Classification and Routine Treatment of Leprosy.

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(*Part of a lecture delivered to medical men in East Africa.*)

THE two chief difficulties which face the medical man when he first begins to take an interest in leprosy are, firstly, the difficulty of classification, and, secondly, that of treatment.

It is at once apparent to anyone beginning to take an interest in leprosy that the text-book descriptions of maculo-anæsthetic, mixed, and nodular leprosy, do not always meet the situation. Workers in India use a classification which was originally worked out by Dr. Muir, and I therefore propose to describe the course leprosy tends to take and then apply this classification to the various stages of the disease. In the first place, I wish to emphasize that each stage of leprosy is more or less closely related to the previous stage. On reading standard text-books, little indication of such a relationship is given, each form of the disease being described separately, and the impression conveyed is that they are entities in themselves. In describing what might be termed a typical leper history, I do not suggest that every subject of the disease goes through this course, for there are many modifying factors. In almost every case, however, previous steps of the disease can be traced, and not infrequently the history of the patient can be followed from the commencement as early nerve leprosy, through the various skin stages, until the case finishes up in the late secondary, anæsthetic stage, the disease having died out.

The country in which the disease passes through the various stages most typically is India, but I have seen enough early cases in Africa to feel sure that the disease does not differ materially from that seen in India.

Leprosy is a disease which does not readily gain a footing in the human body, and unless the patient is very susceptible to the disease, it attacks the most vulnerable tissue, which in this instance is nerve tissue. In the vast majority of cases the earliest signs of leprosy then, are those of nerve involvement. I shall not have time to enlarge on the question of how the mycobacteria of leprosy gain entrance to the nerve tissue, suffice is it to say that the bacilli can be demonstrated around the nerve terminals and in the nerve sheath. The

various changes seen giving rise to the clinical features of the disease are, first, œdema and swelling resulting from the presence of the organism, then contraction as a result of fibrous tissue formation. The signs which indicate nerve involvement are, in the order of importance from the treatment point of view, as follows :

1. Depigmented, or more correctly, hypopigmented patches.
2. Anæsthesia, first to very superficial touch, later pressure sense is gradually lost.
3. Nerve enlargement.
4. Muscular paralysis.

1. *Depigmented Patches.*

The commonest situation for these are the cheeks, outer aspects of the limbs, over the region of the scapulæ, and the buttocks. The patches are light in colour, but in the very dark skin may have a coppery appearance. The condition is more accurately described as hypopigmentation, as the depigmentation is never so complete as in leucoderma.

2. *Anæsthesia.*

This is the commonest and one of the most certain signs of nerve involvement in leprosy. At first the anæsthesia is very superficial, and therefore any method which tests pressure and not tactile sensation may lead to erroneous conclusions. Thermal sense is lost early in the disease, but later pressure sense also disappears. It should be noted that hypopigmented patches, although sometimes anæsthetic, are not generally so. It is along the cutaneous distribution of the ulnar and peroneal nerves that anæsthesia is commonly found.

3. *Nerve Enlargement.*

Associated with anæsthesia there is frequently seen enlargement of the superficial nerves. The usual nerves to become enlarged are the ulnar, peroneal, and great auricular, although I have not seen gross enlargement of the latter nerve so commonly in this country as in India.

4. *Muscular Paralysis.*

This is usually a late manifestation of leprosy, and by the time this is apparent the disease, as a rule, is in the advanced stage. In passing, I might mention that the development of the disease is not necessarily a matter of time. One patient may have had the disease a longer time than another, yet on account of some factor raising his resis-

tance, he may not be in such an advanced stage as the one who has been infected for a shorter period of time. As a general rule, however, the longer he has had the disease the greater the likelihood is that his signs are advanced. While muscular paralysis is usually a late manifestation, an exception to this is the facial nerve, which may become involved early in the disease. As a result of pressure of the bone in the stylo-mastoid foramen, the nerve is speedily permanently damaged, and thus facial paralysis does not tend to recover.

The stage which I have described is classified as early nerve leprosy, and may be denoted by the symbol A1. It cannot be too strongly emphasised that this stage is not contagious, and, where possible, lepers belonging to this group, provided they attend for treatment as out-patients in a dispensary or suitable clinic organised for the purpose, should be allowed to continue their employment.

Skin Leprosy.

If the early nerve leper remains untreated he may sooner or later show signs of skin involvement unless his resistance is high, in which case the disease may become spontaneously arrested, or become localised in a nerve or group of nerves, and never become generalised. Therefore it is at this stage that the leper complains of symptoms which mainly show themselves in vague rheumatic pains, general malaise, and periodic febrile attacks. These attacks may last from a few days to many weeks, and, if severe, may reduce the patient's vitality to such a low ebb that he readily falls a prey to some intercurrent disease, or he may rarely die as a result of the cachexia caused by repeated reactions. The chief signs of this stage of the disease are :

(*a*) Skin rashes.
(*b*) Nodules.

(*a*) *Skin Rashes.*

These are first seen at the periphery of the hypopigmented patches. As a result of a reaction, perhaps during treatment, or because of some factor which lowers vitality, *e.g.*, an attack of malaria, the depigmented patches become red, and the periphery becomes raised, and where no bacilli could be found they can now be demonstrated.*

*At the periphery of the patches take a pair of scissors curved on the flat and snip a piece of the skin out, smear it on a slide and stain the smear as for tubercle bacilli.

(*b*) *Nodules.*

These appear as a result of a blood stream infection, and this stage can be conveniently designated as miliary leprosy, on the analogy of tuberculosis. The skin stage is the contagious stage, for not only can bacilli be demonstrated from skin clippings, but also from smears or scrapings from the mucous membrane of the nasal septum. While the nose is not infrequently positive in this stage of the disease, I believe that the nose is never the primary source of infection. The skin stage of the disease can be denoted by the symbol B. According to whether the patient has few, moderately large, or myriads of bacilli demonstrable in nasal smears or skin clippings, the leper is said to be in the B1, B2, or B3 stage.

In the majority of cases leprosy dies out of the body, leaving the patient mutilated but free of his disease, that is, leprosy can be described as a self-healing disease. Lepers, unless they die of some intercurrent infection, or more rarely succumb during an attack of lepra fever, usually reach this stage. In a certain number of instances the disease may not advance further than the early stages, and the patient become healed of his disease without resulting deformity. In the advanced skin form this process of natural arrest may take many years, and even after all outward signs have disappeared, bacilli can be demonstrated in lymphatic glands on post-mortem examination. Hence the fallacy of giving potassium iodide as a test of cure.

The body overcomes the disease by encapsulating the bacilli in fibrous tissue, this tends to contract, and thus nerve and other tissues are destroyed. As a result of this, the characteristic deformities appear. The absorption of the small bones of hands and feet are largely trophic manifestations. This last stage is styled secondary anæsthetic leprosy, and is denoted by the symbol A2. All the bacilli have disappeared from the skin, and the patient is once again not contagious, but treatment can do little for such cases. The older writers used to refer to this type as " Lepra-mutilans." It is important to realise that trophic ulcers after healing are very liable to recur unless the patient is under constant observation. The breaking down of such ulcers is not necessarily an indication of activity, and is more often due to lack of cleanliness.

I shall now give the stages of leprosy in the form of a summary, and I trust the classification will be apparent.

A1 = Early nerve leprosy (non-contagious).
B1 = Early skin leprosy (contagious).

B2 = Advanced skin leprosy (contagious).
B3 = Very advanced skin leprosy (contagious).
A2 = Secondary anæsthetic or burnt-out leprosy (non-contagious).

There are various intermediate stages which may be denoted as follows :—

A1—B1. Early nerve leprosy passing into the early skin stage.
B1—B2. Early skin leprosy passing into advanced skin leprosy.
B3—A2. Very advanced skin leprosy passing towards the secondary anæsthetic form.
A1—A2. Early nerve form passing to late nerve form and missing the skin stage altogether.