LEPROSY REVIEW

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Principal Contents

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LEPROSY REVIEW

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CONTENTS

Edited by DR. J. ROSS INNES, Medical Secretary of the British Leprosy Relief Association, 8 Portman Street, London, W.1., to whom all communications should be sent. The Association does not accept any responsibility for views expressed by writers.

Contributors of original articles will receive 25 loose reprints free, but more formal bound reprints must be ordered at time of submitting the article, and the cost reimbursed later.

EDITORIAL

I. Correction and Apology

We were sorry to learn from Dr. Chaussinand that in the October number of *Leprosy Review*, we published in error for the second time a letter for Dr. DE SOUZA ARAUJO, who is now deceased. Dr. de Souza Araujo had previously published a letter on the subject, but the one we received and which was published on pages 267 and 268 bore a recent date and we were misled into believing that it was entirely new and failed to notice that it was a repetition. Dr. Chaussinand had already replied to the first letter in our issue of October 1961 on pages 276 and 277. We apologise to Dr. Chaussinand for any distress caused to him by this error.

II. The Forthcoming 8th International Congress of Leprology

A letter has been received from the National Organizing Committee in Rio de Janeiro. This letter is reproduced here in full.

PROGRAM

"Dear Friend,

You will already have heard through *International Journal of Leprosy* that the 8th International Congress of Leprology will be held at Rio de Janeiro from the 12th to 20th September 1963, in the Copacabana Palace Hotel.

1. *The Themes of the Congress*

A slight change has been made in the Themes for discussion in the Congress as compared to those in the last Congress. They will now be as follows:

A. Pathology and Experimental Transmission
B. Borderline and Indeterminate Leprosy
C. Leprosy Reaction
D. Therapy
E. Epidemiology
F. Bacteriology and Immunology
G. Education and Social Aspects
H. Physical Medicine and Rehabilitation, including Surgery and Vocational Training.

2. *The Panel System*

The panel system for detailed discussion by correspondence of each Theme beforehand will be maintained. Arrangements are under consideration for meetings before the Congress of 2 special Round Table Groups, one on Pathology and Experimental Transmission and the other on Borderline and Indeterminate Leprosy (the first two in the above list).

3. *Papers*

Papers from members will be of two kinds:

(*a*) requested papers from members of the panels

(*b*) proffered papers from all full members of the Congress.

Please note that proffered papers must be presented in one of the official languages, viz. Portuguese, English, Spanish or French. This means that if you write in another language you must send also a translation in one of the official languages.

For presentation it is necessary to write the paper in 3 forms – (*a*) an abstract of not more than 200 words which must be received by 12th July 1963, for publication in an Abstract Booklet to be issued to members before the beginning of the Congress; (*b*) a shortened form of the paper *which can be read in* 10 *minutes;* (*c*) the full paper (if this takes more than 10 minutes to read). The last two forms of the paper should be received before 12th June 1963. All papers in their various forms should be sent to Dr. Fausto Gayoso Castelo Branco, President of COCIL, (Rua São Cristóvão no. 1298 – Rio de Janeiro – Brasil).

Projection apparatus is available here and can take the following film and slide sizes. (Details not given).

4. *Registration*

The members will be entitled to take part in all the activities of the Congress, both scientific and social. The fee will be U.S. $10 for members and $5 for persons accompanying and $20 for non-members and $5 for persons accompanying them. These fees should be paid at the time of registration on the day preceding the Congress and the opening day.

5. *Languages*

The working languages of the Congress will be Portuguese, Spanish, English and French. Simultaneous interpretation will be provided throughout the sessions.

I shall be delighted to be at your service for any further information. I shall be glad if you will disseminate this information to your colleagues in your country and elsewhere. Also fill in and send the special registration form for accommodation required, as soon as you have made up your mind to participate. In this way you will contribute to the success of the 8th Congress by allowing of all arrangements to be in good time.

Looking forward to having the pleasure of hearing from you soon.

DR. FAUSTO GAYOSO CASTELO BRANCO,
Presidente da Comissao Organizadora do VIII Congresso Internacional de Leprologia (*COCIL*)."

JAS/1n.

III. Further Information about Membership of Round Tables and Panels

The full list is not quite complete, but it will be helpful if we give the names as far as we know them at present.

A. Round Table on Pathology and Experimental Transmission

Chairman: R. J. W. Rees (U.K.)
Secretary: Prof. H. M. Portugal (Brazil)
Members: M. Bergel (Argentina), C. H. Binford (U.S.A.), Y. T. Chang (U.S.A.), K. R. Chatterjee (India), J. Convit (Venezuela), W. H. Feldman (U.S.A.), W. A. Hadler (Brazil), S. Nishimura (Japan), J. M. Robson (U.K.), C. C. Shepard (U.S.A.), F. F. Wilkinson (Argentina).

B. Round Table on Borderline and Indeterminate Leprosy
Chairman: R. D. Azulay (Brazil).
Secretary:
Members: R. G. Cochrane (U.K.), F. Contreras (Spain), Dharmendra (India), G. L. Fite (U.S.A.) T. Imaedo (Japan), W. H. Jopling (U.K.), V. R. Khanolkar (India), K. Kitamura (Japan), Gay Prieto (Spain), J. N. Rodriguez (Philippines), F. Sagher (Israel), L. de Souza Lima (Brazil), H. W. Wade (President I.L.A. – Philippines).

C. Panel on Reactions
Chairman: F. Latapi (Mexico)
Secretary: D. S. Ridley (U.K.)
Members: E. A. Carboni (Argentina), J. Gomez Orbaneja (Spain), C. K. Job (India), A. R. Mercau (Argentina), A. Rabello Neto Jr. (Brazil), J. Ramos e Silva (Brazil), P. Rath de Souza (Brazil), I. Tajiri (Japan), J. G. Tolentino (Philippines).

D. Panel on Therapy
Chairman: S. G. Browne (Nigeria & U.K.)
Secretary: P. Laviron (France)
Members: A. M. Alonso (Brazil), A. Baccareda Boy (Italy), J. Barba Rubio (Mexico), M. B. Bhojwani (Malaya), T. F. Davey (U.K.), H. Floch (France), Latif K. Hanna (Egypt), Y. Hayashi (Japan), K. Ramanujam (India), K. F. Schaller (Ethiopia), S. Schujman (Argentina), M. F. R. Waters (U.K.).

E. Panel on Epidemiology and Control
Chairman: J. A. Doull (U.S.A.)
Secretary: A. Salazar Leite (Portugal)
Members: E. Agricola (Brazil), D. A. Akintonde (Nigeria), L. M. Bechelli (Brazil), J. A. Kinnear Brown (U.K.), W. M. Candidio (Brazil), O. Diniz (Brazil), C. Kettanurak (Thailand), D. L. Leiker (Netherlands), J. M. Mallac (India), F. E. Rabello (Brazil), C. M. Ross (U.K.), Amado Saul (Mexico).

F. Panel on Bacteriology and Immunology
Chairman: J. H. Hanks (U.S.A.)
Secretary: J. M. Fernandez (Argentina)
Members: J. O. Almeida (Brazil), R. S. Guinto (Philippines), S. W. A. Kuper (U.K.), E. Montestruc (France), N. Olmos Castro (Argentina), A. Rotberg (Brazil), Candido Silva

(Brazil), K. Yanagisawa (Japan), Y. Yoshie (Japan), G. P. Youmans (U.S.A.).

G. Panel on Education and Social Aspects

Chairman: T. N. Jagadisan (India)

Secretary: Luiza Keffer (Brazil)

Members: C. Costa Neves (Brazil), C. I. Crowther (U.S.A.), M. C. Estrada (Mexico), R. Follereau (France), N. D. Fraser (U.K.), K. Hamano (Japan), O. W. Hasselblad (U.S.A.), E. B. Johnwick (U.S.A.), R. V. Wardekar (India), Mrs. E. Weaver (Brazil).

H. Panel on Physical Medicine and Rehabilitation (including Surgery and Vocational Training)

Chairman: P. W. Brand (India & U.K.)

Secretary: J. Arvelo (Venezuela)

Members: N. H. Antia (India), Mrs. Margaret Brand (India & U.K.), J. E. Faggin (Brazil), M. Itoh (U.S.A.), M. Nakita (Japan), Mrs. Nimbkar (India), D. E. Paterson (India & U.K.), E. W. Price (Nigeria & U.K.), D. C. Riordan (U.S.A.), Linneau Silveira (Brazil), D. Ward (India), E. Zamudio (Mexico).

LIMITED PILOT TRIAL TREATMENT OF LEPROSY BY GRISEOFULVIN

By R. E. PFALTZGRAFF, M.D., and R. G. COCHRANE, M.D., F.R.C.P.

September 1962

Because of the interest in Griseofulvin in respect to its fungicidal properties, it was felt that it would be useful to undertake a very limited preliminary trial in order to ascertain whether Griseofulvin has any effect on the *M. leprae*. Early workers, particularly REENSTIERNA, was of the opinion that *M. leprae* was originally a soil fungus and, over the centuries, adapted itself to human tissues. It seems, therefore, to be a reasonable hypothesis that Griseofulvin might have an effect on the *M. leprae*.

The following two cases under a short trial with Griseofulvin are reported:

CASE NO. 1

Ladi Bubwa. *Hospital Case No.* 6945. *Age* 9. Body weight 50 lb., dose 0·5 g. daily for one week and 0·75 g. daily in divided daily doses thereafter.

Classification. Advanced Nodular Leprosy.

History. Both mother and father have leprosy as also have her two brothers. Two sisters are healthy; a half-brother under treatment in the Leprosarium. Onset of disease, 4 years previously (that is 1957). Admission weight: 30 lb., height: 4 ft. 3 in., Hg. index 14 gm. Stools and urine: nothing abnormal discovered. BI highly positive (5+).

Biopsy No. 1 *taken* 2.5.62. *Lab. No.* 4376

H.E. Section. There is a mass of infiltration in the corium occupying 100% of the surface of the corium; the infiltrate does not extend up to the epidermis, but leaves a very clear sub-epidermal zone. The infiltrate consists of closely packed histiocytes with some, but not conspicuous, round celled infiltration. The skin elements are almost completely obliterated by the great mass of infiltration, but there appears to be remnants of a hair follicle around a small focus of lymphocytic cells and a few plasma cells. In this mass of infiltration, individual cells are difficult to recognise. No nerves are seen in the infiltrate and there is some evidence of foamy cell change.

F.F. Stain. Large masses of acid-fast bacilli seen in every field. The bacilli show definite morphological change. Nerves are not easy to recognise because they are distorted as a result of proliferation of the connective tissue (perineurium) and the massiveness of the cellular infiltrate tending to obliterate nerve and skin appendages.

Diagnosis. An advanced lepromatous case, active, but not in reaction.

Biopsy No. 2 *taken* 19.6.61. *Lab. No.* 4419

H.E. Section. There is an intense and massive infiltration under the epidermis occupying the whole of the dermis leaving a narrow but relatively clear sub-epidermal zone. The infiltration consists almost entirely of a mass of closely packed histiocytes with a great many plasma cells scattered among the histiocytes. An occasional nerve is seen, but the nerves are probably compressed completely owing to the massive histiocytic infiltration. In some areas, the skin appendages are almost entirely obliterated except for an occasional hair follicle cut across and remants of sweat ducts.

F.F. Stain. Very large numbers of acid-fast bacilli showing very marked morphological change.

Diagnosis. A very active (? reacting) lepromatous case of moderate severity.

Biopsy No. 3 *taken* 5.9.61. *Lab. No.* 4588

H.E. Section. There is a massive infiltration occupying a hundred per cent (100%) of the corium which does not extend up to the epidermis but leaves a clear sub-epidermal zone. The infiltrate consists almost entirely of histiocytes of the larger macrophage variety; interspersed between the histiocytes is some lymphocytic infiltration, but this is not particularly significant, also here and there, there are collections of plasma cells. In some areas the larger

macrophages have the appearance, but not the setting of epithelioid cells. No nerves are recognisable in the massive infiltration.

F.F. Stain. There are numerous numbers of acid-fast bacilli seen, most of which are crammed in the macrophage cells.

Diagnosis. This is a very active lepromatous case, not in reaction. The fact that no nerves are discernible in the Trichrome Section may or may not be significant. The diagnosis is an active lepromatous case showing influence of therapy. (It should be noted that while the second biopsy report showed marked morphological change in the bacilli, the third biopsy showed no signs of such changes, and that the number of bacilli, if any, had increase for the note is that 'there are enormous numbers of acid-fast bacilli and most of the macrophages were crammed with bacilli'. In these circumstances it was felt that Griseofulvin was having no effect on the disease and, therefore, the treatment was discontinued.)

CASE NO. 2

Merama. *Hospital Case No.* 6954. Body weight 120 lb.

History. Patient gave history of leprosy for four years. The first lesion started on the left leg, where there is an area of anaesthesia. The patient's husband also has leprosy. She has three children. Two died previously, and now one child, boy aged three months. On admission weight: 120 lb., height: 5 ft. 4 in., Hg: 15 gm. Stools and urine: negative. Bacterialogical result: Rt. ear 3+, forehead 2+. The patient clinically shows all the features of a moderately early diffuse lepromatous case and treatment was commenced on 17th May 1961. Dosage given was 0·5 g. daily for 1 week, then 1.0 g. daily thereafter in divided dosage.

Biopsy No. 1 *taken* 17.5.61. *Lab. No.* 4450

H.E. Section. There is a scattered infiltration underneath the epidermis with a relatively clear sub-epidermal zone. In the dermis proper, the infiltration is much more intense, especially in the region of neuro-vascular bundles.

The infiltration generally is lymphocytic and histiocytic; the lymphocytic response, however, is not focalised in any particular pattern. Nerves are most easily recognised because of the intense infiltration, but when they are seen they are not involved.

F.F. Stain. Scattered acid-fast bacilli seen in moderately numerous numbers amidst the cellular infiltrate.

Diagnosis. A moderately early lepromatous case.

Patient continued treatment from 17/5/61 through to 5/9/61—a period of approximately four months. At the end of this period, that is on 5.9.61, a further biopsy was performed following in the histopathological report.

Biopsy No. 2 *taken* 5.9.61. *Lab. No.* 4587

H.E. Section. There is an infiltration underneath the epidermis extending the whole length of the superficial part of the corium leaving a relatively clear sub-epidermal zone. The infiltrate is moderate to gross in intensity, and consists chiefly of histiocytes and lymphocytes: there is no evidence of foamy cell change, neither is there any evidence of the focal distribution of the lymphocytic response. Because the biopsy is too shallow, nerves cannot be readily seen: I think I can see an occasional nerve twig, which is uninvolved but apart from this I cannot recognise any nerve tissue.

F.F. Stain. Moderately large numbers of acid-fast bacilli seen throughout the granulomatous infiltration showing marked morphological change.

Diagnosis. The general impression is that this is a moderately early lepromatous case which, because of the granularity of the acid-fast bacilli would indicate that the case is under the influence of therapy.

General Conclusion

Although during this period, the *M. leprae* showed considerable morphological change, clinically there was no marked improvement, and further, owing to the fact that when the patient was admitted it was noticed that the *M. leprae* showed morphological change. It was thought that this finding was hardly significant. The morphological change seen in the bacilli in the first biopsy possibly indicated that she had received Sulphone therapy elsewhere, for this is regularly available, or it may simply be that the *M. leprae* showed a significant

change in morphology, because it is known that even when a case is not under therapy *M. leprae* can show morphological change, for in the natural course of the disease, there are periods of quiescence and activity. The conclusion, therefore, was that this patient has shown no significant improvement in the four months she was under Griseofulvin therapy, therefore it was not considered justified to continue her treatment. She was put on standard D.D.S. therapy and has since shown quite marked improvement.

Summary

Two cases of lepromatous leprosy. One a moderately advanced lepromatous case, the other a moderately early diffuse lepromatous case, were given Griseofulvin therapy, and in neither instance was there sufficient improvement or indication of sufficient improvement to justify the continuation of the remedy, and therefore, it must be concluded that Griseofulvin has little or no action on the *M. leprae*, and therefore, it is not advised as a treatment for the disease.

The Griseofulvin was supplied as Fulvicin by DR. JOSEF KOLENSKI, M.D., of the Clinical Research Division of Schering Corporation, Bloomfield, New Jersey, U.S.A.

A PROSTHESIS FOR BELOW-KNEE AMPUTEES

By ROY E. PFALTZGRAFF, M.D.
Garkida Leprosarium, Garkida via Yola, Nigeria.

Introduction

Recently there has been considerable material published concerning the problem of the care of the foot in leprosy. These contributions have added materially to our knowledge of the mechanism of foot ulceration; of how to prevent it, and how to treat it when it does occur. However, the problem of ulceration in the anaesthetic foot is not completely solved, and it will be many years before we have seen the last refractory foot ulceration under control. All will agree that at the present time there is a group of leprosy patients with such severe foot abnormalities that no presently known method of management will bring about satisfactory rehabilitation.

Our own management of foot ulceration includes the standard methods of treatment including surgery (carefully conserving any functional tissues), bed rest, walking casts, and a rigid soled prosthetic shoe. Only after several years of persistent treatment without effecting a cure have we resorted to amputation.

In the past two years we have had ten cases where a below-knee prosthesis was indicated. These cases can be divided into three categories. (1) Those who had had previous amputation, of which there were three cases. (2) Those with malignant degeneration in a long standing ulcer. There were four of these. (3) The third group was made up of three patients with ulcers which refused to heal or to stay healed for any length of time over a trial period of at least four years. Two of these were constantly in hospital with ulcerations of the foot.

The results in these ten people are as follows: Eight are fully rehabilitated. One died of metastasis from the original malignancy, and one has not yet been fitted. The only apparent failure is the patient who died, and she too was ambulatory with this type of prosthesis prior to her final illness.

We have had no training in the manufacture of prostheses, and thus much study, thought and experimentation were involved in the preliminary stages of the development of this prosthesis. There were also no preconceived ideas as to how the work should proceed, and thus some revolutionary principles in prosthesis manufacture have been evolved.

In our situation there were certain goals to be met in this project which we list here:

1. The prosthesis must be practical and functional. It must be strong, sturdy, durable and lightweight.

2. It must make the patient ambulatory without crutches so that he or she can do farmwork or housekeeping.

3. It must be manufactured cheaply.

4. It must be simply constructed so that the manufacture can be carried out by a person who has not had technical training in prosthetics.

5. The process of construction must not be time consuming.

Materials Required

1. Woven cotton stockinette of 3 in. and 4 in. widths.

2. One-eighth inch thick foam rubber. 'Rubazote' made by Dunlop.

3. One-quarter inch thick foam rubber. 'Sorbo' rubber, B.F.S. Grade, made by Dunlop.

4. Rubber Cement. Commercial impact adhesives are preferable, such as 'Evostik' Impact Adhesive 528, manufactured by Evode Ltd., Stafford, England.

5. Epoxy Resin of a type suitable for cloth impregnation and lamination technique, such as 'Orthobond A & E', Vernon-Benshoff Co., Pittsburg 30, Pa., or 'Epicote 815' with Curing Agent, Shell Co. Ltd.

6. Lightweight wood, such as kapok or obeche.

7. Other supplies such as cardboard, cord, tyre rubber, etc., which are available anywhere.

8. Simple tools of a type available anywhere.

Technique of Construction

1. The first requirement is that the stump be well healed and shrunk by at least a month to six weeks of constant application of an elastic bandage.

2. *The prosthesis is manufactured directly on the stump itself.* Note that radical departures from customary prosthetic construction are printed in italics.

3. The stump is examined for any pressure points, and if there are any sites which may possibly break down due to pressure, a small pad of cotton is shaped to cover the area and is glued in place directly over the danger site with rubber cement. The most common danger point is the distal portion of the anterior tibial crest. The cotton pad makes a depression in the final prosthesis so that there is less pressure on the tissues in that area.

4. The stump is liberally powdered so that difficulty will not be encountered on removing the prosthesis.

5. A piece of stockinette which will fit the stump snugly is cut of proper length to cover the stump, and with adequate extra length to eventually form the outside layer (covering) of the completed prosthesis (A in Fig. 1). That is, it must be the length of the stump to the apex of the patella *plus* the distance from the apex of the patella to the floor; remembering that as the stockinette is stretched laterally

its length is diminished. The end of the stockinette is then sewn shut in a curve which will make a snug fit over the end of the stump.

6. The stockinette is then slipped on the stump with the extra length pushed up above the knee temporarily. See Fig. 1.

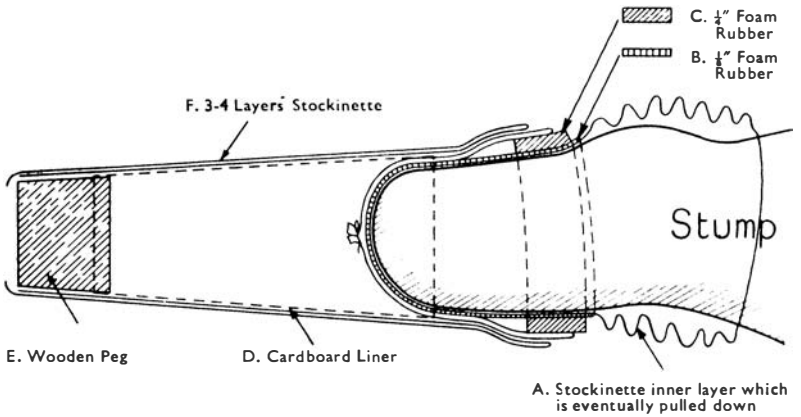


FIG. 1 *Diagrammatic Cross-section of completed Prosthesis showing various layers.*

7. The stockinette beyond the seam over the end of the stump is glued and turned back on itself so as not to produce an elevation on the inside of the prosthesis.

8. A layer of ⅛ in. foam rubber is then measured, cut and glued to cover the entire stump from the apex of the patella downward (Fig. 1-B). It must be carefully fitted over the end of the stump so as not to cause any irregularities. This is accomplished by allowing it to extend about 1½ in. below the end of the stump, and then with the fingers carefully pushing the overlap in, in four quadrants so that they adhere to themselves, leaving four tags which are then cut off flush with scissors. See Fig. 2.

9. A strip of ¼ in. Sorbo foam rubber is then cut 3 in. wide and long enough to go around the stump just below the knee (C in Fig. 1). Leave about ¼ in. of the ⅛-in. rubber extending above the heavier cuff. The ¼ in. cuff is then glued into place under slight tension; care being taken that the underlaying layers of foam rubber and stockinette do not wrinkle.

Note: This is applied with tension in order that on insertion of the stump the prosthesis will grasp it just below the knee, and prevent the prosthesis from slipping. We have not been successful in all instances in getting this sufficiently tight, but where it has been adequately so it has simplified the procedure and is less cumbersome than a retaining strap which is needed if this constricting band is not tight enough.

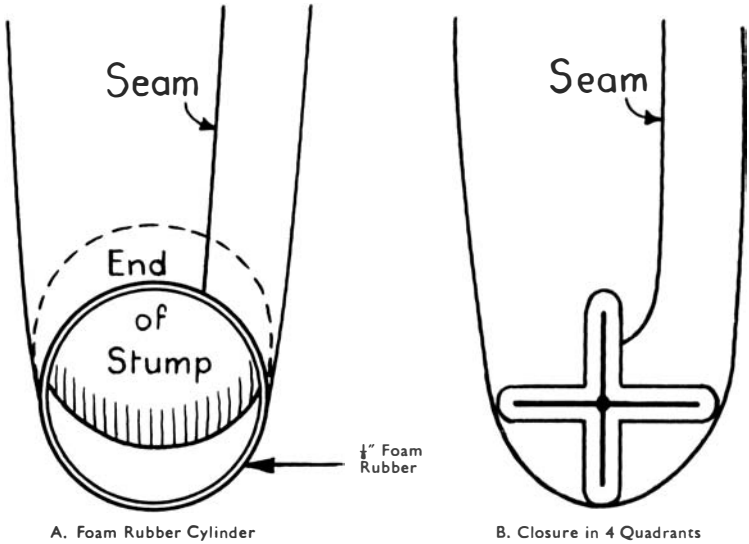


FIG. 2. *Closure of Foam Rubber layer over end of Stump.*

10. The second innovation – *The supporting portion of the prosthesis is a plastic resin which is applied directly over the above preparation while on the limb.*

11. A second layer of stockinette is then cut a bit more than twice the length of the stump. The end is again sewn shut, and it is slipped up over the stump with the second thickness, which will be turned down later, pushed up on the knee.

12. The plastic which is to be used is then prepared for application to the stockinette according to the manufacturer's directions. The impregnation should be higher on the limb anteriorly so that it extends up to the apex of the patella; but posteriorly it should be lower so as to allow for free movement of the hamstring tendons during flexion of the knee.

The plastic must be applied adequately to thoroughly impregnate the stockinette, which will take from 6 to 8 tablespoonsful of the mixture. It is best applied with a spoon. After the first layer is impregnated the short piece of stockinette at the top is turned down on itself and tied shut with a bit of cord over the end of the stump, and this layer also impregnated thoroughly.

13. After the impregnation an ordinary muslin bandage is used to snugly wrap the stump completely. This will take up any extra plastic which may be squeezed out of the stockinette.

N.B. WARNING! Do not use too much plastic at this stage thinking to make a sturdier prosthesis, as the chemical reaction which occurs on the solidification of the plastic produces heat, and it is possible to produce a burn. If the layers are not thicker than recommended above, the heat will be dissipated innocuously. If there is

concern that this will not be strong enough, a second layer of plastic could be added *after* removal from the stump, although we have not found this necessary.

14. The limb should then be supported above the knee so that no pressure is put on the prosthesis so as to distort it during the hardening process. It is then left unmolested for at least two hours; the time depending upon the setting time of the plastic used.

15. When the plastic has become stony hard the entire prosthetic shell is removed from the limb. The anterior border is marked to correspond to the midline of the patella.

16. Any of the muslin bandage which has not taken up plastic and thus been incorporated into it, is now pulled or trimmed off.

17. Measure the length of the other leg to determine the length which will need to be added to the prosthesis to make it equal the other limb.

18. Some sort of jig is now needed to hold the inner shell of the prosthesis (completed in 17 above), and the wooden peg which is used to make the distal end of the prosthesis. This peg is made of lightweight wood and shaped as a truncated cone. The proximal end should be about 3 in. in diameter, and tapered slightly distally. We have made the necessary jig simply from a few blocks of wood, and straps to hold the prosthesis pieces made from inner-tube rubber. (A lathe would make an excellent jig.)

19. The inner shell, and the wooden end piece are now lined up in the jig, and held firmly in the position which is necessary in the final prosthesis. This should be only slightly longer than will be necessary in the final fitted prosthesis. See Fig. 3.

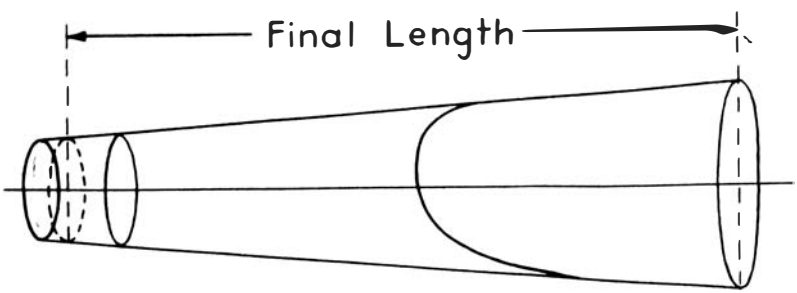


FIG. 3. *Diagram showing line-up of inner shell and end piece.*

20. With rubber contact cement a piece of heavy paper or corrugated cardboard is now glued as a cylinder (conical) connecting these two pieces.

21. This paper is then impregnated with plastic while held in the jig. When it has set, depressions or irregularities can be filled in with some cheap mixture to make the prosthesis cylindrical and symmetrical. (We use sawdust mixed with prevulcanized latex for this). This must be left set to harden and dry before proceeding.

22. Now three or four layers of stockinette are used to cover the entire prosthesis, and are impregnated carefully with plastic layer by layer, remembering to carry the plastic high on the prosthesis anteriorly, and lower posteriorly to allow for the hamstring tendons.

23. Finally the original long length of stockinette which made up the innermost layer of the stump socket is pulled down the outside over all and tied shut over the wooden end plug, and also impregnated with plastic.

24. The cord used to tie this shut is also convenient for hanging up the prosthesis in a place where it will not be disturbed while setting.

25. A careful remeasurement is now made of the required length of the prosthesis, and the end is cut off with a carpenter's saw.

26. A piece of rubber tyre is then glued and secured with a few 1 in. nails to the distal end.

The prosthesis is now ready for use. The majority of our patients have been able to use the prosthesis without further alteration. In some cases, however, the elastic cuff below the knee was not tight enough and it was necessary to add an above-knee supporting strap.

Strap for supporting Below-Knee Prosthesis

This strap is a very simple and unique method of supporting a below-knee prosthesis.

Procedure for making and fitting this Strap

1. The strap is made from rubber sheeting which is cut from automobile tyre inner tubing, which should be cut about 2 in. wide. A flat strap can be cut from an inner tube by cutting the entire tube round and round on a spiral, taking care that at the centre of the tube the strip is of the required width (2 in.). The tube is cut as in the diagram Fig. 4, and the strip obtained looks as shown in Fig. 5, and merely needs trimming of the edges to get a uniform, strong, elastic strap which lies perfectly flat.

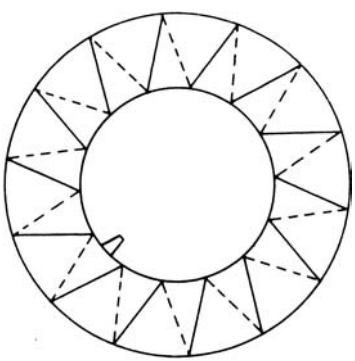


FIG. 4. *Manner of cutting Inner Tube.*

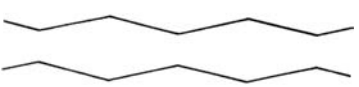


FIG. 5. *Strap obtained from Tube.*

2. The strap is attached to the prosthesis by either two or four $\frac{1}{2}$ in. sheet metal screws inserted on the popliteal side as in Fig. 6.

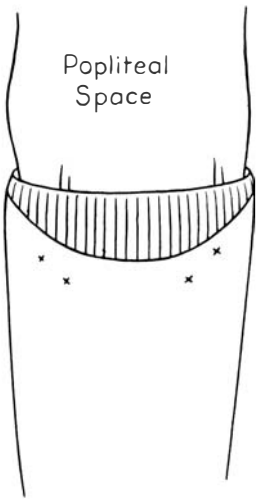


FIG. 6. *Site of fixing Supporting Strap.*

If it is found that the plastic does not extend near enough to the knee or is not thick enough in this area a bit can be added by surrounding the prosthesis near the top with a cuff of plastic impregnated stockinette.

3. The strap is then punched to fit over the one or two medial screws. It runs from there across the popliteal space posteriorly, laterally and superiorly and crosses the anterior of the leg superior to the patella. Here it turns inferiorly and medially and *crosses itself* on the posterior of the knee and is attached to the one or two lateral screws. It is well to rub the heads of the screws with chalk, then pull the rubber strap with some tension across them, thus marking the site for the holes with the chalk covered screw head. The chalkmarks thus show the proper sites for punching the holes in the strap. See Fig. 7.

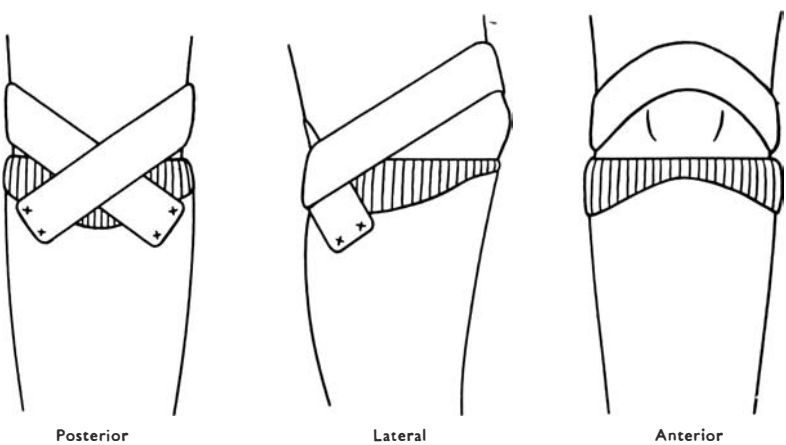


FIG. 7. *Diagram depicting the manner of application of Strap.*

This prosthesis fits very satisfactorily, and most patients have had no trouble walking with it immediately. At first its use must be graduated, and the enthusiastic patient must be curbed or ulceration may occur before the stump is sufficiently toughened to withstand weight-bearing.

THE PREVENTION OF PLANTAR ULCER IN LEPROSY

By E. W. PRICE, M.D., F.R.C.S.E.

The frequency of plantar ulcer is such that, in many places, new cases occur more rapidly than medical care can heal and prevent the recurrence of existing ones. A method of prevention is essential if progress is to be made.

There are several possible approaches to the problem, and their study needs to be correlated with the subsequent ulcer-history of the foot. They can be undertaken by anyone engaged in the care of leprosy patients and, with the exception of those for vasomotor disturbance, no special equipment is used. The purpose of this paper is to encourage interest in one or other method, so as to increase our knowledge of the clinical value of each technique.

Types of Plantar Ulcer

An important distinction must be made between the pliant foot and the rigid foot; the ulcers appearing on each type are of different etiology and are not likely to respond to similar treatment. The difference between the two types may be obvious, as between the foot of the healthy boy and that of an elderly person with subtalar arthritis; but in case of doubt, it can be demonstrated by asking the patient to rise on tip-toe. The ability to do this and the shape of the foot in this position will make the state of the foot apparent (Fig. 1).

The pliant foot will show the '*dynamic type*' of plantar ulcer, on the points of the sole, where the stresses of walking cause the breakdown in the protective mechanisms of the neuropathic foot. (*Lep. Review* 1959, **30,** 98.)

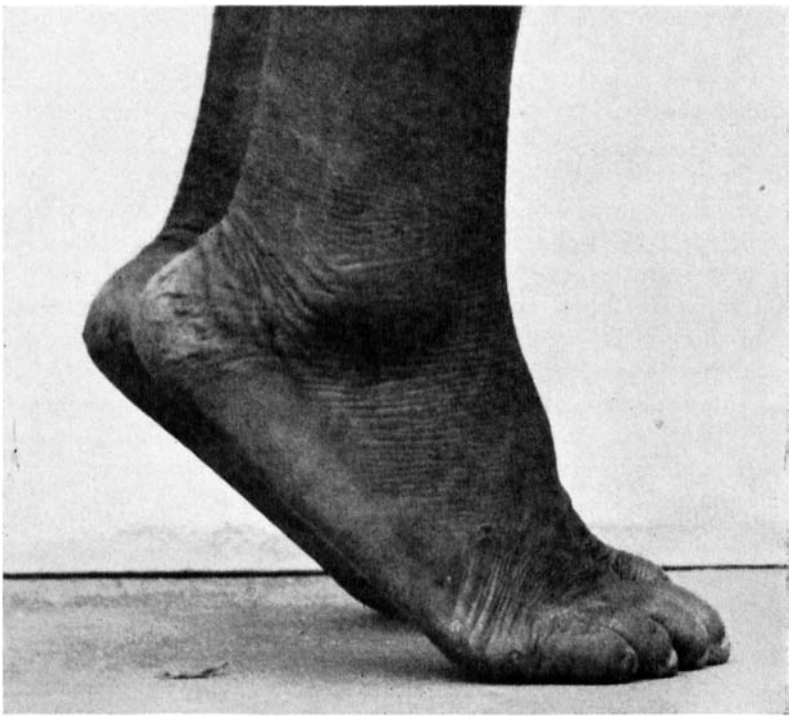
Measures which control these stresses (e.g. rigid-sole footwear) are likely to control the occurrence and recurrence of the lesion.

On the contrary, the rigid foot is more likely to show the '*static type*' of ulcers, which are pressure lesions at whichever part of the deformed foot is most prominent in the sole. The rigidity of the foot is due to the scarring of chronic infection and injury, and to infective or degenerative arthritis. Footwear may be designed to redistribute weight as local circumstances permit; but many cases become candidates for amputation.

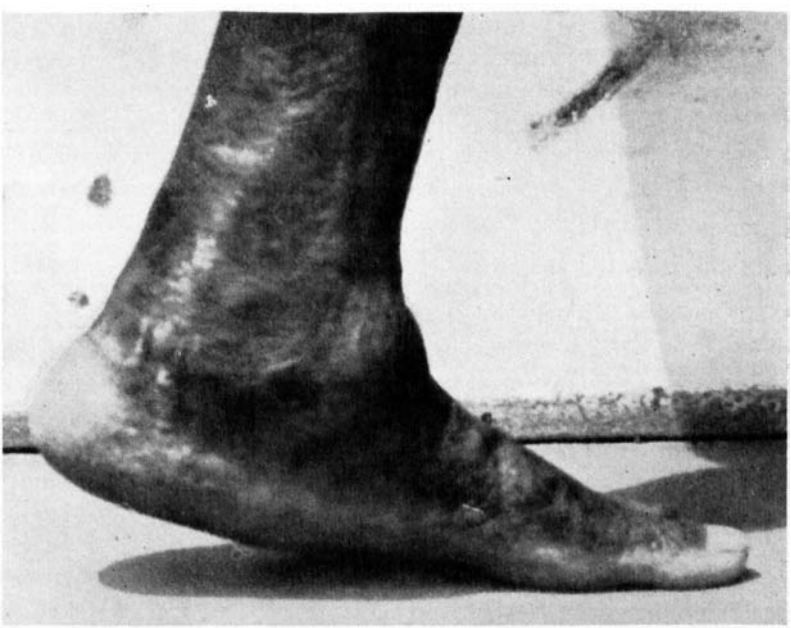
A Notation for Plantar Ulcers of the Pliant Foot

The ulcers to be studied are of the 'dynamic type' and a rapid method of recording the lesion is essential for accuracy of comparison. The following has been used for some years and is found to be satisfactory (Fig. 2.)

The Tip-Toe Test of Foot-Type. Fig. 1.

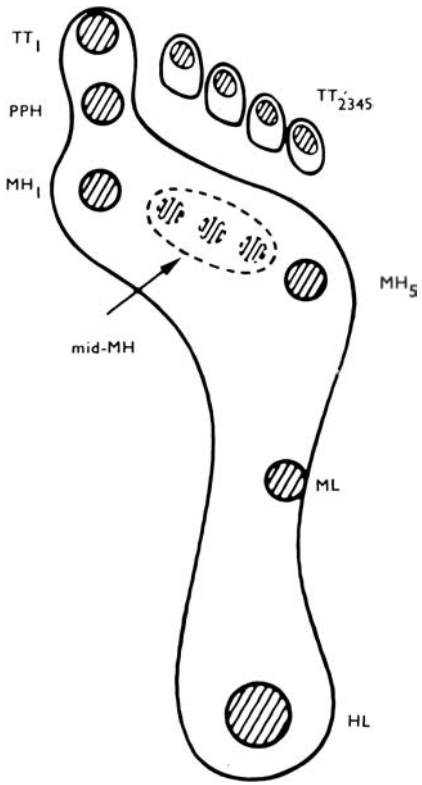


(*a*) THE PLIANT FOOT. *The body rises on 'tip-toe' on to the heads of the metatarsals. If this foot ulcerates, the ulcer will be of the 'dynamic' type.*



(*b*) THE RIGID FOOT. *The foot with scarred plantar tissues, or with diseased bones and joints, is unable to rise on 'tip-toe'. Plantar ulcers of this foot are of 'static' type and are traumatic wounds on whichever part of the sole is most prominent.*

Notation for Dynamic Plantar Ulcers. Fig. 2



Letters refer to underlying bony prominence:

TT = *toe-tip*
PPH = *proximal phalangeal head of big toe*
MH = *metatarsal head*
ML = *mid-lateral*
HL = *heel*

Multiple ulcers on lateral toes are reckoned as a single ulcer spread over more than one toe.

1. The ulcers at impact (heel) and push-off (toe tips) are denoted by HL and TT.

2. The ulcers at points of plantar flexion are indicated by reference to the underlying bony prominence: MH_{1-5}, PPH; or as ML (mid-lateral) in the case of the tuberosity of the fifth metatarsal.

Reference is also made to the side (R or L), and to the diameter of the ulcer, e.g. RMH_1 2 cms = an ulcer 0·2 cm. large under the first metatarsal head of the right foot.

Toe tip ulcers are often multiple, and are recorded as occurring on the big-toe (TT_1) or on the lateral toes (TT_{2345}). Multiple ulcers of the lateral toes are counted as one ulcer spread over several toes. The true plantar ulcer of the toe tip must be distinguished from the terminal ulcer sometimes seen in claw-toes, and from the dorsal ulcer of some cases of foot-drop.

The common ulcer under the interphalangeal joint of the big toe

(PPH) may in special cases (e.g. loss of big toe) be replaced by one under the proximal inter-phalangeal joint of the second toe and is then recorded as PPH_2.

Methods of Study

Possible means of knowing that plantar ulcer is about to develop are:

1. Recognition of early sensory loss.
2. Recognition of early motor loss.
3. Recognition of vasomotor disturbance.
4. Clinical recognition of the pre-ulcerative state.

Stress is laid on the necessity for early recognition. To await complete anaesthesia, obvious foot drop or incipient infection of a callosity is known to be too late. The methods proposed here are designed to detect early loss of neural function.

(*A*) *Tests for mis-localisation of pressure*

In early attempts to recognise pre-ulcerative feet, it was soon found that the criterion of anaesthesia to pain (e.g. test by pin prick) did not reveal some feet that in the event finally ulcerated. Attention has been drawn to this phenomenon by LANGUILLON *et al* (1960), but its significance is not yet known. An earlier stage of sensory loss must be sought, and one such is that of mis-localisation of pressure (MLP).

Recent work (WEDDELL, 1962) has shown that each area of skin is supplied by axons which may enter the spinal cord by as many as four dorsal roots.

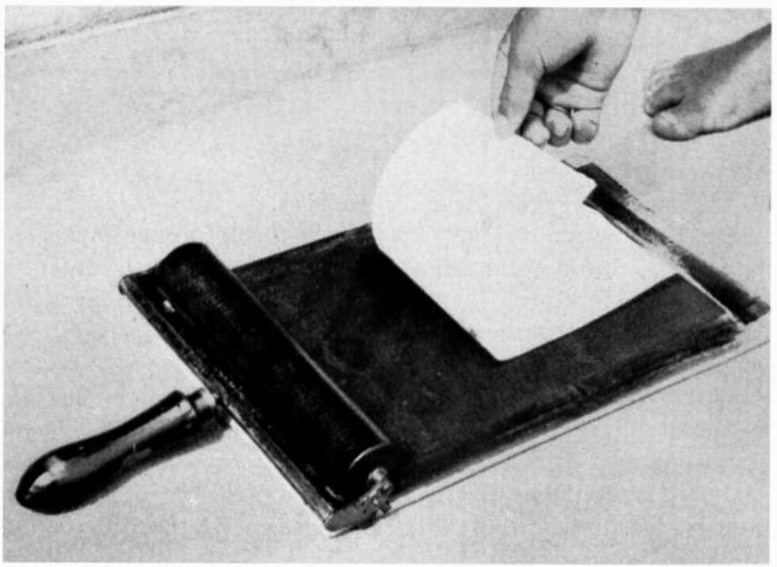
Accurate localisation depends on the integrity of these pathways but as many as 25% may be destroyed without clinical evidence of loss. Further denervation results in a state when the patient is aware he is being touched but is unable to localise accurately. This state offers a field of study that may reveal a significant relation to pending ulceration. Tests already done show that, for each neuropathic foot that is not completely anaesthetic, there is a threshold pressure below which localisation is faulty or absent, but above which recognition with or without accurate localisation is present. This threshold area needs proper investigation with reference to future plantar ulceration.

Before attempting the test on abnormal feet, the reaction of normal feet in the region under examination should be learnt by experience. The test is then done as follows:

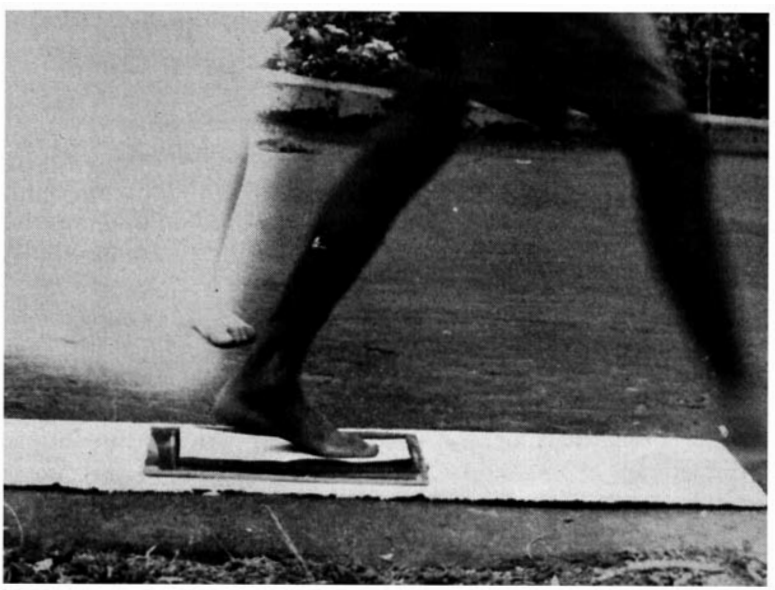
The ulcer-bearing area of the sole is systematically stimulated at random using a nylon thread which projects for 1 cm. from a holder. Pressure is increased until the thread yields. The patient (blindfold) then localises each spot with his finger. Accurate localisation is considered normal.

It has been found wise to allow the patient to do the test first with

The Kindetograph or Dynamic Foot-Print (after Morton). **Fig. 3**



(*a*) THE APPARATUS. *A thin rubber mat with compressible corrugations or scraper-board with embossed pattern is inked with a roller, as in the figure. This is then removed and the mat is covered with paper.*



(*b*) THE METHOD OF USE. *The patient walks across it and the resulting imprint is the total of the sequence of pressures from heel to toes as the step is taken. Imprints are shown in Fig.* 4.

the eyes open and then to cover the eyes completely, and not rely on his shutting his eyes, or covering them with his hand.

The results should be recorded on a diagram of the sole, and should include the pressure used; this can be found by using the same pressure on the pan of a laboratory balance. Cases with loss of localisation should be followed serially without treatment, repeating the test at monthly intervals, to see whether or not ulceration occurs subsequently.

(B) The 'kinetograph' or Dynamic foot-print

Early loss of motor innervation can be demonstrated by the use of the foot-print of the walking foot. The pressures represented by the 'dynamic' foot-print (DFP) are the resultant of muscular action on toes and foot, together with the weight of the body in motion. Disturbance of this balance is seen as an abnormal DFP.

The normal DFP shows that after initial impact at the heel, the pressure passes rapidly to the head of the 5th metatarsal, clearing the intervening area. It then proceeds across the metatarsal pads to its maximum under the first head, and then proceeds forward to the tip of the big toe, also clearing the intervening area. All the toes combine for the final push-off.

The dynamic foot-print is obtained by the use of a rubber mat or MORTON board such as artists' scraper-board (LAKE) on which a fine geometric pattern is embossed.

The print is taken as follows: (Fig. 3)

The mat is inked with printer's ink using a roller. A sheet of absorbent paper, such as newsprint, is laid lightly on top, and the patient walks barefoot across it, stepping on the paper with one foot as he continues onwards.

The mat is then re-inked and the other foot treated similarly. The prints are allowed to dry and are then added to the records. It is important to re-ink between each test, to protect the mat from dust after use, and to note that the print is that of the opposite side to what it first appears to be. As with other tests, familiarity with the normal is essential (Fig. 4*a*).

Interpretation of Foot-prints

The common deviations of interest are:

(i) Lateral displacement of maximal metatarsal-pad pressure (Fig. 4*b*).

The normal maximum at MH_1 may be displaced to MH_2 or even more laterally. Some displacement is normal if the patient uses an intoeing gait ('pigeon-toes'), but otherwise, it indicates failure of the pronators of the foot.

(ii) Appearance of mid-lateral pressure (Fig. 4*b*).

The appearance of an area of pressure mid-laterally between heel and fifth metatarsal pad represents early peroneal failure and is common with the previous abnormality.

(iii) Appearance of pressure area at base of big-toe (Fig 4*c*).

Pressure at the base of the big toe under the inter-phalangeal joint represents weakness of the extensor hallucis longus.

(iv) Areas of concentrated pressure (Fig. 4*d*).

These are common at the metatarsal pads, the proximal phalangeal head of the big toe and the base of the fifth metatarsal. They represent failure of intrinsic musculature, and are often precursors of ulceration.

(v) 'Clutching' toes and 'cocked' toes (Fig. 4*c*).

These examples of muscle spasm due to nerve irritation, and of palsy are appropriately indicated by the DFP, and may precede ulceration of toe tips.

It must always be recalled that other conditions than leprosy can upset the normal balance of the foot. None of the above abnormalities alone represent a lesion of leprosy; but taken with other signs, they may be highly significant.

It will be found that some patients show similar areas of increased pressure on both feet; yet only one side ulcerates. There are evidently additional factors to be elucidated.

(*C*) *Tests for Vasomotor Dysfunction*

The tests for vasomotor dysfunction include those based on variations of skin temperature and usually undertaken in centres of research. These lesions have been fully investigated by several workers such as GOKHALE *et al* (1959) but the results are inconclusive other than demonstrating that there is a loss of vasomotor control in leprosy.

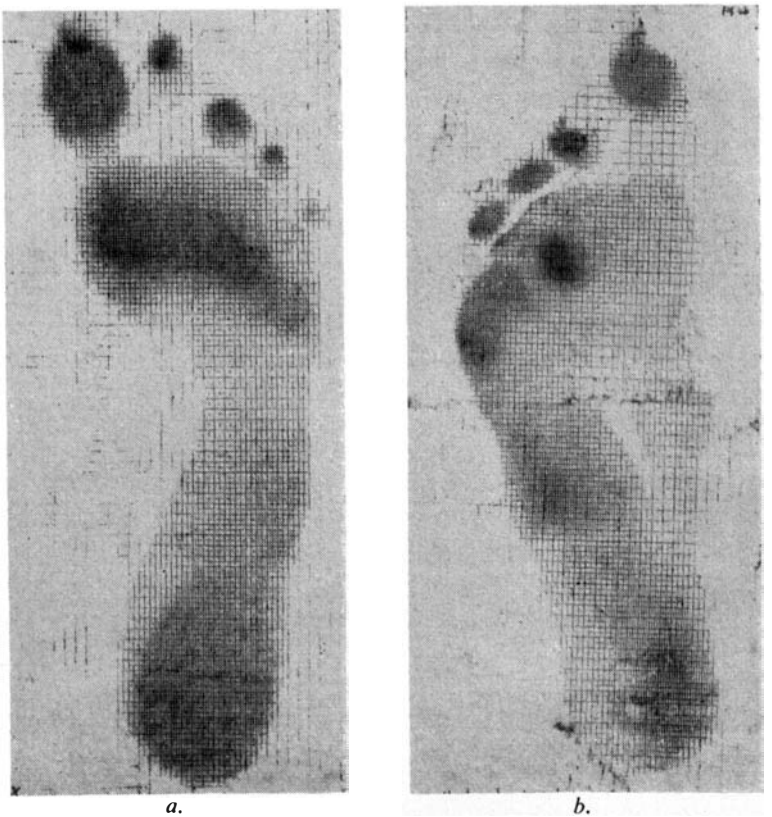
In view of the special apparatus needed, test for vasomotor loss are unlikely to be of wide application in local centres of leprosy treatment and are therefore omitted from this study.

(*D*) *Clinical Recognition of the Pre-ulcerative State*

The early signs and symptoms of the pre-ulcerative state are described in *Lep. Review* 1959, **30,** 180. Further evidence is the condition of 'shiny pads'; the metatarsal pads and toe-tips appear polished and burnished, due to repeated friction with the ground at each step, as the forefoot slips slightly forward at maximal body-weight. This condition discloses failure of the toes to grip the ground and represents early failure of the intrinsic musculature.

It is not always realised that the pre-ulcerative state, of which the signs may be localised plantar swelling and tenderness, splaying of toe, necrosis blister, and shiny pads, is only likely to be seen when weekly foot-inspection is practised. The cycle of events from early deep tissue damage to frank ulcer may occur in ten days, and even fortnightly inspections miss many cases. In a recent series of cases carefully observed for periods up to 18 months in Eastern Nigeria, this state was noted at sometime or other in about half the cases under observation for the pre-ulcerative state; a few displayed it on

Dynamic Foot-Prints (DFP). Fig. 4



a. *b.*

These are obtained by a sequence of imprints beginning at the heel, and passing in sequence to the 5th MT pad, across to the 1st MT pad, and then to the final push-off in which all toe-tips normally co-operate.

(*a*) NORMAL. *Note the gap between heel and 5th MT pad, and between 1st MT pad and 1st toe-tip. Maximal pressure is at 1st MT pad.*

(*b*) PRONATOR WEAKNESS. *Maximal pressure shifts laterally on the MT pads, and mid-lateral pressure appears. Lateral shift on MT pads by itself may be due to an in-toeing gait ('pigeon toes').*

several occasions. In each case, ulceration was avoided by complete bed rest from ten to twenty days without other treatment.

Clinical evidence of the pre-ulcerative state is the last opportunity to avoid ulceration. Failure to recognise it penalises the patient by the development of an open ulcer, which may last for months or years if inadequately treated. It is repeated that the above condition is only likely to occur in the *pliant* foot.

Discussion

It is likely that multiple factors are responsible for the moment of ulceration in a pliant neuropathic foot, though the dominant role of the traumatic effect of walking appears to be established. The

c.

d.

(*c*) FAILURE OF BIG TOE CONTROL. *Pressure appears between 1st MT pad and big toe and may be the maximal. It represents failure of big toe extensors or spasm of the flexors. Note the 'clutching' of 3rd and 4th toes, and the 'cocking' of 5th toe, due to intrinsic disturbance.*

(*d*) LOCALISED INCREASE OF PRESSURE. *The extreme pressures on pads of* MT_1 *and* MT_5 *are obvious and represent intrinsic failure of these digits. They often precede ulceration and are an important danger signal.*

immediate need is for a method of recognising clinically, or by simple tests usable in rural conditions, when a foot is likely to ulcerate or is on the point of doing so. The latter need appears to be met by the recognition of the preulcerative state which precedes ulceration by less than ten days. None of the tests described here is found to disclose by itself the foot which is likely to ulcerate, but it is possible that a combination of tests and clinical observation may be adequate.

Apart from the obvious advantage of knowing that a foot is in the danger period – and when, conversely, it has passed out of that period – there is the practical advantage of avoiding unnecessary, and often monotonous, foot-inspection and the provision of footwear that will have been unnecessary.

In view of the number of workers who see patients at close quarters in leprosaria, and who are ideally placed for following

clinical events and ulceration, it is hoped that this paper will encourage the careful testing and observation of a series against an equivalent number of control cases who are simply observed without testing or preventive treatment. In this way, the value of each method will be apparent.

Summary

1. The importance of prevention of plantar ulceration is stressed.

2. A distinction is drawn between 'dynamic' and 'static' types of plantar ulcer.

3. Methods of determining an early state of neural loss are defined with a view to clinical trials of their value in the prevention of ulcer.

4. Among the multiple factors that precipitate ulceration are noted: sensory loss, motor loss, vasomotor loss, lack of foot-inspection, lack of co-operation from the patient.

The rubber mats can be obtained from Down Bros. Ltd., 70 Grenville Street, Toronto 5, Ontario, Canada (U.S. $8 each); and scraper-board can be obtained from Winsor & Newton Ltd., Wealdstone, Harrow, Middlesex, England. A size and quality suitable are No. 17 white, 19 in. by 12 in. which costs 4s. (U.S. $0.60) a sheet, and will provide two pieces, available two days use each, for any number of patients on one day before it is finally discarded.

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AN INVESTIGATION INTO BACILLAEMIA IN LEPROSY

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Recent literature contains few references to the subject of bacillaemia in leprosy. MANSON-BAHR (1954) states that *Mycobacterium leprae* have been found occasionally either free in the blood or engulfed in leucocytes. IYENGAR (1919) found acid-fast bacilli in the mononuclear leucocytes in venous blood. SHTEIN and TUTKEVICH (1957) detected *M. leprae* in 115 out of 226 thick blood films prepared from finger-prick blood in 59 patients with lepromatous leprosy. Earlier literature has been reviewed by LOWE (1933) who describes a technique for concentrating the bacilli in a sample of venous blood, which was more reliable than the thick film method. By the concentration technique acid-fast bacilli were detected in 2 out of 23 cases of neural leprosy (tuberculoid) while in 51 cutaneous leprosy (lepromatous) 28 were found to have *M. leprae* in venous blood.

LOWE (1933) points out that the thick film method is unreliable and that positive results obtained by this method may be false, bacilli from the skin itself contaminating the smear. He also considered that contamination of venous blood from the skin might also occur during the venepuncture and this might invalidate the results.

This paper presents the results of examination of venous blood in patients at the Alupe Leprosarium.

Methods

Venous blood (2 ml.) was obtained from the median basilic vein in 101 cases of leprosy. The blood was expelled into a McCartney bottle containing oxalate. After thorough mixing, a drop of blood was placed on a slide and a thick smear prepared. The smear was dried at 37° C in an incubator and then stained by the RHODES-JONES (1959) modification of the Ziehl-Neelsen method.

In two patients the vein was exposed surgically before obtaining blood from the vein.

In six patients with a large number of acid-fast bacilli in the venous blood a needle was inserted of a sterile 2 ml. syringe, containing 0·5 ml. normal saline, parallel to the median basilic vein but without puncturing it. The contents of the syringe and needle was washed out into a McCartney bottle; the outside of the needle also being rinsed in the saline. All the fluid was then slowly evapora-

ted drop by drop on to a heated albuminised slide and stained in the manner described. The whole smear was examined.

All techniques were carried out with aseptic precautions, and all bottles and syringes sterilised.

Results

The results of the examination of the venous blood of 101 patients with leprosy are given in Table 1.

In the patients from whom venous blood was obtained after surgical exposure of the vein, seventeen acid-fast bacilli were found in the smear from one, and two in the other.

In the experiment designed to test the possibility of the contamination during the tracking of the needle, no bacilli were found in smears obtained from the two lepromatous subjects and one patient with dimorphous leprosy. A single bacillus was detected in two others with dimorphous leprosy and three bacilli were found in a borderline case.

TABLE 1

Classification	*Examined*	*With acid-fast baccilli in leucocytes*	*Total positive*
Lepromatous	59	8	26
Tuberculoid	22	0	4
Borderline	7	0	4
Dimorphous	9	1	5
Indeterminate	4	0	3
Total	101	9	42

Among the patients examined, several had a history of leprosy for a duration of two years or under and the lepromatous among these showed a higher incidence of Bacillaemia as shown in Table 2.

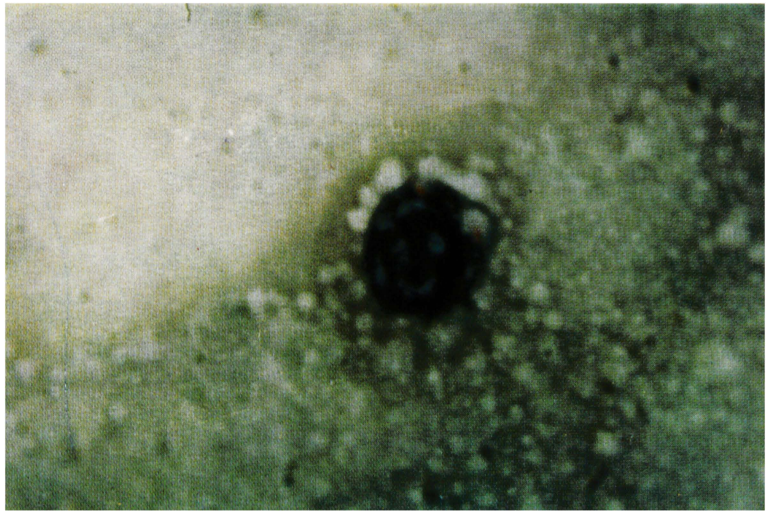
Discussion

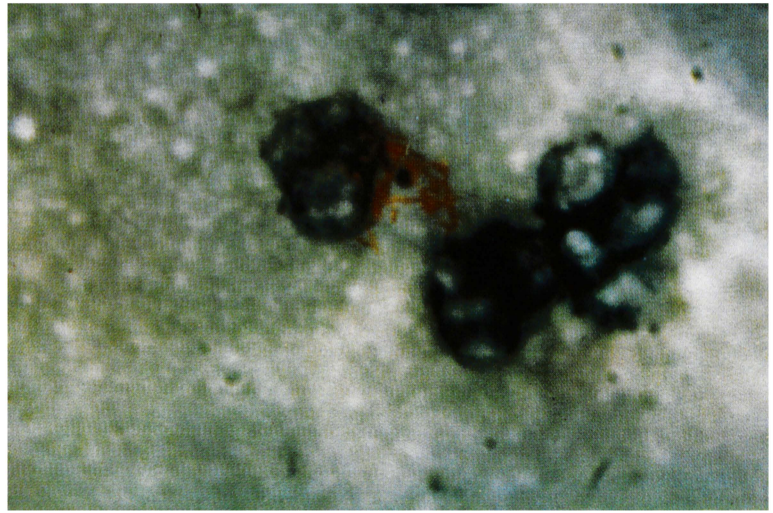
In the early stages of the disease, Bacillaemia is common in cases of lepromatous leprosy and can be present in cases who have been under treatment for over two years. In the four positive tuberculoid cases, only one or two bacilli were detected in each.

M. leprae are scanty or absent in the material obtained by the needle tracking through the subcutaneous tissue.

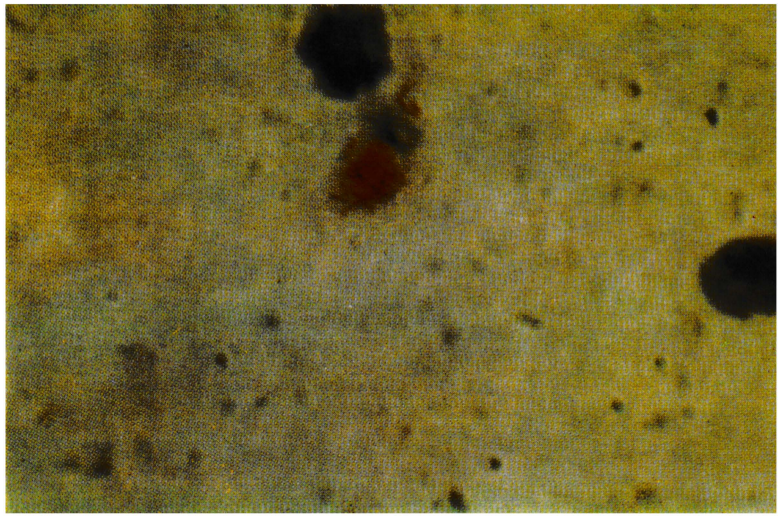
Those patients who had bacilli within the leucocytes, had had little or no treatment at the time of the investigation.

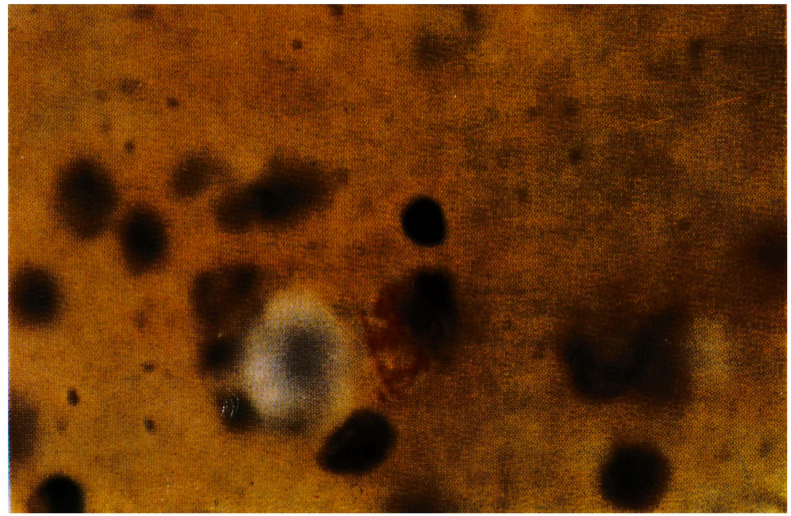
COLOURED ILLUSTRATIONS TO THIS PAPER BY R. RHODES-JONES OVERLEAF





Examples of Acid – Fast Bacilli in Venous Blood of Leprosy Patients.





Examples of Acid – Fast Bacilli in Venous Blood of Leprosy Patients.

TABLE 2

Classification	*Examined*	*With acid-fast bacilli in leucocytes*	*Total positive*
Lepromatous	14	8	13
Tuberculoid	12	0	1
Borderline	1	0	0
Dimorphous	6	1	3
Indeterminate	3	0	2
Total	36	9	19

Summary

Venous blood from 101 cases of leprosy was examined for bacillaemia and of these, 42 were positive.

An attempt was made to evaluate the possibility of contamination of the needle by bacilli in the skin.

Acknowledgments

I wish to thank Dr. John Garrod for his encouragement while carrying out these investigations, Dr. R. F. MacKnight for the surgery, Dr. D. H. H. Robertson for his advice in the preparation of this paper, Mr. Justo Obwa for technical assistance, Mrs. J. Rhodes-Jones for the microphotography and the Secretary-General of the East African Common Services Organisation for permission to publish.

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*SHTEIN, A. A. and TUTKEVICH, L. M. (1957) *Abstract Lep. Review*, 1959, **30,** 263.

* Attempts to obtain the original article were unsuccessful.

THE SO-CALLED "MACULO-ANAESTHETIC FORM" OF THE INDIAN CLASSIFICATION OF LEPROSY

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The recent publication by DHARMENDRA and CHATTERJEE of two articles, almost identical, on the subject of maculo-anaesthetic leprosy [4,5], compels us to expound our point of view with more details than we did in our publication entitled *Classification of Leprosy*[1].

We should like first to make some remarks on terminology though this has not really a prime importance. Nevertheless, in an international primary classification, the use of dissimilar terms should be avoided as much as possible. Therefore we did write in our article[1]: 'It would be unfortunate to use, as the Indian leprologists wish to do, histological definitions for the tuberculoid and lepromatous forms and the clinical definition of maculo-anaesthetic leprosy for the indeterminate form. And the more so, since certain skin lesions of tuberculoid leprosy and sometimes even lepromatous ones, may equally well be described clinically as maculo-anaesthetic*.' This objection was successful enough to excite the interest of DHARMENDRA and CHATTERJEE because we had not understood that they gave a strictly clinical meaning to the terms 'tuberculoid' and 'lepromatous'. But whatsoever particular sense these authors want to give those two words, the meaning of 'tuberculoid' and 'lepromatous', whether they wish it or not, is only a histopathological meaning and we do not think it advisable to use a mixture of histopathological and clinical terms to name the principal forms of leprosy in an international classification.

But leaving aside the question of terminology, is it really worthy of interest to include a supplementary maculo-anaesthetic form in the primary classification of leprosy? This might be justified if the result were to allow a correct classifying of the patients. For DHARMENDRA and CHATTERJEE the maculo-anaesthetic form is a well defined clinical entity. But, when reading carefully what they write, one finds that neither clinical experience, nor bacteriology, nor immunology enables one to see clearly the difference between a maculo-anaesthetic lesion and an indeterminate macule**. Indeed, in

* There is a mistake on our part since DHARMENDRA and CHATTERJEE maintain the indeterminate form in their classification in which it is grouped with 'Borderline' leprosy under the name of 'intermediate' leprosy. However our mistake has no effect on the remarks concerning terminology.

**We would mention that in this article we are using the terms 'macule' and 'macular' in their strict dermatological sense.

each of the two groups, the lesions are flat and hypopigmented, they appear to be more or less anaesthetic, Hansen's bacillus may be present or absent and the lepromin reaction may be positive or negative. According to the Table published by these authors the only point which would permit one to distinguish between the two lesions would be that the maculo-anaesthetic lesion is dry and the indeterminate lesion is not dry. We doubt very much whether this would be sufficient to make a differential diagnosis. As to the histopathological picture of the maculo-anaesthetic lesion it appears identical with that of a pretuberculoid indeterminate lesion (presence of infiltrations of epithelioid cells). Therefore it is surprising that among all eminent Indian and foreign clinicians, attending the meeting of the Indian Association of Leprologists, one epidemiologist only, J. A. DOULL, did make the pertinent following objection: "I would point out however that in some parts of the world the maculo-anaesthetic case would be called 'indeterminate', and that therefore it is necessary to provide a sharper differentiation between maculo-anaesthetic and indeterminate than is given in DHARMENDRA's paper".[6] But is this maculo-anaesthetic form, at least, clearly distinct from tuberculoid leprosy? We do not think so. Indeed DHARMENDRA and CHATTERJEE admit that maculo-anaesthetic lesions may become erythematous and that their hypopigmentation may be masked by hyperpigmentation*. Now erythema and hyperpigmentation are especially to be seen in regressive tuberculoid macules. On the other hand, these authors place under the heading of 'maculo-anaesthetic' cases who are probably typical tuberculoid patients (hypertrophy of cutaneous nerves corresponding to macules; cold abcess in peripheral nerves). Lastly the maculo-anaesthetic lesion can only be distinguished from the macular tuberculoid lesion by a histopathological examination.

From the above it can be inferred that in fact the maculo-anaesthetic form of the Indian classification consists of a mixture of pure indeterminate and pretuberculoid indeterminate cases on the one hand and on the other of macular tuberculoid and regressive tuberculoid cases.

DHARMENDRA and CHATTERJEE consider that the adoption of the maculo-anaesthetic form would help medical auxiliaries to a better classifying of their patients. Personally we are rather sceptical as to the possibility of a correct classifying of patients by most of the medical auxiliaries and that whatsoever may be the classification. Besides we do not understand how it could be easier to class patients suffering from benign leprosy into 'tuberculoid', 'maculo-anaesthetic' and 'indeterminate' instead of just grouping them into 'tuberculoid' (cutaneous lesions more or less elevated or with a surface or a border

* Hypopigmentation 'masked' by hyperpigmentation seems a very curious phenomenon.

slightly wrinkled) and into 'indeterminate' (smooth cutaneous lesions strictly flat).

But what is more serious still the Indian leprologists themselves seem to have some difficulties in classifying the patients into the maculo-anaesthetic form. Thus, in the course of the discussion on MUKERJEE and GHOSAL's communication: *Diagnosis of maculo-anaesthetic cases of leprosy*,[7] DHARMENDRA declared: 'Regarding MUKERJEE's paper, the results of the lepromin test in this series do not fit in with the usual findings in such cases. I doubt there is something wrong somewhere. Either the lepromin used has not been properly standardised, or perhaps there is something wrong in the selection of patients, or something else'.[6] It appears likewise that there is a confusion as far as the clinical aspect of this maculo-anaesthetic lesion is concerned. DHARMENDRA and CHATTERJEE described its surface as perfectly flat, what is chiefly a character of indeterminate lesions. RAMANUJAN notes that these macules have sometimes a wrinkled appearance, which is usually attributed to regressive tuberculoid lesions. BROWN, through the lens, has often seen a uniform presence of micropapules, which is generally considered as a characteristic sign of minor tuberculoid leprosy. Lastly MUKERJEE and GHOSAL even give the name of 'maculo-anaesthetic' to infiltrated lesions elevated sometimes up to 3 mm., that is to say probably typical tuberculoid lesions.[7]

One is therefore justified in concluding that the addition of a maculo-anaesthetic form to the primary classification of leprosy is not to be recommended. Far from making easier a precise classification of patients the adoption of this new hybrid group would only introduce more confusion into the subject.

To influence favourably the admission by all leprologists of a maculo-anaesthetic form in the classification of leprosy, DHARMENDRA and CHATTERJEE declare [4,5]: 'It should be clearly understood that the terms macular tuberculoid (of the Madrid Classification) and maculo-anaesthetic (of the Indian Classification) refer to one and the same type of lesion'. These authors try to strengthen this assertion by denying the macular tuberculoid leprosy of the Madrid classification any tuberculoid character. We do not share in the least that opinion. Really a strictly flat lesion cannot be termed 'macular tuberculoid' unless it shows a histopathological structure of tuberculoid nature. It is therefore absolutely misleading to pretend that 'maculo-anaesthetic' and 'macular tuberculoid' may be considered as synonymous terms.

Personally we are not convinced that there is a dominant need to include a macular tuberculoid variety in the classification of leprosy. This moreover appears clearly from our article *Classification of Leprosy*[1]. In fact macular lesions which are indisputably tuberculoid, showing neither elevation even partly, nor sign of an anterior

elevation, are relatively rare. Besides they can only be detected by medical teams including a histopathologist, competent in leprology, since the diagnosis cannot be made through a clinical examination. If it is deemed necessary to range macular tuberculoid leprosy among the secondary classification, the classifying of the patients belonging to this variety should be only entrusted to teams able to undertake histopathological researches. The others ought to use only the old Havana classification which divided benign leprosy into two types: 'tuberculoid' and 'indeterminate' and only admitted two varieties: 'minor' and 'major' for the tuberculoid type. Lesions thoroughly flat and smooth would be classed into indeterminate leprosy and lesions more or less rising or micropapulous included in tuberculoid leprosy, it being well understood that flat lesions showing a surface or border slightly wrinkled would be considered as regressive tuberculoid lesions. By this procedure macular tuberculoid leprosy could be more thoroughly studied by teams able to diagnose it with certainty whilst physicians who have no histopathologist working with them would not run the risk of introducing great confusion in the variety through including pure indeterminate and pretuberculoid indeterminate cases on the one hand and macular tuberculoid and regressive tuberculoid cases on the other. We also wish to add a few more words about terminology. 'Macular' is a descriptive word, whereas 'minor', 'major' and 'borderline' indicate different degrees of the infection. So if we wish to include this variety in the secondary classification it would be preferable to replace the word 'macular' by a more appropriate term. The adjective 'atypical' might be suitable, since the elevation above the surface of the skin, absent from the macule, is one of the principal clinical characteristics of the tuberculoid cutaneous lesion.

We remain entirely in agreement with the first Expert Committee on leprosy[3] which stated that the basic criteria of the primary classification of leprosy should be clinical and bacteriological, but that when a scientific study of cases is made immunological and histopathological criteria should be fully used to determine certain subgroups. Now the macular tuberculoid variety is precisely one of these sub-groups.

Lastly we should like to repeat what we wrote in our article *Classification of Leprosy*[1]: 'But it should always be borne in mind that there are certain intermediate and transitory stages that exist between different forms and even between certain varieties of leprosy, and which can sometimes be detected only by histological examination. In our opinion these intermediate stages cannot be considered as varieties as we describe them and they ought not, except for borderline leprosy, to be taken into account in the secondary classification. Similarly the reactional states, whether of long or short duration, which alter, for good or for ill, the normal course of

the disease, cannot be classified as different varieties. The use of the terms "pretuberculoid", "tuberculoid reaction", "tuberculoid reactional transformation"[2], "prelepromatous", "lepromatous reaction" and "erythema nodosum" will permit us to describe these transitory stages of the disease'.

Summary

The Indian leprologists consider that a supplemental form named 'maculo-anaesthetic' should be introduced in the primary classification of leprosy. In two similar articles DHARMENDRA and CHATTERJEE even assert that this form constitutes a clinical entity clearly determined. However when studying these authors' text one notices that neither clinical experience nor bacteriology nor immunology allows one to distinguish with certainty a maculo-anaesthetic lesion from an indeterminate macule, from a macular tuberculoid lesion or from a regressive tuberculoid macule. It is therefore erroneous to pretend that the maculo-anaesthetic form constitutes a clinical entity well defined. On the other hand, histopathology shows that this so-called 'form' is really identical with pretuberculoid indeterminate leprosy which means that it represents an intermediate transitory stage between indeterminate leprosy and tuberculoid leprosy which cannot be incorporated in the classification of leprosy either as a 'form' or as a 'variety'.

To favour the admission of that maculo-anaesthetic form in the classification of leprosy DHARMENDRA and CHATTERJEE declare that the expressions 'macular tuberculoid' in the Madrid classification and 'maculo-anaesthetic' in the Indian classification have the same meaning and cover the same type of lesion. These authors try to strengthen this assertion in denying the macular tuberculoid leprosy of the Madrid classification any histological tuberculoid characteristic. In our opinion it is quite erroneous to pretend that 'maculo-anaesthetic' and 'macular tuberculoid' may be considered as synonymous expressions. For indeed a cutaneous lesion of benign leprosy, rigorously flat, cannot be named 'macular tuberculoid' unless it shows a histopathological structure of an unquestionably tuberculoid nature.

It therefore appears that it is not possible to include this so-called 'maculo-anaesthetic form' in the international classification of leprosy. Far from making easier a precise classification of patients, the adoption of this new hybrid group would only bring more confusion.

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AN INTERESTING CASE OF LEPRA REACTION

By DR. DOUGLAS E. HENRY, B.SC., M.B.B.S.
The Mission to Lepers, Chandkhuri Leprosy Hospital and Homes, P.O. Baitalpur, via Bhatapara, M.P., India.

Reactions in leprosy are many and varied. At times some individuals present certain manifestations which are difficult to classify. The following case report is one such example. In this case the clinical picture resembled that of a Lucio-phenomenon. But on closely following the case this idea was over-ruled because, (1) up till now it has not been seen in Indian patients, and (2) the histopathology did not support this view.

However, one peculiarity in this case was the keratin plug in the centre of the lesions which differentiated it from other simple bursting nodules. At first this was not readily explained but on closely following the history and referring the literature it was more or less confirmed that the lesions were modified by the presence of keratosis blenorrhagica due to chronic gonorrhoeal infection.

Case Report

Name of the patient: Jwala Prasad Tiwari S/O Banwali Prasad Tiwari; age – 35 years, sex – male, caste – Hindu Brahmin. Resident of village Maro (which is 7 miles from this institution). Former occupation – village teacher in a Primary School.

Family History: Married 15 years ago. Wife alive and well. Only one son, alive and well. None in the family had suffered from leprosy.

History of Contact: Lived for one year with a friend who was later diagnosed as suffering from leprosy.

Past History: In 1942 (20 years ago) the patient developed gonorrhoea which subsided after some treatment. In 1943 developed burning sensation in the extremities and eyes. He had frequent bouts of spermatorrhoea at nights and this cleared up after some time.

Present History: Started in 1953, when a diffuse hypopigmented patch resembling ringworm infection was seen on the right shoulder and another was seen on the back (5 years after the first patches were noticed) in 1958. The patient noticed some greasiness of the face and started having reddish nodules on the extremities. These nodules became painful and turned into blisters which later burst leaving small ulcer craters. These healed by treatment leaving whitish scars. The patient has been getting these attacks off and on and these have coincided with the recurrence of acute attacks of gonorrhoeal urethritis which seems to be resistant to penicillin therapy. Sometimes the

nodules are numerous and they coalesce making the extremities very thick and very painful due to associated periostitis.

Physical Examination

He is a well developed male patient. The face looks greasy and there is uniform thickening of the skin of the face. The outer parts of both eyebrows have fallen out. There are two big subcutaneous nodules on right cheek and several small ones on both the cheeks. Heart, lungs and abdomen do not show any peculiarity. Extremities are covered with whitish scars and there are some subcutaneous nodules which are reddish and shiny. At other places there are ulcer craters formed by bursting nodules and some are covered with hard brownish crusts.

Laboratory Findings

3+ and 2+ for lepra bacilli in skin smears.

Urethral smear – Extra cellular diplococci seen.

Stool – Hookworm ova present.

Kahn test – ++++

Urine – Acidic, Sp.gr. 1010, albumen and sugar negative.

H.B. – 65% Total R. B.S. 35 millions cu.mm.

Smear of pus from the ulcer craters – Negative for diplococci. Tentative diagnosis – Reaction in leprosy, probably of Lucio type?

Treatment

The patient was treated for gonorrhoea, syphilis and hookworms and the haemoglobin became 80% with 4.14 millions of RBC/cu.mm. Unfortunately this patient could not tolerate dapsone, sulphetrone and thiosemicarbazone. He got acute exacerbations with any of these drugs. But he could tolerate about 150 mgm. of INH per day, Etisul and Ciba-1906 were not tried because they were not available.

For reactions he was treated with PAT, Calcium Gluconate injections, APC, Irgapyrine and Butarin tablets, but relief was obtained only after using corticosteroids like Millicorten and Kenacort or Delta-efcortin.

Progress

In four years of hospitalisation the patient showed very slow but definite improvement. The general health is good and B.I. has fallen from 3.2 to 2.

Biopsies

Two skin biopsies were taken (*a*) one was taken from the forearm and (*b*) was taken from the leg. Each piece of skin included an ulcerated nodule. To make doubly sure about this diagnosis these were sent to two different experts. The following are the reports.

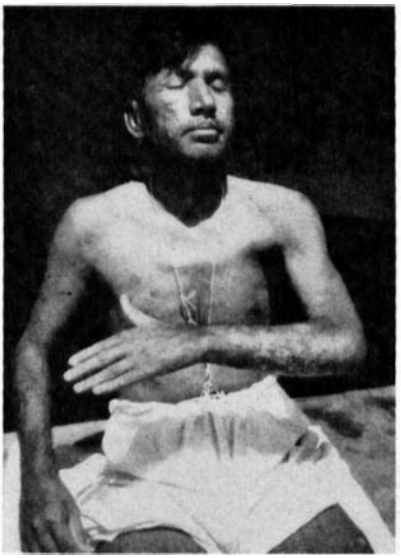

FIG. 1. Front view of the patient showing some loss of the outer aspects of the eyebrows, some subcutaneous modules on the face and some ulcer scars on the left fore-arm.

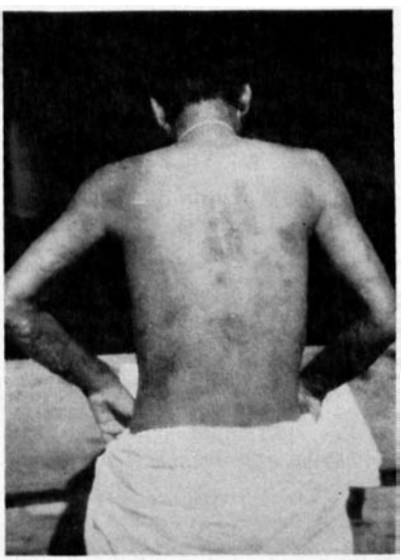

FIG. 2. Showing comparatively smaller number of modules on the back and more on the fore-arms. Earlobes are thickened but are not showing modulations.

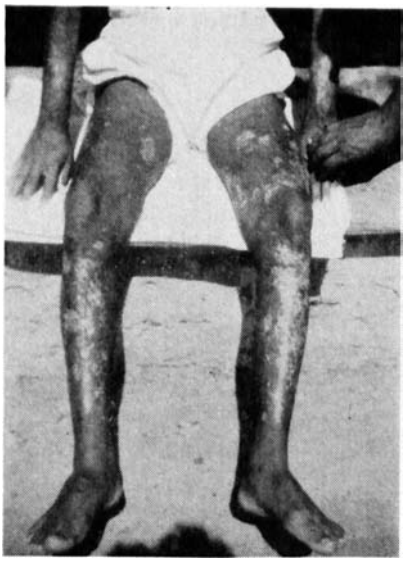

FIG. 3. Whitesh ulcer scars very well shown on the anterior aspect of the legs.

(*a*) Report by Dr. C. K. Job, M.D., Schieffelin Leprosy Research Sanatorium, Karigiri, South India.

'*Macroscopic:* A large piece of skin with a nodule in the middle. The nodule measures 1 cm. across.

'*Microscopic:* This is an interesting case. Section shows epithelium showing pseudocpitheliomatous hyperplasis and a large keratin plug. There is a vessel in the corium which is completely obliterated. Acid fast stain shows a large number of lepra bacilli inside the macrophages.

'I had a similar case two years ago. This is a queer type of exacerbation of leprosy.

'*Impression:* Lepromatous nodule with Keratin Plug.'

(*b*) Report by Dr. George L. Fite, M.D., Chief Laboratory Branch, U.S. Public Health Service Hospital, Carville, Louisiana, U.S.A.

'The lesion is of course lepromatous in nature. It extends throughout the dermis and into the sub-cutaneous area and histologically is typically lepromatous with large number of small microcolonies. In some areas specially at the margins, there are good many plasma cells, which we are inclined to associate with the reactive states.

'In the central part of the lesion the epidermis is hyperkeratotic and there are masses of cells touching the epidermis containing very large numbers of bacilli. This appearance is that of lepromatous granulation tissue forming at the margins of healing lepromatous ulcers which have formed during a reactive episode. There has

probably been some sloughing in this area and healing which is now essentially complete.

'It is my impression that this differs from the Lucio lesion, in which the necrosis stems from sort of vascularitis occurring in the lesion. It is my impression that more than one type of vascular process may occur in the Lucio phenomenon, either (1) Capillary thrombophlebitis, or (2) some allergic type of change with purpuric lesions or with additional endarteritis of small arterioles. Infection of the endothelium of the involved vessel is not essential to the Lucio lesion. The lesion in your case does not show the vascular lesion. Most (if not all) of the Lucio lesions are seen in infiltrative lesions in which the infilbrations are relatively thin and slender although numerous and containing numerous bacilli. In other words they occur dominantly in a somewhat different type of lepromatous leprosy in which there is no coalescence of the infiltrates to form nodules. The necrosis is essentially *infarction* of the skin areas due to loss of blood supply.

'We have seen the Lucio lesion only rarely other than of Mexican origin (from Sinaloa or Jalisco provinces), but they are very rare. However one patient now in the hospital (never been in Mexico, and of Caucasian parentage) shows the typical *lepra bonita* infiltration form characteristically seen in Mexico.

'Thank you again for sending the lesion, and for the opportunity to study this interesting case.'

Discussion

This is a very interesting case because though clinically it resembled the Lucio form described in the literature yet on histopathological examination this was not confirmed.

An interesting thing noticed was the keratin plug found in the centre of the lesion. Its presence was not readily explained. But when we looked again at the history of the exacerbations of the symptoms with the recurrence of gonorrhoeal urethritis, it was presumed that chronic gonorrhoea had some part to play in this. In Sutton's, Diseases of the Skin (Mosbey, 11th Edition, 1956) pp. 301–304, it is shown that gonorrhoeal keratosis blenorrhagica can present similar skin lesions. So it is presumed that in this particular case the lepra reactions were modified by gonorrhoea.

The second point of interest was the occurrence of this disease in a Hindu Brahmin in which this disease is not very common. Yet exposure to V.D. and to a patient in the form of a friend who was a leprosy patient might have given rise to the disease in this patient.

The third point of interest was the close resemblance of this form with that of Lucio phenomenon described by LATAPÍ and ZAMORA in 1948. Since then it has been thought that this form is confined to Mexico only. But it has been clearly brought out by the report from

Carville that other than Mexican can also have this form of leprosy, and we should look for it.

Summary

1. A case of a queer and interesting type of lepra reaction in an Indian patient is described.

2. It is pointed out that the modification of skin lesion may be due to the coincidence of chronic gonorrhoeal infection.

3. It is presumed that the Lucio form may be found in races other than Mexican.

Acknowledgments

I wish to convey my sincere thanks to Dr. V. P. Das, Dr. P. J. Chandy and Dr. R. H. Thangaraj of Mission to Lepers who stimulated my interest in this case and gave encouragement and especially to Dr. C. K. Job of Karigiri and Dr. G. L. Fite of Carville for kindly examining the biopsy specimens and sending the path. reports.

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BALI LEPROSY CAMPAIGN

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The leprosy campaign in Bali has been in progress for five years and the year 1961 was the fifth year. The campaign has been based on the use of the sulphones and has shown ever increasing evidence of the value of these drugs. It has been found that in North Bali during the past two years there has been a great increase in the number of patients coming forward voluntarily, but in South Bali fewer patients are now coming forward and there is no real evidence that patients are hiding, so that it may be taken that the campaign has had a very definite effect on the incidence of the disease. In South Bali there has been a good worker who gained the confidence of the people during the last 20 years, resulting in full co-operation from the people. In 1961, a survey was made in South Bali of 97,198 people, which is 74·5% of the total population at risk, and only 33 additional leprosy patients were found and there was no child case under 15 years of age. This contrasts with the approximate figure of over 200 new patients in North Bali.

For the whole island of Bali the known number of patients in September 1956 was 1,289. By December 1961, 2,016 new cases were added and the total number treated is therefore 3,305. Arrest of the disease was secured in 750 patients.

Treatment of Trophic Ulcers: This matter was taken more actively in 1961. It consisted in a widespread use of applications of gentian violet and brilliant green, and the results were good. There are no funds available for the provision of special shoes.

Deformities: There is an increasing tendency for patients to present themselves before severe deformities. Thus in 1956 29% of patients at one centre presented with minor deformities and 28% with severe deformities, the figures in 1961 were 22% minor and 2% severe.

Results of DDS in Bali: It was found to have a genuine effect, and under DDS the progress to healing was troubled by reactions in only 10%. It was found that some degree of hospital treatment was needed for reactions. As regards the duration of the treatment with DDS, it was found that even after 5 years of regular DDS treatment an estimated 5 to 7% of lepromatous patients remained bacterially positive and this was accompanied by clinical improvement. To secure bacterial inactivity Ciba-1906 and Etisul should be tried, but so far it has not been possible to do this on a sufficient scale.

Drug Resistance to DDS: Over the period of time mentioned in Bali, there has been no evidence of this.

The Effect of DDS in Tuberculoid Leprosy: The eradication of

leprosy bacilli by DDS in a tuberculoid case is undoubtedly too slow, with consequent danger in nerve reactions and other clinical incidents which lead to ultimate deformity.

Hospital Treatment: There is a treatment ward at Balun Hospital which has proved of great value and there cannot be any organised mass treatment of leprosy without such provision for leprosy patients suffering from recurrent illnesses, leprosy reactions, and surgical conditions such as ulcers and deformities. During 1961 there were 91 such admissions of whom 30 suffered from ulcers and other wounds. As was often found, reactions in some patients were very troublesome and inclined to be continuous.

Notes on other drugs used: Ciba-1906 proved invaluable as an alternative to DDS in reaction cases, both as a new basic drug, and as an accompanying drug once the reactional period seemed to be controlled. It was not so convenient in reacting lepromatous outpatients. Supplies of Ciba-1906 are still insufficient. This also applies to the other drug 'Etisul'. This drug does cause trouble in Bali because of its offensive odour. However, it has proved most useful for certain inpatients. In the latest series of reacting lepromatous patients using Etisul for three months in conjunction with Ciba-1906 and other drugs, at least half have shown marked remission of reaction, and clinically a marked clearing of the skin lesions. The remaining half of the patients showed either intolerance of the drug or had no remission of their reactions.

Desensitization of intolerant cases by the injection of 50% *aqueous solution of Sulphetrone:* This remains the accepted method for these cases in Bali, using a slow work up of dosage from 0·1 ml. to 1·0 ml. over 12 months. The results approached a 100% successful return of patients to DDS treatment.

Other drugs used for reaction: These were antimony compounds (e.g. Repodrol), chloroquine, sodium bicarbonate and calcium lactate.

The Outpatient Programme: Treatment is now good at over 135 different outpatient centres throughout Bali, mostly at intervals of two weeks. Motor transport and spare parts for the same is at a minimum. Most patients do not have to walk more than 3 to 5 km. Outpatients maintain regularity of attendance at 90%. There still remains a great deal of fear and prejudice, and attention should be given always to this, but it has been found in Bali that an effective leprosy campaign is a good argument with the people against this fear and prejudice, and is also of great value in preventing a recurrence of new cases in the future. A great deal of education and propaganda is still needed as a part of every leprosy campaign, and this is a matter of the number of personnel available. Many leprosy patients are still careless about so living that they are not infectious

to children. In Bali protective or preventive dosage with DDS is given to such children in selected cases.

Acknowledgment

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ABSTRACTS

Predvatelnie Itogi Lechenya Bolynikh Leproi, Preparation CIBA-1906 (DPT) (*First Results of Treatment of Leprosy with the Preparation Ciba*-1906 (DPT), N. N. TORSUYEVA, Sbornik Nauchnikh Rabot Po Leprologii i Dermatologii, No. 16, 1962, Rostov-on-Don, pp. 40–44.

The author administered this drug to 49 patients of ages 21 to 90 years, with different forms of leprosy. Treatment lasted 2 years, and the drug was found to be effective, because after 5 months of treatment there was a marked clinical improvement in every case. Tolerance was good: there were no complications nor exacerbation of neuritis. Ciba-1906 is a very important anti-leprosy drug, for it is endowed with activity equal to that of the sulphones.

Sostoyaniye i Perspektivi Borbi c Leproi h Rostovskoi Oblasti (*The Progress and the Outlook of the Antileprosy Campaign in the Rostov-on-Don Region*). P. S. GREBENNIKOV and K. K. KHARABADJAKHOV. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don, pp. 3–8

There are 298 leprosy patients registered in the region. Of these 55.3% are treated in the dispensary and live at home, and 107 patients have already completed their treatment. Yearly an average of 9 new leprosy patients are registered and 11 cured cases leave the leprosarium. In the past 14 years the number of new leprosy cases has diminished considerably, and the percentage of lepromatous has reduced from 68 to 50% and the percentage of leprosy patients with initial manifestations has risen from 67 to 77%.

The Infectivity and Mode of Spread of Leprosy. M. F. R. WATERS. The Med. J. of Malaya, **16,** No. 4, June 1962, pp. 251–259. 21 refs.

The author has studied the former opinions of many authors and draws the following conclusions. Until a satisfactory serological test for leprosy is devised, the study of the epidemiology of leprosy will remain difficult and controversial. However, it seems clear that leprosy patients comprise the only known source of infection, and the chief risk lies in smear-positive cases, though the tuberculoid case is not free from risk. Direct contact with a leprosy patient is probably the chief method of transmission, and the more prolonged and intimate the contact, the greater is the risk of infection. It may not be that prolonged intimate contact is necessary in every case. Individual susceptibility to the disease varies greatly. There is a high incidence of childhood leprosy in many highly endemic areas. The infectiousness of patients is rapidly reduced by the specific treatment. From country to country the epidemiology varies. Careful local surveys with good follow-up are needed. The control of leprosy

depends on careful case-finding surveys, along with persistent treatment of those found, and follow-up of contacts.

ACTH, Cortisone and Prednisone in the Treatment of Lepra Reaction. L. S. GARUS. Collected Scientific Papers on Leprology and Dermatology. No. 16, 1962. Rostov-on-Done, pp. 73–77.

He treated 33 patients with lepra reactions with these corticosteroids during 5 years. Each showed itself very effective, especially against febrile reactions, but relapse could not be prevented. The reaction generally declined to a less severe form, and tended to regress in a fairly short time under the influence of repeated injections of the corticosteroids.

Immunological and Allergic Reactions in the Sulphone Treatment of Leprosy. D. K. KANELE. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don, pp. 78–82.

In a group of 46 lepromatous patients given sulphone treatment for two years and observed thereafter for eight years, positivization of the Mitsuda reaction was only noted in 1 patient, but 5 tuberculoid patients with 2 negatives to lepromin before treatment turned strongly positive under the influence of the sulphones, The change in the Mitsuda reaction was not observed in the lepromatous after treatment for five years to eight years. Lepromatous patients not tuberculous do not react to tuberculin, whereas 50% tuberculous leprosy patients are negative to tuberculin. The leprosy infection alone in absence of the bacillus of Koch is incapable of provoking a para-allergy to tuberculin.

Trial of Local Treatment of Leprosy by Injections of 50% *Sulphetrone in the Nasal Mucosa.* G. I. CHIZHE. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don, pp. 51–53.

The injections of 50% sulphetrone were given into the nasal mocosa once in 5 days for a month in 32 lepromatous patients. Of these 23 were rendered negative after a month of treatment. Six months later, bacilli were found in each one of these patients, but they disappeared when a series of injections were repeated. This method could be used as an adjunct to the general treatment of leprosy.

The Lepra Reaction and Erysipelas. E. P. BUKING. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don, pp. 61–65.

In 386 patients observed by the author over ten years, 125 showed erysipeloid reactions. These manifestations were noted especially in older women, with grave or multiple lesions, often with perforating ulcers and osteomyelitis. In analysing his Table of these cases of lepra reaction the author concludes that these 'pseudo-erysipelas' reactions

are provoked by a secondary infection in weakened and cachectic subjects. In these cases, penicillin injections had to be given.

The Influence of the Lepra Reaction (Paralepromatous Fever) in the Prognosis of Lepromatous Leprosy. E. P. BUKING. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don, pp. 66–71.

The author reviews the literature and analyses the evolution of lepromatous leprosy in 289 patients observed for ten years. He divides them into three groups, (1) without lepra reactions, (2) with mild or short lepra reactions, (3) with severe or long reactions. He shows that in the third group it tends strongly to progressive aggravation, more so than in the patients of the other groups.

Contribution to the Study of Proteic Fractions in the Serum of Leprosy Patients. N. N. TORSUYEVA. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962, Rostov-on-Don, pp. 86–89.

The author has studied the sera of 22 leprosy patients and 6 patients with arteriosclerosis, using a modified paper electrophoresis method. The serum of leprosy patients contains less albumen than of healthy subjects or those suffering from arteriosclerosis. On the other hand the serum globulin level is higher in leprosy and especially in the lepromatous form. The albumin globulin ratio does not go beyond normal values in tuberculoid leprosy and in arteriosclerosis. It is below normal in lepromatous and undifferentiated leprosy.

The Treponeme Immobilisation Test in Leprosy. A. V. FLORINSKY, *et al.* Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don, pp. 90–97.

Simultaneously in 242 leprosy patients the authors carried out the Nelson test and the following serological reactions, Wassermann Test with two nonspecific antigens and a cardiolipin antigen; and Kahn and Sachs-Vitebsky flocculation reactions. The authors conclude from these tests that the Nelson test allows of deducing the existence of serological reactions specific in leprosy patients. Since this test gave positive results in 2·8 % of cases of leprosy not attacked by syphilis this confirms the opinion of certain authors who have also noted positive nonspecific results of the test in leprosy. One cannot consider the treponeme immobilisation test to be absolutely specific. The test reads positive especially in lepromatous patients.

Proteinuria in Patients with Leprosy in Malaya. J. A. MCFADZEAN. Transact. of the Roy. Soc. of Trop. Med. and Hyg., **56**, No. 5, Sept. 1962, pp. 404–406.

The early morning specimens of urine were tested from 99 leprosy patients admitted to a leprosarium in Malaya. There was some degree of proteinuria in 73 %, and in most patients the proteinuria was intermittent. The same patients after residence in the

leprosarium for 3 to 4 months on a first class diet, and also after 6 to 12 months, showed a decrease in the incidence and intensity of the proteinuria. Protein deficiency in the diet before admission may well have been the cause of it.

The Skin Reactions of Leprosy Patients to the Intradermal Inoculation of Mycobacterial Antigens. J. A. MCFADZEAN. Transact. of the Roy. Soc. of Trop. Med. and Hyg., **56**, No. 5, Sept. 1962, pp. 407–410.

The author injected 48 leprosy patients in Malaya intradermally with lepromin, tuberculin, antigens derived from BCG, *M. fortuitum*, *M. rhodocrous*, *M. marinum*, *M. phlei* and *M. Smegmatis* and found no correlation between the reactions to lepromin and those to any other antigens. There was a significant correlation between the reactions to tuberculin at 72 hours and those to *M. marinum* at 72 hours, and when the reactions at 72 hours to tuberculin were compared with the reactions at 21 days to *M. marinum*.

The Serological Reactions of Syphilis in Leprosy. G. V. MERTSLIN and V. K. LOGINOV. Collected Scientific Papers on Leprology and Dermatology, No. 16, 1962. Rostov-on-Don.

With the sera of 202 leprosy patients the authors carried out the Wassermann Reaction, using the fixation of complement according to MERTSLIN and the SACHS-VITEBSKY reaction. They observed positive and weakly positive results in 7 patients out of 17 not treated. In 120 patients previously treated they noted 4 positive reactions and 21 weakly positive. Almost all these reactions, save one, were non-specific. All the evidence showed that they were caused by changes in the lipid and globulin content of the serum. At the present time these pseudo-positive reactions are noted less often than during the pre-sulphone era.

The Appearance of Dead Leprosy Bacillus by Light and Electron Microscopy. R. J. W. REES and R. C. VALENTINE. Internat. J. of Leprosy, **30,** No. 1, 1962, pp. 1–9.

The authors made preparations of leprosy bacilli and stained them by the Ziehl-Neelsen method and examined them first by the light microscope and then with the electron microscope. They found by comparing the same individual bacilli that the material which stains red is in the cytoplasm of the bacilli and not in the cell wall, and that every bacillus which appears irregularly stained under the light microscope is shown by the electron microscope to be completely degenerate and dead. There is a close agreement between the proportion of degenerate forms of human leprosy bacilli as shown by the electron microscope, and irregularly staining bacilli with the light microscope.

Before undertaking these comparative studies the authors studied with Ziehl-Neelsen the staining properties of *M. phlei* and *M.*

lepraemurium and found that suspensions containing very large numbers of cell walls do not stain with carbol-fuchsin, and concluded that the constituent of mycobacteria which binds the carbol-fuchsin resides in the cytoplasm and not in the cell wall.

In their subsequent studies on *M. leprae* and *M. lepraemurium* they found a high correspondence between the electron-dense material inside the cell wall and the carbol-fuchsin-staining moiety. The irregular staining of the degenerate form of the bacillus can only be identified by the light microscope when there is sufficient residual cytoplasm to outline the bacillary shape, but their studies clearly demonstrated for the first time that irregularly-stained forms can be identified exactly with degenerate forms seen with the electron microscope, which are likely to be non-viable. All forms of irregularly-stained bacilli whether defined as 'fragmented' or 'granular' or 'beaded' can be considered dead organisms. The observation of DAVEY, T. F. (1960) is therefore supported and is of great importance in experimental chemotherapy and prognosis.

The Culture and Experimental Transmission of M. leprae in Monkeys. A. MUKHERJI. Preliminary Report presented to Indian Medical Assoc., Lucknow, Dec. 1958 and Indian Science Congress Assoc. Jan. 1958. 4 illustrations, 5 references.

The author cultivated *M. leprae* on a medium containing extract of *M. phlei*. The material consisted of 2 g. of tissue from human lepromatous patients, taken from the ears and macerated, mixed with a quantity of water, and digested with 0·1 g. of papain at 60° C for 18 hours. To this were added the well-beaten contents of two fresh eggs, 6 cc. of Douglas broth, and 0·1 g. of asparagin previously dissolved in a little water. Also a 96-hour culture of *M. phlei* 1 g. was well ground with sea sand and mixed with a little water and digested with 0.1 g. papain for 18 hours at 60° C and Seitz filtered. This filtrate was added to 25 ml. of solution of Lowenstein-Jensen medium and well-beaten contents of four eggs. Lepromatous tissue was inoculated in these media and showed signs of bacterial growth about 7–10 days and in some cases not until 4 or 5 weeks. Also of 12 monkeys about 1 g. of lepromatous tissue was inoculated in suspension in 5 intraneurally along the ulnar nerve. Lesions developed in the nerve and the skin of the face, clawing of the hand on the injected side, and the presence of acid-fast bacteria thought to be *M. leprae*. In one monkey both hands and both feet were grossly clawed. The author considers that true leprosy infection was transmitted to the monkeys. Control monkeys who had received only inoculations of *M. phlei* along the ulnar nerves showed nothing abnormal.

The Epidemiology of Leprosy: Present Status and Problems. J. A. DOULL. Internat. J. of Leprosy, **30,** 1, 1962, pp. 48–66 (55 refs.).

The author describes the early epidemiological opinions of HIRSCH 80 years ago and says our knowledge has extended greatly

since then. There is more and more convincing evidence of the contagiousness of leprosy. The evidence is strongly indicative of *M. leprae* being the etiological agent, even though direct proof is still lacking. Much has been learned of the clinical varieties and their relative importance, and the lepromin test is proving a very helpful indication of the individual resistance against the disease. From field studies come an emphasis on the importance of household associations in sustaining the infection, and the importance of sex and age. Much further study is required, but we now have a good working hypothesis.

A New Concept of the Pathogenesis of Leprosy. P. GHOSAL. Indian J. of Dermat. **7**, 2, pp. 1–19.

The author is mainly concerned with the host reaction to leprosy after the entrance of the bacilli through the skin, and resistance by the histiocytes, and describes an allergic reaction against the antigenic protein, an immunity reaction against the lipoid, and a lepromatous reaction against the active bacilli. He describes the meaning of these.

Leprosy Research Programme of WHO is given in The Medical Research Programme of WHO 1958–1961. Report by the Director General, Geneva, 1961. pp. 26–27.

1. *Introduction.* The Leprosy Unit was set up in November 1958. A Scientific Group on Leprosy Research was convened in February 1959 which reviewed the gaps in our knowledge and recommended a wide programme of research. The recommendations of the Study Group were reviewed in the first session of the ACMR. Several projects aimed at obtaining the transmission of human leprosy to laboratory animals and at the growth of *M. leprae* in culture media, have been initiated and some trials of leprosy drugs as well as studies of leprosy prevention, and epidemiological investigations, are now in progress.

2. *Research on the Microbiology of Leprosy* has been given first priority because the transmission of *M. leprae* to laboratory animals and the cultivation of this micro-organism would enable many problems to be solved, and would have considerable impact on leprosy research and leprosy control.

To this end, WHO has:

(i) organized the regular supply of iced biopsies of human leprosy to laboratories interested in the cultivation and transmission of human leprosy to laboratory animals;

(ii) supported research on the transmission of human *M. leprae* to different rodents;

(iii) supported cytological research on the standardization of lepromin and on the serology of leprosy.

3. *Trials of New Leprosy Drugs*, with control groups of patients treated with DDS, have been planned; the collaboration of four centres has been obtained to undertake these trials, on a uniform

basis; statistical analysis has been started on work previously carried out in chemoprophylaxis of leprosy.

The results of the drug trials will provide WHO with definite criteria for the efficacy and tolerance of the many new anti-leprosy drugs and may lead to certain changes in the conduct of mass campaigns against leprosy.

The WHO Leprosy Advisory Team assessing leprosy mass campaigns in progress, confirmed the efficacy of DDS; after one year's treatment, many of the regularly treated lepromatous cases showed definite improvement, clinically and bacteriologically, and after five years' treatment more than 80 per cent of the lepromatous patients were bacteriologically negative. The remaining 20 per cent of bacteriologically positive cases were generally intolerant or reluctant to have regular treatment.

4. *The Epidemiology of Leprosy* has been investigated. Since 1959, WHO has established a Leprosy Advisory Team to assess different leprosy control campaigns in progress and, at the same time, to carry out epidemiological investigations by examination of the whole population by means of the random sampling method. The team has collected reliable information on the prevalence of leprosy, lepromatous rate, prevalence of different types of the disease and its distribution by race, sex and age. It has also collected for the first time information on the frequency and degree of disabilities, also classified by race, sex, age and clinical forms of the disease.

5. *The Problem of Leprosy Disabilities, their Prevention and Treatment*, has also received attention and a Scientific Meeting on Rehabilitation in Leprosy took place in Vellore in November 1960.

As a self-supporting project, WHO obtained the collaboration of a Swiss private organization in the implementation of a pilot project in the Republic of Cameroun, to ascertain the possibility of linking rehabilitation campaigns with leprosy mass campaigns.

LETTER TO THE EDITOR

MINISTRY OF HEALTH,
RURAL HEALTH DIVISION,
ONITSHA LEPROSY CONTROL AREA,
OJI RIVER, VIA ENUGU.

Dear Dr. Ross-Innes,

The Harris Footprint Mat which I mentioned in the article on Footwear in the July issue of *Leprosy Review* is, I understand from Dr. Harris' secretary, to be manufactured in Canada. The address of the manufacturers is:

DOWN BROTHERS LTD.
Surgical Instruments Supplies,
70 Grenville Street,
Toronto, Canada.

Yours sincerely,

W. F. ROSS,
Area Superintendent.

REPORTS

The Leonard Wood Memorial is reported by DR. J. A. DOULL in *Leprosy Briefs* of 1962, for the year 1961:

This is of great interest and encouragement, because it provides details of the widespread beneficial activities of the Memorial, and a list is given of publications by the staff. Perhaps the most outstanding event of the year was the Symposium on Leprosy Research held at Johns Hopkins University. The Report should be studied in detail.

13th Meeting Directing Council PAHO and *Regional Committee WHO.*

The 13th Meeting of the Directing Council of the Pan American Health Organization, 13th Meeting of the Regional Committee of the World Health Organization, was held in Washington, D.C., 3rd to 13th October 1961. The International Leprosy Association was represented by Dr. J. A. Doull, Medical Director, Leonard Wood Memorial.

Dr. Luther L. Terry, Representative of the U.S.A., presided. Drs. Jose Alvarez Amezquita, Mexico, and Doroteo Castillo Rodriquez, Nicaragua, were vice chairmen. All the Latin American republics except Colombia, the United States of America, the United Kingdom and the Kingdom of the Netherlands were represented. Canada sent an official observer. The meeting was also attended by representatives of the World Health Organization and by observers from the Organization of American States, United Nations, United Nations Children's Fund Food and Agriculture Organization, Inter-American Development Bank, International Committee of Military Medicine and Pharmacy and 20 non-governmental organizations.

Leprosy: The report of the Director of the Pan American Sanitary Bureau to the Meeting for the year 1960 which was adopted contains the following paragraph on leprosy:

'The Organization gave special importance to the problem of leprosy. In 1960 consultants were appointed for eight countries in the Americas, and the information from earlier surveys in Bolivia, Ecuador and Peru was brought up to date. UNICEF gave valuable help to some of these programmes. Wherever the programmes were beginning their operations, the number of cases diagnosed – including tuberculoid and lepromatous forms as well as indeterminate manifestations of the disease – was on the rise. That phenomenon was customary in any epidemiological investigation. Agreements were signed with Argentina and Brazil for the extension of their control programmes with the co-operation of UNICEF. Naturally, the currently accepted doctrine had been applied, in which the patients were given ambulatory treatment and kept within their own

social environment, rather than segregated behind the locked doors of the leprosarium.'

The following is included in the comments of the Executive Committee on the Programme and Budget for 1962.:

'*Leprosy* – It was noted that the work of the consultant in Zone III had to be continued, although originally planned for termination in 1961. The Director reported that in a recent meeting of ministers of public health in Tegucigalpa, Honduras, an analysis of the problem showed that as a result of the work of the consultant and the presentation of short courses for the training of professionals in each country, the number of cases discovered in some countries had doubled or tripled, which confirmed the need for continuing the work of the consultant. It was also reported that UNICEF had indicated its readiness to co-operate through the provision of equipment and drugs for leprosy campaigns. In Zone IV it was noted that provision was made for a Zone consultant in leprosy to work in all countries rather than for a consultant for each country as originally planned.'

In Annex II of the final report of the Meeting, which outlines the general programme for the years 1962–65, there is the following paragraph:

'During this period the Organization must devote special attention to research and co-operation with the interested Governments in the execution of pilot projects designed to control onchocerciasis, Chagas' disease, schistosomiasis, hydatidosis, leprosy, and other communicable diseases that can be controlled with a modest per capita investment.'

During the period 1962–65 the Organization will extend its programme of stimulating, co-ordinating, promoting and where appropriate, supporting medical research. Although not so designated, it was learned that leprosy is included among the communicable diseases considered suitable for research by the Organization.

Budget: The budget for the year 1962 was fixed at $5,240,000.

REVIEW

Leprosy, by J. A. DOULL. Tice's Practice of Medicine **4,** 1962, 57 presents a valuable review of modern knowledge and modern thought on leprosy. There are 10 illustrations and 39 references.

The author defines leprosy sensibly as a 'chronic infectious disease of man caused by the leprosy bacillus *Mycobacterium leprae*, affecting chiefly the skin, mucous membranes of the upper respiratory tract, and certain peripheral nerves'. He goes on to give a short but accurate account of what is known of the past history of the disease in the world, and interesting data on its modern prevalence. He then discusses in a balanced manner the etiology, pathology, classification, clinical features, diagnosis and differential diagnosis, treatment by older forms of therapy and the newer forms, which now include prevention of complications and of sequelae by physiotherapy and reconstructive and plastic surgery, and the prevention and treatment of lepra reactions. Prognosis and control are also discussed. In the comparatively short space of 20 pages this article would lay the foundation for any serious reader for an understanding of modern leprosy.

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The letters after the entry have the following significance: Original Articles (O); Editorials (E); Letters to the Editor (L).