the syndrome, their cases however were probably also complicated by infection.

CASE NUMBER 7

Station	Que Que
Date of Admission	30th October, 1950
Name	Jonanu
Age	Adult
Employment	Gold Mining

History

(a) Presenting Symptoms and Duration

On 30th August right leg became swollen, this lasted a week and then disappeared to be followed by pain and swelling of the left leg which has persisted. Painful at rest. No history of trauma.

(b) Questioning

1. Mouth Symptoms - nil.
2. Night blindness - nil.
3. Haematuria - nil.

Diet History.

Rations supplied by employer, viz., meat, mealie meal, no vegetables issued and has not eaten any green food for last three months.

Examination

General Nutritional State - good.

Mental State - normal.

Gums - typical scurvy gums. Several buds present.

Skin -

1. Hyperkeratosis - slight.
2. Pellagra - nil.
3. Phyrnoderma - nil.

Mucous/

Mucous Membranes - well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other vitamin deficiency signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no enlargement, sounds pure.

B.P. - 170/75. Brachial arteries hard and tortuous.

Lungs - no abnormality.

Abdomen - liver palpable, one finger. Spleen not palpable.

C.N.S. - pupils react normally. Reflexes present, equal and brisk, including ankle jerks.

Locomotor System (including gait) - only able to stagger a few steps and suffers obvious pain in the process. Left leg is hot, swollen and tender from just below knee to ankle. Maximum tenderness at ankle joint. Movements of knee normal. Left ankle joint held in extension and movements limited by pain, no adenitis. Right leg painful on pressure over ankle but clinically limb appears quite normal.

Measurements - 4" from lower border of patella, left 14½", right 12"

8" from lower border of patella, left 11½", right 14"

Investigations

Blood on admission (and subsequently) -

Hb 63%, R.B.C. 3.1 M per cmm., W.B.C. 5,200

Treatment/

Treatment

31.10.50 Vitamin C 100 mgms. T.D.S.

Comments and Conclusions

This case of scurvy was only started on treatment the day before my tour of duty expired, therefore no observations as to progress can be recorded. His history of the right leg swelling and subsiding is difficult to account for, a possible explanation could be that when his left leg became affected he forgot about the former discomfort in the right limb. On examination the painful leg was definitely hot and clinically could have been mistaken for a cellulitis. He was apyrexial over the short time I was able to observe him and there was no leucocytosis. I have reason to believe that ascorbutic swellings are frequently confused with inflammatory conditions.

CASE NUMBER 8

Station — Bulawayo.

Date of Admission — 28th November, 1950.

Name — Elu

Age — Adult

Employment — Garden boy.

History

(a) Presenting Symptoms and Duration

1. Pain in right calf of three days duration, onset was slow. Pain only present when walking, not at rest.

(b) Questioning

1. Mouth symptoms - gums swollen for two weeks, gums do not bleed even when brushed.

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Rations supplied by employer, consist of meat, dried beans and mealie meal. He has not eaten any green food since August, 1949. (approximately 14 months).

Examination

General Nutritional State - moderate, not emaciated.

Mental State - dull

Gums - swollen and purplish, most marked scurvy gums seen to date, (only bleed on vigorous pressure).

Skin -

Hyperkeratosis - slight.

Pellagra - nil.

Phyrnoderma - marked on legs.

Mucous/

Mucous Membranes - well injected

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Scrotum - nil.

C.V.S. - sounds moderately well heard, no enlargement, no murmurs.

B.P. - 120/80

Lungs - no abnormality.

Abdomen - tip of spleen (one finger) palpable.

C.N.S. - no abnormality.

Locomotor System - tender area is about 2" square in middle third anterior aspect of right calf and is acutely tender. Affected area very sharply defined, no swelling or induration, slightly hot. No obvious limp, but gait appears stiff.

Investigations

Hb 105%, R.B.C. 4,900,000 per cmm., W.B.C. 9,400 per cmm.

W.R. (Repeated), Negative.

Treatment

1. Multivit. capsules, 1 T.D.S,
2. Vit. C 200 mgm. T.D.S.
3. Vit. C tabs. 6 T.D.S. and 100 mg. intramuscularly.

18.12.50. Cod liver oil ½ oz. T.D.S.

Progress/

Progress Notes

30.11.50 Slight improvement. Tenderness of legs less marked. No change in gums.

4.12.50 Pain has now disappeared from leg. Gait is quite normal. Gums unchanged.

5.12.50 No definite change.

7.12.50 No definite change.

9.12.50 No definite change.

11.12.50 I.S.Q.

12.12.50 Gums still show no change.

14.12.50 I.S.Q.

16.12.50 Gums are unchanged. Patient feels quite fit, possibility of wrong diagnosis must now be considered. Patient denies taking any native medicines prior to admission.

18.12.50 Gums unchanged. Phyrnoderma unchanged.

8.1.51. Gums unchanged. Since admission the phyrnoderma has become less prominent but still definitely present.

Patient discharged.

Comments and Conclusions

It is doubtful whether this case should be included in the series. When originally examined it was observed that although his gums showed considerable hypertrophy with a purplish discoloration, the usual haemorrhagic tendency was not present; moreover, the gum tissue was firmer than usually encountered in scurvy. It was remarked by a colleague that the buccal changes resembled those of epanutin intolerance. Unfortunately the patient denied taking any medicine before coming to hospital. In favour of an avitaminosis C, is the discomfort in the leg which cleared up with treatment very quickly. Early writers describe an inveterate or obstinate scurvy where the disease was irreversible. Lind (quoted from Hess) stated:-

> "I have met with numerous instances not only among the common seamen but of officers with whom it (scurvy) had taken such deep root in the constitution as to prove a lasting affliction to them during the greater part of their lives."

Harvey/

Harvey gives a graphic description of chronic cases.*

It was suggested that the gums could be explained by a fibrosis superimposed on an ascorbutic hypertrophy, however it is difficult to see why this process should occur in a mild case with two weeks history.

The Phyrnoderma was marked. Although there was some reduction during hospitalisation the lesions were still numerous on discharge. Cod Liver Oil did not produce any noticeable change.

* Quoted from Hess:-

> "Patients who have long gone untreated are molested with ambulative, distending, creeping, vellicating or lancinating pains of several parts of the body. They are often loose and subject to falling into violent fluxes of the belly, diarrhoea and lienteries."

CASE NUMBER 9

Station	Bulawayo
Date of Admission	15th December, 1950
Name	Kosamu
Age	Adult
Employment	Tailor's Assistant

History

(a) Presenting Symptoms and Duration

1. Pain in the muscles of both legs and both knees of four weeks duration.

2. Painful and bleeding gums, exact duration unknown.

(b) Questioning

1. Night blindness - nil.

2. Haematuria - nil.

Diet History

Mealie meal supplied by employer. Buys meat and sugar but never vegetables. Occasionally buys milk. Cannot remember when he last ate green food.

Examination

General Nutritional State - moderate.

Mental State - normal.

Gums - scurvy gums, slight hypertrophy and several typical buds present.

Skin -

1. Hyperkeratosis - nil.

2. Pellagra - nil.

3. Phyrnoderma - marked.

Mucous/

Mucous Membrane - well injected.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no enlargement, sounds pure.

B.P. - 105/55.

Lungs - no abnormality.

Abdomen - no abnormality, nil palpable.

C.N.S. - no abnormality. Reflexes (including ankle) present and brisk.

Locomotor System - (including gait), walks with a slight limp, knees held stiff. No abnormality of limbs detected on clinical examination, except tenderness over lower third of right femur.

Remarks - a mild case of scurvy. The minimal involvement of the limbs could be attributed to the "light employment in a tailor's shop".

Investigations

Blood on admission - Hb 78%.

Treatment

15.12.50 Aspirin gr. 10 T.D.S.

22.12.50 Vit. C 300 mgm. T.D.S.

28.12.50 Multivit capsules 2 T.D.S.

Progress/

Progress Notes

18.12.50 No change in gums, considerable improvement in legs; pain no longer present at rest and less marked on walking.

21.12.50 Patient feels better. Pain still experienced when walking, which he does with a noticeable limp. Some improvement can be observed in the gums.

23.12.50 Improvement maintained. Gums show slight reduction of scurvy appearance.

27.12.50 Patient now symptom free. He walks normally without discomfort and is able to run. The gums are normal.

29.12.50 Discharged on Multivitamin capsules with instructions to report in one week's time.

Comments and Conclusions

A mild case of scurvy which showed a very definite improvement on hospital diet and rest. This patient did not report back, although specifically requested and was promised a tip if he did so. This boy was in hospital during an excessively busy spell with an acute bed shortage; the writer was unable to give as much time to studying and recording this patient as would have been desirable.

CASE NUMBER 10

Station	Bulawayo
Date of Admission	15th December, 1950.
Name	Kasoudo
Age	Elderly Adult
Employment	Miner

History

(a) Presenting Symptoms and Duration

1. Pains in both legs (present at rest) of three weeks' duration.

2. Painful bleeding gums for last seven days.

(b) Questioning

Night blindness - nil.

Haematuria - nil.

Diet History

Rations from employer which consists of meat, mealie meal, beans and monkey nuts. No fresh food supplied in rations. He has not eaten any green food for one year.

Examination

General Nutritional State - thin but not emaciated.

Mental State - normal.

Gums - typical scurvy gums with a generalised hypertrophy and buds.

Skin -

1. Hyperkeratosis - moderate.

2. Pellagra - nil.

3. Phyrnoderma - nil.

Mucous/

Mucous Membranes - moderately well injected.

Clubbing - slight.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - slight atrophy of papillae.

5. Scrotum - nil.

C.V.S. - no cardiac enlargement. Sounds pure, no murmurs.

B.P. - 130/85.

Lungs - no abnormality.

Abdomen - no abnormality, nil palpable.

C.N.S. - no abnormality. Reflexes normal, ankle and knee jerks present. Sensation and co-ordination normal.

Locomotor System - patient can only walk with great difficulty and discomfort, limps with the knees held stiff, this abnormality is most obvious in left leg. Pain is experienced in the muscles of both calves and in a circumscribed area of the left foot, immediately proximal to the third metatarsal-phalangeal joint.

O/E Tender areas are sharply defined. The upper third of the left leg is swollen but not hot or indurated. The dorsum of the left foot is swollen but the signs of inflammation are absent.

Measurements taken -

(a) 6" below the lower border of patella,
Left 10 5/8", Right 8 2/8"

(b) 10" below lower border of patella,
left 8 1/8, Right 8 2/8

Investigations/

Investigations

Blood on admission Hb 90%

X-ray of lumbar spine shows osteoarthritic changes.

Treatment

22.12.50 Vit. C tabs. 6 (300 mgm) T.D.S. and 200 mgm. intramuscularly daily.

Progress Notes

20.12.50 Claims to have improved since admission. He is walking more easily. Condition of mouth unaltered.

21.12.50 General condition has deteriorated, he is now complaining of pain in the upper and outer quadrant of the left buttock, this area is hot, swollen and tender.

22.12.50 No improvement.

23.12.50 Patient states pain in buttock is better. Induration and tenderness of affected muscles much less marked. Gums unchanged.

27.12.50 Pain and tenderness improving.

28.12.50 Pain in muscles of legs and buttock have disappeared, now complains of severe discomfort in the lower vertebrae which he states has been present since admission and is now getting worse.

O/E Lower lumbar vertebrae tender and immobile. ? Osteoarthritis which has been aggravated by lying on stone floor. Gait and gums almost normal, walking no longer causes discomfort.

2.1.51 Condition improving. Pain in back very slight, he now walks without a limp. Gums are normal. To be discharged on Multivit tabs. and return in one week's time.

Comments and Conclusions

This patient shows some interesting points. Although crippled with the disease he had no anaemia. In this case, rest and the hospital diet were not curative, in fact made him worse. The involvement of the gluteal muscles which developed during the observation period, was probably caused by fresh haemorrhage in that area. The pain in the back is less easily/

easily explained, considering it developed when the original signs and symptoms were abating, and in view of the X-Ray findings a diagnosis of osteoarthritis seems reasonable. The instructions to report back were not observed.

CASE NUMBER 11

Station	Bulawayo
Date of Admission	20th December, 1950
Name	Chimpandu
Age	Adult
Employment	Labourer

History

(a) Presenting symptoms and Duration

1. Pain in legs for two weeks, pain is present at rest.

2. Pain in chest for 4 days.

(b) Questioning

1. Mouth symptoms - one tooth loose for two weeks, has noticed nothing else.

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Provided with rations by employer, consist of meat, mealie meal, monkey nuts and beans. Twice weekly he is given an onion and a third share in a cabbage. He says he eats his vegetable ration. Patient denies drinking beer. He has been living on this diet for the last two years.

Examination

Patient acutely ill, T.103.

General Nutritional state - emaciated.

Mental state - lethargic.

Gums - typical scurvy gums, viz., generalised hypertrophy and several buds present. The gums bleed on slight pressure.

Skin -

1. Hyperkeratosis - very marked.

2/

2. Pellagra - nil.

3. Phyrnaderma - marked.

Mucous membranes - well injected.

Clubbing - nil.

Oedema - nil.

Dyspnoea - present at complete rest.

Other Vitamin Deficiency Signs -

1. Face - Seborrhoea folliculitis.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no enlargement, sounds pure, no murmurs.

B.P. - 112/60.

Lungs - no abnormality, no signs of pneumonia.

Abdomen - no abnormality, liver and spleen not palpable.

C.N.S. - pupils unequal but react normally to light and accommodation. Reflexes present and brisk, (including ankle jerk). Sensation and co-ordination within normal limits.

Locomotor System - pain is situated in both legs. In the left both calf and hamstring muscles are involved, in the right leg only the calf muscles are affected. Patient is too ill to walk.

O/E Left calf muscles are hot and indurated, the whole of right thigh is swollen. The right leg shows no abnormality.

Measurements taken 8" above upper border of patella - right thigh 13", left thigh 16".

Investigations

Blood on admission (and subsequently),

20.12.50 Hb 65% R.B.C. 3,070,000 per cmm.
W.B.C. 3,900 per cmm.
P.M.L.s 43%
Lymphs 57%

3.1.51 Hb 70%, R.B.C. 3,770,000 per cmm.

22.1.51 Hb 80%, R.B.C. 3,710,000 per cmm., W.B.C. 4,050 per cmm.

29.1.51 Hb 90%, R.B.C. 4,450,000 per cmm., W.B.C. 7,150 per cmm., P.M.L.s 45%, Lymphs 41% Monocytes 2% Eosinophiles 6%

X-ray chest (24.12.50):- Normal heart shadow, no pulmonary disease.

Treatment

20.12.50 Vit. C 100 mgm. T.D.S., this therapy was ordered by a colleague.

21.12.50 Ascorbic acid increased to 900 mgm. daily by mouth and 200 mgm. intramuscularly daily.

9.1.51 Multivit capsules tabs. 2 T.D.S.

Progress Notes

21.12.50 Patient states he feels a little better, temperature has fallen from 103 degrees to 99 degrees.

23.12.50 Patient is feeling better, the discomfort in the legs is diminishing. He is still unable to walk and moves about on his buttocks. Some improvement of gums. There is a reduction in the induration of the left thigh muscles. Patient is now fit to go to X-Ray.

27.12.50 Pain still present but less. He is now able to walk, gums show definite improvement, buds less marked and bleeding tendency reduced. Patient has been a-pyrexial since 24.12.50.

20.12.50 He is now able to stand. Clinical condition otherwise unchanged.

2.1.51 Is now walking much more strongly although still limps with left leg held stiff. Gums almost completely healed. No definite improvement in skin condition.

4.1.51/

4.1.5. Improvement maintained. He now walks briskly, but limp still present.

O/E There is loss of about 20% extension in the left knee joint, which is no longer swollen, hot or tender. There is a complete disappearance of the induration which was formally present over the insertion of the left ham-strings. Gums now normal. Skin condition unchanged.

7.1.5. Gait has improved considerably over last three days, and is now almost normal. The loss of extension at the left knee joint is now only about 5%.

9.1.51 Patient can walk normally, and is able to run. No change in left knee joint. Skin remains unchanged.

10.1.51 Limitation of movement in left knee joint very slight.

12.1.51 I.S.Q.

14.1.51 General condition unchanged, his skin has not shown any improvement since admission.

17.1.51 I.S.Q.

19.1.51 For the first time an improvement can be seen in the skin lesions, the phyrnoderma is disappearing

20.1.51 I.S.Q.

23.1.51 Phyrnoderma has receded since last observed, the hyperkeratosis of the skin is, however, more noticeable today.

25.1.51 Phyrnoderma has nearly gone. Small areas of black discoloration are replacing the waning follicules.

27.1.51 The general appearance of the skin now shows a noticeable improvement. The rough scaliness has gone, and the skin now looks glossy. There is still the same slight degree of limitation of extension in the left knee.

28.1.51 No change.

29.1.51 The hyperkeratosis of the legs has entirely disappeared, and is only faintly present on the arms. Patient is asking for his discharge.

31.1.51/

31.1.51 No further change in skin or knee.

Date of Discharge

31st January, 1951.

Comments and Conclusions

An interesting case showing several unusual features. Like several other scurvies his diet was not devoid of anti-scorbutics. He was emphatic that he ate his vegetables, but he had no idea how his food was cooked.

When first seen he was clinically suggestive of pneumonia, and the diagnosis of scurvy was only made when his mouth was looked into as part of the ritual of routine examination. The possibility that he was pneumonia complicated by avitaminosis C cannot be ruled out. Points against are briefly:-

1) Absence of chest signs

2) Low leucocyte count with a relative lymphocytosis.

3) The rapid response to ascorbic acid (no antibiotics given).

4) Four days after admission his chest was clear radiologically.

Gelfand mentions the tendency for scurvy to develop in the course of an infection; however, the history of painful limbs preceded the onset of pain in the chest by 10 days.

Lind stressed pain in the breast and dyspnoea as common and dangerous symptoms. In the experimental scurvy produced in the recent investigation of the Medical Research Council, two volunteers developed acute cardiac conditions. This patient's general condition and his pyrexia (103, the highest see in any case of scurvy) suggested a lung rather than a heart upset. The possibility of a cardiac lesion cannot be definitely excluded.

This patient's skin condition will receive fuller consideration elsewhere. In addition to the gross hyper-keratosis he had a definite follicular seborrhoea and a marked phyrnoderma. In my opinion he should be classified as a multi-vitamin deficiency state.

CASE NUMBER 12

Station	Bulawayo
Admitted	27th December, 1950
Name	Benard
Age	Young Adult
Employment	Builder boy, his work consists mainly of handing nails to a European carpenter.

History

(a) Presenting symptoms and duration

1. Bleeding gums of two weeks duration.

2. Pain in the legs for about six days, (mouth symptoms started about one week before the legs).

(b) Questioning

1. Mouth symptoms - see above. Teeth do not feel loose.

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Rations from employer, which consist of meat and mealie meal only. He does not receive any of the common foods such as beans, monkey nuts, green vegetables or fruit. This boy claims not to have eaten any green food since 1949, viz., for at least a year.

Examination

General Nutritional State - moderate.

Mental State - normal.

Gums - Typical scurvy gums. There is general hypertrophy with numerous buds. Bleeding occurs on slight pressure.

Skin -

1. Hyperkeratosis - nil.

2. Pellagra - nil.

3. Phyrnoderma - moderate.

Mucous Membrane - well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no enlargement, sounds pure.

B.P. - 130/65.

Lungs - no abnormality.

Abdomen - no abnormality.

C.N.S. - pupils react normally, reflexes present and brisk. Sensation and co-ordination normal.

Locomotor System - pain is situated in the muscles of both calves. Slight tenderness on pressure, otherwise nil abnormal clinically. He walks stiffly without a definite limp.

Remarks - mouth symptoms unusually prominent as compared with the very slight locomotor involvement.

Investigations

Blood Hb 87%, R.B.C. 45 M., W.B.C. 8,650.

Treatment

29.12.50 * Permidine tabs. 2 T.D.S. (Each tablet = .5 gm. of HESPERIDIN).

5. 1.51 Stop Permidine. Start Vitamin C 300 mg. T.D.S. and 100 mgm. intramuscularly daily.

6. 1.51 Multivit capsules 2 T.D.S.

Progress Notes

29.12.50 Pain in legs less severe.

1. 1.51 Legs improved. Gums unchanged.

2. 1.51 No pain on walking, no tenderness elicited on pressure over former tender areas. Gums show a slight improvement, the bleeding tendency especially is reduced.

3. 1.51 Gums show a definite improvement.

5. 1.51 Healing process in the mouth continues. This case has made a very definite response to rest, the hospital diet and Vitamin P.

7. 1.51 Gums still improving.

9. 1.51 Mouth almost completely healed. The patient is agitating for his discharge.

12. 1.51 Gums now normal. Skin condition unchanged.

Date of Discharge

12th January, 1951.

* Vitamin P tablets supplied by Glaxo.

Condition on Discharge

See progress note of 12th January, 1951.

Comments and Conclusions

A mild case of scurvy in which the mouth symptoms were unusually prominent in comparison to the involvement of the locomotor system. A possible explanation is the easy nature of his employment. This patient showed an apparent response to vitamin P; however in view of experience elsewhere the diet of the hospital may have been responsible. Although a definite scurvy this patient had no anaemia. On discharge this boy was symptom free and in apparent good health but the phyrnoderma was unchanged.

CASE NUMBER 13

Station	Bulawayo
Date of Admission	29th December, 1950
Name	Size
Age	Young Adult
Employment	Road Labourer

History

(a) Presenting symptoms and duration

Pain in right leg of long duration (cannot remember time of onset) which is sometimes present at rest.

(b) Questioning

1. Mouth symptoms - nil.

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Buys own food which consists of mealie meal, bread and meat. He occasionally buys vegetables but cannot remember when he last did so, nor when he ate fresh food. Drinks beer twice a week.

Examination

General Nutritional State - good.

Mental State - very dull.

Gums - a slight generalised hypertrophy, several small buds present. Bleeding occurs on moderate pressure.

Skin -

1. Hyperkeratosis - nil.

2. Pellagra - nil.

3. Phyrnoderma - slight on legs.

Mucous Membranes - well injected.

Clubbing - nil.

Jaundice - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no cardiac enlargement, no murmurs detected.

B.P. - 120/70

Lungs - no abnormality.

Abdomen - no abnormality.

C.N.S. - Pupils react sluggishly to light and to accommodation.

Reflexes -	Right	Left
Biceps	++	++
Triceps	++	++
Knee	+	++
Ankle	+	++
Plantar	Flexor	Flexor

There is loss of fine sensation below right knee, but coarse touch and pain unimpaired. Muscular power impossible to estimate because of pain.

Locomotor System (including gait) - pain is situated in middle third of right tibia, affected area is swollen and acutely tender. He also complains of pain in the right ankle which is normal on clinical examination. The patient walks with an obvious limp, the right leg causing him acute discomfort.

Measurements - 7" below patella, right 12½", left 12 1/8"

Remarks - the scurvy appears to co-exist with some lesion of the C.N.S.

Investigation

Blood on admission Hb 102%, R.B.C. 5.3 m., W.B.C. 6,050

P.M.Ls 28%

Lymphocytes 52%

Monocytes 5%

Eosinophiles 15%

W.R. Negative

Treatment

Permidine tabs. 2 T.D.S.

8. 1.51 Stop Permidine, R. vit. C 300 Mgm. T.D.S. and 100 mgm. intramuscularly daily.

24.1.51 Thiamine i cc. intramuscularly daily.

Progress Notes

31.12.50 Condition unchanged.

3. 1.51 Slight improvement of gums, otherwise condition unchanged.

5. 1.51 Further improvement in gums, limp still present. Pain is stated to be less.

7. 1.51 Pain still improving.

O/E No change in gums. The swelling of the right leg has decreased in size, with a reduction in heat and tenderness, limp still present but less marked.

8. 1.51 Patient states, "he feels better". No change in neurological signs.

9. 1.51 Limp improving. No obvious change on examination.

10. 1.51 Gums show a definite change. A further reduction can be seen in the swelling of the leg.

12. 1.51 Walking much better. Gums now normal.

14. 1.51 Still complains of pain in right leg, however, walks without a limp and is able to run.

15. 1.51 Swelling has disappeared from right leg. Fine sensation has returned to the previously affected area, but ankle and knee jerks unchanged viz., still diminished.

16. 1.51 I.S.Q.

17. 1.51 No complaints. Muscular power in both legs equal, condition of reflexes unchanged.

19. 1.51 Definite improvement in the reflexes of the right limb. Ankle and knee jerks now only slightly diminished.

20. 1.51 Legs measured 7" below patella (from lower Border), right $12\frac{1}{4}$", left $12\frac{1}{8}$".

22. 1.51 I.S.Q.

23. 1.51 Neither ankle jerk could be elicited today.

27. 1.51 Knee jerks equal, both ankle reflexes still absent.

28. 1.51 Patient absconded.

Comments and Conclusions

This case, a scurvy of moderate severity, shows several interesting features. All the points raised, either have been discussed in earlier cases or have been reserved for fuller consideration in the general summing up. These problems may be briefly recorded:-

1. No anaemia, the haemoglobin being above the African average.

2. The neurological disturbance.

3. The affected limb improved in the usual manner, but some swelling persisted. As has been experienced with similar cases the reduction is more obvious to the naked eye than with the measuring tape.

CASE NUMBER 14

Station	Bulawayo
Date of Admission	1st January, 1951
Name	Yosefe
Age	Young Adult
Employment	Labourer

History

(a) Presenting symptoms and duration

Pain in the right leg of two weeks duration (only present on walking).

(b) Questioning

1. Mouth symptoms - gums bleeding one week, teeth not loose.
2. Haematuria - nil.
3. Night blindness - nil.

Diet History

Rations from employer, which consist of beans, meat and mealie meal. Has not eaten any fresh food since February, 1950. Denies drinking beer.

Examination

General Nutritional State - good.

Mental State - normal.

Gums - typical scurvy gums, several buds present.

Skin -

1. Hyperkeratosis - nil.

2. Pellagra - nil.

3. Phyrnoderma - moderate severity.

Mucous Membranes - well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Lips - nil.

2. Eyes - nil.

3. Face - Follicular seborrhoea.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no cardiac enlargement, no murmurs, B.P. 130/72,

Lungs - no abnormality.

Abdomen - no abnormality.

C.N.S. - no abnormality.

Locomotor system - pain is situated in the muscles of the medial aspect of the lower third of right thigh. Affected area is tender and indurated but not hot.

Measurements -	Right	Left
At upper border of Patella	14 5/8"	13 5/8"

Walks with a definite limp, the right leg held stiff.

Investigations

W.R. - Negative

Blood — Hb 77%, R.B.C. 4,850,000 per cmm.
P.M.L.'s 40%
Lymphs 58%
Eosin 2%
W.B.C. 3,300 per cmm.

30. 1.51 — Hb 100%,

R.B.C. 4,850,000 per cmm.
P.M.L's 37%
Lymphs 58%
Monocytes 2%
Eosinophiles 2%

W.B.C. 8,550 per cmm.

Treatment

2. 1.51 — Permidine Tabs. 2 T.D.S.

7. 1.51 — Vit. C Tabs. 6 (300) T.D.S. and 100 mgm. intramuscularly daily. Stop Permidine.

15. 1.51 — Stop I.M. vitamin C.

27. 1.51 — Multivit capsules 2 T.D.S.

Progress Notes

3. 1.51 — No change.

5. 1.51 — Gums show slight improvement, otherwise I.S.Q.

7. 1.51 — Condition has deteriorated. The area of tender induration which originally only involved the muscles of the medial aspect of the lower third of right thigh has spread to the calf muscles of the same leg. On admission although limping he could move about quite easily, now he can only walk with the greatest difficulty and discomfort.

In other words he is crippled. No further improvement to be seen in the gums.

8. 1.51 No definite change in the induration of the leg. This patient has been running a continuous pyrexia (of about 100 degrees) since admission.

9. 1.51 Patient states leg is feeling better.

O/E Area of induration is much softer, tenderness much less. Gait shows considerable improvement. Mouth remains unaltered.

10. 1.51 No definite change in gums. Leg much improved.

12. 1.51 Induration still present but no longer tender. Oral manifestations are slightly improved.

14. 1.51 Induration decreasing. Patient states he has had no pain since yesterday. Gums definitely better. He is still walking very badly.

15. 1.51 Gums show further improvement. Induration has almost disappeared. Limp still very noticeable but said to be painless. The range of movement of the right knee joint has increased, it now lacks about 5 degrees of full extension.

17. 1.51 Mouth almost normal. Gait shows great improvement. Induration unchanged.

19. 1.51 Gums now normal. Limp very slight. Induration unchanged.

20. 1.51 Walks normally and is able to run. Indurated area still present. Measurements taken at upper border of patella - right 13 7/8", left 13 1/2".

22. 1.51 A further reduction in induration. Right knee is capable of full range of painless movement.

23. 1.51 No change.

25. 1.51 Induration has disappeared from calf muscles, but is still palpable in the popliteal fossa in a

roughly triangular area of about 4" above the flexure of the knee. Phyrnoderma still present but shows a real improvement.

27. 1.51 No changes in induration.

29. 1.51 Condition of right leg unaltered since 25.1.51. Phyrnoderma folicules are now much less numerous and less obvious. Patient is asking for discharge and is anxious about losing his employment.

31. 1.51 Further reduction in phyrnoderma. Induration unchanged.

Comments and Conclusions

A case of scurvy of moderate severity, who was not crippled when first seen. This case did not respond to the hospital diet or Vitamin P. In fact the condition deteriorated rapidly making him well nigh bed-ridden. These therapeutic measures were either impotent or insufficient to stay the progress of the malady. At the end of 5 days treatment with Vitamin P, large doses of Vitamin C were substituted and a very good response was seen within forty-eight hours, and he was symptom free in less than two weeks. The signs in the right leg although responding to treatment lingered on for over three weeks and were detectable on discharge. The circumference had decreased by 3/4". I would have liked to have kept this boy under observation until the induration had either disappeared or become permanent. Unfortunately I felt it was unfair to subject him to continuous financial worry and perhaps even loss of his employment, moreover the possibility of absconding was very real and the bed shortage pressing.

CASE NUMBER 15

Station	Bulawayo
Date of Admission	5th January, 1951.
Name	Siambaba
Age	Young Adult
Employment	Steel Worker

History

(a) Presenting symptoms and duration

Pain in the right knee and right calf of one week's duration. Pain present at rest.

(b) Questioning

1. Mouth symptoms - gums slightly painful. Has not noticed any bleeding.
2. Night blindness - nil.
3. Haematuria - nil.

Diet History

He is only given mealie meal by his employer. Buys meat only. Never buys any vegetables, milk or fruit. Last ate vegetables in February, 1950. Does not drink beer.

Examination

General nutritional state - moderate.

Mental state - normal.

Gums - purplish, several buds present. Bleed profusely on moderate pressure.

Skin -

1. Hyperkeratosis - nil.

2. Pellagra - nil.

3. Phyrnoderma - moderate.

Mucous Membranes - moderately well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Other Vitamin Deficiency signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - nil.

5. Scrotum - nil.

C.V.S. - no enlargement, no murmurs heard.

Lungs - no abnormality.

Abdomen - no abnormality.

C.N.S. - pupils react normally to light and accommodation. Reflexes present and equal (including the ankle jerks).

Locomotor System (including gait) - Walks with considerable difficulty. Knee flexed. Pain only felt in Right leg.

O/E Knee held in flexion. Knee joint and upper two thirds of right leg are swollen but not hot and only slightly tender. Tenderest area being at the insertion of the right hamstrings.

Measurements -	Right	Left
Knee joint at upper border of patella	17"	14½"
6" from lower border of patella	14"	12½"

Investigations

Blood on admission (and subsequently) -

No Malarial parasites.

Hb 70%, R.B.C. 3,520,000 per cmm., W.B.C. 6,880.

Hb 88%, R.B.C. 4,400,000 per cmm., W.B.C. 6,850.
P 52%, L 48%
W.R. Negative.

Treatment

15. 2.51 Vit. C tabs. 300 mg. T.D.S. and Vit. C 100 mgm. 1 m. daily.

Progress notes (N.B. date of disappearance of specific lesion)

6. 1.51 Gums unchanged. Patient states pains in legs improving.

7. 1.51 Gums unchanged. Pain improving. Today the affected area is hot (vide condition on admission). No change in gait.

8. 1.51 No change in gums. Patient claims to be improving.

O/E No change in leg or gait.

9. 1.51 Gums show slight improvement. Pain in leg has decreased and is now able to move the right knee more easily.

O/E Tenderness less, and affected area feels softer. No definite improvement in gait.

10. 1.51 No change in gums. Induration improving. Slight improvement in gait.

17. 1.51 Gums unchanged. Induration of left leg improving, now has increased range of movement of left knee and is walking more easily.

13. 1.51 No change in condition.

15. 1.51 Slight improvement in gums.

15. 1.51 Patient states that leg is much better. Pain now limited to right knee joint.

O/E Very definite softening of induration of calf muscles, but induration unchanged in popliteal fossa. Gait shows slight improvement. Since admission on 5.1.51 patient has shown an unmistakeable improvement on rest and hospital diet.

16. 1.51 Very definite improvement in gums. Reduction of induration of calf muscles continues. No change in gait. Phyrnoderma still marked.

17. 1.51 Further improvement in gums. Patient volunteered the information that his leg was now soft.

O/E Reduction in the swelling of the calf muscles and further reduction in the induration of same. Today for the first time a reduction in the induration of the popliteal fossa was observed.

19. 1.51 Gums normal. Pain only experienced when walking.

O/E Obvious reduction in the swelling of the right leg. No definite change in induration and no improvement in gait.

20. 1.51 I.S.Q.

21. 1.51 Paracentesis of right knee unsuccessful.

22. 1.51 No change in condition.

23. 1.51 No improvement to be seen in the right leg. The knee is still held in flexion and the range of movement is approximately from 100 degrees of extension to 70 degrees flexion. He is not walking any better.

24. 1.51 I.S.Q.

25. 1.51 A definite improvement today, the movement of the right knee has improved by about 20 degrees extension. (Flexion is still about 70 degrees). The swelling and induration are unchanged. His gait has shown only a very slight improvement since admission, however he says walking is less painful. The phyrnoderma has not altered since admission.

27. 2.51 Improvement continues. He is walking better and extension has increased by 5 degrees.

Measurements -	Right	Left
At upper border of patella	15.3/4"	14.1/4"
6" from lower border of patella	12.3/4"	12½"

2. 3.51 No alteration in limp or induration. Extension improved by another 5 degrees. Phyrnoderma unchanged.

3. 3.51 I.S.Q.

5. 3.51 Very definite improvement in gait. He is now bending the right knee, previously he has always walked with the joint stiff. Patient is now capable of about 145 degrees extension. The induration is unaltered.

6. 3.51 Improvement sustained. The limp is less obvious. Another 5 degrees added to extension. There is a slight reduction in the induration of the right calf muscles, the thigh is as before. The phyrnoderma remains unchanged.

8. 3,51 A possible improvement in extension otherwise I.S.Q.

12. 3.51 A general improvement today, the right knee only lacks about 10 degrees of full extension. He is walking much better although a limp is still present.

O/E The right knee is still swollen, this would appear to be partly effusion and partly periarticular.

13. 3.51 The limp can now be described as slight. Extension is still short by about 10 degrees. The Phyrnoderma is unaltered.

16. 3.51 The gait is almost normal, the limp is very slight, and requires careful observation to be detected.

N.B. The writer was transferred to an outstation today to relieve a Medical Officer injured in a motor accident. Only one more chance of examining the patient presented itself 6 days later.

22. 3.51 Gait as recorded on 16. 3.51. The right knee still swollen, but is now capable of 180 degrees extension. There is some reduction in the phyrnoderma, which is however still definitely present. Patient is still in hospital and receiving treatment.

Comments and Conclusions

Many of the features of this patient have already been commented upon in earlier cases, e.g. the anaemia and the improvement without treatment. The main interest lies in the crippling severity of the leg lesions, which after seven weeks in hospital were greatly improved but not cured. Exigencies of the service prevented further observation, but in the writer's opinion, the swelling would have persisted and the right knee will suffer permanent disability. The phyrnoderma only improved after 6 weeks on large doses of Vitamin C. The significance of this interesting skin condition will be discussed elsewhere.

CASE NUMBER 16

Station	Bulawayo
Date of Admission	12th January, 1951
Name	Million
Age	Young Adult
Employment	In Cement Factory

History

(a) Presenting symptoms and duration

Pain in both legs of two weeks duration, this pain is present at rest.

(b) Questioning

1. Mouth Symptoms - for last two weeks, gums painful, teeth not loose, has not noticed any bleeding.

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Buys own food, which consists of meat, mealie meal and bread. He ate two oranges last month and can recall having oranges in September (patient admitted in January). He never buys vegetables and is emphatic that he is a teetotaller on religious grounds.

Examination

General Nutritional State - moderate.

Mental State - normal

Gums - typical scurvy gums of moderate severity.

Several buds present. Profuse bleeding occurs on slight pressure.

Skin

1. Hyperkeratosis - nil.

2. Pellagra - nil.

3. Phyrnoderma - marked on legs.

Mucous Membrane - very pale.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.

2. Lips - nil.

3. Eyes - nil.

4. Tongue - smooth and atrophic.

5. Scrotum - nil.

C.V.S. - no enlargement, no murmurs

B.P. - 138/55.

Lungs - no abnormality.

Abdomen - Tip of spleen palpable.

C.N.S. - pupils react normally to light and accommodation.

Reflexes -	Right	Left
Biceps	+	+
Triceps	+	+
Knee	Nil (Painful area)	++
Ankle	+	++

Sensation and co-ordination within normal limits.

Locomotor System, (including gait) - patient only able to stagger a few steps with greatest difficulty. Pain is felt in right knee joint and upper third of right calf muscles, also at the insertion of the left hamstrings.

O/E Right knee joint and lower third of thigh hot, swollen and tender, no induration present. Patella tap elicited in right knee joint. Apart from pain at the insertion of the hamstrings, no clinical abnormality could be found in the left leg.

Measurements -	Right	Left
At mid point of Patella	15"	14½"
2" above upper border of Patella	15.2/8"	14.7/8"

Investigations

Blood On admission (and subsequently) -

13. 1.51 Hb 36%, R.B.C. 2.4 M per cmm., W.B.C. 4,150 per cmm.

Differential Count PML's - 40%
Lymph - 58%
Eosinophils - 2%

12. 2.51 Hb 85%, R.B.C. 4.3 M per cmm.

26. 2.51 Hb 96%, R.B.C. 4.9 M per cmm., W.B.C. 7,050 per cmm.

W.R. Negative.

TREATMENT

12. 1.51 1. Vitamin C 300 mgm. T.D.S.
2. Vitamin C 100 mgm. intramuscularly daily.

16. 2.51 Multivit capsules 2 T.D.S.

Progress Notes

13. 1.51 No change.

15. 1.51 Patient states pain is less. No change in clinical condition.

17. 1.51 Pain in legs is improving. Gums feel more comfortable and do not bleed.

O/E Slight improvement in gums, no change in legs or gait.

19. 1.51 Pain improving.

O/E Cannot walk any better. Tenderness of painful areas less marked. Gums show definite improvement.

20. 1.51 Pain is now only experienced when walking.

O/E Gums much better, buds have almost disappeared. Gait is still grossly abnormal but moves more easily and says walking is much less painful. No obvious change in the condition of the legs.

23. 1.51 Gums nearly normal, the buds have disappeared and the bleeding tendency reduced. The heat and tenderness in the right knee are much improved. The swelling is still present and has changed in character. The patella tap has disappeared and there is now an increase in the periarticular tissue which was not present before. Patient still hobbles.

25. 1.51 Mouth almost normal, however the abnormal purple colour still persists, only bleed on vigorous pressure. No change in Phyrnoderma. Very definite improvement in gait.

28. 1.51 Gums now quite normal. Gait improving. No change in the legs.

29. 1.51 No change.

31. 1.51 Patient states pain has disappeared from the knee, but the swelling interferes with walking.

O/E No change in the character of the swelling. There is considerable limitation of movement. Patient can only flex right knee to a right angle and lacks about 20 degrees of full extension. Pain is felt at extremes of both movements. Patient now walks with a slight limp.

2. 2.51 40cc. of blood withdrawn from right knee.

5. 2.51 Right knee shows some reduction in swelling. Pain still present in movement. Gait improving.

7. 2.51 Slight reduction in swelling of right knee. Pain still felt at limit of flexion. No improvement in flexion but only lacks about 10 degrees of full extension. The right knee joint no longer feels hot. Patient can now walk normally.

9. 2.51 Very definite improvement. The swelling of the soft tissue of the right knee is smaller. He now only lacks about 5 degrees extension and 20 degrees flexion.

10. 2.51 Improvement continues.

12. 2.51 No further change in size of soft tissue swelling of right knee, now only lacks about 10 degrees of full flexion and about 5 degrees of full extension.

13. 2.51 Now has full flexion at knee (although this is painful). Extension unchanged.

15. 2.51 I.S.Q.

16. 2.51 No change in size of soft tissue swelling. Extension still lacks about 5 degrees. Phyrnoderma has improved since admission, but numerous follicules still present on both legs.

17. 2.51 Condition unchanged. When this patient is specifically requested to walk, he does so normally. However, it has been noticed that at other times he walks with a definite limp. On questioning he admits to having a recurrence of pain if he walks fast or for any distance.

20. 2.51 Soft tissue swelling persists and there is no change in the condition of the right leg.

22. 2.51 Patient states that he is now entirely free of pain even after walking a considerable distance or running, the soft tissue swelling of the right leg remains unaltered. He still has a very slight limitation of extension of the right knee.

24. 2.51 No further improvement has been observed in the phyrnoderma. For some time now the patient has been agitating for discharge.

25. 2.51 I.S.Q.

27. 2.51 Condition static.

Measurements -	Right	Left
(a) Mid point of patella	14½"	14.1/8"
(b) 2" above upper border of patella	14½"	15"

2. 3.51 Condition of right knee unchanged. Since admission there has been a definite reduction in the phyrnoderma which however is still present and has shown no further improvement over the last two weeks.

Date of Discharge

2nd March, 1951.

Condition on Discharge

Patient claims to be in excellent health, and fit for his work. The swelling of the right knee has not changed over the last three weeks. The inequality between the knee joints is much more striking to the eye than the slight discrepancy revealed by the measuring tape would suggest. The slight abnormality of gait referred to in the progress note of 17.2.51 is still noticeable.

Comments and Conclusions

This patient showed several interesting features. Although he was crippled by the disease, the mouth changes were only of a moderate severity and no worse than sometimes seen in ambulant cases.

The anaemia was severe (36% Sahli), the response to Vitamin C can be described as dramatic and occurred without any other haematinic drugs.

The soft tissue swelling after an initial decrease became static in size. A possible explanation is, the original scorbutic haemorrhage into the tissue became organised. The haemarthrosis is interesting as a classical sign of the disease and has been encountered before in this series.

The behaviour of the phyrnoderma is worthy of note but difficult of explanation. A definite improvement did occur but was not noticeable until after 5 weeks treatment, and did not improve further during the last 16 days in hospital, in spite of the exhibition of multivitamin capsules. Phyrnoderma is discussed more fully elsewhere.

CASE NUMBER 17

Station — Bulawayo

Date of Admission — 12th January, 1951.

Name — John

Employment — Labourer (Porter's Cement)

Age — Young Adult

History

(a) Presenting symptoms and Duration

Pain in the right leg of three weeks duration, (this pain is present at rest).

(b) Questioning

1. Mouth symptoms - gums painful, (vague as to exact duration).

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Buys own food, viz., mealie meal, meat, nuts and beans. He does not buy vegetables or fruit. He cannot remember when he last ate any antiscorbutic food. Drinks beer occasionally.

Examination

General Nutritional State - moderate.

Mental State - normal.

Gums - slightly hypertrophic and purplish, several small buds present. Bleeding occurs on light pressure, which also causes pain.

Skin

1. Hyperkeratosis - slight.
2. Pellagra - nil.
3. Phyrnoderma - moderate on legs.

Mucous Membrane - well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Dyspnoea - Present on slight exertion.

Other Vitamin Deficiency Signs -

1. Face - nil.
2. Lips - nil.
3. Tongue - atrophic.
4. Eyes - nil.
5. Scrotum - nil.

C.V.S. - no enlargement. No murmurs.

B.P. - 100/60

Lungs - no abnormality.

Abdomen - no abnormality. Liver and spleen not palpable.

C.N.S. - pupils react normally. Reflexes present, equal and brisk, (including ankle jerk). Sensation and co-ordination within normal limits.

Locomotor System - pain is situated at the insertion of the hamstrings and upper third of Rt. calf muscles, affected area is swollen, tender and indurated but not hot.

Measurements - 1. Upper border of patella,
Left 14 7/8", Right 14"

2. 3" below lower border of patella
Left 11½", Right 13".

Patient stated he was unable to walk, but was able to staggar a few steps.

Remarks - gums signs of slight degree compared to involvement of locomotor and cardio-vascular systems.

X-Ray of painful limbs.

Blood - Hb 50%, R.B.C., 2,580,000 per cmm., W.B.C., 7,050 per cmm.

Treatment

18. 1.51 Permidine tabs. 2 T.D.S. (1.5 gm. Hesperidin per diem).

Progress Notes

14. 1.51 Patient states pain is improving. No change in clinical condition.

15. 1.51 I.S.Q.

17. 1.51 Pain in legs has improved, nothing to report on examination. He is still very ill and is only able to stand with the greatest difficulty. Slight activity causes dyspnoea. Unless specifically requested, he does not attempt the upright posture and drags himself along on his buttocks.

19. 1.51 Patient states pain in legs is less severe.

O.E Gums improving, no change in gait or dyspnoea.

20. 1.51 Patient died during the night. He suddenly developed dyspnoea and acute pain in the chest and died within a few minutes.

Comments and Conclusions

This African's death must be described as tragic and avoidable. The following conclusion may be drawn. In at least the more severe cases the hospital diet does not contain enough vitamin C to have a curative action. Death occurred after two days treatment with vitamin P. Although it would not be justifiable to state that this substance had no connection with African scurvy, the writer feels the patient would not have died if large doses of vitamin C had been exhibited instead of permidine. A possible contributory factor was making him walk on the afternoon of the 19th January, 1951, after this exertion he fainted but quickly recovered. At this juncture it was debated whether it was justifiable to with-hold ascorbic acid any longer.

The sudden collapse is a well known complication of scurvy. Lord Anson writing of his voyage round the world mentions sudden death occurring after the slightest exertion,

> "such as moving a patient in his hammock from one part of the ship to another."

Post Mortem Report

Heart and Pericardium

No free fluid in the pericardial cavity. The heart was not enlarged, measuring 4½" at the broadest part. No abnormality could be found in the chambers or the great vessels.

Respiratory System

The lungs were an unusual purple colour and felt very tense. When cut a frothy brown fluid exuded,

C.N.S.

The brain was unusually pale. No haemorrhages were found on section.

Liver

Normal.

Spleen

Enlarged and hard, typical malarial spleen of an African, (the writer failed to palpate this organ during life).

Intestine

The last part of the sigmoid colon and upper part of rectum showed a continuous line of haemorrhage which was situated subperitoneally.

Bladder

Normal, no evidence of bilharzia.

Limbs

The intra-muscular fascia showed extensive haemorrhages. The muscles were darker in colour and contained more blood than usual. These abnormalities were patchy in distribution. The periosteum of the lower end of Rt. femur was infiltrated with blood. Just above the insertion of the Rt. knee joint capsule there was a subperiosteal clot roughly triangular in shape being about 11/2" in its longest axis.

Histology

1.	Small bowel	Haemorrhages into muscle.
2.	Lung	Interstitial oedema and haemorrhages.
3.	Skin	Hypertrophy of collagen with degeneration of that substance.
4.	Muscle	Large numbers of red cells seen between the fibres.

Dr. Tulloch (pathologist, Bulawayo General Hospital) inspected the slides and agreed with my conclusions.

CASE NUMBER 18

Station — Bulawayo

Date of Admission — 15th February, 1951

Name — Daniel

Age — Young Adult

Employment — Labourer.

History

(a) Presenting symptoms and duration

Pains in left leg of three weeks duration. Present at rest.

(b) Questioning

1. Mouth symptoms - teeth loose and painful. Gums bleeding. Duration 3 weeks.

2. Night blindness - nil.

3. Haematuria - nil.

4. Pains in limbs - see above.

Diet History

Rations supplied by employer, meat, mealie meal and beans. Vegetables issued every second day, cabbage and spinach which is cooked in factory kitchen. Does not eat milk or fruit. Denies drinking beer.

Examination

General nutritional state - good. Patient is well covered.
Mental State - satisfactory

Gums - hypertrophic, purplish, several buds present, bleed easily on moderate pressure.

Skin

1. Hyperkeratosis - nil.
2. Pellagra - nil.
3. Phyrnoderma - nil.

Mucous Membranes - well injected.

Clubbing - nil.

Oedema - nil.

Jaundice - nil.

Other vitamin deficiency signs

1. Face - nil
2. Lips - nil
3. Eyes - nil
4. Tongue - nil
5. Scrotum - nil
6. Dypsnoea - nil

B.P.- 150/70 (nervousness). See progress note of 16.2.51.

C.V.S. - no enlargement, no murmurs.

Lungs - N.A.D.

Abdomen - N.A.D.

C.N.S. - pupils react normally. Reflexes present and equal (including ankle jerk). Sensation and co-ordination within normal limits.

Locomotor System (including gait) - walks with marked limp. Left knee held stiff in flexion. Marked swelling of calf muscles of upper two thirds left leg, swelling hard and tender but not hot. Also swelling of periarticular tissues of left knee.

Treatment

2. 3.51 (1) Multivit Tabs. 2 T.D.S.
(2) Vit. C mg. 100 T.D.S.

Progress Notes (N.B. date of disappearance of specific lesion)

15. 2.51 Patient states leg is less painful.

16. 2.51 Patient claims that there is slight improvement in mouth and leg.

O/E A very definite and dramatic improvement in the induration of the calf muscles, which has almost disappeared. Patient walks much more freely and is no longer afraid to bend his knee. Gums unchanged. B.P. 130/62 today.

17. 2.51 Gums unchanged. No definite improvement in leg. Gait shows improvement.

19. 2.51 Gums unchanged. Induration of calf muscles has almost disappeared. Swelling of knee joint persists, definite patella top elicited today. Gait unchanged.

20. 2.51 Gums show slight improvement. Limp now very slight. No change in condition of left leg.

21. 2.51 Improvement of mouth continues. Patient now walks without a limp, and is able to run without discomfort. Induration of calf muscles very slight. The swelling of the periarticular tissues has disappeared. The left knee is capable of full range of painless movement.

22. 2.51 I.S.Q.

23. 2.51 Gums show further improvement. Left knee slightly less swollen.

24. 2.51 Gums improving. No change in leg.

26. 2.51 A further slight improvement of the mouth. Induration has disappeared from the left calf muscles except for an area in lateral aspect of the gastrocnemius. A marked reduction in the swelling of the left knee can be seen today.

27. 2.51 Some improvement of the gums. Induration of left leg has disappeared.

28. 2.51 I.S.Q.

2. 3.51 Gums normal apart from slight sponginess.

2. 3.51 Slight sponginess of gums persists. The swelling of left knee joint has almost completely disappeared.

6. 3.51 Slight sponginess of gums still persists. The swelling of the left knee has not yet completely disappeared. Despite the persistance of the above sign, for all clinical purposes the patient is now cured.

7. 3.51 No change in condition. Discharged.

Comments and Conclusions

A mild routine case of scurvy, a point of interest was the general impression of health and his apparently excellent state of nutrition.

The patient may be regarded as a therapeutic control. A clinical cure with disappearance of the scorbutic manifestations was obtained on rest and the hospital ration. In assessing any method of treatment the beneficial effect of hospitalisation per se must be considered. While this certainly holds good for milder cases, severe scurvy would seem to require ascorbic acid for recovery.

As has been recorded in other patients his diet included vegetables. The response to hospital food excludes a defect of absorption. The explanation probably lies in cooking, or possibly like quite a few natives he does not eat his greens (although he did not admit to this).

CASE NUMBER 19.

Station — Gwanda

Date of Admission — 5th April, 1951.

Name — Saluzika

Age — Young Adult

Employment

History

(a) Presenting symptoms and duration

Pain in right calf for one month, this pain is present at rest.

(b) Questioning

1. Mouth symptoms - gums have been bleeding since childhood, teeth do not feel loose.

2. Night blindness - nil.

3. Haematuria - nil.

Diet History

Rations supplied by employer, viz., mealie meal, meat and dried beans. Part of a cabbage supplied once a week. Does not partake of milk, fruit, wild vegetables or Kaffir beer.

Examination

General Nutritional State - moderate.

Mental State - normal.

Gums - generalised hypertrophy with marked purple discolouration. Bleed very freely. No typical buds but two areas are suggestive.

Skin

1. Hyperkeratosis - nil.
2. Pellagra - nil.
3. Phyrnoderma - slight.

Mucous Membranes - well injected.

Clubbing - nil.

Jaundice - nil.

Other Vitamin Deficiency Signs -

1. Face - nil.
2. Lips - nil.
3. Eyes - nil.
4. Tongue - nil.
5. Scrotum - nil.

C.V.S. - no enlargement, no murmurs, sounds regular and pure.

B.P. - 140/80.

Lungs - no abnormality.

Abdomen - nil abnormal, liver and spleen not palpable.

C.N.S. - pupils react normally to light and accommodation. Reflexes in upper limb normal and equal.

Lower Limb	Left	Right
Knee	+	++
Ankle	+	++
Plantar	Flexor	Flexor

Sensation and co-ordination within normal limits.

Locomotor System, (Including gait) - walks with a definite limp and onviously has discomfort in his right leg. Painful area consists roughly of the upper third of right calf.

This region appears swollen and is tender on firm pressure.
No sign of inflammatory change.
Measurements, taken 4" from lower border of patella,

Right 12½", left 12".

Blood on Admission - Hb 81%

Remarks - the neurological changes are slight but definite.

Treatment -

10. 4.51 Vit. C 300 mgm. T.D.S.

19. 4.51 Multivit Capsules 2 T.D.S.

Progress Notes

8. 4.51 Patient says the pain is unchanged.
O/E No change in gait, gums or leg.

9. 4.51 Much better today, requested discharge.

10. 4.51 Pain has now disappeared entirely while walking.

O/E Gait is now normal, patient is able to run, gums show no definite change.

11. 4.51 I.S.Q.

12. 4.51 Gums show definite improvement, bleeding tendency still present but reduced. Right calf still swollen.

C.N.S. Reflexes of the lower limbs now equal and normal, there is no difference of power in the two legs.

16. 4.51 Owing to attendance at court in Bulawayo no observations have been possible over the last four days. Gums now show a very definite improvement. The spongy appearance is less noticeable and the bleeding tendency is less marked. On questioning the patient states the gums no longer bleed even when he is cleaning his teeth. He also volunteered the information that he feels much stronger in his body.

17. 4.51 Gums improving.

18. 4.51 Gums show further improvement, now almost normal.

19. 4.51 Gums still not quite normal, as a purplish sponginess persists.

Measurements - taken 4" from lower border of patella -

Right 12½" Left 12"

20. 4.51 Gums now normal.

23. 4.51 Measurement of legs unchanged. No definite change in phyrnoderma.

Date of Discharge

23rd April, 1951.

Condition on Discharge

See progress note of 23rd April, 1951.

Comments and Conclusions

It was debated whether this patient should be included as a scurvy. The gums although strikingly abnormal were not completely typical of the disease, particularly in the absence of buds. However his progress in hospital, in the writer's opinion clinched the diagnosis.

Features worthy of comment are:-

1. His diet included vegetable. Interrogation as to the amount of cabbage and the method of cooking same, was exhausting and unprofitable.

2. The neurological signs were rather puzzling and will be discussed elsewhere. His C.N.S, was examined by a colleague who concurred with the findings.

3. This patient was symptom free within 6 days of admission without treatment. This response to hospitalisation must be considered when judging the merits of any therapy especially in milder cases.

4. The phyrnoderma and the swelling of leg were unaltered on discharge. It was felt to be unfair to prolong this case's sojourn in hospital and deprive him of his wages, merely for investigation.

ACKNOWLEDGMENTS

I welcome this opportunity of thanking my colleagues of the Southern Rhodesian Medical Service for their friendly interest and co-operation. In particular, I wish to thank Dr. Baker-Jones, who has given me help and advice on many occasions. It is in no spirit of facetiousness that I express my gratitude to the African patients for their invariable good humour, patience and toleration.

REFERENCES

Annual Reports of Southern Rhodesian Nutrition Council, 1950, 1951, 1952.

Anson, Lord George (1748): A Voyage Round the World (Compiled by Walter).

Baker-Jones, E. Nutrition Officer, Southern Rhodesian Medical Service (1952): Personal communication.

Baker-Jones, E. (1952): Some Facts and Figures of Bantu Nutrition in Southern Rhodesia. (Report made to F.A.O.)

Baker-Jones, E. (1952): Economic Aspects of African Malnutrition in Southern Rhodesia.

Berman, C. (1940): South African J. Med. Science. 5, 54.

Bicknell, F. & Prescott, F. (1946): The Vitamins in Medicine.

(1)Bullock, G. (1931): The Mashona.*

Bronte-Stewart, B. (1951): Personal communication.

Cameron, D.G. & Mills, E.S. (1942): Canad. Med. Ass. J. 46, 548.

Crandon, J.H. & Lund, C.G. (1941): J. Amer. Med. Ass. 116, i, 663.

Crandon, J.H. Lund, C.G. & Dill, D.B. (1940): New England J. Med. 223, 353.

Davies, J.N.P. (1948): Lancet i,317.

Frazier, C.N. & Hu, C.K. (1936): Arch. Dermat. Syph. 33, 825

(1) Mashona are one of the principal tribes of Southern Rhodesia.

Fox, F.W. & Goldberg, L. (1944): South African Food Tables

Gelfand, M. (1948): The Sick African.

Gelfand, M. (1950): Schistosomiasis in Central Africa.

Gelfand, M. (1952): Personal communications.

Gilder, S.S.B. (1950): Brit. Med. J. i, 341.

Gillan, R.U. (1934): East African Med. Journal. 11, 88.

Gillman, J. (1951): Personal communication.

Gillman, J. & Gillman, T. (1951): Perspectives in Human Malnutrition.

Goodwin, G.P. (1934): Brit. Med. J. ii, 113.

Harrington, L. (1951): Physician, Bulawayo Native Hospital: Personal communication.

Higgs, B. (1951): Medical Superintendent, Government Mental Hospital, Bulawayo: Personal communication.

Hess, A.F. (1920): Scurvy Past and Present.

Howman, R. (1946): The Native Labourer and His Food.

Hughes, W. (1950): Brit. Med. J. i, 634

Ibbotsen, J. (1942): Survey of Urban African Conditions in Southern Rhodesia.

Israels, M.C.G. (1943): Lancet i, 170

Janisch, M. (1941): A Study of African Income and Expenditure in 987 Families in Johannesburg (quoted from the Gillmans; no reference given).

Jennings, G.H. & Glazebrook, A.J. (1938): Brit. Med. J. ii 784.

Keys, A. (1948): J. Amer. Med. Ass. 138, 500.

Laurie, N.R. Moore, T. & Rajagopal, K.P. (1941): Biochem. J. 35, 825

Lind, J. (1772): A Treatise on the Scurvy.

Lowenthal, L.J.A. (1933): Arch. Dermat. Syph. 28, 700.

Martin, A.P. (1952): Late Medical Director: Personal communication.

MacDowall, R.J.S. (1950): Handbook of Physiology and Biochemistry.

McHenry, E.W. & Perry, H.M. (1940): J. Nutr. 19, 12.

MacMillan, R.B. & Inglis, J.C. (1944): Brit. Med. J. ii, 233.

Meiklejohn, A.P. (1949): Practitioner 163, 353.

Meiklejohn, A.P. (1950): Personal communication.

Medical Research Council (1948): Vitamin C Requirements of Human Adults. Lancet i, 853.

Medical Research Council (1949): Vitamin A Requirements of Human Adults. Spec. Rep. Ser. No. 264 London.

Montgomery J. (1953): S. Rhod. Medical Service: Personal communication.

Morris, R.M. (1952): Director, S. Rhodesian Medical Service: Personal communication.

Nicholls, L. (1933): Indian Med. Gaz. 68, 681.

Pemberton, J. (1940): Lancet i, 285.

Platt, B.S. (1945): Brit. Medical Bull. 3, 179.

Popper, H. (1944): Arch. Path. 31, 766

Proctor, R.A.W. (1926): E. African Med. J. 3, 284.

Public Health Report of Southern Rhodesia (1950).

Report of Social Security Officer (1944): (S. Rhodesian Government publication).

Scarborough, H. (1940): Lancet, ii, 644.

Shafar, J. (1949): The Vitamins in Medical Practice.

Smith, D.A. (1950): London School of Hygiene and Tropical Medicine. Personal communication.

Stannus, H.S. (1911): Trans. Roy. Soc. Trop. Med. Hyg. 5, 112. Ibid. 1913, 7, 32.

Stannus, H.S. (1944): Lancet ii, 359. Ibid. ii, 105.

Squires, B.T. (1950): Colonial Medical Service, Bechuanaland Protectorate. Personal communication.

Sparrow, C.H. (1952): Ophthalmologist, S. Rhod. Medical Service. Personal communication.

Sydenstricker, V.P. (1944): J. Amer. Dietetic Assoc. 20, 4.

Trowell, H.C. (1940): Trans. Roy. Soc. Trop. Med. & Hyg. 33, 389.

Trowell, H.C. (1948): Medical Examination of 500 African Railway Workers. (Reprinted from East African Medical Journal, vol. XXV, No. 6, June, 1948, No. 8, August, 1948, No. 11, November, 1948).

Trowell, H.C. (1950): Problems Raised by Kwashiorkor. (Reprint from Nutrition Reviews, vol. 8, No. 6, June, 1950)

Trowell, H.C. (1951): Personal communication.

Tulloch, B. (1951): Pathologist, Bulawayo General Hospital. Personal communication.

Vilter, R.W. & Woolford, R.M. (1945): J. Lab. Clin. Med. 30, 388.

Vilter, R.W. Woolford, R.M. & Spies, T.D. (1946): J. Lab. Clin. Med. 31, 609.

Whitby, Sir Lionel E.H. & Britten, C.J.C. (1946): Disorders of the Blood.

Wiltshire, H. (1919): Lancet ii, 564.

Williams, C.D. (1933): Arch. Dis. Childhood. 8, 423.

Williams, C.D. (1935): Lancet ii, 1151.

Witts, L.J. (1947): B.M.J. i, 1. Ibid (1947) 1, 45.

Wright, G. (1951): S. Rhod. Medical Service: Personal communication.

Wright, S. (1934): Applied physiology.