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INDIA OFFICE.

SCHEME

FOR

OBTAINING A BETTER KNOWLEDGE

OF

THE ENDEMIC SKIN DISEASES

OF

INDIA.

PREPARED BY

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SCHEME

FOR

OBTAINING A BETTER KNOWLEDGE OF THE ENDEMIC SKIN DISEASES OF INDIA.

GENERAL REMARKS.

A.—Objects and Mode of the Inquiry.

Two main objects are proposed:—

1. To obtain and then to circulate a better knowledge of the more important endemic skin diseases of India, or such as principally attack the skin; and thereby
2. To bring about an agreement, which is far from existing at present, between the profession in India and in England as to the nomenclature, the typical character, the varieties, and the probable or demonstrated causes of the diseases in question.

At present very considerable confusion arises from the want of an agreement as to the use to which particular terms should be put—elephantiasis for example—the same disorder being spoken of by different persons under different names, and *vice versâ*. The prevention of such confusion would itself be a great gain to science, as it would enable observers to conduct investigations relative to diseases with infinitely less labour and unquestionably with greater certainty of obtaining reliable data.

One special though indirect result of the inquiry would be, that those who were training in this country for medical service in India would be enabled to acquire with no little readiness a satisfactory knowledge of a certain class of diseases of the commonest occurrence in India, the components of which class they would have at once to treat on their arrival in that country, and of which no great amount of clinical experience can be obtained in England. Further, the inquiry would be of great use to the English, and indeed the Continental practitioner, in furnishing him with valuable guides for the more speedy recognition and the better treatment of the numerous cases of peculiar skin diseases that are imported into this country and elsewhere from India, a locality that abounds in material for scientific investigation as regards the influence of climate, &c. upon cutaneous disease.

It is also believed that the inquiry may tend to show not only what are the special diseases due directly to special climatic influences, but also the character and extent of variations in the same disease induced by differences of climate: for the same disease does undoubtedly vary in its characters in different countries.

The mode in which it is believed the objects above stated may be easily secured is by seeking the assistance of the scattered and able medical officers of India in the collection of facts, and by utilising and unifying the experience and opinions of these gentlemen.

The details of the scheme are so arranged as to remove, it is believed, some of the special difficulties under which the Indian medical officer labours in the prosecution of careful investigations into the nature and causes of disease in India; though in the face of very special obstacles it is surprising how good is the work done by Indian observers from time to time. Reference is specially made to the few opportunities afforded Indian medical officers of consulting libraries, of obtaining access to the multifarious periodical publications in which are recorded the most recent

researches of European pathologists, the difficulty of carrying about with them the necessary apparatus for minute and experimental inquiry, and the like.

The difficulties referred to will be in great measure lessened by giving a resumé of the latest researches and the opinions of European dermatologists relative to the various diseases to which it is thought desirable to direct attention, and by indicating the points of doubt which require to be cleared up, and the line of investigation which should be pursued in the future, for the further elucidation of the nature and causes of particular diseases.

By indicating the several points upon which information is specially needed, not only will the main objects of the inquiry be promoted, but the time and labour of Indian medical officers will be greatly economised.

The endemic skin diseases of India deserve to be studied for themselves for several reasons. They are a great source of distress to sufferers from their attacks. They are the indications in many cases of morbid action, affecting the body profoundly and arising from preventible causes, and when closely studied they afford valuable information as to the causation of disease. They crucially test the action of remedies, whilst a more correct and a wider knowledge of their causation will greatly tend to increase the confidence of man, be he European or native, in the power of hygiene and medicine to relieve suffering. It is not at all unlikely, moreover, from concentration of attention to this, till recently, but slightly cultivated branch of medical science, important facts may be gained as to the nature and action of remedies which are successfully employed by European and native medical men in India, but with which we are unacquainted in this country. Some of these remedies are frequently mixed up with inert or dangerously active substances which compose the lengthy prescriptions of the hakeem, in such a way as to render it a matter of difficulty to determine what the active powers of these medicines individually are. Already something has been done to define them by medical officers in India, who would be glad to see them attract the attention of the therapeutic student in Europe. Many good drugs have still to be recognised, and they are considered too valuable to be disclosed by the native practitioner. With the progress of education, however, it is to be hoped the prejudices that lead to secrecy will be overcome, and the real or pretended powers of many undisclosed remedies be fully understood.

B.—Scheme of the Inquiry.

The scheme is, as before indicated, to give under the head of particular diseases, a brief statement of the views of the leading European dermatologists as to the nature of these diseases, to indicate doubtful points and the chief questions to be now determined in regard to them, and to ask for answers to precise questions.

The following heads indicate the kind and extent of information sought :—

a. The accurate observation of cases, especially with reference to the exact mode of origin of disease.

b. The microscopic characters of morbid products.

c. Precise information, inasmuch as climatic influences have much to do with the genesis of disease in India, touching the name and character of particular localities in which particular diseases prevail, with exact statements as to the nature and alliances of those diseases. [Situated as medical men are in India, in such varying climates and among such different races acted upon by such a variety of circumstances as to food and clothing, &c., they might gather valuable information about the geography of diseases of the skin. It is notorious that sharp lines seem to cut some diseases off from different parts of the country. This is the case as regards severe forms of itch, leprosy, Madura foot, Delhi sores, &c.]

d. The nature and peculiarities of the food and water supply of the affected population.

e. The tribes or castes in which particular diseases occur, and the habits of these tribes, ex. migratory or otherwise. [The influence of certain religious practices, such as that of covering the body with ashes and other earths, as followed by fakeers and religious medicants, deserves notice.]

f. The occupation of the attacked.

g. The dress of the attacked, especially with regard to the exposed or unexposed nature of the seats of disease. [It is an interesting question, for example, to determine whether the diseases of the scalp are more frequently developed in races that cut short their hair and shave their head, or among those who keep their hair long. Many of the Hindoos shave all or the most of their scalp, head, arm-pits, &c. The Mahommedans, as the rule, cut the hair close. The whole Seikh race never allows a razor or scissors to touch a hair. Many millions of Indians go about bareheaded; others wear only skull caps; others wear great masses of cloth like table-cloths on their heads. The influence of these practices on disease it is important to know.]

h. The observed differences between diseases as seen in the darker races of the East and in Europeans, and the differences in the same disease as observed in India and in Europe.

j. The connexion between season and disease. [Where the variety of climate is so great as in India, the observation of this point might lead to important results.]

The following are the diseases to which it is thought desirable that attention should be directed:—

1. Morphœa.
2. Scleroderma.
3. Frambœsia.
4. Delhi sore.
5. Keloid.
6. Fibroma.
7. The Elephant leg, or Elephantiasis Arabum, or tropical big leg.
8. The Fungus foot of India, or Madura foot.
9. The true Leprosy, or Elephantiasis Græcorum.
10. Leucoderma.
11. Pityriasis vesicolor in unusual forms.
12. Burmese ringworm.
13. Malabar itch.
14. Lichen tropicus, or prickly heat.

N.B.—The information which is particularly asked for, is essentially such as is precise. Mere general statements or replies to questions of a wide and general character are specially undesired. Special forms for note-taking and for tabulating cases are given with the accounts of certain of the diseases.

When sufficient data have been collected a special summary report will be made on the whole subject. Each observer will have full credit given him for any contribution he may furnish towards the report.

Morbid specimens properly preserved, if sent home, would be thoroughly investigated and the results embodied in any report made. Representations of diseases in the way of photographs, sketches, and coloured drawings will be very useful.

MORPHŒA.

This disease is in all probability of pretty frequent occurrence in the East, though mostly unrecognised. It is not unlikely that it is confounded with other affections of a similar nature, leucoderma to wit. There are no data in our possession at the present time to show that the disease is either rare or common in India; but *à priori* considerations, especially the asserted alliance of the disease with and the similarity of certain of its characters to leprosy, would lead one to expect that it will be found to be of not infrequent occurrence in that country.

Use and relation of the term Morphœa.—Morphœa signifies form. It is in reality the same disease as that described by Dr. Addison as Keloid, as in fact "Addison's Keloid," a disease wholly different from the Keloid of Alibert. It is most unfortunate that Dr. Addison should have employed the term Keloid to describe it, and that certain writers should have continued to the present time to designate it as "Addison's" Keloid. The latter term is now appropriated by general consent to

signify, in accordance with the original use to which it was put by Alibert, a fibrous outgrowth of the skin, *e.i.*, Keloid. As it is impossible to apply the term to two diseases of a totally different kind, the innovation of Addison must certainly give way to the priority of Alibert. The term Morphœa, therefore, as used by Mr. Erasmus Wilson, for the disease about to be described, is much the best.

Typical characters.—Morphœa in the European is characterised by the presence of circumscribed patches, varying in size from that of a pea to two, three, or more inches, whose surface is non-elevated, white like alabaster, polished, smooth, dense, and feeling and looking as though it were the seat of a deposit like white, or faintly yellow-white, wax. The disease is not an outgrowth, not a mere discoloration, but due to organic change in the skin structures. In well-marked cases the centres of the patches are more or less anæsthetic. There is generally a distinct halo of redness at the circumference, in the form of a lilac-coloured vascular ring, and this redness may be at times only very faintly marked, but at others more distinctly visible. The cuticle does not actually desquamate, but it shrivels up, being slightly discoloured and dirty-looking at times, but not always. The skin around the patches is often somewhat more pigmented than usual, and the whiteness of the patch may not be so distinct from such a cause: hence the terms *morphœa alba* and *morphœa nigra*. There may be only one patch or several. In a case now under observation the whole trunk is marked by large patches that look at a distance like large wheals with their white centres and red circumferences, but these patches feel firm and are constant and not itchy, so that their true nature is at once seen on careful inspection. The disease begins by slightly red spots, the centre of which presently levels down a little below the ordinary surface, then becomes white from the presence of the commencing deposit, the lilac ring gradually widening out in all directions as the latter augments. The seats of the patches are, in order of frequency: the back of the neck, the upper part of the chest, the mammary region, the abdomen, the upper part of the thigh and the arm, the forehead, and the cheeks. When the deposit has existed awhile, a stage of atrophy may ensue; the spots become thinned from the removal of the peculiar deposit and they waste, a scar-like state of tissue being left. This is the so-called morphœa atrophica. But the disease only follows the rule of certain new growths, ex., syphilitic tubercles, leprosy, and the like, in this respect; the atrophy is much more speedily produced in some cases than in others, and under these circumstances it may seem to be the most marked feature in the disease. Morphœa is mostly unilateral, it occurs in females especially, and such as are of weak constitution; and it is said oftentimes in pregnant women. It is very chronic, and disappears slowly after having lasted, it may be, a good many years.

Nature of the disease.—Morphœa is held to be a fibroid degeneration, involving the whole thickness of the skin; but more information is needed on this point, and specimens taken from the affected would be especially interesting objects for microscopic studies.

Diseases liable to be confounded with Morphœa.—The following have to be distinguished from morphœa: (1) In the first place, Leucoderma, which is merely white skin, resulting from deficiency of pigment in a particular spot or spots, *without any textural alteration* whatever. If the colour only be attended to, and the deposit feature be overlooked, error is likely to occur. (2) Secondly, Keloid, but this is an actual outgrowth of contractile fibro-cellular tissue, in which the elastic elements are unusually abundant; and (3) Thirdly, the early eruptive phase of Leprosy, as seen in the anæsthetic form. This is particularly referred to in the next paragraph.

Points to be cleared up.—These are chiefly two; (*a.*) the connexion between Morphœa and Scleroderma, *i.e.*, the hide-bound disease; and (*b.*) between Morphœa and Leprosy.

In regard to the first point, it is important to observe that in England the morphœa is observed as the early stage of scleroderma (which will be described next in order), not always but not infrequently. Some think that morphœa is in reality a circumscribed scleroderma, or scleriasis, as it is sometimes called. With reference to the second point, it may be observed that in leprosy certain anæsthetic patches with depressed centres, and it may be vascular edges, arising out of erythematous rednesses, or after bullæ or brown discolourations, are present, as described so admirably by Dr. Vandyke Carter. These discoloured anæsthetic

patches have been called morphœa, and the question arises whether these patches, and true morphœa, are one and the same in nature. It is probable that there is only similarity, not identity, between the two; in each case there is a new deposit that destroys the skin, and alters the pigmentation, atrophy following; but in leprosy we do not have the white waxy deposit from the outset, and as the whole disease, but clearly a new deposit of different character, accompanied by evidence of general nutritive disorder, involving especially the nerve trunks. We have a case under our care now in a lad from Demerara, who has large brownish patches on the face (both sides), and the arms, and extensive tracts of disease extending from the middle of the thigh to near the ankle, the boundary of the patches being slightly raised, dull brown, somewhat scaly, the general area dry, shrivelled-looking, dullish white, and feeling somewhat thinned: the sensibility being blunted over the whole of the diseased patches. The follicles of the skin appear as if congested, and as though they had been the seat of deposit, which had been almost entirely absorbed and had left behind a certain amount of atrophy. It is only when a pin is thrust deeply into the skin that the boy feels pain. There are one or two marked anæsthetic spots. There are little masses of deposit in the pharynx. The disease commenced by brown stainings, and appeared in the face nine months since. The boy has had "fever and ague" in Demerara. First came brown stainings, then thickening, then a certain "paling and shrivelling" of the skin, with a dark scaly extending edge, and the blunting of the sensibility of the affected parts. The disease is not psoriasis. There is no hyperæmia, no hypertrophy of the papillary layer of the skin, no free scaliness, no affection of the elbows and knees. The disease is clearly "leprous."

Questions suggested for replies.—The following queries are specially applicable to the case of morphœa:—

1. Is the disease common or is it ever observed in your district?
2. How many cases have you yourself observed?
3. What is the mode of origin and course of the disease?
4. The age, sex, peculiarities in the occupation and the food of the attacked?
5. The seat of the disease, unilateral or general? One patch or more?
6. Does the disease usually exist by itself as the sole disease?
7. Is it associated with scleroderma, *i.e.*, hide-bound disease?
8. Is it identical or only similar with the white anæsthetic patches of leprosy?
9. Is it accompanied by any disease of the nervous system, such as paralysis? and if so, is this only coincidently so?
10. Does it present any peculiarities when it occurs in the dark as compared with the whiter races?
11. What are the microscopic characters of the deposit?
12. What name do the hakeems give the disease, and to what cause do they attribute it?

N.B.—No attempt has been made as regards morphœa to prove or disprove its connection with the Leuke of the Greeks or the Vitiligo of Celsus. We have used the word morphœa as the modern and accepted designation, in a modern sense, for a well-known form of disease, the object being to get at the experience of modern observers, and not the views and opinions of the past.

N.B.—Any portions of skin affected with the disease may be preserved in a weak chromic acid solution (1 or 2 per cent. strength).

SCLERODERMA.

In the year 1854 Thirial first drew attention to this disease in a paper entitled "Du Sclerème chez les adultes." The subsequent names given it have been Scleroderma (Kretschmar, Schmidts Jahrb., vol. cxxvi); Scleriasis; Hide-bound disease; Scleroma, etc. Scleroderma seems to be the best, as it implies hardened skin, whereas scleriasis signifies rather the act or process of hardening.

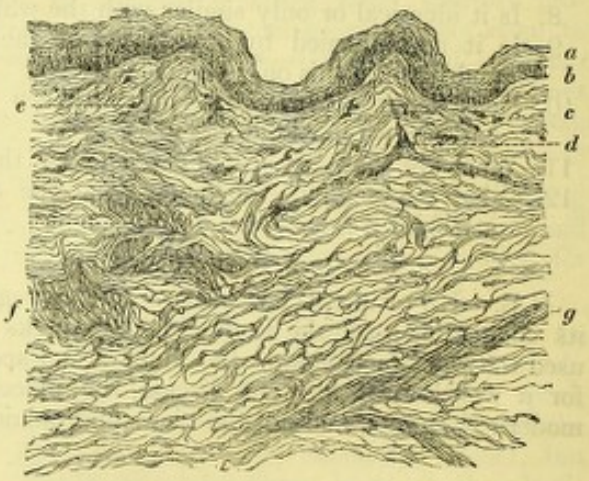
Typical characters.—These are readily given. At first there is stiffness in a part whose movements are thereby somewhat interfered with, ex., in the nape of the neck, where the disease frequently begins. Then come hardness or horny induration,

denseness but not pitting on pressure. The stiffness and induration may come on suddenly over a large area, and subsequently extend in bands or raised lines to a greater or less distance. The skin cannot be pinched up, and it is more or less immoveable over the subjacent parts. The bands or plates of induration may run down the whole back or along the entire length of a thigh or an arm. The diseased surface is yellowish or waxy-looking, and the hue fades away in colour through a dull white into that of the surrounding healthy integuments. There may be a partial boundary line of vessels at the edge of the disease. The deposit contracts somewhat, the skin becoming dryer, denser, more parchment-like, whilst there is much deformity produced, especially about the face and joints, when these are the seats of the disease. Sensation is not at first but only subsequently impaired. Several parts of the body are successively affected. Females are attacked more than males. There are sometimes one or more patches of morphœa present in addition, or the edges of the band or the indurated area may present the aspect of morphœa, being of a whitish hue, though raised. Scleroderma, when in bands or ridges, is distinctly raised. One writer remarks that "the appearance is not " that of a tumour, but rather as if the arm had been burnt and had left a leather- " like hardness, which required surgical operation as after a burn to remove it, or " it seems as if a bad erysipelas had become turned into cartilage and bone." (Lancet, 1855.) Dr. Fagge has found the liver tough and thickened, and its fibro-cellular elements increased. The induration in this disease oftentimes after some time disappears.

Nature of the disease.—The disease is supposed to be "fibroid hypertrophy of the " skin;" the papillary layer is intact, but bands of closely-packed connective tissue extend into it. The distinction between the corium and subjacent cellular tissues is lost. All the fibro-cellular elements are increased in amount, the fat is absent, the capillaries fewer than natural, the glands unaltered, and the nerves imbedded in the hypertrophied tissue. Now and then are seen collections of connective tissue cells in different parts of the corium.

The accompanying illustration is from Neumann's work, for the use of which we are indebted to Dr. Pullar.

Relation to Morphœa and Leprosy.—It is thought that morphœa and scleroderma are essentially one and the same disease, the one being the diffused, the other circumscribed form. They occur together in the same subject, hence this opinion is probably true. But we want more evidence on this point; see questions under the head of Morphœa. It is further a most interesting question whether there is any connection between leprosy and scleroderma. We need information on this point also, we have nothing at present but assertion to deal with.



a. epidermis. *b.* pigmented Malpiglian layer. *c.* dense fibrous bands. *d.* isolated vascular strand (pigmented). *e.* minute clump of pigment. *f.* transverse section of muscular fibres. *g.* muscular fibres.

The following queries are specially applicable to the case of scleroderma:—

1. Is it common or rare in India? Have you observed it?
2. Age, sex, and occupation of the attacked?
3. The usual seat of the disease?
4. Does it usually exist *per se?* Is it often preceded by morphœa? or conjoined with it?
5. Is it ever a stage of leprosy, or is there any reason to believe the two are related?
6. Does it offer any peculiarities as it occurs in the dark races?

7. In any post-mortem examinations made, what has been the condition of the internal organs?
8. The microscopic appearances presented by the diseased skin?

N.B.—In relation to morphœa and scleroderma, it is of the highest importance to remember in making a diagnosis, the admitted distinction between leprosy and these two diseases. Morphœa may be mistaken for the white anæsthetic patches of leprosy, and vice versâ, and scleroderma for the general thickening of the skin in leprosy. We assume, however, that leprosy on the one, and the two other diseases on the other hand have no relation to each other.

FRAMBŒSIA OR YAWS.

It will be interesting as well as important to learn whether this disease, which is common in Africa and Guinea, and from whence it is conveyed to the West Indies and America, is observed in any of our East Indian possessions. It is quite possible that the disease may have reached India lately through the return of the emigrant coolies who have resided for some time in the West Indian islands, and if any cases occur at the English dispensaries, it will be desirable to inquire into the probabilities of the conveyance of frambœsia from locality to locality by human agency.

A good descriptive history and representations of the disease, as regards its mode of origin, course, naked-eye and minute characters, are much wanted, and if Indian medical officers would furnish the same they would be doing medical science a great service.

General features and nature of the disease.—Frambœsia is said to be an eruptive disease of zymotic origin, commencing with malaise weakness and pains like those of rheumatism, followed by the development about the face, axillæ, arms, and genitals of small red papular spots more or less grouped, and varying in size from that of a pin's head to half an inch. The spots are slightly raised, and in some cases speedily disappear, whilst in others, the papules become pustular, give out a thin ichor, then enlarge, and become crusted over, the crusts hiding a raw surface remarkable for the exuberance of its granulations. The skin around is harsh and dry. Troublesome ulcers succeed certain of the papulæ, but are always superficial and heal at length without any scarring being left. Most of the granulating surfaces however take on a peculiar morbid action, whereby fungoid elevations acquiring a resemblance in a few weeks to raspberries (hence the term frambœsia) are produced. There are generally several of these sprouting masses together, joined by their bases as it were, but free at their apices, and usually one of them becomes larger, and is more prominent than the rest, and receives the name of mama or mother-yaw. It presently ulcerates and leaves behind a foul ulcer. The disease attacks children particularly, and lasts with them from three to six months, and in the adult six to twelve months. There is a somewhat curious but very general belief in Africa, that frambœsia takes the place of measles and scarlatina, which notion is in all likelihood merely an indication of the commonness of the disease. Frambœsia attacks the darker races more readily than the fair-skinned. It is said to be communicable from person to person through the inoculation of the discharge accompanying it, and so may be transmitted from individuals through the agency of flies. It is said moreover to occur but once in a lifetime. It is never fatal. Its nature is not understood.

Remarks.—One cannot help seeing that such diseases as Delhi sore or boil, Moultan sores, Biskra-bouton, and Aleppo evil, to be noticed in detail presently, have many points in common with frambœsia, provided we allow for the tendency in certain of the dark races to hypertrophous growth of the white fibrous tissue of the skin in and about wounds and ulcers, a tendency well marked in the case of frambœsia. If we recognise this tendency, it is not difficult to see in the origin of frambœsia from an indolent papule or a pustule, and the subsequent occurrence of unhealthy ulceration, the probable operation of some deteriorated blood condition, caused by climatic influences, as in the other diseases named.

In the Aleppo evil, when the tumour is single, we have the male variety, when there are several smaller tumours surrounding a large one, the latter is, as in frambœsia, called the female. But in the Delhi and Moultan sores there is no hypertrophy of the fibro-cellular tissue. Is it because a different race is concerned,

as compared with that which is attacked by frambœsia. Observers should not forgot the importance of bearing in mind, in the conduct of investigations, that the same skin affection, especially such as is of an ulcerative kind, may vary very considerably in external features, according as it occurs in different races.

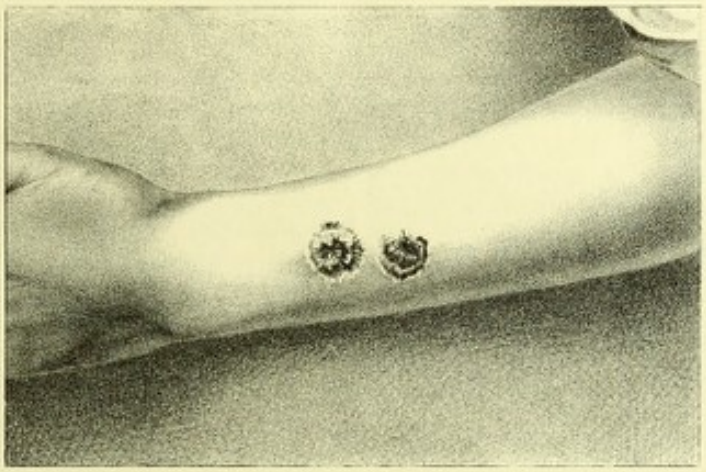
DELHI BOIL OR SORE.

Syn. Aurungzebe or Bulkea.

The Aleppo evil, Biskra-bouton, and Scinde boil will be noticed under this head.

The attention of India medical officers has been so frequently and distinctly directed of late to the disease, Delhi boil, that the present is a peculiarly fitting time to institute a specific inquiry into its nature, and to direct the attention of observers categorically to the more important and particular points in its history demanding investigation. The name Delhi boil is to some extent an unfortunate one, inasmuch as the disease is not peculiar to, though perhaps most prevalent at Delhi, but is known to occur in many localities in different parts of the East, ex., Scinde, Lahore, Moultan, Agra, Aden, Meerut, Roorkee, Umballa. The so-called Lahore, and it may be the Scinde boils, though there is doubt on the point to be presently noted as regards the latter, the Moultan sores, probably the Aleppo evil, and the Biskra-bouton (Algeria), are it would seem the same disease. It would not, however, be well to change the name before we are quite convinced by further investigation that these several diseases are of the same nature, and more light is thrown upon their pathology and cause. Some such term as Oriental sore or pustule might then be employed to designate the disease as it occurs in various parts of the world. The word Delhi sore is, however, preferable to Delhi boil.

Typical characters and cause of the disease known as "Delhi boil."—The disease has been described as commencing by itching, followed by the development of a reddish spot, in the centre of which appears a papule or two, giving rise to the aspect of a wart, or as it has been described, "a small hard pimple, which when first "seen has desquamating epithelial scales on its top." Dr. Fleming (Army Medical Report, 1869) depicts the original appearance as resembling "a musquito bite with "the skin slightly elevated, on examination a number of blood vessels are seen "radiating to the centre of this little red spot, which gradually enlarges without "any pain, throws off its epithelium, becomes smooth and flat on the surface, "assumes a shining appearance and a relative degree of transparency. The growth "slowly increases in size and often spreads irregularly to a considerable distance from "the centre by little ridges of smooth skin, and it would appear to attack the roots "of the hair and sheath first whilst it is extending. The growth or any of its "prolongations, pits on pressure and causes a stinging sensation, contrasting with "the healthy skin around." Others have described the enlargement of the original spot to be in part produced by the development of new papules around the original one, these papules being seated at the hair follicles. These new spots coalescing with the original one and themselves, and producing as above described an inflamed, brownish-looking, shining induration. When matters have advanced thus far, ulceration is imminent, and the surface may be seen to be studded over with deeply seated yellowish-white points, which have been regarded as points of suppuration, and ova, but are in reality altered and inflamed hair and gland sacs. Presently a scab forms by the aggregation of epithelial scales and a certain amount of ichor discharged from the soft centre of the tumour, and then ulceration begins beneath the scab, especially if the "boil" is irritated. There is some slight variation described by observers in the early condition of the boil; for example, it is said that before the scabbing takes place the papule may suppurate or give place to a small abscess, and this we can easily understand. What is always found present is the ulceration going on beneath a crusted pustule. As before observed, the discharge and crusting rapidly augment with irritation. The sore itself is surrounded by a zone of redness, and new papules develope around it, whilst the sore enlarges by ulceration, is very indolent, and fails to show for a long time any tendency to heal. In some cases the disease is altogether of a less marked kind than we have now described, there is no suppuration, less ulceration, and scarcely a cicatrix left behind after cure. The ulcerated surface itself, when present, is red, flabby, and irregular, being studded over by fungoid granulations that bled freely. The surface discharges a thin ichor and it is painful. Its edges are hard.



"DELHI ULCERS" produced by inoculation of peculiar brownish cells.—Plate III figs C & D.

22nd March. 1870.

Vincent Brooks, Day & Son, Lith.

Dr. Fleming (Indian Medical Gazette, Nov. 1869) particularly calls attention to the fact that during the growth of the tumour and up to the period when ulceration begins, and when the "boil" is relatively transparent and shiny, the small yellowish or yellowish white bodies before referred to as the hair sacs, may be detected with a lens. If these be cut out they will be found to be altered hair sacs, and they will sometimes come away attached to adherent scabs which are forcibly detached (See Dr. Cleghorn, Medical History of the Bengal Native Army for 1868, by Surgeon-Major Ross). As the ulceration advances, signs of healing show themselves in the centre of the original seat of disease, and cicatricial tissue springing up gradually spreads farther and farther outwards as the ulceration extends, and the sore finally heals after two or three months, a scar remaining. The accompanying Pl. I. is Dr. Fleming's representation of Delhi boil (Army Medical Blue Book, 1869).

The Aleppo evil, endemic about Tigris, the Euphrates at Aleppo, and Bagdad, begins, as far as we know, by a papulation which presently takes on a pustular character, then scabs over whilst ulceration goes on beneath, exactly as in the Delhi sore or boil.

Biskra-bouton, especially prevalent and endemic in the districts of Constantine in Algeria (see Paynter, Army Medical Reports for 1867, p. 438), and in other parts, ex., Morocco, commences by itching, then a small tubercle very superficially seated appears, and this enlarges, whilst the epidermis scales off, the centre often discharges a thin ichor, scabs and ulcerates, so that a disease like anthrax is produced. It is said to be like the Aleppo evil. This is what we are told.

Now in comparing Delhi boil, Biskra-bouton, and the Aleppo evil together, certain analogies are recognized. In the first place, they all attack the exposed parts, ex., the backs of the hands, the uncovered arms and legs, the backs of the feet, the nose, cheeks, and ears, &c. They all last about the same time, from several months to a year, or a little more. They similarly attack all ranks, ages, and classes, but especially new-comers, after a three or four months' residence; they all leave cicatrices, and so on. The Delhi boil attacks dogs, but this has not been stated of the Biskra-bouton nor of the Aleppo evil, though horses are attacked by the Biskra-bouton. However the inter-relationship of these diseases is a matter that requires investigation. Delhi boil and Biskra-bouton seem to be especially prevalent after rains. In each disease the general health does not suffer greatly, if at all, and the three diseases are known to have oftentimes a long period of incubation. They break out now and then a long time even after the removal of the patient from the places where the diseases are endemic.

[It may be here stated, in reference to the observation that Scinde boil is probably Delhi boil, that Dr. Farquhar's experience leads him to conclude that the ordinary Scinde boil is very different from Delhi boil. There may be the Delhi boil in Scinde, he allows, but the Scinde boil is, according to his opinion, a true "furunculus," a severe form of the boils that are so very frequent in the rainy season all over India. These boils are seldom met with till after the first fall of rain, and are in many places believed to be connected with the eating of mangoes. This supposition is apparently, however, a mistake, and arises from the fact of mangoes getting ripe and fit for eating just after the first fall of rain. Dr. Farquhar has seen these "rain boils" occur as frequently in districts of India where no mangoes were to be had, as where they are plentiful. The boils appear to be of a malarious origin: their strange frequency in the legs being explicable probably by the dependant position rendering the circulation torpid.

Europeans suffer more than natives from these boils, which are sometimes very trying to the general health from the pain they occasion. The inflammation will sometimes cover half the leg below the knee, and the induration be as large as a crown piece. At other times these boils are about the size of a sixpence. Dr. Farquhar has counted as many as five-and-twenty on an adult's leg in the middle of October, all more or less in an active state, and he has also known a Scinde boil kill a strong man through continuous sloughing of the core and edges; erysipelatous attacks supervening and exhausting the patient. Poultices favour the reproduction of these boils tenfold at times.

It is important to gain the views of others on the question of the nature of Scinde boil, and as to whether Delhi sores occur as a distinct disease in Scinde.]

[The two following are extracted from Dr. Tilbury Fox's work of Skin Diseases.]

Fig. 1.

Fig. 2.

Morbid Anatomy.—Most interesting observations have lately been made by Surgeon-Major Smith (Army Med. Rep., 1868, vol. x.) and Dr. Fleming (Army Med. Rep., 1869), as to the microscopic characters of the tumours and ulcers of Delhi sore. The former made out the presence of "a large number of peculiar "bodies, varying in shape from an elongated oval to that of a kidney or crescent-"form." These were of a dark chocolate brown colour as seen by transmitted, and of a bright orange red as viewed by reflected, light. Their average size was probably equal in length to 5 or 6 blood discs by about 2½ to 3 in width. They had distinct cell walls and were filled with minute dark granules, and varied much in transparency. They abounded not only in the discharges but all over the skin. Other cells were found in the discharge from open ulcers like *distomata*, full of granules in some cases, and in others having one end transparent, as though "being "thinned by protrusion and consequent tension at the moment when the spot was "first distinctly visible." On one occasion a curious animalcule was believed to have been discovered in the boil. The cellular bodies, as represented by Surgeon-Major Smith, are seen in Series I., Pl. II.

These bodies are probably the ova of distomata from impure water, according to Prof. Aitken, but it is not unlikely that some may be altered epithelial growths pigmented more than usual. They do not appear to have been generally observed.

Dr. Fleming has more recently done much to throw light upon the nature of the diseased processes in Delhi boil. When a section is made of the tumour before ulceration has commenced, the appearances represented at Fig. A., Pl. III., and Fig. R., Pl. IV. will be observed; and we beg to say here how greatly we are indebted to the courtesy of the Army Medical Department for the use of the plates.

The normal structures are replaced by a fibro-cellular tissue, enclosing in its interstices a large number of cells in masses, the sebaceous glands and sweat glands being destroyed, as well as the papillary layer of the skin. The cells make up the chief part of the boil at this time, *i.e.* before ulceration has commenced. They are seen at C and D, Pl. III., acted upon by acetic acid, and in Series III., Pl. II. They are oval or roundish, yellowish brown, the cell wall being soon destroyed by pressure, and they contain two or more nuclei. The cells are regarded by Dr. Fleming as the essential and peculiar growth of Delhi boil. But then there are very curious changes in the hairs. They appear to be the seat of cystic formations. The epithelial layer is so arranged as to give rise to an appearance of a fibrous envelope, and this encloses a finely granular matter. *See* F, G, H, Pl. III., and K, L, and M, N, in Pl. IV. Series II., Pl. II., are other representations of the altered hair-balls referred to by Dr. Fleming.

In commenting upon these appearances we would be understood as offering suggestions for the guidance of future inquirers. What is there, it may be asked, inconsistent with the idea that the cell growth is but a proliferation of the connective tissue corpuscles, an arrest in their growth, which gives rise to the formation of a kind of granulation tissue, which presently degenerates to a greater or less extent into pus; for the cells have the appearance of pus cells in many cases, so far as their characters are portrayed by Dr. Fleming in his drawings. One can easily understand that such a change may be induced in the tissue of the cutis as the

SERIES I.

These bodies are probably the ova of distomata from impure water according Prof. Aitkin but it is not unlikely that they may be altered Epithelial growths more than usual. They do not appear to have been generally observed

SERIES II.

——— = 100ths of an inch × 30

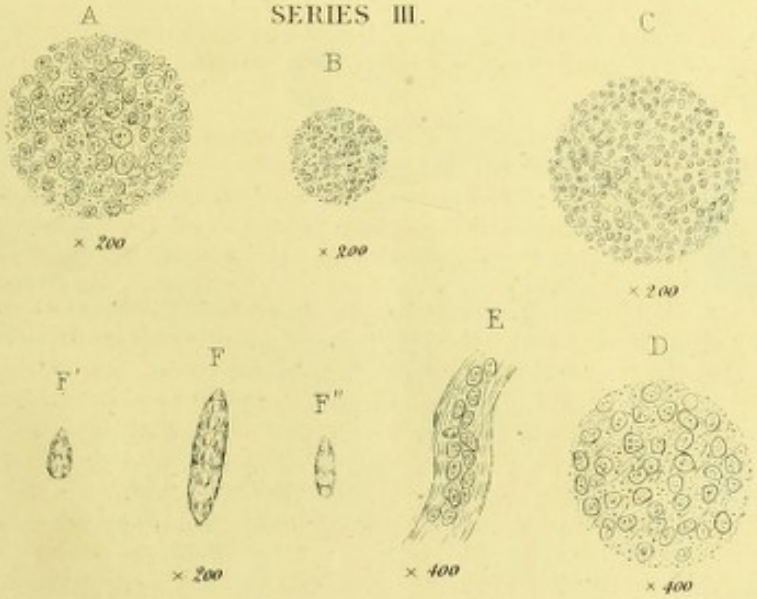
(Drawn by aid of the camera)

Figs. a and b, extracted from Delhi sores, January 1866

Figs. c and d, extracted from Lahore sores, May 1869

The above are representations of the altered hairbulbs referred to by Dr. Fleming These drawings are from specimens in the possession of Dr. Fleming and were obtained from different persons.

SERIES III.



——— 100ths of an Inch × 200

Fig. A ... *Pus from a "Delhi Ulcer" produced by Inoculation.*

" B ... *A + Acetic Acid.*

" C ... *A Section of the Tumour before Ulceration.*

" D ... *Part of C highly Magnified.*

" E ... *Shows a disposition of the Cells, observed in some Sections.*

Acetic Acid has been added in each Case Produced by Inoculation.

" FFF" *Bodies found in the examination of Pus from Chronic "Delhi Ulcers" magnified 200 diam^r.*

Vincent Brooks Day & Son Lith.

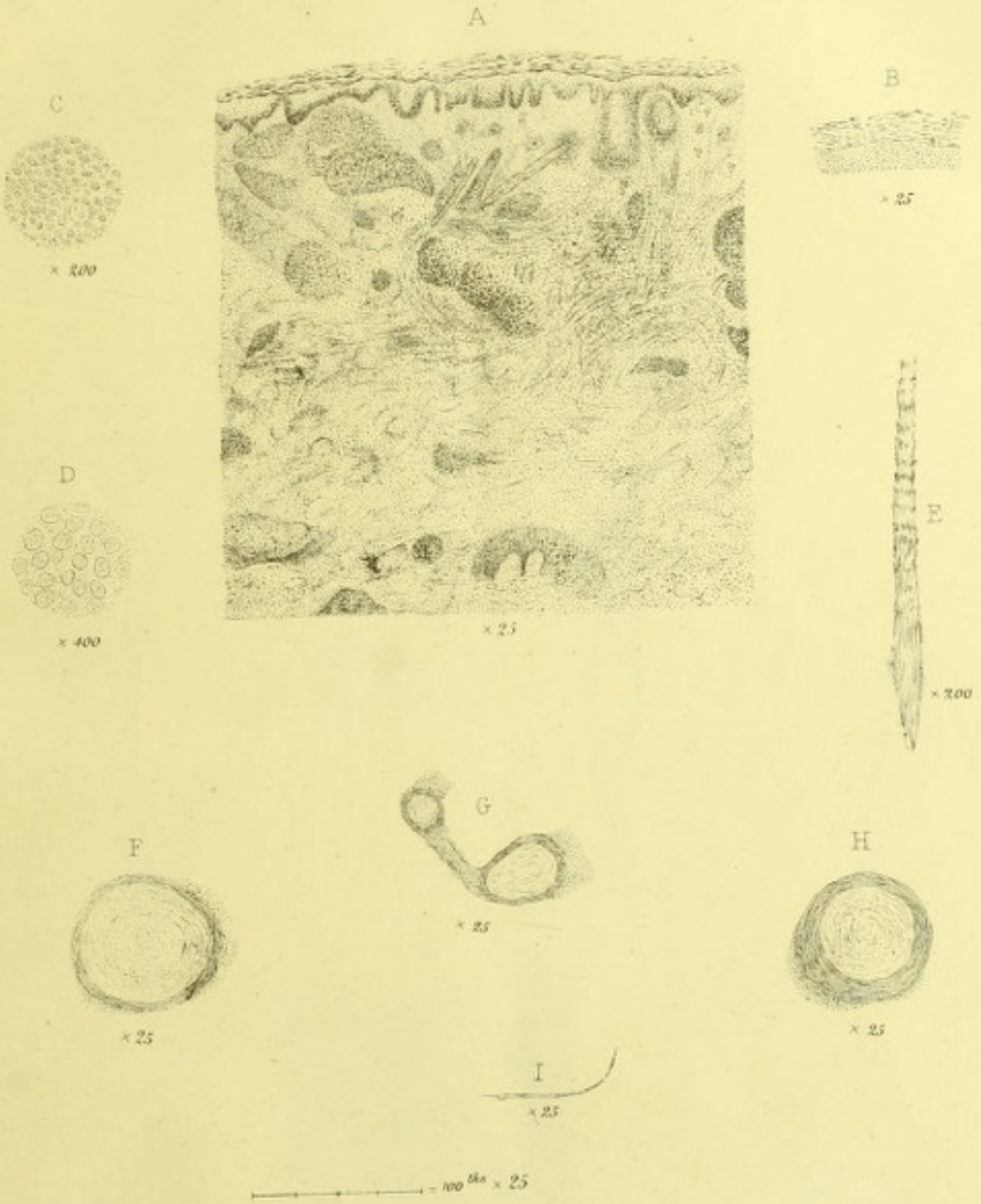


Fig. A..... *Vertical Section of a Dehli Tumour before Ulceration commenced*

B..... *The same in an advanced stage, showing the Papillæ destroyed*

C & D..... *Section of a Dehli Tumour after the addition of Acetic Acid, showing the peculiar Cell structure*

F G & H. *Cystic formations of the hair bulbs & sheaths*

E & I. *A fine hair from the interior of F, a Cystic formation.*

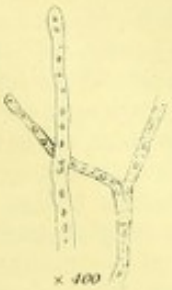
Vincent Brooks, Day & Son, Lith.

K

× 25

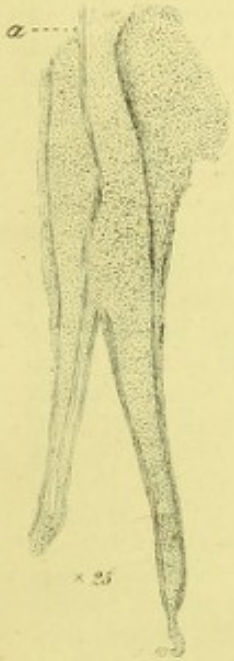
L

× 25



× 400

M



× 25

N

× 25

P

× 400

R

× 25

S

× 200

T

× 300

= 100th of an In: × 25

Vincent Brooks Day & Son, Lith.

result of impared nutrition. The so-called cystic formations in the hair are evidently due to immaturity of the cells that form the pith, the cortical part being less abundant than usual, a condition seen in other cases where the nutrition of the body is much interfered with, as for instance in syphilis.

It would appear to be a very important point to distinguish clearly, as a means of throwing light upon the true pathology of the disease, between the microscopic appearances observed *before* and *after* ulceration has occurred. There can be little doubt that after ulceration has occurred, and under the peculiar circumstances met with in India, ova fungi and other foreign bodies may readily be conveyed, by means of impure water and the like, to open sores, and so be found in the discharges therefrom, and it would only be the fact of finding these bodies or organisms in the tumours before they ulcerate that could be worth a moment's notice in proof of their being the cause of the disease. The finding ova and the like in the discharge of Delhi sores shows nothing more than that these have probably gained access from without to the discharging surface. Now Dr. Fleming's researches help us greatly upon the point in question. He gives us the characters of the tumour—a new granulation tissue—before there is any ulceration, and he shows that the new cell growth or tissue, if inoculated, will reproduce the disease. But if the cells were pus cells this might be explained by their possessing specific contagious properties, as much as those of gonorrhœal or syphilitic pus. The pus, however, from the Delhi boil will not, if inoculated, induce the disease; there must be with it some of the cell growth described by Dr. Fleming. But after all, the latter may be an early stage of pus, and it may be that in syphilis the inoculable material is not actual pus, but granulation tissue, which is present in chancres and syphilitic ulcers.

Pathology and cause of Delhi boil.—Firstly, it would be well to know the exact seat of the disease at its origin, if the disease affects the hair follicle, or a sebaceous gland, or is situated in the rete or papillary layer. Secondly, we want more information as to the characters of the cell growth described by Dr. Fleming, and as to its origin from the connective tissue corpuscles, escaped white blood cells, or the spindle-shaped cells existing normally in the rete as described by Biesiadecki, and which play such an important part in all inflammatory skin diseases. Specimens of Delhi boil might with advantage be sent home for examination. The disease would seem to be widely existent not only in India, but in Eastern cities generally, so that we must look for a common cause in operation over a wide area, not in anything specially peculiar to Delhi. It is clearly not connected with poverty—Dr. Murray's report seems to show this conclusively; nor does it appear to depend on malaria directly, since it is found to be absent from some of the most malarial districts. Then it has been ascribed to the bite of an insect, but of this there is no positive evidence. Delhi, it is true, is remarkable for its flies, but then Delhi sore is rare, whilst flies abound amongst the suburban population. Then the water has been blamed for the occurrence of the disease, and in two chief ways; firstly, in regard to its impurities, which it is said, taken internally, induce the disease, and secondly, in that the disease is averred to be caused by ova of insects, introduced beneath the skin from the water used for washing. The Biskra-bouton and Aleppo evil are said to be caused by bad water. If the cause be in the water, we must find some condition common to all cases of the three diseases, and the districts wherein they occur. In Dr. Murray's official report, reference is made to the remarkable immunity of a detachment of native cavalry drinking excellent water which they obtained outside the Lahore gate of Delhi. Can similar facts be observed elsewhere, viz., immunity of certain sections of the community who are using a special water supply? Dr. Fleming's observations on the microscopic appearances of Delhi boil before ulceration, in which nothing like ova were observed, would seem to set aside as untenable the doctrine that the disease was due to any parasite, and, as before observed, the fact of parasites being found in ulcers is no evidence that they are the cause of them, and it would be surprising if they were not so found in India. Surgeon Major Smith is the one who inclines to the opinion that the disease is caused by some parasite, and argues that they come from the water used for washing; but Mr. Alcock (Med. Times and Gazette, No. 22, 1870), meets this by saying that the disease does not prevail amongst the water-carriers, as in the case of the guinea-worm disease, which it would do if it were an animal parasitic disease, occasioned by the contact of certain waters with the skin.

But let us notice some other facts. Many observers agree in stating that Delhi sores are very liable to be immediately developed in the seats of abrasions, and that

small sores take on in India an ulcerative character like Delhi boils. Dr. Smith speaks of this as occurring in the chafed surfaces which occur in winter, in connexion with the wound of a dog's leg, &c. Mr. Cleghorn notices the same thing (Sketch of Medical History of the Native Bengal Army, 1868), and so does Mr. Alcock (Med. Times and Gazette, loc. cit.), who has seen "an accidental abrasion become "a specific sore within a fortnight." Whether similar occurrences are observed generally in connexion with the development of Delhi boils is a point to notice in future. There can be little question that disorder of the general nutrition induced by climate is one element in the production of Delhi boil and its allies. The parts attacked are those most exposed to musquito bites. After all it may turn out that simple boils, wounds caused by musquitoes, &c., because of the disordered state of health, take on the morbid action observed in Delhi boils. We are not without analogical evidence of disease being induced in like manner in this and other countries. There is a disease of the skin of common occurrence in England—*contagious impetigo*, in which the same kind of thing is observed. In those who are attacked by the disease, the slightest scratch is followed by the development thereon of the characteristic pustule, and other members of the same family, who are free from the disease at the time when that is epidemic, will often be attacked by it in abrasions of the surface. In the West Indies simple sores take on not a suppurative or ulcerative action, though they do this sometimes, but frequently are succeeded by an hypertrophous growth of the fibrous tissue as the result of climatic influences.

Again, from French sources we learn that the French in China suffered from a species of severe ulceration (to which the term Cochin China ulcer was applied), which was ascribed to climatic causes. It attacked at all ages, both sexes, and men of all kinds of constitution. It consisted in "ulceration following some lesion of "the skin, often the most trivial," the legs being most affected, the ulceration not deep as a rule, but occasionally severe and rapid. These and similar facts suggest the question whether after all Delhi sore is not a species of furunculus modified by climatic influences. But there are two other considerations that militate against the doctrine of its local, and in favour of its essentially constitutional nature; the one is the immunity which is the lot of old residents in districts where the disease is endemic—an undoubted fact; and the development of the disease a long time after removal from those places in which it occurs. It may be said, if the disease was of parasitic origin, one might expect a certain period of incubation, but certainly not so long as is recorded of Delhi boil and Aleppo evil, viz., a year or more. We can explain the attack specially of new-comers to a district upon the supposition of its being a constitutional disease, as well as upon the ground of its being a local affair. It by no means follows that because the disease can be cured by the destruction of the new growth described by Dr. Fleming, that therefore the disease is local, for the same happens with scrofulous and syphilitic sores, &c.

Points to be specially attended to in future Investigations.

A.—To define the localities in which the disease and its allies are found, with a view of defining their prevalence.

B.—Pathological cosiderations, viz.: The anatomical seat at the outset; the microscopic characters of the disease *before ulceration*; the nature and origin of the new cell tissue, whether it arise from the rete, the root sheath of the hair or the connective tissues: its exact relation to pus.

C.—Causation. Source and microscopic characters of the water used for drinking and washing; nature of the district, malarial, low, high, &c., in which the disease occurs. The prevalence of flies, musquitoes.

The following would, from what has already been said, appear to be the most important questions to be always specially answered in recording facts relative to Delhi boil and its allies. They may be ranged under two heads. A, as to locality and cause. B, as to the pathological nature of the disease.

A.—*Locality of Occurrence and Cause.*

1. Is the disease observed in your district; what is the name of the place or district; its situation and sea level? Is it a city, if so, the population and its character? Is it malarious? Are there any heavy rains at particular seasons? Is it at all drained?

2. Is the disease endemic or sporadic in the locality? Is there any evidence that it is imported? What is its relative frequency?

3. What are the prevailing diseases? Are ordinary sores, wounds, or ulcerations prone to take on an unhealthy action which *à priori* one is not inclined to expect?

4. The source and nature of the water supply used by the population? The peculiarities as to source or kind in that used by those who are attacked or who peculiarly escape?

5. Are the attacked new-comers; and if so, what is the average duration of residence before attack? Are long residents secure against attack? The age and rank of the attacked? Are water-carriers specially attacked?

6. Are animals attacked, as the dog or the horse?

7. The character of the grain used as food?

8. Do you note the existence of flies or musquitoes in abundance in the neighbourhood?

9. Are furunculi common in the district and among the population, and do they show any differences from those observed in England?

B.—*As to Pathological Nature of the Disease.*

1. The seat of the Delhi sore or boil in order of frequency?

2. Does the development of the disease follow the receipt of injuries or bites, and in what proportion of cases have you observed this? What is the nature and extent of such injury?

3. Describe briefly the first stage and the general course of the disease?

4. What are the microscopical appearances of the boil before ulceration, as compared with those observed after?

5. The nature of the peculiar cell growth observed by Dr. Fleming? The differences of the cells from pus cells? Are there transitional forms between these and pus cells found in the discharge?

Table for recording Observations relative to Delhi Boil.

Locality. (*a*) Name? (*b*) Situation? (*c*) Malarial? (*d*) A city or not. (*e*) Population, character of? (*f*) Heavy rains at particular seasons.	Is the disease endemic? Any evidence of importation. Relative frequency. Is it epidemic after heavy rains?	Prevailing diseases. Are ordinary wounds prone to unhealthy action? Are furunculi common?	Source and character of water supply of affected portions of the population.	Average duration of residence of the attacked? Are they always new-comers? Occupation of attacked?	What, if any, animals are attacked?	Character of food (grain) of district.	Common seats of the disease? Does it follow injuries? Kind, and frequency?	Early stage of disease.	Microscopical characters of the disease before and after ulceration has commenced.	The nature of the cell growth observed by Dr. Fleming?	Other remarks.

N.B.—Each observer would prepare an enlarged sheet according to this plan and number the several cases in order.

KELOID.

Very little need be said in reference to this disease, because its characters are so very well defined, and there is no reason to think that it is liable to be confounded with any other affections. It must be distinguished carefully from scleroderma and morphœa on the one hand, and fibroma on the other. So-called Addison's keloid is the morphœa before described. We are now dealing with Alibert's keloid.

It is usual to describe two forms, the true or idiopathic; and the false or traumatic keloid or keloid of cicatrices. In true keloid, or kelis as it is termed, but more properly idiopathic keloid, the seat of the disease is not as in scleroderma, the sub-cutaneous tissue, but the corium itself, the morbid change consisting in hypertrophy of the white fibrous tissue of this part, forming a distinct, raised, well-defined tumour; at orst pale, and then pinkish and shiny, and oval in shape, possessed presently of fiffshoots of fibrous tissue like the claws of a crab, which contract and produce distortion. Kelis does not ulcerate, is unaccompanied by enlargement of the glands, and is not destructive to life. There may be one or several growths, and they may be small and scattered. False or traumatic keloid springs up in the cicatricial tissue of all wounds, as after flogging, burns, scars left by rupia, the application of acids, syphilitic ulceration, &c. The disease is simply hypertrophy of the white fibrous tissue with condensation.

It will be interesting to know the circumstances under which keloid occurs in India; with what frequency, in what races, and if there be any real differences observable between the true and false varieties, except in the presence of a traumatic exciting cause in the latter.

The representation, Fig. 3, Pl. VI., page 28, is that of an individual affected with traumatic keloid, or the keloid of cicatrices, and as the particulars connected with the case illustrate the nature of the so-called keloid diathesis, I give them in detail, as reported by Dr. Maury, in the Photographic Review of Medicine and Surgery, a new publication of Philadelphia, from which the illustration is taken. The collar-like growth round the neck is of course most unusual, and altogether an out-of-the-common occurrence. The structural characters are those of a dense and contractile tissue, quite different from the lax pendulous folds of pachydermatocele.

The patient, F. J., coloured, æt. 28 years, was admitted to the surgical wards of the Philadelphia Hospital, 26th May 1870.

When eight years of age a small abscess made its appearance on the anterior part of the neck, which, on being opened, discharged an ounce of pus. As the result of this abscess a well-marked induration followed at the original seat, which gradually extended in both directions around the neck. After nine years' growth it had half encircled the neck, and was about two inches in width. Professor N. R. Smith, of Baltimore, at that time removed the growth. The resulting wound healed kindly in six weeks. The line of the cicatrix, however, was speedily occupied by a hard, rounded ridge, which slowly extended and enlarged. Eighteen months later an accidental wound was inflicted by an axe on the posterior part of the neck. This wound also soon presented a hard, nodulated cicatrix, which crept around the neck to join the one from in front.

His master now carefully avoided punishment, as the least incisions seemed prone to take on this morbid action. Four years after this date a band of soldiers whipped him severely; each gash on healing was succeeded by the hard, elevated ridge, the result of the previous wounds.

Seven years subsequent to Dr. Smith's operation, the tumour was again removed, at which time it had extended round the entire neck. Three months were occupied in the healing of the wound, which also assumed the same morbid action.

At the present time there are 37 tumours of variable size. The large one resembles, in a marked degree, the ruffles worn in the time of Queen Elizabeth, The two original growths are now thoroughly blended and form one solid mass, touching at the posterior part of the neck. It measures 28 inches in its greatest circumference, and five inches in its perpendicular diameter. It is plicated, and has deep fissures separating the folds, from the bottom of which is exuded a thin, yellowish, offensive fluid. The skin is intact, not having undergone ulceration. There is little or no elevation of temperature, no pain when pressure is instituted, the mass being only painful from the weight. The entire mass can be moved without difficulty, thereby indicating only a cutaneous attachment. The sensibility of the skin is perfect, the presence of a fly being at once recognised.

The other tumours are situated on the back and right arm, and vary from the size of a pea to that of a medium-sized tomato.

In July 1870, two of these growths were removed for the purpose of microscopical examination, and also to observe the rapidity of recurrence. For one the ecraseur was used, for the other the knife; in both instances a portion of healthy skin tissue was removed. At this date, almost three months afterward, there is a well-marked tendency to the development of new growths in the same place.

The following careful microscopical examination was kindly made by Dr. L. A. Duhring:—

"To the naked eye, upon section vertically through the tumour, the cut surface "presented a structure close and compact in appearance, of a yellowish-white colour. "To the touch it was tough, resisting, and firm, with a certain amount of elasticity, "and upon pressure exuded a thin, pale straw-coloured liquid."

Microscopical examination.

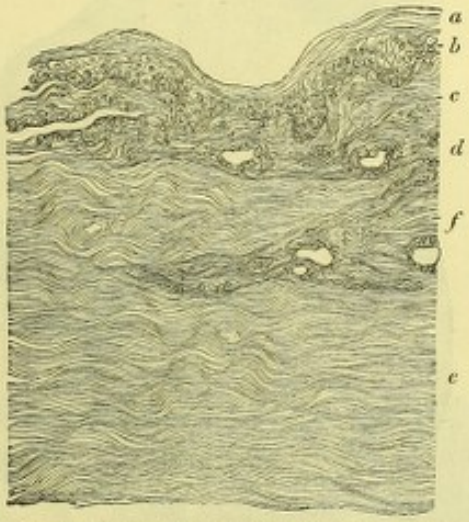
"After being prepared in solution of bichromate of potassa and alcohol, vertical "sections were made and examined in glycerine. The horny layer of the epidermis "was thin and scanty, the cells themselves being well broken up, and many of "them having undergone granular degeneration. The cells in the upper layer of "the rete mucosum seemed closely packed together and unusually numerous, while "the deeper layer contained the pigment cells well coloured.

"The mass of the tumour was composed principally of connective and elastic "tissues, the former being disseminated throughout, while the latter appeared here "and there in the form of good-sized, well-developed elastic bands, running both "transversely and vertically. Fat was found in some parts in fine globules. "Long, wavy bundles of connective tissue were seen running in striæ transversely, "just beneath the papillary layer. Here and there a cut sebaceous gland was "found.

"In some of the fields a loose network of connective and elastic tissue inter-"mingled, was present, with globules of fat. Connective tissue cells, long and "twisted, were to be seen, sometimes approximating each other and again "scattered."

The following is a representation from the work of Dr. Neumann of Vienna:—

The disease is supposed to begin by cell infiltration about the vessels of the corium, the change in the adventitia being marked especially at the edge of the growth, and at the part at which the arteries send offshoots into the papillæ, says Neumann.



a. epidermis. *b*. rete malpighii. *c*. tissue of the cutis. *d*. remains of the cutis. *e*. dense fibrous tissue of the keloid. *f*. cell infiltration around the adventitia.

Some special remarks on the pathological analogies and relationship of scleroderma, keloid, and other diseases will be found to follow the description of Fibroma.

The following queries are applicable to keloid:—

1. Is it observed in your locality, and with what frequency?
2. At what age?
3. As an idiopathic disease or secondary to lesions of the surface, and in what proportions respectively?
4. In what races is it observed?
5. Has keloid any relation to other forms of fibrous outgrowths from or degeneration, of, the skin? Is it found to be associated with the lax pendulous outgrowths of fibroma molluscum or the "scrotal tumours" so common in India?

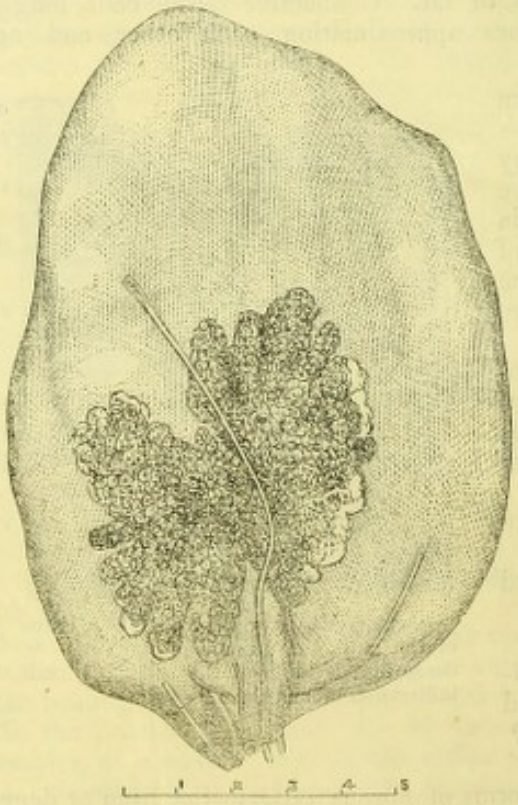
FIBROMA.

This is called by some writers Molluscum fibrosum.

General characters.—The disease consists in its fully developed state of little outgrowths from the surface, having all the characters of lax integument; they are

flabby, generally more or less pediculated. These tumours vary in size from that of a pea to that of a large fig and more. Sometimes the tumours are sessile. These fibromata are soft to the feel, and as before observed have the aspect of ordinary integument, but they are at times corrugated. The neck, chest, back, and more rarely the limbs, are the special seats of these tumours, which occur in elderly people usually. Mr. Wilson accurately describes the feel of these fibromata when he says that "taken between the fingers they often give the idea of a loose bag of integu- "ment, the looseness of the contained areolar tissue permitting of the inner walls "being rolled the one upon each other." These tumours are now and then flattened from the pressure exerted by the clothes. There is no contractibility about the growths as in keloid. The palms of the hands and the soles of the feet are almost always free from the disease. There was a very well-marked case of this form of disease of the skin in the Leper Asylum at Calcutta two or three years ago, we are informed. The accompanying representations, Plate V., sent to me by my friend Dr. Izett Anderson, of Jamaica, give a good idea of the varying size of the tumours and the extensive way in which they may be sometimes distributed over the body. The little tumours look, in the illustration, like those of keloid, but they are to the feel soft, flaccid, and flabby, and unlike the hard dense tumours of keloid.

The Pathology of Fibroma.—The disease would seem clearly to be an hypertrophous growth of the fibro-cellular tissue of the skin, especially that part of it which constitutes the dermic layer of the hair follicle. Dr. Beale settled this latter point as long ago as 1855. (Path. Soc. Trans., vol. vi., p. 313). The mass of the growths on section presents a surface with the aspect of fibrillating material enclosing collections of cells, as shown by Dr. Beale many years since. Mr. Howse, of Guy's Hospital, has lately carefully examined the tissue, and states "that there is not any "particular arrangement of this nucleated connective tissue, except that here and there "it was disposed in bars across the preparation; these bars were also occasionally seen "in transverse sections as circles, looking something like gland tubes or vessels, from "which, however, they were readily "distinguished by their structure "and the absence of any central "canal." Dr. Beale concluded that neither the sebaceous glands—which might be involved and destroyed—nor the sweat glands were concerned in the formation of these tumours, but that the cells at the deepest part of the hair follicle and of the follicle itself were principally concerned, and Mr. Howse confirms this, and locates the anatomical seat of the disease in the first instance in the two external layers of the dermic coat of the follicle. Dr. Fagge thinks that the sebaceous glands which are involved in the tumours are hypertrophied (see figure opposite from *Med. Chir. Trans.*), but neither Dr. Beale nor others allow this. Dr. Howse says, that in Dr. Fagge's case the glands were more sacculated than usual, and the acini more separated, but this was due to the growth of tissue between them, dividing them one from another.



Diseases resembling Fibroma. — There are instances of very lax pendulous outgrowths in which the integuments hang in loose folds, and in which the fibro-cellular tissue is increased, which resemble fibroma, except that the growth is not in distinct circumscribed pedunculated tumours. Valentine Mott called this disease *Pachydermatocele.* Wilson calls it *Dermatolysis.* The hard contractile sessile outgrowths of keloid could not well be mistaken for the lax, flabby, pedunculated tumours of fibroma which have the aspect of normal integument.

Plate V.

A single tumour

Back view

to face page 18

The points upon which information is needed as regards fibroma are very few and simple, and may be dealt with in the following queries :—

1. Is fibroma common in your district ?
2. At what age, in what sex, and it what races does it occur ?
3. Is it ever associated with morphœa, scleroderma, keloid, or scrotal tumours, or the "elephant leg" ?
4. Is it distinguishable, in your experience, from keloid by its anatomical seat or by commencing in the walls of the follicle and involving the glands, and by its structure, *i.e.*, by being composed of fibro-cellular tissue in which are loculi enclosing masses of cells, leaving the corium and cutis more or less unaffected, and not, as in keloid, being purely fibrous, originating in the corium, and having much elastic tissue in its composition ?
5. Does it appear to follow lesions of the skin ?

General Remarks on Fibroid Growths and Degenerations.

There are several of the preceding diseases—in which the changes in the skin apparently constitute the sole disease, and in which alteration in the character and quantity of fibro-cellular tissue is all that is present—that have a close relationship. We are much in want of information as to the relative prevalence of these diseases in India, for some observers have declared that there is a relationship or have tried to trace a resemblance between certain of these fibroid diseases and leprosy. Hebra even hints as much as regards leprosy and fibroma. If there be any connexion between these fibromata and leprosy it should be readily traced in India. European authorities generally disallow any connexion between the same.

A rough glance at the external features of these fibroid degenerations discloses in them a gradational aspect. In morphœa we have the whole derma replaced by a low-typed fibro-cellular tissue, which is not elevated above the natural surface, but which becoming absorbed leaves behind atrophy. In scleroderma the corium and cellular tissue beneath are involved ; there is more condensation ; distinct hyper-production of tissue, but as an infiltration with general elevation, and though it would seem that the kind of deposit is much the same, it does not destroy the skin, but becomes absorbed without leading to atrophy. In keloid the corium is specially affected, there is a more distinct outgrowth of fibroid tissue which has a contractile quality about it. In fibroma we have the same outgrowth, but originating about the hair sacs, and of a laxer kind altogether than in keloid, containing more cells in its composition and a less amount of elastic tissue, and this lax form of tissue reaches its climax in dermatolysis.

Then again there is one more relationship to be traced out, if it exist, in the case of fibroma, as illustrated by the patients whose photographs were given under the head of Fibroma. The scrotum in that case was hypertrophied, and presented, says Dr. Anderson, of Jamaica, the same appearance as seen in the common form of Elephantiasis arabum as it occurs in this island (Jamaica). It is heavy, rugose, semi-elastic, and slightly fluctuating to the touch, and if strong and long-continued pressure is made with the point of the finger a slight pit is formed. The orifices of the cutaneous ducts are very much enlarged, and would permit the entry of a fine probe. The penis is affected in a similar manner, and at its root is nearly three inches thick, and the glans is bent at a right angle to its body. This angular condition of the penis was produced only about nine months ago by the patient drawing back the prepuce and being unable to return it, a permanent condition of paraphimosis being thus produced. The prepuce thus reflected is much hypertrophied, and a portion of it is more than an inch thick. On the pubes several tumours exist distinctly defined, and also on the upper part of the scrotum ; but lower down they appear gradually to merge into the general elephantoid condition of the part. The fibromata were developed all over the body.

We might conclude from this account that the two diseases, fibroma and elephantiasis arabum (scrotal tumour) were related ; but I think that the concurrence of the two morbid conditions admits of another explanation, namely, that the two diseases occurred together accidentally in the same subject. Dr. Anderson further observes that some of the tumours were not lax and pendulous, but others hard and solid, the skin being adherent to the general mass beneath. He further states that, though not all, yet some of the tumours developed in the seat of scratches and cuts, though we are not told whether these were the soft or hard tumours. Dr. Anderson

concluded that the case was one of keloid diathesis, he did not appear to think there was fibroma present besides, for he styles the case Multiple keloid tumours; but if we have the characters of keloid, we have also those of fibroma in certain of the tumours, and those of "scrotal tumour," elephantiasis arabum, also. We give no opinion on this remarkable case, but ask for facts for or against the connexion, *quoad* coincidence or identity, between these fibroid degenerative ailments. We believe true leprosy to be wholly disconnected with them.

AINHUM.

The name "Ainhum" signifies "to saw," and is the term applied to a disease which is said to exist amongst the Africans; but Dr. Collas affirms that it occurs in India, and for that reason a note is given here. The disease consists of spontaneous amputation of the little toes, with hypertrophy of the amputated part, as shown in the figures given.

Fig. 1.

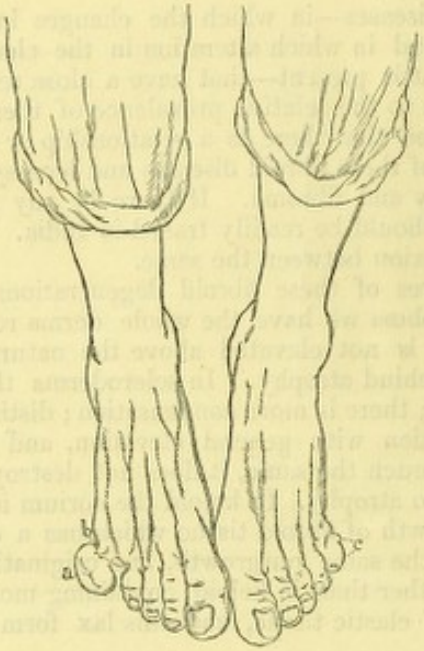


Fig. 2.

Fig. 3.

Fig. 1 shows the feet with the appearance of appendages to the little toes, copied from a woodcut in Dr. J. F. Silva Lima's paper in the *Gazeta Medica di Bahia*, for 1867, p. 149.

Figs. 2 and 3 show the appearances presented on section of the appendage.

Fig. 2. *a.* cicatrix of separation; *b.* joint between terminal and middle phalanx; *c.* remains of matrix of nail; *d.* bursal cavity; *e.* adipose tissue; *f.* pigment of rete mucosum.

Fig. 3. *a.* ulcer left by amputation leading down to bone *d*; *b.* remains of matrix of nails; *c.* ungual phalanx, joint structure unaltered; *d.* middle phalanx; *e.* adipose tissue of ball of toe unaltered; *f.* thickened cutis and rete mucosum.

A small semicircular furrow first appears in the digito-plantar fold, which gradually increases, without pain or inflammation, the toe enlarging twice or thrice its size, and getting loose and in the way. If the toe is cut off, the wound left heals very speedily. The cause is unknown. The general health does not suffer. The disease is symmetrical. The amputated toe shows fatty change of the tissues, enlargement of the areolar spaces of some of the bones of the phalanges, the bone tissue between the middle and proximal phalanges being replaced by fibrous tissue, the separation of the toe taking place at the proximal inter-phalangeal joint and not the metatarsal phalangeal joint: the cartilage and articular end of the middle phalanx being removed and replaced by fibrous tissue, which looks like an ordinary cicatrix. Information relative to this disease is greatly needed.

ELEPHANTIASIS ARABUM.

Syn. Elephant leg, Bucnemia tropica, Barbadoes leg; called also by Erasmus Wilson, after Mason Good, and so named in the Museum of the College of Surgeons of England, Spargosis.

Nomenclature.—There is a very unhappy confusion in the use of the term Elephantiasis at the present time. Most Indian officers, when they use the term "Elephantiasis," refer to the elephant leg, whereas in Europe, dermotologists signify thereby true leprosy, or Elephantiasis Græcorum, as distinguished from Elephantiasis Arabum, and the term is so used in the new nomenclature of the Royal College of Physicians. We may ask our Indian brethren not to use the term "Elephantiasis" without qualification, but to append to it the additional term Arabum when the "elephant leg" is meant, and "Græcorum" when true leprosy is signified. Much confusion will be avoided hereby. The use of the term Bucnemia tropica, or "tropical big leg," and when the disease attacks the scrotum, "scrotal tumour," would banish all confusion, and is to be commended, so as to get rid of the word elephantiasis altogether. Leprosy being called leprosy.

Description of the disease.—It is scarcely necessary to give a description at any length of a disease so well known. The disease usually attacks the lower limbs and is mostly confined to one, but may affect the scrotum, belly, breast, pudendum, and other parts. It is characterised by hypertrophic growth of the cellular tissue of the skin, giving rise to general enlargement and alteration in the aspect of the skin, so that it becomes tawny, hard, dark, livid, thickened, often scaly, and perhaps fissured, whilst by and by warty points appear, so that the skin looks and feels like that of an elephant. The general swelling results as the direct consequence of attacks of inflammation of the lymphatics, each attack being accompanied by "fever," increased swelling of the affected part, which does not subside with the disappearance of the lymphatic inflammation, but remains as a permanent increase of the disease. The malady consists of an hypertrophic state of the derma and the subcutaneous cellular tissue, both of which are infiltrated by a fluid rich in fibrine; the lymphatics being obliterated and the veins obstructed.

Points to be noted.—The circumstances under which the disease occurs, in reference especially to the question of cause which is believed to be malarial. The intimate connexion of the disease in its development with attacks of "fever" is remarkable, but more information is needed as to the nature of this fever. The habit of "squatting" in the damp is blamed in Bengal for predisposing to scrotal tumours. But this, together with malarial fevers, are so universally prevalent that we may perhaps look to other influences as the true cause of bucnemia tropica.

The following queries seem all that are necessary to be put forth here:—

1. Is the disease common or not in your district? Is it sporadic or endemic? Does it occur in residents and natives of the place?
2. The sex, and occupation of the attacked? The part of the body attacked?
3. Is the district malarial?
4. Character of the water supply as regards organic matter, especially such as is used by the attacked?
5. Do febrile attacks always precede the onset of the local swelling or its augmentation, and is the latter proportionate to the frequency and severity of the febrile attacks? The nature of those febrile attacks?
6. Do keloid and fibroma ever co-exist with bucnemia tropica or scrotal tumour, in your experience; and if so, with any frequency?

MADURA FOOT, OR FUNGUS FOOT OF INDIA.

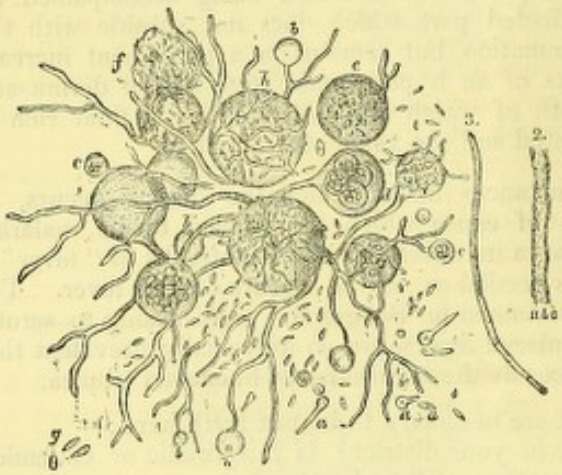
Synonyms.—The other terms applied to the disease are Mycetoma, signifying the causation of the malady by a fungus; Ulcus grave; tubercular disease of the foot; Morbus tuberculosis pedis, and Podelkoma.

Description and general remarks.—A good deal has been written and said about this disease, from time to time, by Eyre of Madras, Ballingal, Bagunjee Rustomjee, Day, Vandyke Carter, Minas, W. J. Moore of Rajpootana, and Dr. Bidie, and sufficient to make us well acquainted with its character. Dr. Carter was the first to discover a fungus in the disease, and to suggest that as the cause, but it is difficult to gather from his writings in how many instances he has detected the fungus; and other Indian officers, save Dr. Bidie, do not appear to have met with it. We have had the opportunity, through the great courtesy and kindness of Dr. John

Shortt, of Madras, of carefully examining several excellent specimens of the disease which he has sent home, and have in only one case detected the fungus. The disease when fully developed consists of marked swelling of the affected part, generally the foot, though it may be the hand or the shoulder even, it is said, which is studded over with little soft buttony elevations about the size of a pea, having a central aperture leading into a sinus. The buttony enlargements are studded over themselves with little black grains or masses like fish-roe, which also collect about the openings of the sinuses. From the sinuses are discharged black and white particles, with thin sero-purent fluid. The accompanying representation gives some idea of the appearance presented by the foot attacked by the disease.

Now if we make a section of a diseased foot, what do we find? Dr. Carter thus sums up the appearances:—"General confusion of parts owing to absorption of the "bones and fibrous thickening of the soft parts; often the presence of granules, sepa- "rate or aggregated in mulberry-like masses of a yellow or brown colour, lodged "in spherical cavities excavated in the bone or in the soft parts, or in tunnels or "channels leading from the cavities to the apertures on the surface, also lined by "membrane. These granules are "present in the discharge; some- "times there is a deposit of "fleshy (may be reddish or dark- "coloured) substance, contain- "ing numerous minute particles "(white or red) and occupying "the same localities as the above- "mentioned granular deposit. "Lastly in the same localities "we find black granules, sphe- "roidal tuberculated masses of "the same colour, radiated in "structure, which have been "mistaken for melanosis or blood "clots." These black masses are the fungus. The accompanying is Dr. Carter's description of the red fungus.

Chionyphe Carteri, from Dr. Fox's Work.



The original drawing of Carter reduced to $\frac{1}{4}$.

1. *Red Fungus* which grew on the surface of the fluid covering the portions of a foot affected with the "Black Fungus," magnified to show its development from the germinating sporidia, *a*, *a*, *a*, to the formation and bursting of the spore *f*.

a, *a*, *a*, *a*. Germinating sporidia. *b*, *b*, *b*. Commencement of spore-cells containing nucleus. *c*. Nucleus and contents of spore-cell further advanced. *d*. Apparent quadruplication of contents of spore-cell with further subduplication of their interior. *e*. Spore and sporidia formed. *f*. Spore bursting. *g*. Sporidia more magnified to show shape and nucleus. *h*. Spore embraced by a condensation of small filaments, very common, if not constant.

2. Felt-like form of the layer of Red Fungus as it grows in the bottle. *a*. Filamentous layer. *b*. Layer of spores. *c*. Filamentous layer below.

3. Filament to show that it is composed of cells with a nucleus in the upper end of each.

Filaments $\frac{1}{2400}$ in diameter; sporidium, $\frac{1}{2400}$ long.
Spore, largest piece, $\frac{1}{1200}$ in diameter.

We can quite confirm Dr. Carter's description as applicable to certain cases, but we have very carefully examined other specimens in which there was one feature entirely wanting, viz., the presence of the black granules and masses in the spherical cavities, all else being the same, even to an abundance of roe-like particles. Such a specimen we presented to the Pathological Society of London on the 19th October 1870, and it was referred for a detailed examination and report to Dr. Moxon, Mr. Hogg, and ourselves. We very carefully examined the foot and made the following report:—

"The soft parts of the foot are swollen; but the muscles are degraded and wasted, so that it is difficult to recognise them. The swelling arises partly from increase of the subcutaneous fat, and partly from the size and numbers of the canals. The several tissues are traversed in all directions by these canals, which branch and intercommunicate. The bones as well as the soft parts are pierced by them, but the tissue of the bone, even close to the walls of the channels, is quite healthly looking. The walls of the channels are composed of a soft greyish filmy substance, continuous with and not separable from the tissues around. Microscopic examination does not reveal any structure in this substance, except a few fibrils and a defaced

nucleus here and there. The contents of the channels are not connected with their walls. They correspond to the descriptions of fish-roe-like substance which is described as filling these canals in the second form of fungus foot, except that they do not show any pink colour. . . . There was no trace of structure that could be set down as that of fungus. The cells and fibres that Dr. Carter has described in the black matter of his first form of fungus foot we could not see any sign of. His opinion that the rounded bodies composing the fish-roe-like substance are made up of defaced fungus structure, coated with fringes of fat crystals, may be correct; but we must remark that if so, the defacement of the fungus character is curiously complete. On the other hand, these rounded masses (*see* Fig. 1, after Carter, natural size, colour, and appearance; Fig. 2, one of the masses magnified, ½-inch object glass), with their covering of subfilamentous material, have a very uniform appearance, such as suggest to us a less accidental nature than that attributed to them by Dr. Carter. The substance of the little rounded masses is softly granular, and has in some instances a texture of fine fibrils in it, like those of coagulated fibrine. The surface of each mass is rounded and its curve is perfect, but we cannot see any nuclei or cells upon it. The sub-filamentous material presents at first sight the appearance of a ciliated epithelium, as its component matter gathers itself into masses about the size of the cells, and these masses will separate and float about, but in them, when separate, there is no nucleus to be seen, but only faint fibrillation; in some instances these filaments are separated from each other. They are not acted upon by acetic acid, caustic soda, or potash of moderate strength. The filaments bend in a wavy manner, and appear entirely devoid of rigidity such as characterise crystals (*see* Fig. 2, above).

Fig. 2.

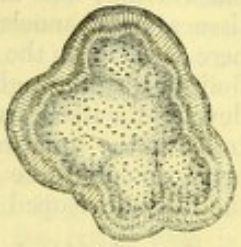
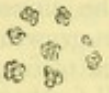


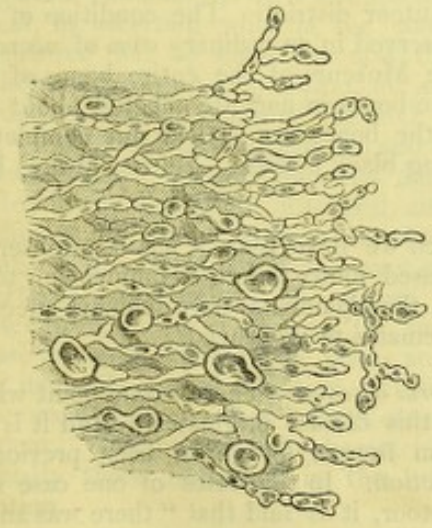
Fig. 1.



"We are of opinion that the nature of these remarkable structures requires further investigation, directed rather to their stages of development or of further transformation than to their minute structure. We think that their very constant and peculiar form, and especially the sub-filamentous covering of them, marks them as something more definite than perished fungus."

In a second specimen of the disease which we still more recently exhibited to the Pathological Society of London, black masses in abundance were present in

Fig. 3.



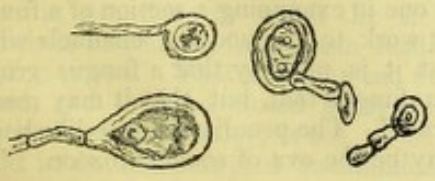
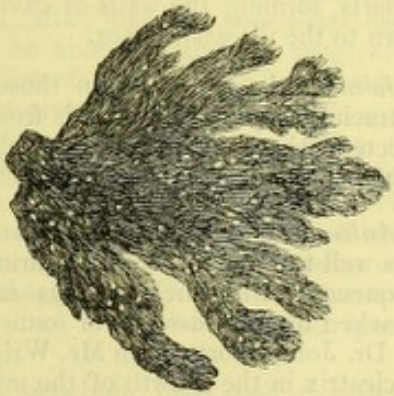


Fig. 4.



loculi in the foot, and these black masses were carefully examined by Dr. Bristowe, and found to be made up of fungus elements, having the characters of oidium. Fig. 3 is Dr. Bristowe's representation of the fungus as he saw it after boiling in potash; Fig. 4 gives the appearance of a piece of the black masses magnified 10 times. Fig. 3 is one of the terminal points of Fig. 4, magnified 420 times. There still remains the fact that in one specimen where the foot was disorganised there was no fungus at all. So that after all it may be that there are two aspects of the disease, one in which fungus is a complication, and the other in which it is absent.

The most striking difference between the two phases of fungus foot would appear to be the absence in one of the loculi (above and beyond the channels) filled with black truffle-like mass of fungus. The similarity is the perforation of the whole tissues by channels giving out the fish-roe-like masses. The question suggested here is this: Is the presence of the fungus an accidental phenomenon; and does it find its way through the sinuses running from the surface, and there luxuriating, develop for itself by its growth, loculi in the tissues? To determine this we need to learn the appearance of fungus foot in its earliest condition. But there are one or two points to be still further considered in regard to the appearances presented by the fully-developed disease.

Absence of black matter.—It does not follow that because we in England have found no black masses in certain cases, that they may not have existed to some extent in specimens prior to their being sent to this country; for in the instance which Dr. Moxon and we ourselves examined, and in which no trace could be found, black matter was discharged from the sores in the foot before it was amputated, so we were informed, and when the amputation was performed it was noticed that "the "medullary part of the bones just above the ankle was infiltrated with a black "fluid, the disease having extended up into the tibia." The nature of this black matter is uncertain. Is it fungus or blood, because some is certainly altered blood, and blood is sometimes discharged from the openings in the foot? And this leads to another very important point.

State of the bones.—In some cases where the disease has appeared to be confined to the bones of the foot, and where no sign of disease has existed in the integuments of the leg, it has been noticed that when the leg has been amputated just below the knee, "the bones were unusually soft and yielded readily to the "saw," and it is in such cases that grumous black fluid is found infiltrating the medullary substance of the bone shaft upwards towards the soft part. In other cases this accompaniment of the disease has not been observed. We especially refer to Mr. Wright's observations in the Guntoor district. The condition of the bones themselves is sometimes simply that observed in an ordinary case of necrosis. There is a fine specimen in University College Museum of the entire bones of the foot illustrating this point. In other cases the bones in part are affected about the neighbourhood of the joints; and in others, the bones are shelled out completely in parts, forming the walls of cavities enclosing black masses. Sinuses always lead down to the diseased bones.

Joints and cartilages.—In those cases which we have seen, it has been observed in tracing the disease upwards from very diseased joints to joints commencing to be affected, that the articular surfaces about the ligaments were chiefly affected. Whether this is always so in the early stage remains to be proved.

Antecedents as regards attack of Guinea-worm disease.—There is one point which it is well to refer to before inquiring how all this disease originates. And it is the frequency with which patients suffering from fungus foot have been previously attacked by guinea-worm or some similar affection. In the notes of one case sent by Dr. John Shortt, from Mr. Wright of Guntoor, it is said that "there was an old "cicatrix in the growth of the middle of the skin and a small fistulous opening on "the outer side of the calf of the leg, half way up. He had previously suffered from "guinea-worm." The idea that often strikes one in examining a section of a fungus foot is, that some entozoon must have been at work to produce the channels which riddle the foot through and through, and that it is unlikely that a fungus growth could produce such appearances as are seen in fungus foot, but that it may readily lodge and luxuriate in the channels already formed. The peculiar fish-roe-like bodies have given rise to the surmise that they may be the ova of some entozoon. The

point is worthy of close attention. It is different from anything we know at present, we may say, to find a fungus deeply penetrating solid tissues, channelling out sinuses, piercing bones, and producing necrosis, with destruction of joints, cartilage, and ligaments.

Mode of origin and cause.—How does all the disease above described arise? This is a most important question indeed. We are told that Mr. Bagunjee Rustomjee (Dr. Carter's paper), found "in the early stage little or no swelling of the foot; "the integments are natural in colour, or slightly congested and hot, having in the "surface elevations, which when burst or opened, allow a thin yellowish puriform "discharge to exude, containing granules like poppy seeds. The skin in the "plantar surface is irregularly thickened, and converted into knots at intervals, and "gives, on handling, the feeling of lumps." Another surgeon says, "the disease com-"mences by small irregular and somewhat painful swellings on some part of the "foot; it slowly increases, suppurates, bursts, or discharges a thin purulent matter." Mr. Moore, Rajpootana, says, "when the black variety of the fungus is present the "skin has a blue mottled appearance;" and again, "it appears as a small nodulated "swelling, presenting black particles beneath the integument, as if gunpowder or "Indian ink had been pricked into the foot." The fungus in the latter case is supposed to find entrance to the skin by a wound, as from a thorn.

We have the history of four of Mr. Wright's cases. In one, "about ten years "before the patient noticed a small boil or pimple on the sole of the right foot near "the toes; a few months after others appeared, but no further change occurred for "eight years, that is two years ago," when the whole foot swelled and became painful, and discharged a blackish matter. In the second case, the man "noticed "two years before a small pimple on the sole of the foot near the smaller toes, "which came into an open sore. Other sores then appeared and the foot swelled "generally with discharge of black matter." In case three, the man "noticed a "small blister on the inner side of the left foot, when the ankle began to swell and "sores broke out in different parts of the foot, discharging a glairy fluid and blood, "but no black matter it seems." In the fourth case, about 18 months before admission, he "noticed a small sore between the big and second toe of the right "foot; the toes then began to swell, fistulæ formed." Mr. Minas noticed in the case of the hand the first appearance to be a bluish discoloured swelling.

Now it will be observed here that we have no proof that the surface disease is not an evidence of deeper-seated disease—we do not affirm it is; but what we want is a careful examination of a whole foot when the early stages above described are present, to ascertain what is the condition of the deeper parts; for it will be remembered that we stated just now, that very serious disease of bone may exist, to be discovered during amputation, for instance, when no evidence of its existence is afforded by the condition of the soft textures covering it. If the disease begins from without, and travels inwards, and is produced by an external cause, then in the earliest stages we should find the deeper parts healthy and the superficial parts affected in the way above described, and we must be able to trace the progress of the disease in more advanced cases, from without inwards. It is clear that a serious amount of disease of the bones may exist without giving evidence of its presence by external appearances, and it has yet to be actually disproved, that when the sinuses form, and then open on the surface, this is not the result of serious deep disease primarily affecting the bones and the joints with their component parts. This point demands careful attention. It does not appear that the discharge in all cases is necessarily black, or that it must contain black granules, for it may be it seems very purulent. The microscopic characters of the discharge, and of the black granules, especially those imbedded in the integuments at an early date, demand special attention.

From what is known of fungi generally, we conclude that such free development of fungus, as is sometimes found in Madura foot, does not take place unless in situations to which the air has free access, or in other words, under conditions in which a fungus can readily gain access from without to the locality in which it grows. That a fungus can first find its way to the skin, and through some wound, and then be closed in by the healing process, lie dormant a certain time, and then rapidly develop, is not to be expected. We should anticipate that there would be an uninterrupted communication between the external air and the locality

in which the fungus develops, but our ideas may have to be modified. At any rate, we ought to be able to trace the first stage of the disease before the existence of free channelling through the tissues caused by the luxuriating fungus; for granting the existence of channels, we can readily explain the presence of the fungus as a frequent accidental phenomenon; we ought therefore to trace the earliest stages in order to determine whether the disease is due to the attack of a fungus.

There are no doubt certain considerations that lead, in some cases at least, directly to the inference that the disease is caused by a fungus. The receipt of wounds which subsequently become, as asserted, the seat of fungus growth, the free discharge of masses of fungus during the progress of the disease, and apparently *from the moment of formation of the sinuses*, the formation of the sinuses being apparently dependent upon the free growth of the fungus masses, the existence of black nodular swellings before any sinus has formed, the large amount of fungus found in the disease sometimes, the infiltration of the bones by a black fluid in the seats of the advancing disease, a fluid *supposed* to contain fungus and the occurrence of the disease in an unsymmetrical form and in a part of the body specially liable to come in contact with moulds of various kinds in the cotton fields, and the like. But yet it may be shown that very serious disorganisation may exist without a particle of fungus. It is on this point we want more evidence.

Points to be specially attended to.—These have been pretty clearly indicated in the preceding remarks. The first is, are there two distinct forms or aspects of fully developed fungus foot with extensive destruction of bone and tissue, but the one with and the other without the truffle-like masses embedded in loculi and tunnels? and if so, then this would go very far to show that the disease was non-parasitic, but only complicated more or less frequently by fungus growth which excavates for itself loculi? In order to determine this question, we want, secondly, exact information as to the constant appearance seen at an early stage, and on section of the deeper parts before the formation of actual sinuses opening upon the surface, which would enable us to determine whether the *deep* parts or the *superficial* parts are *primarily affected*, the skin and the subcutaneous tissue or the cartilage and medulla of the bones; in other words, whether the disease travels from without inwards or *vice versâ*. Thirdly, we want information regarding the minute character of the discharge as it *first* comes from the openings that appear on the surface, and of the small black particles that stud the integuments in an early stage, with a view to show whether fungus is present at the early stage, or only appears shortly after the fistulous openings occur, and also the power of the fungus to "bore" into the integuments. Suppose we do find fungus present at an early stage, and superficially placed with sinus, it may even then be accidental; and therefore the real point to determine is the second; that is to say, to disclose the existence or not of deep disease first of all without fungus; or the origination of the disease in the superficial parts, and its travelling from without inwards, in connexion with the presence of fungus elements. We append a form for "case-taking," in the observation and recording of cases, which seems to us most likely, if adopted, to secure the information needed.

Madura Foot, or Fungus Foot of India.

(Table for recording cases.)

A.—Clinical Facts.								B.—Pathological Observations.		
Number of case? By whom recorded? In what district? Date of observation.	Name, Age, Sex, Diathesis.	Employment in cotton fields, or swampy or damp places? Race and peculiarities? Are the feet of attacked habitually exposed?	Antecedents as to wounds directly followed by the disease, or as to prior occurrence of guinea-worm disease?	Is the disease symmetrical or unsymmetrical? What part of the body attacked?	First symptoms as stated by the patient? When did they occur, and what was the order of their sequence?	First signs as observed by the medical man from personal knowledge, and date of duration of disease up to time of present record.	Describe briefly the condition of the diseased parts.	Give the microscopical characters of— (*a*) The black specks in the skin. (*b*) The discharge at the earliest period when the sinuses first open on the surface, and subsequently noting any differences. Are the black specks present in all cases? N.B.—The character of the discharge at different periods is important.	The appearances on section of the diseased part in reference to— (*a*) The nature of the small discoloured nodular elevations that exist before the formation of sinuses, when such elevations occur. (*b*) The general aspect of the parts, whether it is true that in some cases there is free channelling through the tissues and bone, with great disorganisation, and free collection of fish-roe-like (white) grains, but not dark granules or masses in loculi, or large excavations filled with truffle-like masses. (*c*) The presence of a black-like fluid in the bones about the joints in the site of extending disease, and its nature (see next query). (*d*) The state of the joints and ligaments, and cartilage in the earliest stage of disease. (*e*) The condition of the bones in parts unconnected with channels. (*f*) The existence of black matter in parts absolutely without communication with the external air, as contrasted with parts directly communicating through sinuses with the exterior?	The microscopic characters of— (*a*) The fish-roe-like masses. (*b*) All black granules. (*c*) All black fluid, as showing that it is fungus or altered blood?

N.B.—The ninth column contains queries referrable to the discharge at different periods; the tenth to the minute changes in the diseased part; and the eleventh to the nature of foreign bodies. The object being to determine whether the general disorganisation of the fungus foot, that is to say, the infiltration of the tissues, the bone disease, the channelling of the tissues, &c., may be present without the fungus itself.

LEPROSY, OR ELEPHANTIASIS GRÆCORUM.

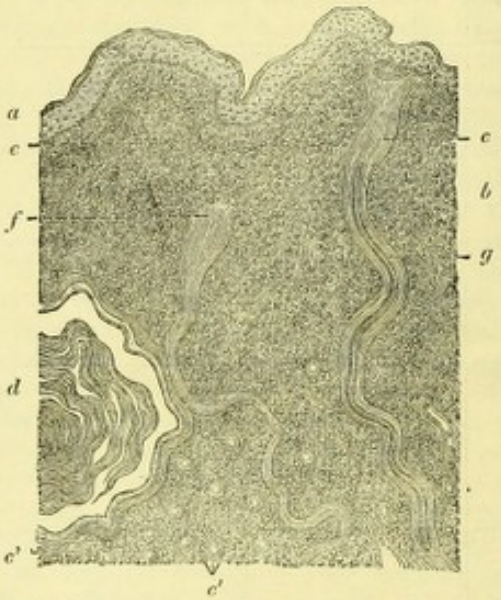
The Report on Leprosy issued by the Royal College of Physicians, and compiled from answers received from all parts of the world, has made us more perfectly acquainted than we hitherto were with the characters, the course, and the varieties of leprosy; and it has confirmed the prevailing opinion that leprosy exists in two principal forms, the tubercular and the anæsthetic, and that these are the same all over the world, with the exception that the one prevails more plentifully in one than another district. The pathology of the disease is fairly understood.

The accompanying figures, Plate VI., illustrate the features of leprosy. Fig. 1 is an eastern picture of a leper. Fig. 2 represents the face of a native of Jamaica, with well-marked tubercular leprosy. The illustration was sent to me by Dr. Izett Anderson of that place.

In the disease a granulation tissue is found deposited in the integuments and in the fibrous textures, and about the nerves and at times the nervous centres: more plentifully, and particularly in the nerve structures of the body in the anæsthetic, and in the skin and mucous surfaces in the tubercular form, a circumstance that sufficiently explains the peculiarities of the two forms.

Neumann has given an excellent illustration of the minute characters of a leprous papule. We give his figure, for the use of which we have to thank Dr. Puller.

Neumann found the papillary body elevated, the cutis thickened, the normal tissue replaced by minute cells, slightly expanded by acetic acid; so that in some parts there only remained a slight amount of normal tissue. The fat goes. At first it seems there are colloid cells in the corium, then aggregated colloid globules, and then the small cells infiltrate the whole cutis. Many observers believe the new growth begins about the hair follicles, and take the form of strands. "There is a continual production of small rounded "cells, between which the intercellular "substance becomes gradually more "scanty, so that between the cells "(arranged in groups and rows) are "seen only narrow strands, of somewhat striped substance, the nuclei of "which are rendered opaque by acetic "acid." In fact the cell growths invade the fibrous textures gradually to more or less defacement of them.



a. is the epidermis and rete. *b*. cutis with cell infiltration. *c*. groups of colliod granules. *c'*. colloid globules. *d*. disorganised and dilated sebaceous glands with accumulated contents. *e*. coiled hair follicle with atrophic hair. *f*. *g*. sinuous connective tissue strains, formerly hair follicles (?).

But the cause of leprosy is as obscure as ever, and upon this particular matter the "Leprosy Report" gives us very little satisfactory explanation, beyond illustrations of the general statement that leprosy disappears *pari passu* with an improvement in the hygienic condition and diet of a people, and the cultivation of land in districts where it has abounded.

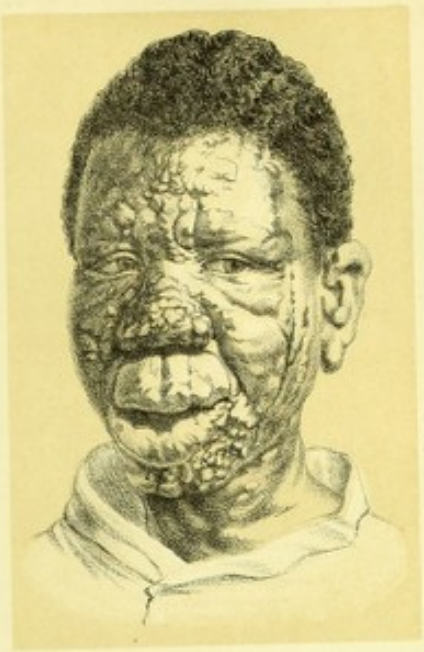
It is especially with a view of seeking information and promoting the collection of facts, touching the immediate cause of leprosy, that the following observations are offered. We have enjoyed unusual opportunities of seeing leprosy in its native haunts, and also have had many cases under our care in this country, and have paid no little special attention to the disease, so that we speak from a practical acquaintance with the disease and its surroundings.

Now in estimating the cause of leprosy we must be very careful to distinguish between its *production* and its *propagation*. This distinction is a vitally important one, for we may have leprosy merely *propagated*, and that extensively, in certain districts and under conditions, whilst we attempt to seek for its *origination* in the action of some malarial poison, or some peculiarity in the food of the people, or something outside the individual, and are so led completely astray and to wrong inferences. We might, in discussing for example the explanation of the cause of

to face page 28.

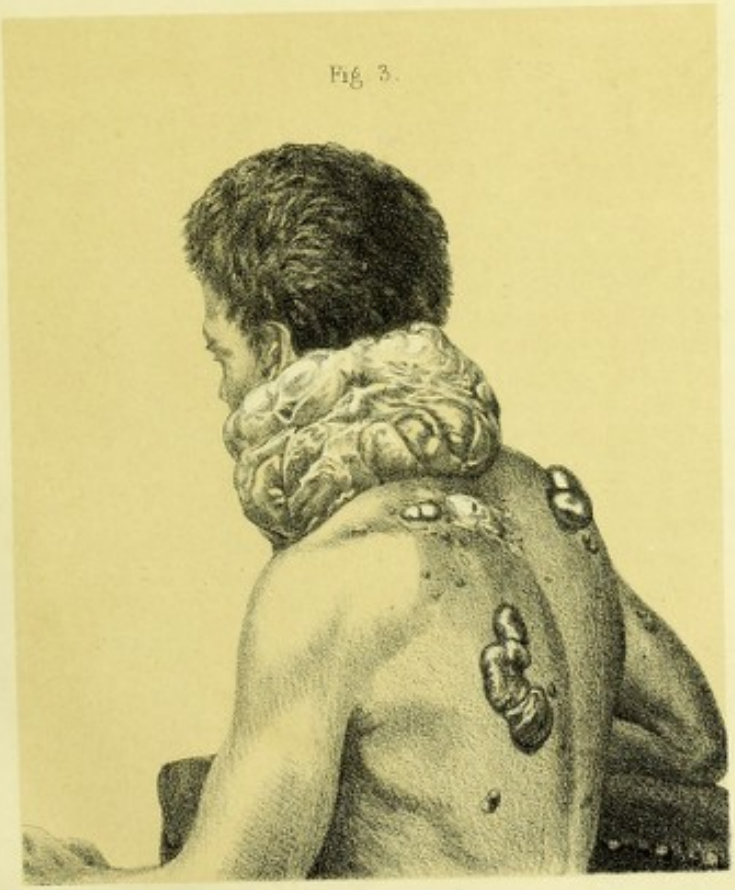
Fig. 1

Fig. 2.



LEPROSY

Fig. 3.



KELOID (TRAUMATIC)

leprosy in the fish-eating habit of the people of a certain locality, argue that this could not be the *cause* of the disease, because the habit was not observed in other districts where the prevalence of leprosy is common; but then leprosy might really be accounted for in these places by importation, or by the intermarriage of lepers or the leprous with the healthy. In fact, we might very erroneously come to reject a peculiarity of life or diet as an element in the causation amongst certain leprous communities, because it is not operative in other cases where the presence of the disease is really to be explained by hereditary transmission or importation. We have no doubt that in Syria the leprosy is mainly propagated by the intermarriage of the leprous or those hereditarily tainted by the disease. It seems to us then, in searching for the actual cause of leprosy, most important to determine in the first instance, in regard of any given place, whether the disease is only propagated or produced, and if partly propagated and partly produced, to what extent relatively.

A.—*Propagation of Leprosy.*

Now, the causes of propagation are mainly three:—

1. Intermarriage of the leprous or with the leprous.
2. Hereditary transmission.
3. Inoculation and cohabitation.
4. Vaccination?

First. As to intermarriage little need be said. It sufficiently accounts for the occurrence of a large number of cases of leprosy in the offspring of lepers. We mention it specially here in order to impress upon observers the necessity of making due allowance for its influence; and this leads us to refer—

Secondly, to hereditary influence, which is most marked in children who are begotten by lepers far advanced in the disease. Of 623 cases to which reference is made in the Leprosy Report of the College of Physicians, 287 were known to be hereditary, and it is no doubt probable that this is not a correct proportion, since leprous taints in families are as much as possible concealed.

Thirdly. As to cohabitation and inoculation. Of course these are not such potent causes as intermarriage and hereditary tendency in spreading leprosy, but still it is probable that they account for a certain numer of cases. The Leprosy Report of the College of Physicians tells us that "the all but unanimous conviction of the "most experienced observers in different parts of the world is quite opposed to the "belief that leprosy is communicable by proximity or contact." In a general sense and under existing conditions the view here taken may be correct; but there is by no means a slight body of facts which seem to show that the inoculation with matter from a leprous sore, and this may occur in cohabitation and constant contact and in vaccination (?), may give rise to the disease. It is certain that at present there exist certain conventional impediments to the occurrence of contagion which, so to speak, has no fair chance of operation. We have no right to conclude that leprosy is not contagious because it does not show this quality under present circumstances. As we have elsewhere observed, in order that we may conclude with certainty that the disease is not contagious, it would be necessary to remove all the impediments which have been raised by tradition, popular prejudice, and legal enactments, and which have kept lepers practically in an isolated world of their own, and to secure the freest intermingling of lepers with the healthy of the community (which does not at present take place), and then to observe no increased spread of the disease, before we could fairly say that leprosy cannot spread by contact. As Mr. Macnamara very pointedly observes, "That leprosy does not spread by contagion among "the natives of India is in itself an hypothesis, but presuming it to be a fact it "may be explained; for although lepers move about among their countrymen, they "are to a great extent isolated from them. Who ever saw a healthy native touch, "much less eat, with one afflicted with leprosy? In many parts of India the fact "of admitting a leper to a general hospital is sufficient to drive away every other "person out of it. The wealthy leper may purchase immunity from some of the "social evils to which his poorer brethren are exposed. But even he is frequently "obliged to leave house and home and to wander as an outcast over the face of "the earth, visiting shrines and holy places in expiation for his sins, which he "believes has been punished by the infliction of leprosy. Rich and poor lepers, "however, though living and moving among their fellow-men, are as a general rule "as isolated from them as were those condemned to the lazar-houses in the middle

" ages." But we may appeal to positive facts, showing that leprosy is apparently spread by the free contact of the healthy with the leprous in districts in which its appearance and spread can only be explained apparently in this way, and where in some cases the diet and *morale* of the people has marvellously improved and leprosy is not endemic in the district. Dr. Davidson, in speaking of leprosy in Madagascar, remarks, " It certainly deserves notice, that while the laws of Madagascar excluded " leprous persons from society, the disease was kept within bounds, but after that " this law was permitted to fall into disuse, it has spread to an almost incredible " degree. This is no doubt due in part to lepers being allowed to marry without " hindrance; but the natives are also strongly impressed with the conviction that " the disease is inoculable " (Lep. Rep. p. 221). It may be said that this is the result of intermarriage. Be it so. Then we refer to another very remarkable series of facts, which are contained in the appendix to the excellent pamphlet of Mr. Macnamara on leprosy, and are contributed by Dr. Hillebrand, of Honolulu. The disease was thought to be unknown in the Sandwich Islands till 1859, and, on close scrutiny, cannot be traced further back than the year 1852, or at the earliest 1848. Dr. Hillebrand has been at Honolulu since 1851. A recent census numbers the lepers at 250, or nearly 3½ per thousand of the natives, and he thinks this is below the average. The disease seems to have been brought by the Chinese in 1848. Here, then, the influence of hereditary transmission is out of the question. The disease arises in a clean nation; is unnoticed at first, and spreads slowly. And in no case can we better study the question of contagion. It so happens that the hygienic state of the natives and colony has improved, and not deteriorated. Animal food is within the reach of all. Labour is in great demand and well paid for. The natives are clad now like Europeans; formerly scantily, if at all. The climate is, perhaps, the finest in the world. Taxation is light. Yet, notwithstanding, leprosy spreads, and has spread from and around known lepers as from centres of contagion. Dr. Hillebrand saw the first leper in 1853, about twenty miles from Honolulu; in 1861 he had got very bad, and six other persons in his neighbourhood had become affected. The same thing was observed, in 1864, in another village, the tax-gatherer of which had been for years the only leper in the place. Dr. Hillebrand observes " that the natives are of a very social disposition; " much given to visiting each other; and that hospitality is considered as a sacred " duty by them. . . . About one-fourth avow contact with other lepers as a cause." Dr. Hillebrand gives the details of several very interesting cases. As candid and scientific inquirers, we cannot overlook the significance of such facts as these and the attacks of those who dress the sores of the leprous. Of course in such a case as that of Honolulu, where the disease is *propagated* apparently and not *produced*, it is no use looking for the *de novo cause* of leprosy.

It has been said that leprosy may be communicated by vaccination, but if so it must be infinitely rare and scarcely worthy of being taken into account. It appears, then, that in searching for the cause of leprosy we must make allowance for a large amount of *propagated* disease, through intermarriage, hereditary transmission, and contact with the affected; for, in fact, disease propagated from individual to individual. Having first, in regard to any particular district, determined its amount, we are then in a position to investigate the *production de novo* of the remaining mass of disease. Again we repeat it is of vital importance to make a clear distinction between the *propagation* and the *production* of leprosy, otherwise we shall be sure to miss or obscure the cause of leprosy, because we shall be trying to trace the operation of a like cause in the case of the two different classes of leprosy, and this will only make us miss the real cause of that produced *de novo*.

B.—*The Production of Leprosy.* (The cause.)

Now what may we conclude from a survey of the circumstances under which the cessation of leprosy in certain parts, as in England, took place? The Leprosy Committee of the College of Physicians in their report say: " This happy change " (the disappearance of leprosy) perhaps may have originated, and been continued, " from the much smaller quantity of salted meat and fish now eaten in these " kingdoms; from the use of linen next the skin; from the plenty of better bread; " and from the profusion of fruits, roots, legumes, and greens, so common in every " family. Three or four centuries ago, before there were any inclosures, sown " grasses, field turnips, field carrots, and hay, all the cattle that had grown fat in " summer, and were not killed for winter use, were turned out soon at Michaelmas

" to shift as they could through the dead months, so that no fresh meat could be " had in the winter or spring. Hence the marvellous account of the vast stores of " salted flesh found in the larder of the eldest Spencer in the days of Edward II., " even so late in the spring as May 3. In Lent, too, the poor used to consume " large quantities of fresh and salt fish, and the bread was made of barley and " beans;" which means that the cause of leprosy was one of diet.

But we may reply, why was not leprosy *produced* by the bad conditions above described? The quotation assumes that the peculiarity of diet was the *cause* of leprosy in England in the middle ages; whereas the presence of the disease was clearly, in greatest part, if not entirely, to be accounted for by its introduction through the Crusaders from the East, and its propagation by inter-communication of the leprous with the healthy. The extinction of leprosy was effected, we believe, in all probability by the enforced segregation of lepers, so sedulously ensured by the Church and State. The same events repeated themselves on the Continent. It is unsafe to draw final conclusions from our knowledge of past occurrences, and we had better study those of to-day to ascertain the cause of leprosy. The influence of climate and diet are the two points to which attention is and should be perhaps particularly directed.

First as to climate. Dr. Hobson, speaking to this point in commenting on leprosy in China, says very pertinently that the disease "exists in Norway and " Hindoostan, in the Arctic Circle and China, Iceland and New Zealand, the Cape, " Morocco, Mexico, Sandwich Islands, Borneo, Batavia, throughout Asia Minor, " parts of Russia, and Carthagenia," therefore in all kinds of climates, at all elevations, both inland and on the sea-board; hence it can scarcely be that climate, *per se*, has much influence. Of course leprosy is propagated not produced in some of these places, ex., the Sandwich Islands, but making due allowance for this, it is clear that leprosy is seen in all kinds and varieties of climates. We must look well to concomitants to explain the genesis of leprosy. But still the influence of climate does sometimes appear to accelerate or favour the occurrence of, if not produce leprosy. It will be noticed that those who are affected with the disease have had frequent attacks of "fever," and their general health has been much impaired. But the evidence we at present possess does not warrant us in saying more than this, or in asserting positively that the disease is of malarial origin. The connexion between leprosy and malarial poisoning may however be of the closest kind. Attention needs to be specially given to this point.

Next as to diet. This is of prime moment. It has been the fashion to ascribe the origin of leprosy to the consumption of fish in abundance, and as the chief article of diet, and fish moreover which is stale or bad. Others again have looked upon the consumption of rancid oil, others that of bad cereals, as the cause of the disease. Now as regards the influence of a fish diet, leprosy is very abundant in certain sea-coast districts and amongst fish-eating people. There is no question of this. In Egypt the natives feed on a beastly compound of semi-putrid fish called "fasciah;" in Norway again the consumption of fish is large, as also at the Cape, and in parts of India, &c. But on the other hand there are many exceptions in places where leprosy is endemic, in disproof of the theory of the causation of leprosy by ichthophagic habits. It is very advisable that we should have more facts on this point and with reference to the influence of the large and constant consumption of oil of a rancid kind. Another peculiarity of diet which may have great influence on the genesis of leprosy, is the absence of such vegetables as contain a large amount of potash. Mere poverty of diet will not suffice, as the case of Ireland very clearly shows, to produce leprosy, for in this country the wretched state of the population has not produced leprosy, probably in great measure on account of the abundant consumption of the potato. It is a curious fact, worthy of mention in this place, that leprosy has much diminished in Iceland since the introduction of the potato into that country. This statement is made on the authority of Dr. Hjaltelin, the chief physician of Iceland.

The use of grain grown on uncultivated land is a matter that demands every consideration. In England it is usual to hear the people of that great tract of country, India, spoken of as of one race, but it would be perhaps more appropriate to speak of the various nationalities of Europe as one people, than to believe that the Bengalee near Calcutta, the Rajpoot of Oudh, and the Puthan of the Punjab were one people. As to climate and modes of living, these races are also very differently situated. We find the Bengalee living in a humid, steamy, and malarious atmosphere all the year round, and feeding almost exclusively on rice and fish.

The Rajpoot lives on a hot level plain, which is dry and comparatively non-malarious for nine months in the year, but for the other three flooded with heavy rains, and feverish. His food consists of wheat made into unleavened cakes, and dhal, a sort of pea. The Puthan again lives in a more northern and elevated country, the winter of which is colder and more prolonged, while the rains are later and more uncertain. He lives principally on wheat, but eats butcher meat whenever he can afford to have it. Now amongst these people there is one frequently successful means of improving all the symptoms for a time or permanently holding the disease in abeyance, and that is a liberal supply of nourishing food; we are therefore led to look to the character of the general food used. In relation to this matter Dr. Farquhar's observations made in the Punjab some years ago, and subsequently followed up in other parts of the Bengal Presidency are of interest.

A grain merchant who came to Dr. Farquhar's dispensary in the cantonment of Sealkote, suffering from leprosy, stated a fact in regard to his food of much significance. The village he lived in was surrounded by cultivated land, the wheat grain from which he was in the habit of receiving for sale. In the neighbourhood, however, there were vast tracts of uncultivated land, over which a nomadic race, living in tents, wandered with their flocks. When rain fell plentifully, in any particular spot, at a propitious season of the year, these people set about turning up the soil, to the depth of an inch or two, with primitive ploughs, consisting of stout sticks burnt at one end and tied on to bullocks by strings.

The wheat grain was then cast into the ground, covered over, and in due time yielded corn. The area thus sown was too great for the few people to reap the straw from, so only the ears of corn were plucked from the stalks and gathered into sacks. The grain seller added, that he bought this inferior-looking grain from these people, and because his customers, as a rule, disliked it, he lived upon it himself, and sold all his good grain. The idea struck Dr. Farquhar that this uncultivated and poor grain might have something to do with the leprosy, and following up the thought, he found in other parts of the country, what appeared abundant proof of leprosy being associated with the consumption by the population of inferior grain. He found the disease to be common in another district in the Punjab, where a large uncultivated plain was near to a long line of villages, near a cultivated district. This plain was sown only at intervals of a year or two, when the rain fell, and no manure or other care, beyond the ploughing and reaping, as above described, was bestowed on the soil.

Leprosy Dr. Farquhar noticed was comparatively absent in those districts of India, where there was long-established cultivation of a higher order, where the fields are properly cared for and manured, and where man lives industriously by "the sweat of his brow."

In the rice country of Bengal, where cultivation has been long established, there appeared to be an argument against the "uncultivated grain" theory of leprosy; on inquiry, it is found, however, that the Bengal ryot exhausts his soil, by drawing yearly, sometimes three crops from it, and that the grain (rice) produced at one season of the year is known among the people to be unwholesome. New rice is also known to be deleterious, and is eaten only by the poor-ryots themselves, who live in extreme poverty, through the rack-renting practised by so many of the native landlords. The facts too, that diseases similar to those produced by ergot of rye, as by Kessaree dhal, &c., are common in India, suggests the advisability of our studying the character of disease in relation to different kinds of food. We see, for instance, in the Upper Provinces of India, where unleavened cakes of wheaten flour form the staple of the food of 60 or 80 millions of people, *calculus vesicæ* is a very common disease, while in the rice-feeding 30 millions of Bengal Proper, this affection is comparatively very rare.

The extraordinary statement also that in the "Patna" district of Bengal, leprosy exists in a large proportion of the population in a slight form, should be further and specially investigated and reported on.

It may turn out after all that the use of grain grown on uncultivated land is not of much moment in regard to the cause of leprosy; and that it is only coincident with residence in an undrained and marshy (malarious) locality, which also may have nothing to do with the genesis of leprosy. Some observers declare that the subsidence of leprosy and ague go on *pari passu* with the introduction and extension of drainage in localities in which leprosy has been endemic.

The late Dr. Kinlock Kirk supposed, as the result of his observation, that the use of the leguminous seeds, common in India under the name Dâl, is capable

of giving rise to something like leprosy, and especially in the case of the dhal derived from the cytisus cajan, and called "urhur." This is consumed by the poor under the idea that it enables them to bear great labour; it gives rise as an occasional meal to general disturbance of health and rheumatic pains. Some eat it constantly, and the final results are urticaria, sense of heat in the stomach, redness of the mucous surface of the mouth, bronzing of the skin, sponginess of the gums, burning of the hands and feet, dryness, harshness and cracking of the same parts, rheumatic pains, white spots indicating a leprous taint about the body, and lastly confirmed leprosy. Another dâl, the lathyrus sativus, we know, induced paraplegia. How far the use of dâl may be the cause of leprosy requires to be determined. But it must be recollected after all that leprosy may result not from the operation of any positive poison in climate or in diet, but negatively from the absence in the diet of certain principles, such as nitrogen and potash, and that it is accelerated by bad residence, uncleanliness, poor diet of all kinds, fever, and the like.

These remarks it is to be hoped will suffice to indicate the direction in which we should attempt to make out the *causa vera* of leprosy.

The following particulars, embodied in questions, seem to require elucidation in regard to the presence and cause of leprosy in different districts and individuals:—

A.—Leprosy in Districts.

1. The name and situation of your district?
2. The nature of the district, inland or sea-coast?
3. Is the disease endemic?
4. Is it an importation and only sporadic? How many forms do the natives recognise?
5. What is the nature of the climate? Malarial or not?
6. Is the disease on the increase or decrease? Give statistics if possible.
7. In what races and castes does it exist? and by preference? Is it frequent in Europeans?
8. The general character of the diet of the population as regards fish-eating, dâl-eating especially?
9. What is the extent of the cultivation of the soil? Is the grain used obtained from the district, or imported? If imported, is it of bad character? How much of the grain used by those in leprous districts is grown on uncultivated land?
10. Give any facts showing a gradual and related decline of leprosy in districts which have gradually undergone cultivation or in localities or communities into which the use of fresh vegetables, ex., the potato, has been introduced?
11. Give any facts showing that leprosy and "ague" have decreased in amount *pari passu* with increased cultivation and drainage of districts?
12. What other forms of disease are considered as akin to leprosy?

B.—Leprosy in Individuals.

1. What reason is there to believe that the disease in your district is propagated by intermarriage, cohabitation, and hereditary tendency, as distinguished from its being produced by some cause external to the individual, especially some peculiarity of diet or climate? What is the proportion of *propagated* to *produced* cases?
2. Will hereditary taint often account for the disease where it is supposed to be due to some dietetic mal-influence, or where this is not to be found as a probable cause?
3. In what proportion of cases has severe and perhaps repeated attacks of malarious fever occurred in the individual prior to the onset of leprosy?

LEUCODERMA, OR WHITE SKIN.

This affection, common in India, is simply a disorder of the pigmentation of the skin. It consists in the development of white patches, due to deficiency of pigment *without any textural alteration whatever*, no tubercles, no anæsthesia, being present. It has no relation to leprosy, nor to morphœa. It may be partial or general; when extensive it gives the individual a piebald appearance. The hairs of the affected part are often white. Several other diseases are confounded with leucoderma, because in them localised deficiency of pigment is observed, but they are associated with serious structural alteration. Such confusion should be avoided. The well-to-do natives of India suffer from leucoderma, and the question they put the doctor for solution is:—Is this leprosy? The hakeems are divided in opinion

about it. Leucoderma, as before observed, has nothing to do with leprosy. It may be as well to add that the deficiency of the pigment in the white circular spots of leucoderma may be accompanied by an excessive accumulation in the part around, so that we have a white centre and very dark areola,—a very unequal distribution of pigment in fact, but still *no structural alteration*. The general features of the disease are well illustrated by Plate VII., for the use of which we thank Mr. Hutchinson.

Is the disease more common in your district among the higher classes or the fair-skinned than amongst the poorer or dark-skinned people?

[Dr. Farquhar's observations lead him to conclude that the fair-skinned individuals among the natives are most subject to this change of colour, and that leucoderma is very common about Peshawur, where the inhabitants have very fair skins, being for the most part immigrants or the children of immigrants from the fairer tracts of Central Asia.]

PITYRIASIS VERSICOLOR, OR TINEA VERSICOLOR (Chloasma).

We do not mean to say that this is an endemic skin disease of India, and do not notice it as such, but only because it would seem to offer certain peculiarities when it occurs in India, and chiefly in that it is much more severe and extensive, than in England. As seen in England and in whites, the disease is characterised as a fawn-coloured discolouration of the skin, in small or extensive patches, of an itchy nature, giving off branny scales when scratched. These scales are invaded by fungus elements, as seen under the microscope. The explanation of the disease being more extensive and severe in India, is probably to be found in the fact that conditions in that country, the heat and moisture which abound more or less, are particularly favourable to the luxuriant growth of parasitic fungi. If the fungus grows at the outset of the disease rapidly and freely, in this country we observe the disease to consist of bright red erythematous rings. The red ring extends as the mycelium sprouts, whilst the centre pales, becomes fawn-coloured and furfuraceous, and this fawn-coloured area increases whilst the circumferential redness declines. It will be interesting to know if this is often observed in India. Again, we have noticed that the disease, when it has originated in India, has been so severe and extensive as to have been mistaken for syphilis on several occasions, the patches being more generally scattered and raised than usual, and being accompanied by redness and a deeper tint in consequence of the greater amount of fungus and irritation present. It should be remembered that syphilis never gives rise to extensive discolouration as the only development of the disease. Lastly, in pityriasis versicolor of hot climates, there is sometimes a good deal of pigment deposited, and it would be an interesting question to know whether the so-called "pityriasis nigra" is not so produced. We have seen one such case. These remarks are only intended as suggestive.

BURMESE RINGWORM.

In various parts of the East many local designations are given to ringworm of the surface of the body (tinea circinata as we have called it, and as it has been called in the new nomenclature of the College of Physicians). Hence there would appear to exist in different places peculiar diseases apparently different, but in reality one and the same in nature. Chinese, Burmese, Tokelau ringworm and Malabar itch are examples in question. It is highly probable that these affections are nothing more or less than ordinary ringworm of the body, such as we have in Europe, determined in their occurrence to certain parts of the body by peculiar circumstances, and assuming characters somewhat different from those observed in the disease as it exists in colder climates, in consequence of the greater luxuriance of the parasite, consequent upon the presence in the one case of a greater amount of heat and moisture, which are favourable to the development and speed the growth, of fungi.

Burmese ringworm, as far as we have observed, and we have had many cases under our care, is in reality the "erythema marginatum" of the Germans, which has now been shown by abundant proof to be nothing more than a modification of tinea circinata (or the old-fashioned herpes circinatus of the surface). It occurs about the fork of the thigh chiefly, where heat and moisture are more influential than elsewhere. It is a vegetable parasitic disease. In England we see the disease, in those who have returned from India, in two chief forms, or rather in two different degrees of extensiveness. In the one the disease consists in red itchy rings affecting the pubic region, the fork of the thigh, extending over the buttocks, and more or less about the axillæ, the front part of the chest, and the parts covered by hair about the navel. The rings vary in size from that of a shilling to that of

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Plate VII.

LEUCODERMA IN A PORTUGUESE WOMAN.

From the Catalogue of the New Sydenham Society's Atlas

Vincent Brooks, Day & Son Lith.

the palm of the hand nearly; the colour is bright, the rings are itchy, and their surface is to some extent raised, and they leave behind furfuraceous surfaces. The aspect may be altered by scratching, so that the integuments become excoriated and infiltrated. All this means that the fungus is made up of actively-growing mycelial threads that sprout freely and forcibly amid the epithelial layers. Sometimes the disease seems to disappear, and only slight scaly, itchy, scurfy patches remain behind. Again it lights up and reappears in all its intensity.

In the other form or degree, the disease is less erythematous, does not take on the ring form, and appears to be limited to the fork of the thigh and the parts about it. There is a red, scaly, itchy surface which festoons a greater or less distance down over the thigh in front, and attacks the perinæum and the buttocks to some extent. The disease begins as a small itchy scurfy spot—that is to say, the fungus does not luxuriate so freely and so produce red rings—and as this spot spreads the centre pales or rather gets brownish; the red edge extending. The edge is sometimes distinctly papular, and very well defined. The papules are mostly abortive vesicles, but even vesicles may be visible. If we pass the hand over the patches they feel thickened, dry, and harsh. If the disease is much scratched and irritated it may appear eczematous, or small boils may appear, or there may be a certain amount of lymph infiltration as the result of the irritation, and in such a form as to give the patch an uneven, somewhat knotty aspect and a very rough feel. The disease may after a while break up into islets—one part getting better, another becoming worse, or remaining *in statu quo*. The disease as a whole, often, if left undisturbed, gets "better and worse." It is always itchy, especially with the warmth of the bed, and the skin is, in chronic cases, much discoloured. The fact that the disease occurs where heat and moisture are present accounts for the amount of change induced, and also the variation from ordinary tinea circinata, from which it differs mainly in being accompanied by more infiltration.

Nature of the Disease.—Hebra now admits in "Eczema marginatum" the presence of a fungus, but thinks the disease is tinea engrafted upon an eczema. But it begins as tinea, and not eczema. Kobner, in 1864, described the fungus, and produced tinea circinata on his own arm by inoculation with it. Pick holds that the disease is an intertrigo and a tinea circinata, but as we affirm, it is a tinea circinata modified by the presence of ample heat and moisture, which causes the fungus to luxuriate, and consequently the tissue changes to be greater than in ordinary ringworm of the surface.

Now it is easy to see that the occurrence of bright red rings is only a stage of the disease, occurring when and where the fungus happens to find itself in such a condition as to be able to sprout beneath the epithelial tissues far and wide, and that very rapidly. We sometimes see the same red rings in the onset of tinea vesicolor. But in most cases we have a more gradual growth of fungus, and the production of scurfy patches. We suspect that Burmese ringworm is common in India. When erythematous itchy rings occur about the hairy parts and gradually increase whilst their centres become pale and branny and brownish, or when red itchy patches appear with a well-defined papular edge, and presently become furfuraceous, the scales of such places should always be carefully examined for a vegetable parasite.

The fungus may be detected by scraping off some of the scales of a patch, placing a very thin layer under the microscope—after adding weak liquor potassæ and allowing it to soak for some time—and making careful examination. We must take very thin portions of scale. Sometimes very fine moniliform chains of spores are seen, at other times freely branching mycelial tubes of fungus (trichophyton). The following is a representation of the mycelial threads of the fungus as seen in the scrapings from a case recently under our treatment.

The disease under notice is very unhappily called "eczema marginatum." It is not an eczema at all. It is not a serous catarrh of the papillary layer, and is not characterised by sero-purulent discharge drying into thin yellow crusts and stiffening linen.

Trichophyton tonsurans, from a case of Burmese ringworm.
U. C. H. Skin Clinique, April 1869.

It is, however, satisfactory to be able to simplify matters by including Burmese ringworm under the term Tinea circinata.

It is only necessary to add that Indian medical officers will do great service if they will investigate the nature of Burmese and all other "ringworms" bearing "topographical" or "popular" names, with a view to discover if they be not one and all referrable to "tinea circinata," their peculiarities or rather minor differences being accounted for by the influence of climate and the like.

MALABAR ITCH.

We know nothing of this disease in England, but are informed that its characters are those of Burmese ringworm. Information touching Malabar itch would be very acceptable, interesting, and valuable.

N.B.—Microscopic observations, with sketches, of vegetable parasites found in "ringworms" are specially asked for, and preserved specimens mounted in glycerine or balsam, if possible.

LICHEN TROPICUS, OR PRICKLY HEAT.

The pathology of this disease is well worth the attention of Indian observers. The disease is regarded as a lichen, but every red pimple is called lichen. True lichen means a disease in which solid lymph papules are formed in the skin, and these undergo no further change when once produced, except absorption and resolution. True lichen is rare. In lichen tropicus, the skin is studded over with red papules, but these are vascular, and evidently formed at the sweat ducts. In some cases vesicles are interspersed with the red papules, showing that a certain amount of sweat has been produced and has collected in such a way as to uplift the cuticle and to give rise to sudamina. The cause of the disease, the heat, acts generally upon the surface, and the eruption is general. The itching is not primary, it is the consequence of the failure of the sweat function to relieve the skin, and of the retention of the sweat. The anatomical seat of lichen tropicus in our opinion is the sweat follicles. These are called upon to perform an excessive amount of work, congestion is the result, with failure of the sudoriferous function; the surface is not properly cooled, the sweat products are retained, and morbidly stimulate the nerves of the skin, hence the pricking and burning, which is of course aggravated by everything that increases the cutaneous circulation, such as warm clothing and warm drinks. This matter is one worthy the attention of the Indian medical officer.

Note.—The influence of malarious poisoning has been alluded to in speaking of several of the diseases referred to in the foregoing pages. This influence may be on the one hand merely one which leads to prostration of the patient and disturbed nutrition of a general kind, so that the individual is more susceptible to the attack of disease in general; or on the other hand the influence of malarial poisoning may be of a specific kind. The questions have been framed with a view to determine the exact influence of malaria (if any) in the production of the diseases under discussion.

Doubtless other skin diseases that are considered peculiar to India find no place in this document. It will be a matter of much satisfaction if the circulation of this paper should be the means of eliciting information relative to such diseases, to the benefit of medical science generally and dermatology in particular.

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