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Leprosy and the Community

TEACHING AIDS IN LEPROSY

The Institute for Child Health, London, has been inspired, particularly by Dr David Morley, to produce 23 sets of colour slides for teaching purposes on various aspects of tropical medicine, especially as it applies to children, together with authoritative commentaries. Among these sets is one on leprosy, recently prepared by Dr Colin McDougall, Leprosy Consultant, The Slade Hospital, Headington, Oxford, and formally Leprosy Specialist, Ministry of Health, Lusaka, Zambia. The 24 slides cover all important aspects of leprology, and are of excellent quality. The illustrations have an African background, but this in no way restricts the usefulness of this series of slides for teaching purposes. A tape recording of the commentary is available. The set is produced at a very low price, and further information may be obtained from 'TALC', Institute of Child Health, 30, Guildford Street, London WC1N 1EH, England.

PRIZE ESSAY

In order to stimulate interest in leprosy among the younger members of the medical profession, LEPRA instituted an annual prize essay competition on some aspect of leprology. The winning entry for 1973 came from a medical student, Miss Celia Moss, whose essay is here presented. The views expressed are those of the author, and publication of the essay does not mean that they are necessarily the views of the Editorial Board.

The subject of the 1974 Essay is announced under Notes and News in this Journal.

THE TRANSMISSION OF HUMAN LEPROSY

CELIA MOSS

It seems probable that a necessary requirement for the transmission of leprosy is the passage of the bacillus *Mycobacterium leprae* from one person to another. An obvious way of studying the transmission of leprosy would therefore be to study how *Myco. leprae* passes from one individual to the next. However, the validity of this approach is doubtful since Koch's Postulates have not been fulfilled for *Myco. leprae,* i.e. this organism may not be the sole and sufficient cause of leprosy. *Myco. leprae* is certainly identifiable in every case of the disease; leprosy can be introduced into experimental animals by injecting bacilliferous material from patients; and the organism can be recovered after serial passage through several animals. But *Myco. leprae* has not yet been isolated in pure culture: it has been grown in certain tissues *in vitro* with variable success, but nobody has yet developed a reliable cell-free culture medium.

There are several plausible explanations for the notorious obstinacy of *Myco. leprae* in satisfying Koch's Postulates. It could be simply that some chemical constituent of susceptible cells is absent from all media so far tried; alternatively, since *Myco. leprae* is an obligate intracellular parasite, perhaps some specific feature of the intact cell is necessary (e.g. membranes) which would preclude growth in a cell-free medium. On the other hand, it is conceivable that some symbiotic organism is required, i.e. *Myco. leprae* alone cannot cause leprosy.

For this reason alone, there must be more to the study of leprosy transmission than bacteriological studies: but besides this theoretical objection there are more obvious practical ones. Many people have tried to piece together the route of transmission by searching for *Myco. leprae* in various sites. The trouble with this apparently sensible approach is that it has been embarassingly productive: nasal mucus, ulcer exudate, hair follicles and milk have all been shown to contain viable bacilli. The louse, the flea, the cockroach and the mite have all been incriminated. In short, if *Myco. leprae* is to be found almost everywhere it is sought, how can we ever unravel its route of transmission? Susceptibility to leprosy seems to involve factors other than exposure alone to the bacillus. Some people contract leprosy after minimal exposure, while others may remain healthy after living for years with an actively lepromatous patient. To take into account other variable factors such as human resistance and bacterial pathogenicity, and thus to clarify the relationship between exposure to *Myco. leprae* and transmission of leprosy, one must use epidemiological methods.

In conclusion, neither the bacteriological nor the epidemiological approach alone can answer the question "How is leprosy transmitted?" but together they can be applied to the problem formulated in the following way:

(a) What epidemiological variables determine the distribution of leprosy?
(b) How can the effects of these variables be explained in terms of a mechanism of transmission of *Myco. leprae*?

Epidemiological Studies

Dr T. W. Meade has discussed in detail the application of epidemiological studies of leprosy to the identification of high-risk groups, i.e. people to whom leprosy is easily transmitted. There are four essential conditions for a useful study:

(i) The study must be prospective, not retrospective.
(ii) One should measure incidence, not prevalence, of leprosy.
(iii) A multivariant technique of analysis should be used.
(iv) The population studied must not have been subjected to prophylactic measures.

These requirements present great practical problems which no study to date has overcome. In addition there is the theoretical difficulty of knowing what data to collect. One must clearly be selective, and yet not hampered by preconceived ideas. For instance, the classical view of transmission of leprosy is that skin to skin contact is necessary, so investigations have focused on that rather than nose-blowing, eating and defaecation habits, which may be equally relevant to transmission.

Therefore, epidemiology has so far cast little light on the mode of transmission. What is needed is an intensive survey satisfying the requirements laid down by Meade. This would require a large insular and static population where leprosy is endemic and where there have been no control measures. After exclusion of all

those already affected, the rest should be examined for signs of leprosy at least once a year. Each person should be asked a series of questions, with multiple-choice answers suitable for computer analysis. Information collected should include:

(1) Data concerning factors which might influence host resistance to infection: i.e. age, sex, race or tribe, past illnesses, lepromin reaction, etc.
(2) Opportunity for contact with the bacillus: relationship to known patients, nature and duration of contact with them, level of hygiene in the household, e.g. presence of parasites and domestic animals; whether overcrowded, sanitation, treatment of minor skin wounds, method of disposal of nasal secretions, etc.
(3) Examinations of bacilli from known patients for differences in pathogenicity.

In addition, those who develop leprosy during the study should be asked *about contacts,* the site of first lesion, and whether they have had ulcers and if so whether they were dressed. Bacillary load of nasal mucus, as well as skin, should be assessed quantitatively. Finally, values for all these variables could be fed into a computer and correlated independently with the occurrence of leprosy. Those environmental factors thought to influence transmission of leprosy by affecting the opportunity for transfer of bacilli, rather than either of the other variables, might then be revealed. Their effects could then be interpreted in terms of theories of the route of bacterial transmission.

This ideal experiment is clearly ambitious. Much preliminary work would have to be done: sociological studies, as well as a preliminary estimate of incidence, so that a suitable population size could be defined. However, in view of the gravity of the situation at present, and the uncertainty of the pattern of spread (e.g. sudden epidemics arising in non-endemic areas) it is clearly a matter of great urgency to identify the people at risk, and to analyse the predisposing factors, so that the mechanism of transmission can be recognized and blocked.

Direct Studies on the Mechanism of Transmission of *Myco. leprae*

There is a very wide range of possible routes of bacterial transmission, and these can be investigated in several ways. One can search for the bacillus in various sites and try to piece together the route of transmission; or one can select the more likely routes and test them on experimental animals (Rees *et al.,* 1967; Rees, 1969, 1970). When lepromatous material is injected into the footpad of a normal mouse *Myco. leprae* will multiply locally for about six months and then die. If an immunologically deficient (thymectomised, irradiated) mouse is used, the disease becomes progressive: after initial local multiplication bacilli spread, via lymphatics and blood, to cause lesions in many other sites. There is a remarkable similarity between the histological manifestations of *Myco. leprae* infection in the mouse with the disease process in humans.

(A) ROUTE OF EXIT OF *MYCO. LEPRAE* FROM AN INFECTIVE PERSON

Most work on this, the first stage in transmission, has been on lepromatous patients. There is obviously a potential exit from the heavily bacilliferous dermis when the overlying epidermis is cut or ulcerated. An interesting question is whether viable bacilli can also find their way out of unbroken lepromatous skin, as patches usually cover a far larger area than ulcers.

Pedley (1970*a,b*) found very few *Myco. leprae* on lepromatous patches (only 20 on 300 cm^2) using his Composite Skin Contact Smears (CSCS) technique. The method involved pressing a glass slide firmly on ten different areas of skin, heat-fixing it after every second or third smear, and then examining it for bacilli, suitably stained. As a control he showed that the CSCS method would pick up bacilli (a) from nasal mucus on the skin, (b) shed from nearby sores, and (c) from skin smeared with tissue from positive slit scrapings. His conclusions were that *Myco. leprae* does not emerge through intact epidermis, and that therefore skin to skin transmission is unlikely.

Periaswami (1968) found considerable numbers of acid fast bacilli on the skin of lepromatous patients. His method was to smear a few drops of egg-albumin on a closed lesion and transfer it to several other similar sites, thus concentrating anything lying on the surface. Control smears were taken from healthy individuals: no bacilli were found in these.

These positive findings of Periaswami are incompatible with the common observation that bacilli are hardly ever present in the epidermis, even when the whole dermis is replaced by bacilliferous granuloma. Reasoning that there are three possible routes out of the skin (sweat duct, sebaceous duct and hair follicle), Periaswami went on to show how bacilli can emerge through intact skin, by electron-microscopy. In skin biopsies of lepromatous patches he found a concentration of bacilli in the hair follicles, far less in the sweat glands, and hardly any in the sebaceous glands. He also showed, in transverse sections, bacilli apparently spilling out on to the skin around hair follicles, and concluded that infection could occur by this route. Seabra Santos (1965) made the same observation, and concluded that the apparent affinity of *Myco. leprae* for hair follicles could account for alopecia in leprosy. Desikan and Iyer (1972) have confirmed this finding in skin biopsies from 100 lepromatous and borderline patients: they found bacilli in nerves in all cases, in the epidermis in 8 cases, and in hair follicles in 38. There were none at all in sweat or sebaceous glands.

The nasal mucosa may remain bacteriologically positive even after skin smears have become negative, so bacilli might be disseminated in sneezes, nose-blows, sputum or exhaled droplets. Recent experiments of Rees on hospitalized lepromatous patients, in which a 24 h sample of nasal mucus was collected in a sputum-pot left at the patient's bedside, showed that the total bacillary content was of the same order as that of *Myco. tuberculosis* in sputum from patients with active pulmonary T.B. This supports the idea that infected nasal mucus may transmit leprosy.

Milk could also be a medium for transfer of bacilli (Pearson, Rees and Weddell, 1965; Pedley, 1967) provided they can be absorbed from the gut. However, this cannot be more than a subsidiary route: the index case in a family is by no means always the mother. The same applies to findings of *Myco. leprae* in seminal fluid: the frequency of conjugal infection is very low indeed. In conclusion, the predominant route of exit of bacilli is likely to be via nasal secretions or sputum, ulcer exudate, or the hair follicle. Only in the case of nasal mucus is there evidence that this could be quantitatively adequate.

(B) ROUTE OF ENTRY OF *MYCO LEPRAE* INTO SUBSEQUENT HOST

Theoretically, *Myco leprae* might enter the body at any interface with the environment. It is generally assumed that there is a single site of entry and the

discussion below is based on this assumption. However, there could be multiple routes. Perhaps people susceptible to tuberculoid leprosy are more vulnerable to bacilli on the skin, while people tending towards the lepromatous form are more vulnerable to bacilli in the lungs or gut. Another possibility is that the site at which bacilli enter predisposes the subject to one form or the other.

Some light might be cast on the portal of entry if one knew how the bacillus is subsequently disseminated throughout the body. Conversely, the mode of dissemination might be elucidated if one knew the portal of entry. Unfortunately neither of these problems has yet been solved, so inevitably this field of leprology is fraught with circular arguments.

(1) Evidence for entry through the skin

The main reason for the popular belief that leprosy is transmitted by skin to skin contact is, of course, that it is a predominantly cutaneous disease with a peculiar affinity for the peripheral nervous system. This alone does not prove the cutaneous route of entry: it could be that chemotactic influences attract *Myco. leprae* from other parts of the body, or, particularly in tuberculoid leprosy, that bacilli are not particularly concentrated in the skin, but tend to cause damage in sites exposed to cold or other trauma. Some arguments for cutaneous entry are:

(i) Epidemiological evidence, especially site of first lesion. But the inadequacy of these studies has been stressed above.

(ii) Evidence for the primary involvement of cutaneous nerves. The nerves most frequently affected (ulnar, peroneal, greater auricular, etc.) appear to be those containing mainly sensory fibres. Even when large mixed trunks are involved, the long time-lapse between the appearance of sensory and motor changes suggests that at first bacilli are confined to sensory nerves. *Since both motor and autonomic nerves are eventually affected it would seem that these fibres enjoy no special resistance: it merely takes longer for the infection to reach them. This again supports the idea that the infection starts in the skin.

(iii) Evidence for the centripetal spread of bacilli (Lumsden, 1964). Gerlach (1891) showed that the earliest changes are in the most peripheral branches of cutaneous nerves. Soon sensory branches in the larger nerve trunks are affected, and the process can advance as far proximally as the spinal cord. He concluded that the earliest and most extensive degeneration occurs at the site of entry of bacilli (i.e. the skin). This has been taken a step further by recent histological studies of Ridley (1970). In the earliest recognisable tuberculoid lesion there is infiltration of the epidermis by lymphocytes and epithelioid cells, before any infiltration of nerves occurs or any AFB's can be seen. This picture is interpreted as a stage of maximum immunity, *Myco. leprae* being arrested on entry into the epidermis. If resistance is lower they may reach the dermis and get a more secure hold on nerve and muscle: in this case a few AFB's are seen and there is a non-specific cellular infiltration around nerve bundles and other dermal structures.

The most potent argument against cutaneous entry is that *Myco. leprae* is very rarely found in the epidermis. This makes inunction through the skin unlikely, so if *Myco. leprae* does enter through the skin it must be through breaks in the epidermis. Experimental attempts to transmit leprosy to volunteers have been remarkably unsuccessful, but there is circumstantial evidence that leprosy can be transmitted in this way. There are one or two cases of lesions developing around

* Autonomic nerves may be involved very early. Ed.

the site of a pin-prick, and although many of these might be explained in terms of unrecognised contact with a leprosy patient, there is still the classic case of two sailors who were both tattooed on the forearm at the same place, and both developed leprosy around the site of the tattoo some years later (Porrit and Olson, 1943).

Since the theory of cutaneous entry of bacilli rests partly on evidence for centripetal neural spread, another counter-argument is that haematogenous spread of bacilli undoubtedly occurs. Visceral foci are common post-mortem findings in humans and in irradiated mice injected with leprosy bacilli. However, supporters of the theory of neural spread claim that it is the relative infrequency of internal (compared with cutaneous) lesions that calls for explanation, not their occasional occurrence.

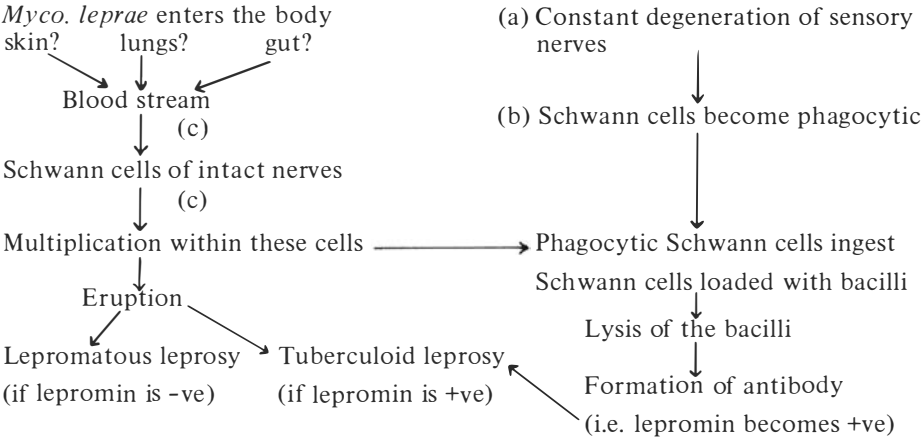
(2) Evidence for entry through respiratory and gastro-intestinal tracts

If one could show blood-borne bacilli at an early stage in the disease, and a positive attraction of *Myco. leprae* to cutaneous nerves from other sites, it would not be necessary to postulate cutaneous entry of bacilli to account for the findings mentioned above. More positive evidence for entry at other surfaces would be the demonstration of early accumulation of bacilli around capillaries in these sites.

(i) *Evidence for haematogenous transport.* The occurrence of visceral foci in all clinical forms of leprosy has already been mentioned, and frank bacillaemia is often found in the lepromatous form. In some cases lymphatic transport may be responsible for internal lesions (Desikan and Job, 1966). However, there is often no lymphatic connection between cutaneous and visceral foci, and transport must be via the blood stream. It would be interesting to know whether visceral lesions can precede skin lesions. Further support for haematogenous transport is provided by observations on mice: even in immunologically intact mice the capillary endothelium is a site of preferential accumulation and multiplication: sometimes electron-micrographs show bacilli apparently bursting out of endothelial cells into the lumen. In irradiated mice, bacilli injected intradermally proliferate locally and may cause alopecia at that site: 8 weeks later they are found in lymphatics draining that area, and involvement of the viscera, particularly the spleen, is found commonly within 18 weeks.

(ii) *Evidence for the attraction of* Myco. leprae *to the skin from other sites.* If it could be shown that the injection of *Myco. leprae* could produce a typical cutaneous lesion at a completely separate site, all the evidence for cutaneous entry would be invalidated. This has not yet been carried out in a healthy human being. However, Cochrane once demonstrated, in a single splenectomized monkey, that implantation of lepromatous tissue into the splenic stump followed by daily intradermal injections of lepromin, resulted 1 year later in a tuberculoid lesion on the forehead. This has not been repeated. In contrast, it is a common finding that intravenous injection of bacilli into thymectomized, irradiated mice causes lesions predominantly in cutaneous sites: the skin of the ears, paws, nose and tail.

Dr Weddell has proposed that *Myco. leprae* is attracted by the Schwann cells of cutaneous nerves (Weddell and Palmer, 1963; Weddell, Jamison and Parker, 1964). His theory of transmission, and some of the relevant experimental observations are summarized below.



(a) There is normally continuous degeneration and regeneration of all cutaneous nerves.
(b) The debris is removed by macrophages which originate from within the perineural sheath. Carbon particles injected into the skin of lepromatous patients are found, 5-7 days later, in Schwann cells related to degenerating axons. Moreover this phagocytosis shows some degree of specificity for *Myco. leprae:* heat-killed bacilli injected into the radial nerve of an infected person are found later in Schwann cells whereas *Myco. lepraemurium* injected into the other side as a control are found in the connective tissue sheaths of nerve bundles rather than inside Schwann cells.
(c) In lepromatous leprosy, bacilli have been found in Schwann cells of intact nerves suggesting that *Myco. leprae* is attracted to, and can multiply within, non-phagocytic Schwann cells without lysis.

This theory is attractive in that it explains quite simply several odd characteristics of leprosy: the long incubation period, changes in lepromin reaction with exposure (it becomes positive) and the apparently higher susceptibility of children. More important in this context, it accounts for the sites of predilection of leprosy: the nerves usually affected are just those which are most exposed to trauma and undergo frequent degeneration and regeneration, i.e. contain a high proportion of phagocytic Schwann cells. If it is correct, there is no need to postulate that the peripheral sites of lesions reflect the sites of entry of bacilli (although cutaneous entry would not be ruled out completely).

(iii) *Positive evidence for entry via the lungs or gut epithelium* in humans is so far lacking. With regard to the gut, it could easily be shown whether bacilli remain viable in gastric juice: if not this route would be ruled out. Proof of entry at either of these sites would require demonstration of bacilli in the epithelial cells and capillaries some years before the manifestation of leprosy—clearly a difficult experiment to arrange. However, it would be feasible to collect samples of gastric, intestinal and respiratory mucosa from autopsies in an endemic area: if the bacillus is as ubiquitous as is commonly suggested, one would surely find *Myco. leprae* in a few sections, particularly those of close relatives of a leprosy patient.

This is a field where animal experiments could be very useful. In fact Rees has already attempted an experiment in which three groups of immunologically deficient mice were exposed to bacilli in different ways. One group received *Myco. leprae* in their food, a second in nose-drops, and a third in an aerosol spray. Unfortunately the results were largely negative because the experiment was terminated too soon: it would surely be worth repeating. Three questions need to be answered: firstly, do these mice acquire lepromatous lesions at all? Secondly, which method is the most successful in producing lesions? Thirdly, in which cells of the mucosa concerned do *Myco. leprae* settle and multiply?

(C) EXTRA-HUMAN SURVIVAL OF *MYCO. LEPRAE*

Any site where *Myco. leprae* can remain viable for long periods must be considered as a possible reservoir for infection, if not an essential step in transmission. Bacilli should be sought in fomites, droplets, nose-blows, sneezes, etc., and their viability estimated after various intervals by injection into mouse footpads. Studies on the bacillus *in vitro*, e.g. chemical and physical conditions incompatible with viability, are clearly relevant to this phase of transmission, but there is no room to discuss them here. However, this sort of data is inconclusive on its own. For instance *Myco. leprae* have been demonstrated in the gastro-intestinal tract of the flea which may be found in large numbers in endemic areas. Some have concluded from this that arthropods contribute to the transmission of leprosy, but this may well be just an incidental finding: considering the vast numbers of bacilli in a single skin smear it would be remarkable if a flea, which spends its life biting human skin, did not contain *Myco. leprae* in its gut.

Conclusions

No experiment has yet proved or disproved any theory of transmission of leprosy. Bacteriological studies can, at best, only rule out impossible modes of transmission. Epidemiological studies have contributed no concrete facts because the ideal conditions are so difficult to fulfil. It is disappointing to finish with no definite conclusions, although this can be rationalized in terms of the question set at the beginning: until the variables determining the distribution of leprosy are known (from the suggested large-scale epidemiological survey) their effects cannot be interpreted in terms of a mechanism of transmission.

In view of the likelihood that nasal mucus of infectious patients contains enough bacilli for transmission of leprosy, it might be worth emphasising this route a little more in research. In epidemiological studies one should record nasal load of *Myco. leprae* instead of just skin load. In the laboratory the viability of *Myco. leprae* in nasal secretions should be studied, and more attempts made to transmit leprosy to mice by methods other than injection, e.g. nose-drops and aerosol sprays. Finally, *Myco. leprae* should be sought in lung and gut epithelial cells and capillaries.

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Field Workers' Forum

INDICATIONS AND CONTRAINDICATIONS FOR RECONSTRUCTIVE SURGERY

GUIDELINES FOR FIELD STAFF

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Social and Psychological Considerations

Preference should be given to
(1) children and young people;
(2) people for whom reconstructive surgery may mean a definite improvement in their economical or social conditions;
(3) patients who actively desire reconstructive surgery;
(4) all patients with endangered vision.

General Medical Considerations

(1) The patient should have been on stable treatment for not less than 3 months prior to surgery.
(2) The bacterial index (B.I.) should be + 2 or less.
(3) The morphological index (M.I.) should be O. Inherent in No. 1 is the claim that
(4) the patient must have shown NO evidence of "reaction" during the last 3 months prior to surgery.
(5) The patient must have received NO corticosteroids or ACTH during the last 3 months.

Surgical Considerations

(A) INTRA-OCULAR OPERATIONS

(e.g. iridectomy, cataract extraction, intro-ocular drainages, etc.) Intraocular surgery in leprosy presents a number of difficult and potentially dangerous situations. Such patients should be treated either by an ophthalmologist with training in leprosy, or by a reconstructive surgeon with training in ophthalmology.
(1) B.I. and M.I. must have been O for at least 6 months.
(2) No clinical activity must have been detected during the last 5 months prior to surgery.
(3) No septic conditions must be present anywhere in the body.

(B) SURGERY OF THE TEAR CANALS

This is usually required in cases with chronic infection of the tear canals. Since it frequently is associated with corneal/conjunctival anaesthesia, its correction is a matter of urgency.

No special contraindications are recognized.

(C) SURGERY ON THE EYELIDS

This is usually required for entropion or for lagophthalmos. Since both conditions frequently are associated with corneal/conjunctival anaesthesia, and since both conditions, even in the absence of this complication constitute a serious risk to the preservation of an unimpaired vision, their correction is a matter of urgency. If the surgical considerations are fulfilled, patients may be referred to plastic surgeon or to ophthalmolgist for correction, or preferably to reconstructive surgeon. If the surgical considerations are NOT fulfilled they must urgently be sent to reconstructive surgeon, who in consultation with the medical team will decide on the plan of treatment.

(D) RECONSTRUCTIVE SURGERY OF THE FOOT

(1) Weakness of dorsiflexion or dropfoot of less than 3 months duration requires referal to reconstructive surgeon (the treatment may in certain cases be conservative).
(2) Dropfoot of more than 3 months duration requires referal to reconstructive surgeon for operative treatment.
(3) Clawing of toes will in most cases be associated with dropfoot at the moment when the patient presents his complaints. The surgical correction may be undertaken simultaneously with dropfoot correction.
(4) Conditions related to plantar ulceration and scarring: see guidelines on ulcers and scars.

(E) RECONSTRUCTIVE SURGERY OF THE HAND

(1) Clawing of fingers, paralysis of the thumb, and wrist drop of less than 3 months duration require referral to reconstructive surgeon (in many cases attempts at conservative treatment may be made).
(2) Uncomplicated clawing of fingers, paralysis of the thumb, and wrist drop of more than 3 months duration require referal to reconstructive surgeon.
(3) Hands with severe absorption, deformed and twisted fingers, contractures, or extensor tendon damage can with careful surgery and competent physiotherapy in many cases yield surprisingly good results. Consider the social and psychological situation, and if the patient is suitable, refer to reconstructive surgeon for assessment and possible surgery.

(F) PLASTIC AND COSMETIC SURGERY OF THE FACE

This is usually required for madarosis, correction of collapsed nose, correction of pendulous and and deformed ears, and correction of excessive wrinkling of the face.

(1) Consider the social and psychological situation.
(2) B.I. and M.I. must have been O for at least 6 months prior to surgery.
(3) No clinical activity must have been detected during the last 6 months prior to surgery.

It should be recognized that with increasing urbanization, more and more people will come to depend on jobs where their appearance is of great importance.

(G) NON-SPECIFIC SURGICAL CONDITIONS

(1) Gynaecomastia and hyperthelia may be treated surgically either at local general hospital or by reconstructive surgeon.
(2) Any other condition that may require surgery: (I) if general medical conditions permit, refer to local general hospital; (II) if general medical conditions do NOT permit, refer to reconstructive surgeon or to medical leprologist for advice.

(H) SPECIAL CONSIDERATIONS FOR PREGNANT OR LACTATING WOMEN

It is particularly important under these conditions to ensure an adequate diet, particularly in respect of protein intake. Treatment with iron preparations and multivitamins should invariably be given.

(1) If the woman is under regular treatment: (I) stop further increase in DDS dosage; (II) refer to medical leprologist for advice.
(2) If the woman has discontinued treatment for whatever reason: (I) do NOT initiate or re-start treatment with DDS; (II) refer to medical leprologist for advice.
(3) If the woman has never had anti-leprosy treatment (I) do NOT initiate anti-leprosy treatment; (II) refer to medical leprologist for advice.

Since reconstructive surgery in leprosy was initiated by Mr P. W. Brand, 25 years ago, tremendous advances have been made in the surgical techniques and in the related and essential field of physiotherapy.

It was fairly early recognized that the medical and social conditions of the patients who submitted themselves to surgery were of great importance. One should not brush aside the occasionally seen provocation of reactions by the surgical trauma. The very nature of this problem has rendered the surgeons wary of initiating double blind studies to ascertain the exact nature of indications and contraindications in this field. We, therefore—all of us—tend to work from experience and impressions, coupled with a knowledge of certain basic facts of biology.

The guidlines set out here represent the experiences of the author from a number of years of work in this field in various parts of the world and under varying conditions. Naturally discussions and information from many colleagues have influenced this. Equally naturally different surgeons may differ in details of opinion.

In different social, economic and cultural settings the relative priorities of surgery of the hand and of the foot tend to vary. However, vision-preserving surgery must always have the highest priority. Cosmetic surgery—such an apt and often misused term—tends to assume higher priority with increasing urbanization.

On all points the individual surgeon in consultation with his medical colleagues and local social services must make up his own mind.

It is the hope of the author that these guidelines may help to a better understanding and helpful service to the leprosy patients.