GANGOSA IN NEW GUINEA AND ITS ETIOLOGY

BY

ANTON BREINL

FROM THE AUSTRALIAN INSTITUTE OF TROPICAL MEDICINE

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PLATES XIV-XVII

Gangosa, or Rhinopharyngitis mutilans, is a disease of limited geographical distribution, being, according to our present knowledge, confined mainly to the Pacific Islands.

According to Castellani and Chalmers (1910), cases of this disease were described first in 1828 by a Spanish Commission to the Marianne or Ladrone Islands, under the name of Gangosa, meaning 'Nasal Voice.' Fordyce and Arnold (1906), and in the same year Leys, gave a clinical account of a similar disease in Guam, and were followed by Mink and McLean (1906) who discovered cases of Gangosa in the Ladrone and Caroline Islands.

Later, Stitt (1907) reported the occurrence of Gangosa in a white man, who was treated in the United States Naval Hospital at Canacao. This patient had contracted his infection during a stay in Guam, where he had become intimately associated with natives in whose families Gangosa had been known.

A further case with complete post-mortem findings was described by Musgrave and Marshall (1907) in a native of Santo Domingo de Basco, Batan Islands, being the first case reported from the Philippines. Careful examination of the blood was made by these authors. According to their findings the blood did not show any marked pathological changes; the differential count proved that the relative numbers of the various types of leucocytes were normal.

In 1906 N. T. McLean observed two patients suffering from what he believed to be Gangosa on the occasion of a visit

to the Hospital at Gonaives, Haiti, and stated that according to information that he collected numbers of such cases were supposed to have occurred at Port au Prince.

A case was also published by Stitt in a young Filipino who had never been outside Cavite Province (Philippine Islands).

In 1910 Garrison pointed out that there were 327 cases of Gangosa in Guam, and pronounced the opinion that according to his experiences from a clinical, and particularly from a therapeutical standpoint, Gangosa was to be considered a very late form of Syphilis—perhaps a fourth stage. Odell (1911) reported a positive Wassermann reaction in 82% of his cases. Anti-syphilitic treatment seemed to benefit all cases considerably.

Rossiter (1912) described a disease in a two year old child where the destruction of the hard and soft palate were the most prominent lesions. In specimens taken from the ulcerated surfaces numerous *Spirochaeta pertenuis* were found. Two months previously the child had suffered from typical framboesia.

Also in 1912 Ziemann drew attention to the fact that a disease resembling Rhinopharyngitis mutilans occurs not infrequently amongst the negroes of Kamerun.

I observed cases of Gangosa for the first time in the Torres Straits Islands, whilst on a short journey during the latter end of 1910.

Then I was inclined to consider this disease either as a peculiar manifestation of syphilis or as a hitherto undescribed disease occuring on Murray Island. Elkington referred to most of these cases in the yearly report of 1912, adding a number of cases which he discovered on Darnley, Boigu, Saibai and Dauren Islands. The disease must have been prevalent on Murray Island for nearly a century, as it is, according to Elkington, referred to by Dr. Wilson, R.N., in an account of a visit made by him to Murray Island in 1822.

The occurrence of cases of Gangosa in New Guinea has been noted previously, but these cases had been regarded by Sir William Macgregor as lupus.

During my short journey in New Guinea typical cases of Gangosa were seen in villages on the south coast, especially in Kerapuna, a fairly large village, where as many as ten cases were

examined. Furthermore a number of natives suffering from Gangosa were encountered in the western parts of British New Guinea and in the Mekeo District, and there the earliest stages of the disease were seen, showing peculiar manifestations which did not altogether coincide with the description given by previous observers elsewhere.

CLINICAL ACCOUNT OF THE DISEASE

According to Castellani and Chalmers the disease 'begins as a sore throat, when on examination a nodule can be seen on the back of the pharynx, the posterior pillars of the fauces or the edge of the soft palate. This ulcer usually spreads rapidly at first, but more slowly later, and destroys the soft palate first, later the bony parts of the palate and the nasal septum. In consequence the skin of the nose falls in, and the cavity of the nose and mouth are connected.

'In consequence of the destruction of the soft and hard palates the ulceration may extend afterwards to the face or lips or affect the pharynx. As a rule a pungent odour is given off, whilst a slight discharge of granular and necrotic debris is present. The ulceration may progress continuously for a period of 10-35 years, or it may advance at certain times and be quiescent at others, or it may cease at any time, leaving a chronic ulcer.'

So far as observed, the cases of Gangosa met with in New Guinea may be conveniently divided into three groups.

Firstly. Early cases in which the patients had been ill for a few months only and where a small area of the skin of the face was found to be affected. The cartilaginous septum had been destroyed but the disease had not caused any extensive destruction of the bone.

Secondly. Cases in which the ulceration had advanced further. Large areas of the face were found to be the seat of extensive weeping sores. The bone near the affected parts had become implicated, and the disease had led to the destruction of the skeleton of the nose and the hard palate.

Thirdly. Cases in which the active ulcerating process had come to a standstill. Extensive cicatrisation had taken place [illegible]

seat of the ulceration, as a rule the nose had disappeared, and was represented by a smaller or larger opening. In one extreme case the opening was so small that the woman in question was able to smoke comfortably a cigarette through the small opening—the only remains of the nose. In another case the whole of the back of the pharynx could be seen without any difficulty through the large opening in the middle of the face.

The uncertain etiology of Gangosa and its similarity to tertiary syphilis in its clinical manifestations makes it advisable to refer in greater detail to the history of a few of the typical cases met with.

EARLY CASES

Only a comparatively small number of early cases were seen.

Case 1. (Pl. XIV, fig. 1.) Apau Kaianga was an unmarried girl of about 12 years of age. Her parents as well as four brothers and sisters were alive and well. Her disease began about one month previous to my visit as a sore below her nose which spread rapidly, affecting especially the septum, the left ala of the nose, the upper lip and the left corner of the mouth, and had already destroyed part of the cartilaginous septum.

The examination revealed the nose considerably flattened, and also thickened on account of oedematous swelling. The left ala was the seat of a large raised dry ulcer, covered by a thin scab, below which there was a reddish uneven granulating surface. This ulceration had extended to the upper lip, being confined to its central part, just below the nostrils. The left corner of the mouth was the seat of a similar ulcer which showed an uneven surface. The upper lip around the ulceration was swollen and oedematous and its surface was intersected by purplish irregular raised lines simulating scar tissue. The cartilaginous septum had been partly destroyed by the ulceration, whereas the osseous septum was still intact. The hard and soft palate were of normal appearance.

Case 2. (Pl. XIV, fig. 2.) Ame Aua was a woman about 25 years of age, belonging to the same village. Her father was still living, her mother had died. Some time ago she was married, her three children were alive and well, except for manifestations of Yaws.

According to her own statement she had been ill for four months only, the disease in her had begun as a small sore on the tip of her nose which had spread gradually into the nasal cavity. The examination revealed a fairly large ulcer on the nose, a yellowish greasy scab covering the raw granulating surface. The cartilaginous septum of the nose had been destroyed by ulceration and the nose was in consequence flattened. A similar ulcer below the nostrils extended into the nasal cavity. On the right cheek was a narrow raised line resembling oedematous scar tissue. The hard and the soft palate and the pharynx did not show any pathological changes.

Case 3. (Pl. XIV, fig. 3.) The woman belonged to Kerapuna, a large village on the south-east coast of New Guinea. She had been married and was the mother of six children. Four of them were healthy and two were in the secondary stage of Yaws.

Her upper lip showed the same scar formation as described in the two previous cases, the scars being oedematous and raised in the centre. The free edge of the skin of both the alae showed ulceration and both nostrils were closed up by yellowish scabs.

Case 4. (Pl. XIV, fig. 4.) Ame Kwipa, a young girl, belonged to Veipa, a large village in the Mekeo District. She had had small sores on her face, especially on her right cheek for about one year, and these had shown a tendency to heal during the last two months. Her brothers and sisters were healthy.

When examined her nose appeared broadened, its skin swollen and oedematous. The upper lip, especially the right side, was swollen, oedematous, and showed raised irregular lines resembling scars. The right cheek was the seat of a number of small pustules which contained whitish pus.

Case 5. (Pl. XV, fig. 5.) Ainye, a girl about 14 years of age, belonged to Bebeo, a small village in the Mekeo District. Her father was alive and well, her mother and one brother had died. Her illness commenced about one year previously with a small sore on the upper lip below the nose, which had spread slowly and gradually to the nose and downwards to the mouth.

When examined her nose was flattened, showing an uneven surface; the mucous membrane of her nostrils was covered by a yellowish scab. The upper lip was swollen and succulent in appearance, whilst the left angle of her mouth was drawn up by the formation of scar tissue. Her body was clean.

Case 6. (Pl. XV, fig. 6.) The patient was a young unmarried girl, Wabuagi, belonging to Kaile, a village on the south-east coast of New Guinea. Her mother was alive, her father dead. Her disease began about two years previously as a small sore on the upper lip just below the nose, which had spread gradually to the cheeks and into the interior of her nose.

At the time of examination her nose was flattened and had sunk in, as the cartilaginous septum had completely disappeared. The surface of the nose was uneven and rough; the right half of the upper lip was drawn up on account of extensive scar formation. The same scar formation was seen on both cheeks, more extensively, however, on the right side. The skin of the affected parts was very thin and shiny, intersected by raised streaks of apparently dense scar tissue. Here and there on the nose and cheeks were small ulcerations covered by a yellowish scab. The hard and soft palate did not show any pathological changes.

Case 7. (Pl. XV, fig. 7.) Was a young unmarried woman, belonging to Kerapuna. Her parents as well as two younger sisters were alive and well. Her disease began when a small child with sores on her face which had since spread, implicating the nose. She showed large and extensive ulceration on the face, the nose had completely disappeared and was represented by a small opening in the middle of the face surrounded by irregular ulcers covered by yellowish scabs. The upper lip was partly destroyed by extensive ulceration. A large ulcer on her left cheek had a raised granulating surface and irregular edges, covering copiously a slightly yellowish clear, foul smelling discharge. The left cheek showed scar formation. The hard and soft palate were normal.

LATER CASES

Case 8. The patient, a man about 22 years of age, belonged to Hula, a village on the south-east coast of New Guinea. His mother had died, his father was alive and healthy. He was married and had three normal children. According to information the disease had commenced about five years previously with a sore on the forehead over his left eye-brow, which had spread gradually until the greater part of his face was affected. His face, when examined, was badly disfigured; the nose was flattened and was sunk in. Deep scar formation on the right side of his forehead caused the upper eyelid to be drawn up. The skin on the left side of his forehead showed extensive ulceration which had spread downwards and caused the destruction of his left eye. The central part of his upper lip was partly destroyed. The hard palate was perforated, showing a round hole of about 2 cm. in diameter, with smooth rounded edges.

Case 9. Was a woman, Kila Alukma, of about 40 years of age, living in the same village as the previous case. Her parents were dead, her two daughters were married and apparently well.

Her disease had begun about ten years previously with a pain in the stomach which gradually worked up to her mouth and came out through her nose. She pointed out that there had been a sore in her mouth before her face became affected. This case was very similar in appearance to Case 8. There was extensive scar formation on the face round the nose, the upper lip was drawn up on account of dense scar tissue. A stinking discharge from the nostrils was noticed; the hard palate was perforated, the oral and nasal cavity communicating through a round opening of about 3 cm. in diameter.

Case 10. Was an unmarried woman of about 35 years of age belonging to Kaile on the south-east coast of New Guinea. Her mother was still alive, her father was dead. About ten years previously a small ulcer had started on her nose and soon began to spread. At the same time ulcers appeared on her legs.

When examined the cartilaginous septum of her nose had disappeared and extensive scar tissue formation was observed around the nose, on both cheeks and on the upper lip, the mouth and eyelids being normal. The hard palate was perforated. Her legs, as well as her left forearm were the seat of deep ulcers of varying size some of which extended as far as the bone, whilst some of them were covered by a greyish greasy scab.

Case 11. (Pl. XV, fig. 8.) Was a man, Aiaba, about 40 years of age belonging to Kivori village. Both his parents were dead. He was married and the father of six healthy children. His illness had begun about four years previously with a small sore on the upper lip just below the nostrils, which spread at first into the nasal cavity and later to his face. He had lost the toes of his right foot in an accident when he was a small boy.

The patient's face was of horrible appearance. The nose had completely disappeared, leaving a large hole in his face through which the posterior wall of the pharynx could be seen. A large irregular granulating sore on his forehead was covered by a yellowish dry scab under which thick pus had collected. A similar sore was on his right cheek showing here and there recent scar formation. The upper lip was the seat of extensive scar formation. The skin of his left cheek was swollen and oedematous.

The hard palate was perforated and the oral cavity communicated with the nasal cavity by means of a large round opening with sharp edges.

THIRD STAGE OF THE DISEASE

Case 12. (Pl. XVI, fig. 9.) A woman, Ugre, about 25 years of age, who lived in Kerapuna, a village on the south-east coast of New Guinea. She had been ill, according to information, for about ten years; her nose had been affected long before her mouth became involved. When she fell ill her husband married a second wife. Both were seen and were in excellent health. When examined the skeleton of her nose had disappeared and there was a hole in the centre of her face, the surrounding skin as well as the skin of the upper lip and forehead showed extensive scar formation, indicating that the whole face had been previously affected by the disease.

Case 13. (Pl. XVI, fig. 10.) An unmarried woman, about 32 years of age, was seen in the same village as the previous case. Her father had died, her mother was still alive and in good health. Her disease had started when she was quite a small child. When examined the bony skeleton of her nose had completely disappeared, the nose was sunk in so that the nostrils were only represented by a small opening. Both the upper and the lower lips had disappeared, the skin of the face directly joining on to the gum, so that the teeth were unprotected; the skin of her face was one mass of scar tissue, the right lower eyelid was drawn down by scars giving rise to an ectropion. The left side of her throat and her left shoulder were of the same appearance as the skin of her face, showing extensive scar formation. Her soft and hard palate did not show any pathological change.

Case 14. (Pl. XVI, fig. 11.) An unmarried woman of about 35 years of age. She had been ill for quite a long time, in all probability between ten to fifteen years. Both her parents were alive and in good health. Her disease began on the 'outside of her nose' in the form of a small sore which had spread slowly implicating the lips and mouth. Some time ago the ulcerated parts had begun to heal up. Her face was completely disfigured. The whole outer nose had disappeared as well as the anterior part of her hard palate, so that the oral and nasal cavities communicated extensively. The posterior part of the hard and soft palate were still intact. The central part of the upper lip and the lower lip had disappeared, the outer skin joining immediately on to the mucous membrane of the gum. There was extensive scar formation on the skin of her face and on the neck which closely resembled the scars after deep burns.

Case 15. A woman of about 35 years of age belonged to Ififu (Mekeo District). Her parents were dead, two brothers were alive and well, one of them showing scar formation around the mouth, most probably the remainder of Yaws. She was married and mother of a healthy girl. Her illness began when she was a girl, in the form of a small sore on the upper lip. Subsequent to the infection of the face a large ulcerating sore developed on the abdomen, around the navel which, however, soon healed up. Her mouth was small and deformed, the left angle drawn up by scar tissue. Extensive scar tissue formation was seen on the upper lip and both cheeks, the skin otherwise being smooth, shiny and very thin.

Case 16. (Pl. XVI, fig. 12.) Was an unmarried woman, Kaiyo, about 35 years of age. Her parents were dead. The disease began at a very early age with a sore on the left foot soon after she had begun to walk. Later on a small ulcer appeared on her upper lip below the nostrils which gradually spread eating away the whole upper lip and the nose. At the time of examination her nose as well as her upper lip had completely disappeared, a small round hole representing

the nasal opening. Extensive scar formation was noticeable around the mouth and on the cheeks. Her left eye had been destroyed by the disease. The hard and soft palate did not show any pathological changes.

Case 17. Was a middle-aged woman. Her parents were dead. She had four brothers and sisters, two of whom had died. She had never been married. Her disease began some time ago with an ulceration inside her nostrils. Her nose had been destroyed by the disease, and the back of her pharynx was visible through a large triangular opening in the face. The anterior part of the hard palate was destroyed, but the posterior part was still existent. Her mouth was deformed, the right angle drawn up by scar tissue.

The case histories recorded above describing a few typical cases out of the many examined, prove that Gangosa, or Rhinopharyngitis mutilans, is a definite morbid entity, and can be differentiated from other diseases causing similar lesions, such as Syphilis, Lupus and Leprosy. It is a very chronic but very rarely fatal complaint. A great number of the patients recover without specific treatment, mostly, however, after the morbid process has brought about extensive destruction of the face.

From the number of cases observed in the different stages, Gangosa in New Guinea seems to differ in its earliest stages from the disease described by Castellani and Chalmers.

According to my experience in New Guinea, Gangosa usually begins as a small ulcer on the upper lip, just below the nose. Only in a small number of cases did patients state definitely that the palate was affected before any other part of the face. This small ulcer soon begins to spread, destroying at first the fleshy parts of the face, such as the lips, the alae and the skin of the cheeks surrounding the nose. As a rule, the tissue in the near neighbourhood of the ulcer is swollen on account of the oedematous infiltration, the surface skin is glossy and intersected by reddish, raised lines, resembling in some respects a relief map of mountains.

In the earliest stages the ulcer may spread gradually or sometimes rapidly, finally implicating the bones of the infected regions, destroying at first the cartilaginous parts of the nose, and later on the bony skeleton and the soft and hard palate.

The ulcers are raised, possess irregular edges, and are not well defined from the surrounding tissues. The surface of the ulcer is formed of granulating tissue, which secretes a malodorous yellowish discharge, and is often covered by a dark yellowish scab, underneath which pus accumulates. Very soon, however, some of the

ulcers show a tendency towards healing. The granulation tissue begins to discharge less, and after a varying period new smooth skin grows over the ulcer from the surroundings, often, however, breaking down again, and giving rise to renewed ulceration.

Some time afterwards dense scar tissue is formed, leading to the deformities of the face so characteristic of the disease.

Now and again only a single primary ulcer is seen, at other times a number of ulcers appear simultaneously.

The morbid process may come to a standstill at any stage of the disease, even before it has led to extensive destruction.

The tendency to scar formation is a marked feature of the disease, and in this respect Gangosa simulates Syphilis, so that in some of the late cases a differential diagnosis would be practically impossible.

Some of the patients who showed typical symptoms of Gangosa in their faces were suffering from large ulcerations on other parts of the body, especially on the legs. It is impossible to decide whether these ulcers corresponded only to Ulcus tropicum, or were due to the same parasites as the face lesions.

It is interesting to note that the great majority of cases seen were women, only a comparatively small number of men being affected.

THE ETIOLOGY OF THE DISEASE.

As the etiology of the disease is unknown, material was collected for microscopic examination. Special attention was paid to the early lesions with a view of finding a possible parasite in the oedema fluid. It may well be understood that the examination of the secretion of open sores of advanced cases did not seem to offer much opportunity for the forming of a definite opinion, smears taken from the secretion of open wounds always contain enormous numbers of bacteria and spirochaetes of varying shapes and sizes, which are to be considered as secondary infections.

From the earliest cases showing the marked oedematous swelling of the affected parts as described above, oedema fluid was obtained by pricking the skin over the lesion with a needle in the same way as serum would be obtained from a leprous nodule. The specimens

were stained with Giemsa's stain. In the oedema fluid of five early and two later cases, in addition to a small number of red and white corpuscles, small bodies were found resembling yeast cells. The majority of them occurred free, only a small number was contained in leucocytes (Pl. XVII, figs. 1 and 2). These cells showed a marked polymorphism; some of them were round, others oval or pear-shaped (comp. figs. 3 and 4), and were seen either singly or in groups.

The cells measured from 0·75 μ to 4 μ, and consisted of a lightly bluish staining cytoplasm showing a typical network structure, a great number of them contained a distinct and sharply-defined vacuole (Pl. XVII, figs. 5-7 and small roundish masses which resembled chromatin in their staining reaction. These chromatin-staining masses occurred either singly, being more or less sharply defined, roundish or oblong in shape (figs. 7, 8, 13), or as number of similar bodies enclosed within the cell (figs. 9, 10, 14, 15).

Budding of the cells in its various stages could be frequently observed.

In the earliest stages only a small protuberance on the periphery of the cell was seen, consisting of cytoplasm only (figs. 3*a*, 6, 14).

In the more advanced forms this protuberance had increased in size, and at this stage a small chromatin-like staining mass was often seen at the base of the bud (figs. 3*b*, 16). Sometimes the bud itself contained one or more chromatin-like staining particles (figs. 7, 13).

The bud increased in size, and usually became detached before it reached the same size as the parent cell (figs. 9, 10, 16), and in this case invariably contained chromatin-like staining particles and often a distinct unstained vacuolic area (fig. 18).

In many instances only one bud was formed, not rarely, however, two, three or even more buds were seen still connected with the cell (figs. 7, 15).

Forms as seen in fig. 17 were noticed only very rarely. In these forms the cell consisted of a central portion stained lightly reddish by Giemsa's method, containing one or more distinct chromatin-like particles, the whole being surrounded by a thick bluish pellicle.

From their morphological appearance there is no doubt that the

cells described are to be considered as parasites belonging to the genus Cryptococcus of the family Saccharomycetes. The characteristic features of Cryptococcus is the reproduction by budding only and the absence of endospores and ascospores.

In these parasites only multiplication by budding was observed, endospores have never been seen, although carefully sought.

The parasite itself has all the typical features of yeast cells. Unfortunately circumstances only permitted of the collecting of dry films, which were stained by Giemsa's method, and nothing definite can be said about the nuclear details. When stained by Gram's method the cells did not become decolourised.

Parasites of the genus Cryptococcus are well known to produce granulating sores in man and beast; the best known and most studied disease due to Cryptococcus is probably epizootic lymphangitis in horses, occurring in different parts of the world.

Gilchrist and Stokes (1898) describe a similar parasite in man from a case of chronic ulcerative dermatitis, resembling Lupus, under the specific name of *Cryptococcus dermatitis*. Busse (1894) isolated *Cryptococcus hominis* from a purulent periostitis of the tibia of a woman.

On the other hand, yeast cells occur very frequently in open sores of every description.

These parasites were found in great numbers in the oedema fluid of the early cases of Gangosa, where open sores had not formed. Moreover, specimens of the oedema fluid did not contain any other microorganisms besides the yeast cells and a small number of red and white blood corpuscles.

In the seven cases from which oedema fluid could be obtained these parasites were found, most numerously in Case 11, where early and late manifestations were present at the same time, and in which the disease seemed to be progressing most rapidly.

The finding of a Cryptococcus in seven cases out of eight examined, suffering from Rhinopharyngitis mutilans, justifies the conclusion that one may regard this parasite as the cause of the disease in question.

The fact that blastomycetes are known to cause similar skin lesions in man and animals is an additional argument in favour of the Cryptococcus being the etiological agent of Gangosa.

therefore propose for this parasite the specific name of *Cryptococcus mutilans*.

Unfortunately, circumstances did not allow attempts at cultivation. Animal experiments have been performed at our instigation by Dr. Elkington whilst visiting Murray Island. Two monkeys were inoculated on the eyelid and intra-nasally with the secretion of open sores, but in spite of prolonged observation none of the animals in question showed any lesions whatsoever.

CONCLUSION

Rhinopharyngitis mutilans is a disease which occurs not infrequently amongst the native population of the south coast of British New Guinea, and is a blastomycosis due to *Cryptococcus mutilans*, n.sp.

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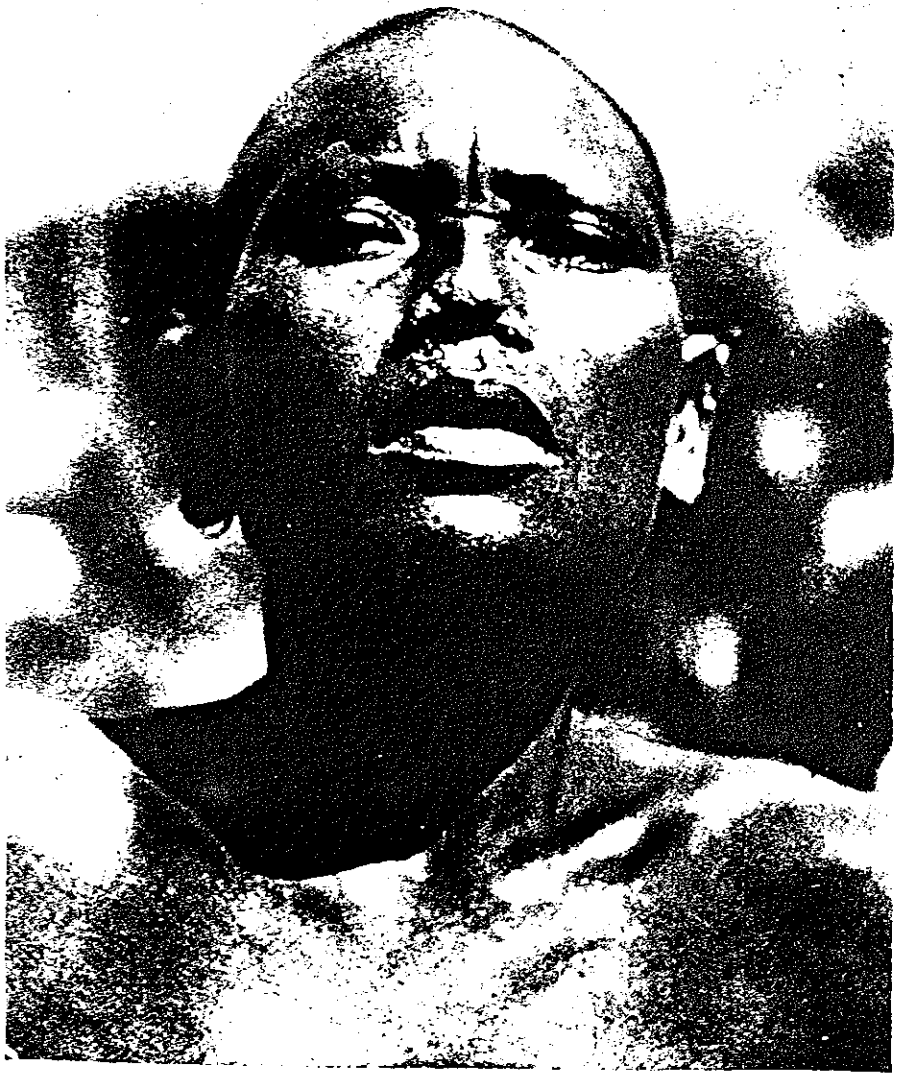
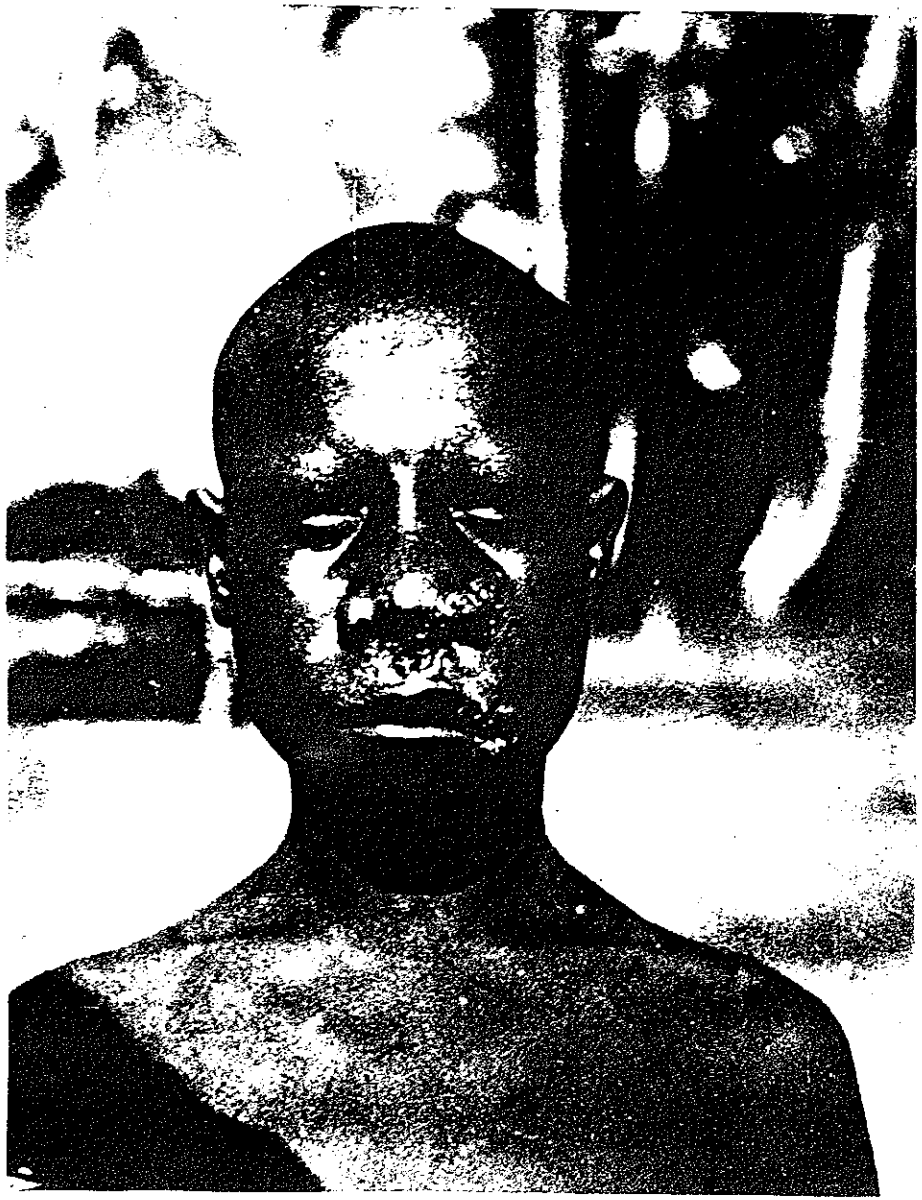
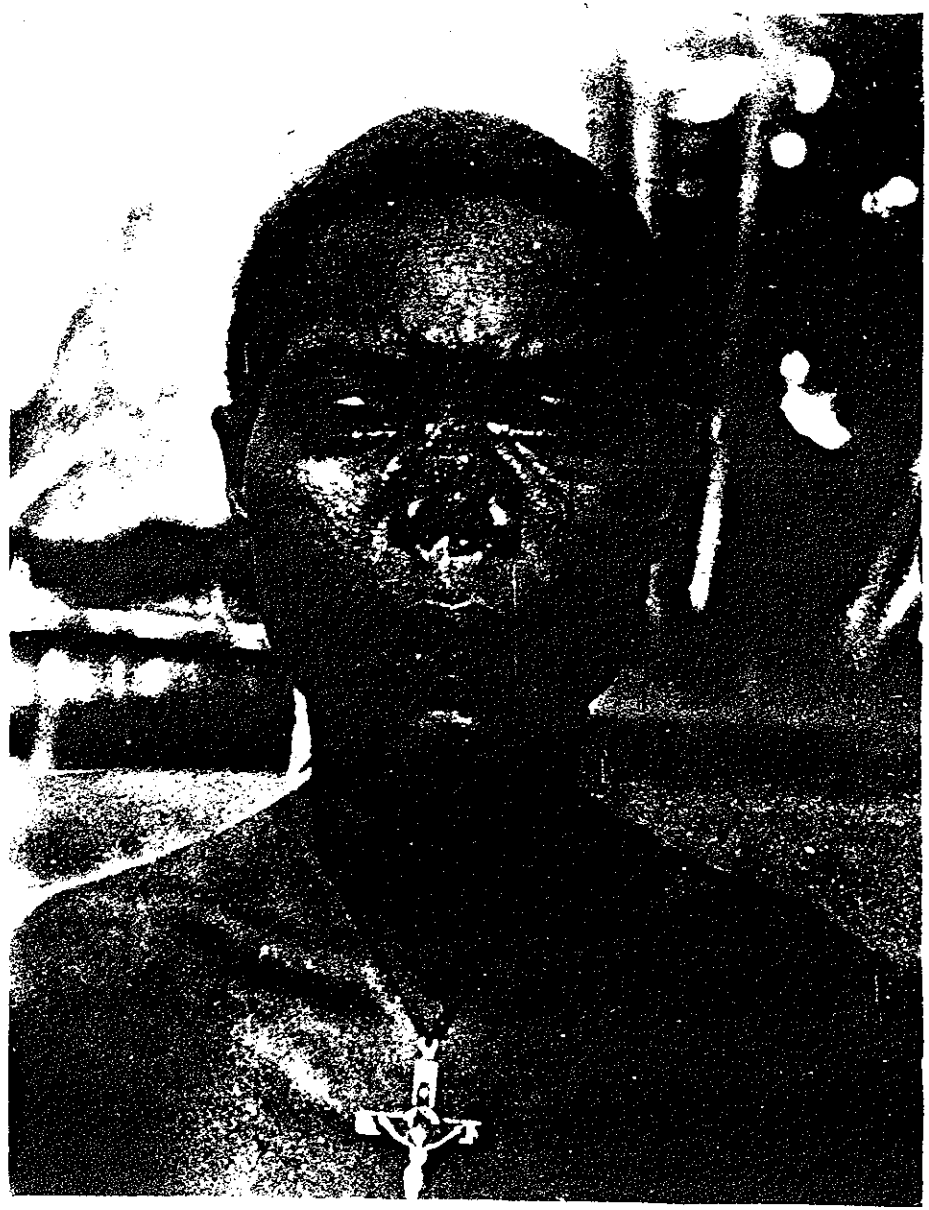
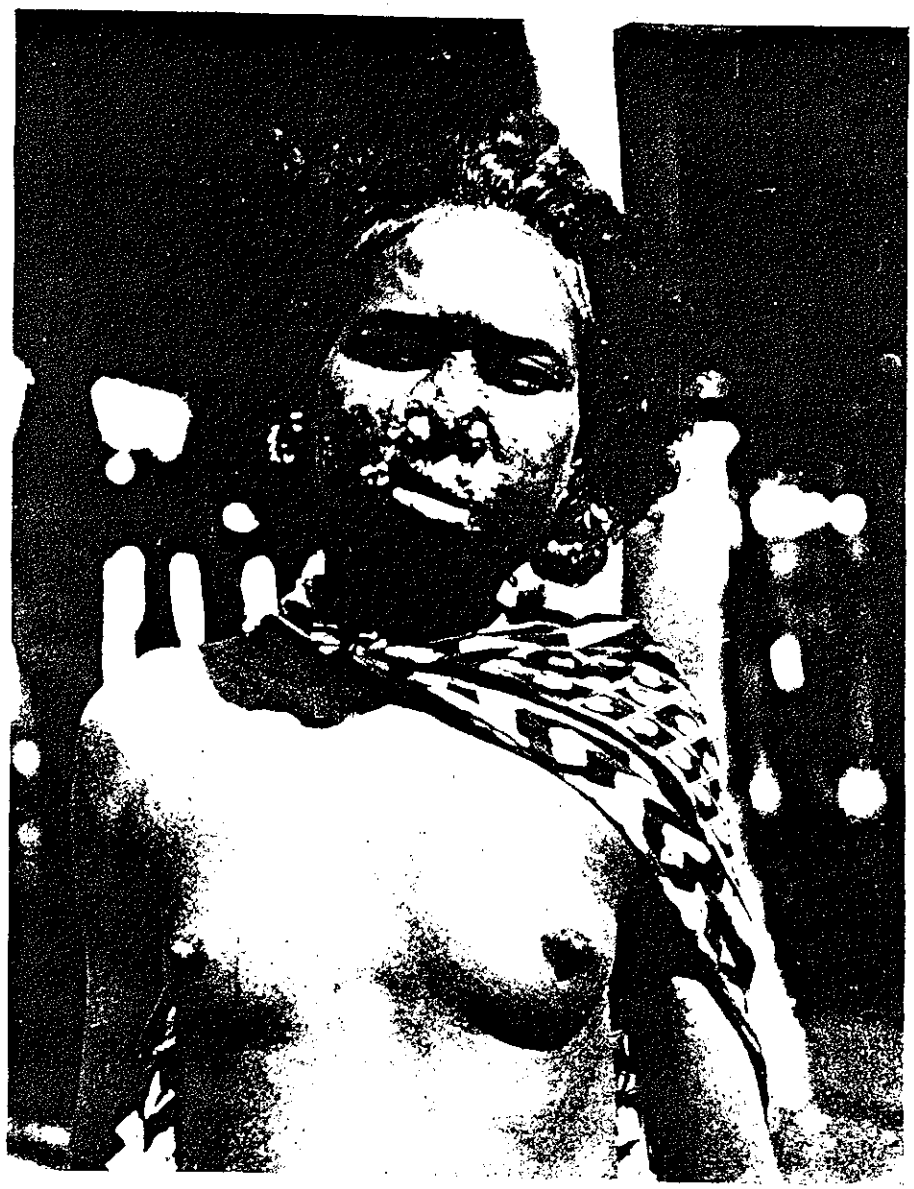


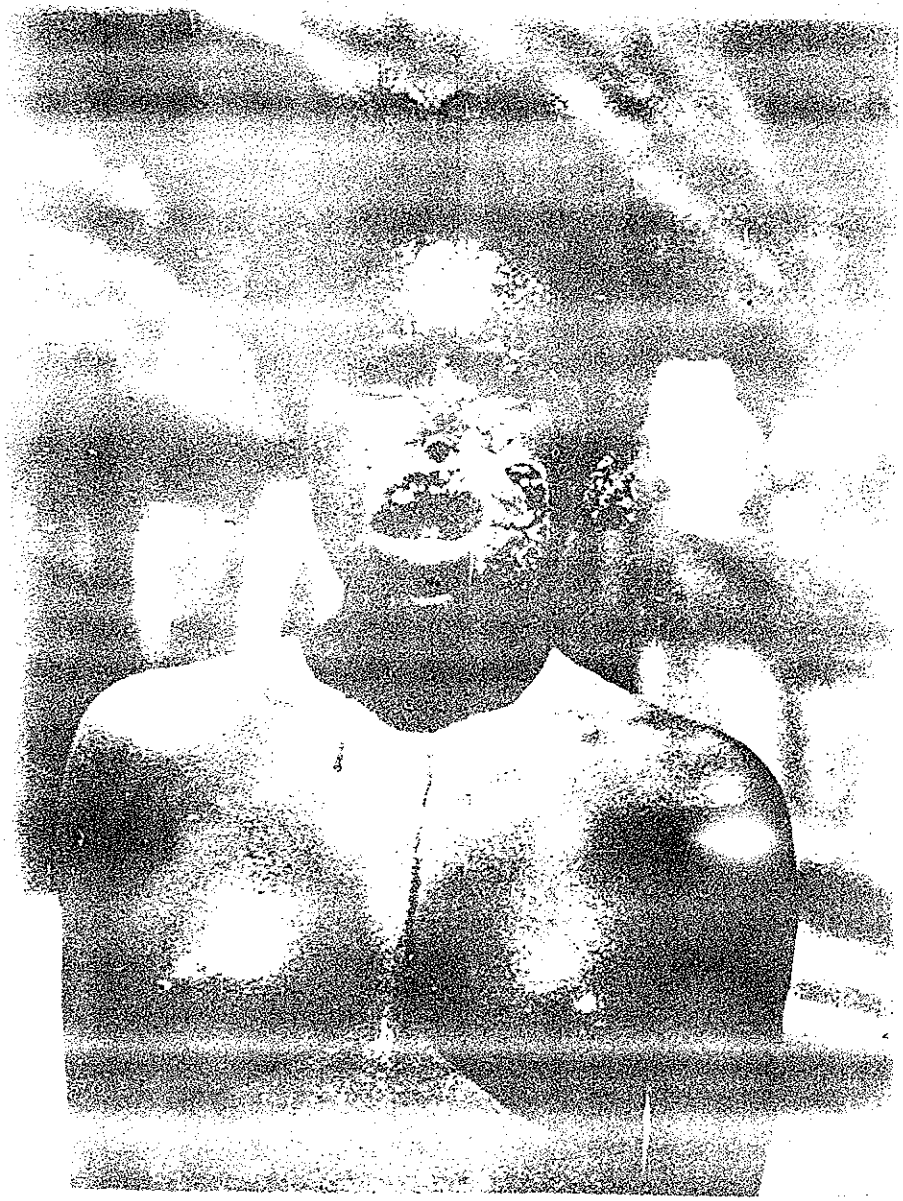
Fig. 3

Fig. 4









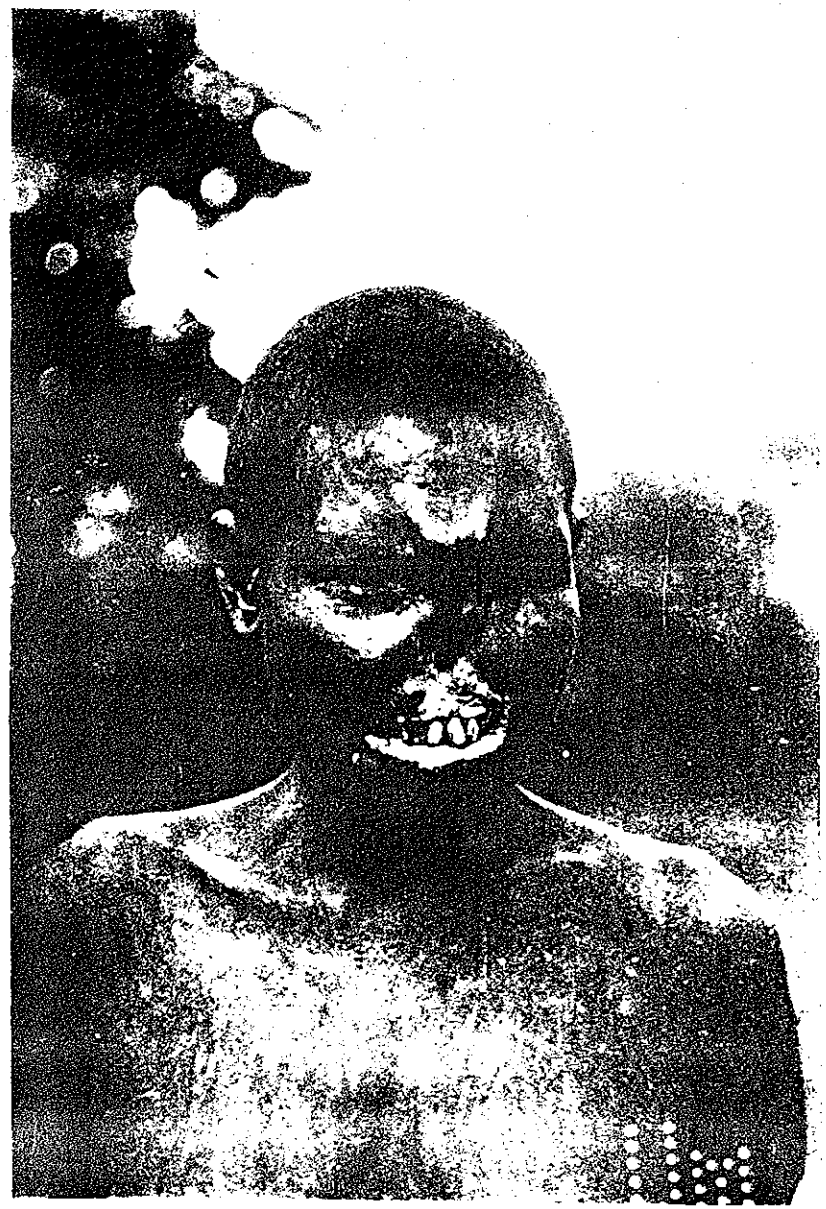


Plate XVII

All the figures were drawn with a large Abbe drawing apparatus, using 2 mm. apochromatic oil immersion.

For Figs. 1-2. Compensation ocular 4 was used.

For Figs. 3-18. Compensation ocular 18.

The specimens were fixed in alcohol and stained by Giemsa's method.

Figs. 1-2. Intracellular forms of *Cytozoon antillense*.

Figs. 3-4. Groups of parasites.

Figs. 5-7, 9, 10, 12-16, 18. Different stages of budding.

Figs. 8, 11. Large forms.

Fig. 17. Apparently encysted form, rarely seen.

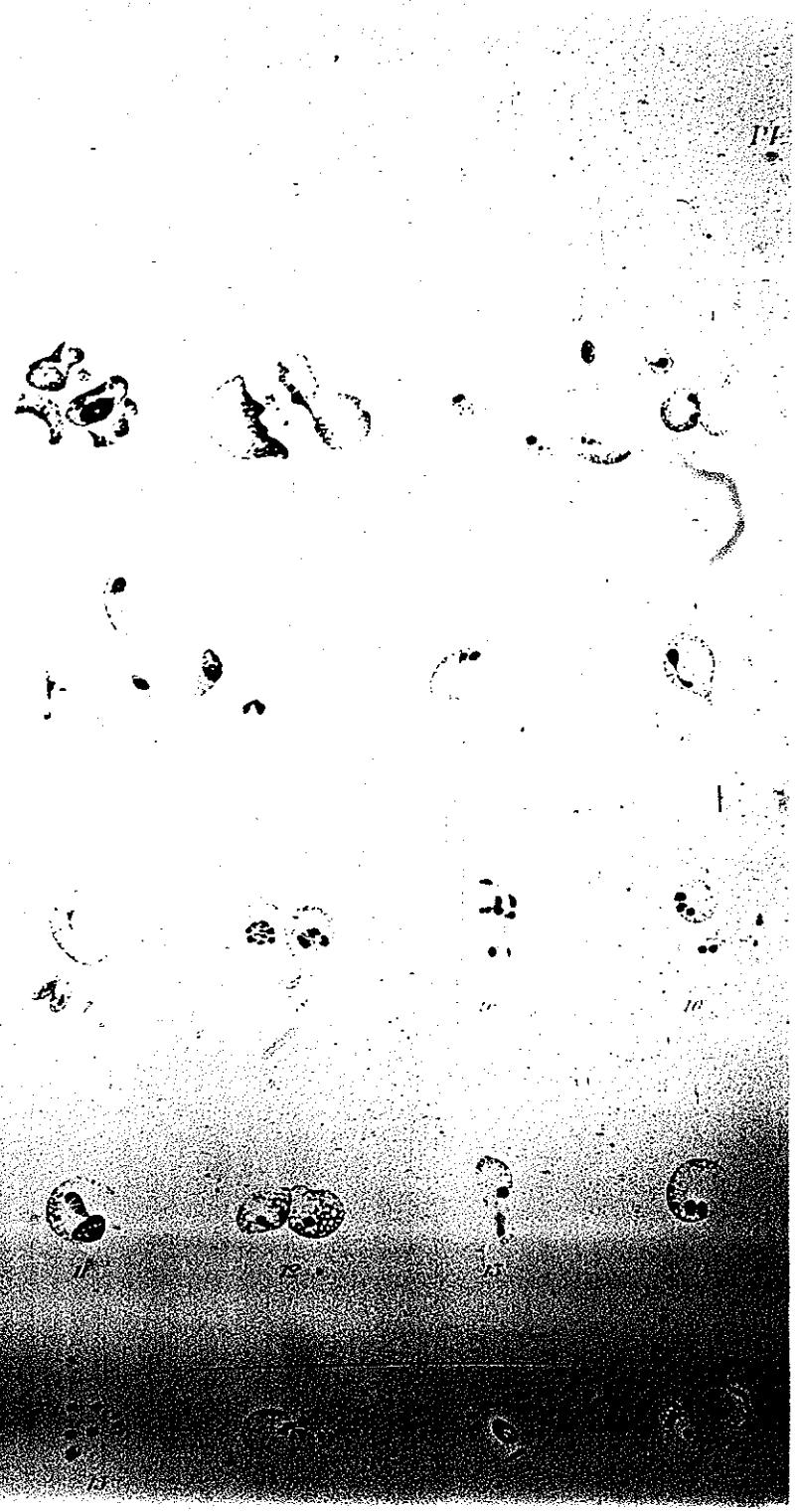


PLATE XVI

Figs. 9-12. Cases of Gangosa (Rhinopharyngitis mutilans).

FIG 9.

FIG. 10

PLATE XV

Figs. 5-8. Cases of Gangosa (Rhinopharyngitis mutilans)

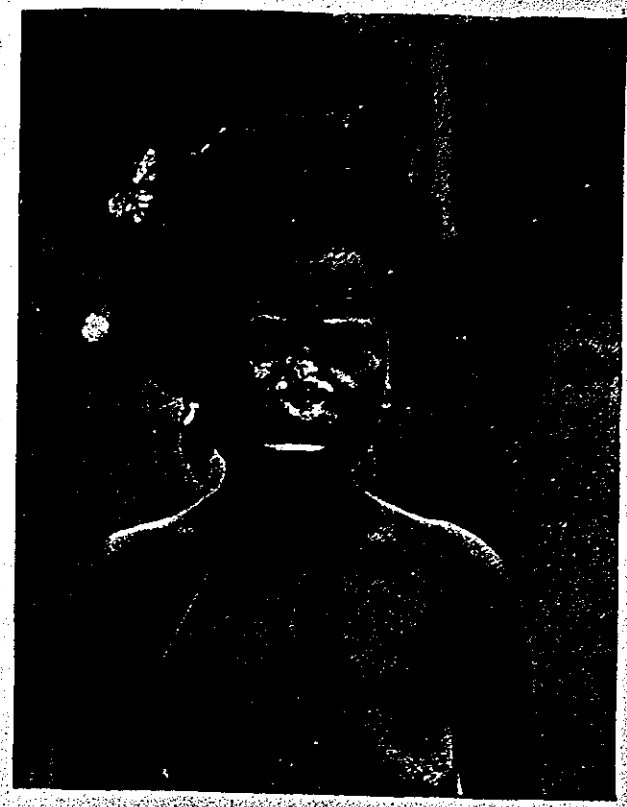


FIG. 5

EXPLANATION OF PLATES

PLATE XIV

Figs. 1-4. Cases of Gangosa (Rhinopharyngitis mutilans).

FIG. 1

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DR. H. B. FANTHAM

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