LEPROSY REVIEW

Volume 52, Number 2, June 1981

Published Quarterly for the
British Leprosy Relief Association

ISSN 0305-7518

Lepr Rev (1981) **52**, 111–119

Editorial

THE EYE AND LEPROSY

All those who study or work with leprosy are aware of the major problems that ocular complications present in the long-term management of the disease. Over a hundred years ago Bull and Hansen drew attention to the very high incidence of ocular involvement, commenting that 'there is no disease which so frequently gives rise to disorders of the eye, as leprosy does',[1] and the disturbing consequences of these complications were re-emphasized by Margaret Brand in her pamphlet on the care of the eye: 'Blindness in the individual who has normal skin sensitivity is enough of a handicap, but in one who has lost that faculty it is disastrous. Few have the resources, material, mental or spiritual, to live with it'.[2]

Despite this long awareness of the large variety of ocular changes that can occur in the disease there is still considerable ignorance of the underlying pathological processes and their consequences, and this ignorance stems mainly from two causes. First it derives from poor coordination of research into the clinical and pathological changes that affect the eye in all stages of leprosy, and secondly it relates more particularly to the lack, until recently, of an experimental animal that could be used to study the evolution and pathogenesis of the disease in the ocular tissues.

The problems of communication and coordinated research are mainly logistic and will always remain. The leprologist, working often in difficult circumstances with limited facilities and equipment, may not have the opportunity and ophthalmic experience to make detailed assessments of ocular disease and may not have the time to carry out the important longitudinal studies that are required to understand the natural history of the ocular changes. Evidence from the world literature on ocular complications of leprosy over the last 40 years shows how fragmented the knowledge of this important subject really is. There are many survey reports on groups of patients in scattered parts of the world, but little in the way of long-term follow-up studies which are so essential for the understanding of the disease process. Where these examinations have been carried out by ophthalmologists with specialized knowledge and sophisticated techniques and equipment, the information has proved more valuable but, with few exceptions, such colony surveys

0305–7518/81/052111 + 09 $01.00

and case reports serve merely to build up a general picture of the immense problems of ocular damage throughout the world and provide only isolated glimpses of the pathology of a condition that has to be considered equally in terms of its evolution and duration.

The development of an experimental animal model ought to prove a turning point in the understanding of ocular pathology in leprosy, and evidence from animal and clinical studies may make it necessary to rethink some of the traditional ideas of the disease process and its therapy – particularly in lepromatous leprosy. The purpose of this paper is to draw together some of the clinical and experimental data that is now emerging in order to indicate lines of clinical and pathological research that could be followed, with the intention not only of solving some of the many unanswered questions about ocular complications and methods of preventive therapy, but also to remind leprologists that they have in the eye an organ that lends itself above all others to the clinical, pharmacological and pathological study of the neural involvement of the disease.

Some of the statements to be made will be hypothetical, some will be provocative, but all are intended to stimulate renewed interest into the effect of leprosy on the eye – an interest that has, with a few exceptions, remained sadly fallow over the past decade.

Incidence of blindness in leprosy

It is possible that there are between 500,000 and 750,000 patients in the world who are blind due to leprosy, but estimates are difficult because the information on which they are based is so variable and erratic. These estimates rely heavily on isolated reports from scattered leprosy communities, with differing ethnic populations, taken at different times, so that the extrapolated global figures are almost meaningless, except that they reveal the magnitude of the ocular problems. Certain general conclusions can be drawn from a closer analysis of the available data from these reports – that the ocular complications have a much higher incidence in the lepromatous form of leprosy, and therefore certain races, particularly Asiatics, are more susceptible to ocular disorders, and that ocular involvement is more prevalent in leprosy in temperate climates; thus the major ocular complications are to be expected in the more northerly parts of the Far East rather than in Africa. In the latter continent leprosy may just be one of a variety of endemic conditions that can cause blindness – these include trachoma and onchocerciasis – and the shorter life expectancy of the African patient means that many succumb before the inevitable late ocular complications have had time to develop. In addition most of the reports from leprologists working in the field stem from Africa where the ocular complications are relatively lower, and this therefore tends to understate the global significance of eye disease.

Ocular complications

It is not the purpose of this article to classify all the ocular complications of leprosy, many of which develop as manifestations of facial nerve paralysis and secondary infection causing a variety of degenerative corneal conditions. Primary involvement of the eye is demonstrated in Table 1 in a simplified form with the clear distinction being made between tuberculoid and lepromatous leprosy. Borderline leprosy has variable features depending on the stage and state of the patient's immunity.

Table 1

Tuberculoid	Lepromatous
V neuropathy	V neuropathy
VII neuropathy	VII neuropathy
	Episclera – nodules
	Cornea – 'beading' → keratitis
	– leproma
Iris – acute iritis	Iris – acute iritis
	– iris pearls → *chronic iritis*
	– iris leproma
	Ciliary body – ? phthisis
	Lens – ? cataract
	Choroid – ? peripheral lesions

The importance of paralysis of the facial nerve cannot be overstressed as a major cause of blindness. It occurs in all forms of leprosy and may result in a chronic exposure keratitis and subsequent corneal scarring and degeneration. The role of trigeminal neuropathy is less established; certainly corneal sensation is often badly affected in leprosy with an accompanying diminution in the normal protective responses, but several studies have shown that complete corneal anaesthesia is rare,[1–6] and it is probable that the effect of Vth nerve paralysis on metabolism and nutrition of the cornea is equally important.

All forms of leprosy may give rise to an acute iritis which, if left untreated, may lead to a profound and permanent loss of vision. In lepromatous leprosy the inflammation may be spontaneous and is believed to represent a reaction to the deposition of circulating immune complexes within the eye.[7–8] This reaction, also known as erythema nodosum leprosum, involves many tissues including the skin, eyes, peripheral nerves and sometimes the kidneys.[9] The iritis does not differ in its clinical presentation from any other form of acute iritis and requires intensive therapy with local mydriatics and steroids.

The blinding conditions produced by facial nerve palsies and by acute iritis are common to all forms of leprosy and are potentially preventable. Training of leprologists and paramedical workers in the management of these

two conditions is already being undertaken in several centres in the world, notably Carville in the United States, and timely tarsorrhaphies or even more extensive plastic surgery, together with encouragement of patients to report for treatment if an acute painful red eye develops, are already contributing significantly towards preventing the late corneal and intra ocular complications of these disorders.

Primary corneal involvement in lepromatous leprosy takes the form of infiltration and later destruction of corneal nerves followed by a chronic superficial stromal keratitis to which may be added pannus formation or corneal degeneration. These corneal changes are usually not serious for the vision unless corneal deposits are substantial,[2,10] and may take many years to evolve. A true leproma of the cornea or iris may occur but is rare. It is the so called *chronic iritis* of lepromatous leprosy that is responsible for the major visual impairment in the disease, a condition aptly described by Weekeroon[11] as 'the cause par excellence of blindness', and considered by most authors to be the prime ocular complication of leprosy, and the remaining part of this article will be directed towards an examination of existing knowledge of the pathogenesis of this interesting condition and its significance in the disease process.

Chronic iritis

The condition was described by Bull and Hansen[1] as 'iritis without violent symptoms with exudations around the borders of the pupils and adhesions to the capsule of the lens in patients who have not complained of pain or derangement of sight'. The iritis has many features that sets it apart from other forms of chronic inflammatory disease and it shows several characteristics of a basically degenerative process of slow evolution. An interpretation of the clinical signs, and of the few pharmacological and pathological studies that have been carried out, strongly suggests that the chronic iritis of lepromatous leprosy is *neuroparalytic* in origin, and the evidence for this and its diagnostic and therapeutic significance will be discussed.

Clinically the condition develops quietly a few years after the onset of the disease. It causes no symptoms and very few signs initially, the eye is quiet, without discomfort and with little overt evidence of inflammation. The iritis is usually bilateral and iris pearls are frequently seen in the early stages as a transient phenomenon. Many authors have reported that conventional mydriatics have little effect on pupil dilatation despite the fact that synechiae are uncommon,[12–13] although these may form if an acute iritis supervenes. The changes in the anterior chamber are described as showing a faint flare and fine atypical keratic precipitates often pigmented and the condition may drag on for years, and does not respond to local steroid therapy. Eventually the

iris shows progressive signs of atrophy and disintegration accompanied by increasing miosis, and it is this miosis associated with corneal or lenticular changes that is responsible for the severe visual impairment that inevitably ensues. The advanced iris atrophy has been noted for long by surgeons who have attempted to undertake cataract operations or optical iridectomies in these patients and have found the tissue extremely friable.[14–18]

This clinical pattern does not suggest a typical inflammatory disease, and some other underlying pathological mechanisms must be sought. The preference of the leprosy bacillus for areas of low body temperature has been noted by clinicians for many years,[19] and the involvement of superficial nerves in the generalized bacteraemia that occurs in lepromatous leprosy is responsible for the major neurological complications through its destruction of fine nerve endings in the cooler parts of the body. Temperature measurements in animals have demonstrated that the anterior segment of the eye is three degrees cooler than the general body temperature,[20] and this suggests that the nerves in the cornea and iris are some of the coldest nerves in the body. In the experimental animal bacilli were found to be concentrated at the front of the eye,[8] and clinical evidence reveals that it is rare for the posterior segment of the eye to be involved in the disease. Of additional interest is the fact that anterior segment temperatures are related to environmental conditions,[21] and this might explain the higher incidence of ocular complications in temperate areas.

The assumption can certainly be made that iris and corneal nerves are susceptible to attack from leprosy bacilli during the bacteraemia that occurs in lepromatous leprosy and there is ample evidence to support this supposition.

The sequence following corneal nerve involvement is well-established clinically and histologically. Leprous infiltration of the corneal nerves produces the typical transient beading followed by disintegration that leads to stromal disease and is associated with irregular loss of corneal sensation. Histology shows round cell infiltration of the Schwann cells with bacilli contained within characteristic foam cells. A parallel clinical situation seems to exist in the iris with the early transient appearance of iris pearls which are pathognomonic for lepromatous leprosy followed by progressive iris atrophy, and the resemblance to corneal beading has already been commented on by Choyce.[16] Histology of these iris pearls also shows tightly-packed bacilli inside mononuclear cells with no accompanying inflammation or foreign body reaction.[22] The distribution of the iris pearls conforms to the anatomical distribution of the autonomic nerves plexuses in the iris, these nerves contain small non-myelinated sympathetic and parasympathetic fibres supplying the sphincter and dilator muscles of the pupil – nerves that by their position and size would be expected to be particularly susceptible to leprous infiltration. The evidence suggests that iris pearls are the visible manifestations of the breakdown of the iris nerves (cf. corneal beading) and that the autonomic nerves are primarily involved.

Further support for this hypothesis can be obtained from clinical and pharmacological observations. The non-reacting pupil of lepromatous leprosy has already been noted, paralysis of the autonomic nerves would certainly produce no dilatation of the pupil after Atropine or its derivatives, and it would require a direct sympathomimetic drug such as Phenylephrine to achieve this, provided the dilator muscle was functioning. An intriguing study by Swift and Bauschard[23] on patients with early lepromatous leprosy showed that the pupils of a significant proportion (55%) of cases dilated with 1% L-epinephrine whereas only 5% of normal controls showed a similar dilatation. The results were interpreted as a manifestation of denervation hypersensitivity and suggested that early iris involvement was more common than previously reported. The authors concluded that the leprous eye was a model of peripheral post-ganglionic denervation and further studies were recommended. Slem, reporting his studies in Turkey,[24] also concluded that the progressive iris atrophy of leprosy was likely to be neurotrophic in aetiology.

Deprivation of the autonomic and therefore the motor supply of the iris muscles would be expected to be followed by progressive tissue atrophy such as occurs elsewhere in the body. Disintegration of muscle fibres over a long period of time sets up local inflammatory changes consisting mainly of round cell infiltration and these have been observed in iris tissue examined pathologically,[13, 22, 25] and the chronic low-grade flare and pigmented cells noted by slit-lamp examination of the anterior chamber could well be manifestations of this destructive process and account for the lack of clinical signs and symptoms and the failure to respond to local steroids. Hashizuma and Shionuma examined the iris in lepromatous leprosy by electron microscopy and found lepra cells infiltrating the stroma and non-myelinated nerves although they considered the primary disturbance to be in the muscles, but no further specific electron microscopical examinations of the iris nerves have been reported. The later stages of this evolutionary process are characterized by increasing iris atrophy and miosis. Studies on the histology of the sphincter and dilator muscles in lepromatous leprosy has shown a differential atrophy with the dilator more affected than the sphincter and in 15 specimens recently examined, 11 showed signs either of degeneration or complete dilator atrophy whereas in only 7 out of 15 was the sphincter muscle degenerate.[13] This differential atrophy could be explained by a difference in the innervation and morphology of the two muscles with the dilator more thinly spread throughout the iris, and its consequent atrophy gives rise to the increased friability of the tissue and the persistent and troublesome miosis.

The question of the relationship between leprosy and cataract remains unanswered. No figures are available for the overall incidence of cataract in the disease as the condition is common in most parts of the world where leprosy is endemic. Many authors consider there to be no true leprosy cataract and that the lens changes that are observed are part of the changes that are

seen in a normal ageing population. Certainly there is a higher incidence of cataract after acute iritis and it may be a major complication in untreated patients, but its relationship to the chronic iritis of lepromatous leprosy has not yet been satisfactorily established. A study by Prabhakaran[26] considered the affinity that *Mycobacterium leprae* is known to have for DOPA, and it is of interest that this substance is found in large quantities in iris tissue. Some of the breakdown products of DOPA metabolism, notably the quinones, are known to be cataractogenic in animals, and a current study by Clayton *et al.*[27] suggests that the biochemical profiles of cataracts in leprosy differ from those of other senile lens opacities.

Conclusion

Cochrane[28] described leprosy as 'the most thrilling and exciting adventure on which any medical man can embark' and certainly the ocular problems posed by the so-called chronic iritis of lepromatous leprosy will provide the leprologist and the ophthalmologist with a fascinating and rewarding exercise in medical detection. If this condition is truly a neuroparalytic iritis then it is one of the few situations in which this occurs clinically – it may be present in Herpes zoster ophthalmicus and in heterochromic cyclitis – and the diagnostic and therapeutic repercussions are far-reaching. The use of pharmacological pupil tests in the early stages of ocular involvement will need to be evaluated, but more important is the possible role of pupil testing in the detection of the systemic neurological manifestations of the disease, to augment and perhaps even replace some of the existing clinical and laboratory tests. The therapeutic implications of contemporary ophthalmic management will also need to be reassessed. For example, whether the conventional use of steroid and mydriatic drops has any value in the early stages of chronic iritis, since the use of a paralysing agent such as Atropine in an already paralytic situation has little to commend it on pharmacological grounds. On the other hand it might be possible to preserve the function of a failing dilator muscle by the administration of direct sympathetic stimulants in the same way that physiotherapy preserves the function of denervated voluntary muscle. The role of DOPA and its breakdown products in relationship to cataract formation and perhaps chronic iritis also merits further study. All these aspects of the condition require investigation and clinical research on a coordinated and if possible international basis. Long-term longitudinal studies are essential in the understanding of the natural history of the ocular manifestations, and pathological material is needed particularly in the study of the iris nerves which should be examined by both light and electron microscopy. Furthermore, it is hoped that studies on experimental infection in animals will provide some of the answers to the questions posed by clinical leprologists and pharmacologists.

Finally a cautionary note must also be sounded – 'the history of leprosy is strewn with the wrecks of so-called cures',[29] and the literature is full of noble aspirations that have come to nothing. This should not, however, be allowed to impede the combined efforts of experimental and clinical research in the hope of at least advancing some way towards alleviating the intolerable situation that the blind leprosy patient must experience.

Acknowledgements

I would like to express my gratitude to Dr Mia Topple and all the staff of the Wilson Leprosy Centre and Rehabilitation Hospital, Yosu, South Korea for their help and kindness, and to thank LEPRA for advice and encouragement. I am also indebted to Gwen Brookman for secretarial assistance.

T. J. ffytche
St Thomas' Hospital
London

References

[1] Bull OB, Hansen GA. *The Leprous Diseases of the Eye*. Christiana: Albert Cammermeyer, 1873.
[2] Brand Margaret. *The Care of the Eye*. Carville, Louisiana: The Star, 1980.
[3] Soto MC, Zubelzu AB. Corneal sensitivity in leprosy. *Arch Oftal B Aires,* 1963, **38**, 273–81.
[4] Krassai A. Corneal sensitivity in lepromatous leprosy. *Int J Lepr*, 1970, **38**, 422–7.
[5] Weekeroon L. Ocular leprosy in West Malaysia. *Brit J Ophthal*, 1972, **56**, 106–13.
[6] Shields JA, Waring GO, Monte LG. Ocular findings in leprosy. *Amer J Ophthal*, 1974, **77**, 880–90.
[7] Grove DI, Warren KS, Mahmoud AAF. Algorithms in the diagnosis and management of exotic disease. XV. Leprosy. *J Infect Dis*, 1976, **134**, 205–10.
[8] Hobbs HE, Harman DJ, Rees RJW, McDougall AC. Ocular histopathology in animals experimentally infected with *Mycobacterium leprae* and *M. lepraemurium. Brit J Ophthal*, 1978, **62**, 516–24.
[9] Holmes WJ. The eyes in leprosy. *Trans Opthal Soc UK*, 1961, **81**, 397–420.
[10] Allen JH, Byers JL. The pathology of ocular leprosy. I. Cornea. *Arch Ophthal*, 1960, **64**, 216–20.
[11] Weekeroon L. Ocular leprosy in Ceylon. *Brit J Ophthal*, 1969, **53**, 457–65.
[12] Barros M de. A clinical study of leprous iritis. *Int J Lepr*, 1940, **8**, 353–60.
[13] ffytche TJ. The role of iris changes as cause of blindness in lepromatous leprosy. *Brit J Ophthal*, 1981 (in press).
[14] Wood DJ. Ocular leprosy. *Brit J Ophthal*, 1925, **9**, 1–4.
[15] Prendergast JJ. Ocular leprosy in the United States. *Arch Ophthal*, 1940, **23**, 112–37.
[16] Choyce DP. The diagnosis and management of ocular leprosy. *Brit J Ophthal*, 1969, **53**, 217–23.

[17] Sivasubramanian P. Some aspects of ocular leprosy in Ceylon. *Trans Ophthal Soc Ceylon*, 1971, **10**, 13–21.
[18] ffytche TJ. Cataract surgery in leprosy patients. *J Royal Soc Med*, 1979, **72**, 826–30.
[19] Sabin TD. Temperature-linked sensory loss. A unique pattern in leprosy. *Arch Neurol*, 1969, **20**, 257–62.
[20] Schwartz B. Temperature gradients in rabbit eye. *Invest Ophthal*, 1962, **1**, 513–21.
[21] Schwartz B. Environmental temperature and ocular temperature. *Arch Ophthal*, 1965, **74**, 237–43.
[22] Allen JH. The pathology of ocular leprosy. II. Miliary lepromas of the iris. *Amer J Ophthal*, 1966, **61**, 987–92.
[23] Swift TR, Bauschard FD. Pupillary reactions in lepromatous leprosy. *Int J Lepr*, 1972, **40**, 142–8.
[24] Slem G. Clinical studies in ocular leprosy. *Amer J Ophthal*, 1971, **71**, 431–4.
[25] Hashizuma H, Shionuma E. Electron microscopic study of lepromatous changes in the iris. *Int J Lepr*, 1965, **33**, 61–82.
[26] Prabhakaran K. Cataract in leprosy; a biochemical approach. *Lepr Rev*, 1971, **42**, 11–13.
[27] Clayton HM, Cuthbert J, Phillips CI, Bartholomew RS, Stokoe NL, ffytche TJ, Reid J, Duffy J, Seth J, Alexander M. Analysis of individual cataract patients and their lenses. (Unpublished data.)
[28] Cochrane RG. Leprosy in Korea. *Lepr Rev*, 1956, **27**, 1–19.
[29] Muir E. The sulphone treatment of leprosy. *Brit med J*, 1947, **1**, 798–801.

Lepr Rev (1981) **52**, 121–126

Hormonal changes in human leprosy

GH RÉE,* FIR MARTIN,† KATHERINE MYLES† & IVANA PELUSO†

**The University of Papua New Guinea, Box 4820, Papua New Guinea; †Department of Endocrinology & Department of Medicine, University of Melbourne, Royal Melbourne Hospital, Victoria, Australia*

Received for publication 23 June 1980

Summary In an attempt to define the extent of disturbances of testicular–pituitary function in leprosy, a study has been carried out on an unselected group of male patients attending a leprosy treatment centre, who were not obviously suffering from testicular atrophy or gynaecomastia.

Introduction

Involvement of the testis in male patients with leprosy is well documented and may be associated with impotence, sterility and gynaecomastia.[1] Testicular histology shows atrophy of seminiferous tubules, with hypertrophy and clumping of Leydig cells and hyalinization of the small and medium sized vessels.[2] The hormonal functions of the testes have been studied by a number of workers, usually in patients with testicular atrophy and gynaecomastia.[3–5] In these particular patients, androgens are generally diminished and gonadotrophins increased. The pathogenesis of the testicular damage is uncertain, though Wall and Wright[6] found that testicular germinal cell antibodies were present in 75% of lepromatous patients, and postulated that autoimmunity, Erythema–Nodosum–Leprosum immune complex damage and direct invasion by *Mycobacterium leprae* may all be contributory.

It is generally accepted that a classical feedback system operates between the testis and the pituitary gland.[7] The interrelationship between androgen and luteinizing hormone (LH) levels appears to be reasonably straightforward but may operate following conversion of testosterone to oestrogen. The testicular factor which controls follicle-stimulating hormone (FSH) levels is probably a poorly characterized non-oestrogenic, non-adrogenic substance known as

0305–7518/81/052121 + 06 $01.00

inhibin. Some synergism between these two gonadotrophins in both Leydig cell function and spermatogenesis may occur,[8] but in general plasma levels of LH reflect the function of interstitial cells, and of FSH the seminiferous tubules.

Patients and methods

The patients were randomly selected males attending the Port Moresby Treatment Centre. Prepubertal boys and patients with obvious gynaecomastia were excluded from the study, but otherwise no specific clinical examination for testicular function was made. In Port Moresby, but not as a rule elsewhere in Papua New Guinea, leprosy patients are classified on the 5-point scale of Ridley and Jopling.[9] For the purpose of this study, the patients were classified into 2 groups only, lepromatous (Ridley and Jopling LL and BL) or tuberculoid (BT and TT). Patients with pure borderline (BB) or indeterminate (I) leprosy were also excluded from the study.

Venous blood was taken from the patients into heparinized tubes between the hours of 9.00 and 10.30 a.m.: plasma was immediately separated, and stored at − 20°C until being flown to Australia, packed in dry ice, for LH, FSH and testosterone estimations.

FHS and LH were assayed by a double-antibody radio-immunoassay using LER-907 obtained from the National Pituitary Agency, USA as standard for both FSH and LH and antisera supplied from the same agency. I^{125}-labelled FSH was obtained from Sorin Nuclear, Brussels, and commercially obtained goat anti-rabbit gamma globulin used for precipitation.The normal male range for both FSH and LH is 2–10 m IU/ml.

Testosterone was measured by radio-immunoassay using pure standard and tritium-labelled testosterone obtained from the Radiochemical Centre, Amersham, UK, and New England Testosterone rabbit anti-serum NEA-042A. Samples were extracted with ether before assay, and incubated at 4° C overnight. The normal male range is 10–35 nmol/l.

The significance of differences between means was estimated by Student's 't' test. The correlation coefficient, r, was computed automatically on an HP32E calculator: the significance of r was calculated from the equation:

$$t = r\sqrt{\frac{n-2}{1-r^2}}$$

Results

There were 97 patients in the study: their ages and type of leprosy are listed in Table 1. The mean age of the lepromatous patients (L) (33.7 ± 10.8 years) was significantly higher than that of the tuberculoid (T) patients (28.4 ± 10.1 years,

Table 1

	Age distribution of the patients	
	Lepromatous	Tuberculoid
15–19	4	6
20–29	25	9
30–39	21	11
40–49	9	3
50+	7	2
Total	66	31

$t = 2.32$, $p < 0.05$): and the *L* patients had been on treatment significantly longer (9.34 ± 7.3 years) than the *T* patients (5.94 ± 5.7 years, $t = 2.473$, $p < 0.02$).

In the *L* patients, 36 (54.5%) had significantly elevated FSH levels (greater than 12 m IU/ml), compared with 3 (9.7%) of the *T* group. Using a χ^2 test with Yate's correction, this gave a value of 15.8457: for two degrees of freedom, a *p* value of less than 0.001. Likewise 13 (19.7%) of the *L* patients had elevated LH, against none of the *T* patients. The mean FSH and LH levels in the *L* patients were significantly higher than in the *T* patients (Table 2 and Figure 1).

Table 2

Group	No.	Mean FSH (mIU/ml)	Mean LH (mIU/ml)	Mean testosterone (nmol/l)
Lepromatous	66	17.75 ± 12.9* (3.6 — 5.4)†	7.52 ± 7.6 (0.8 — 54.6)	13.7 ± 10.01 (0.5 — 50.3)
Tuberculoid	31	7.91 ± 3.99 (2.0 — 23.8)	3.69 ± 1.25 (1.2 — 7.2)	19.5 ± 6.4 (9.3 — 37.1)

*± 1 Standard deviation.

†Range.

Difference between lepromatous and tuberculoid patients: FHS $t = 6.006$, $p < 0.001$; LH $t = 3.95$, $p < 0.001$; testosterone $t = 3.402$, $p < 0.001$.

Table 3. Effect of duration of treatment on FSH and LH levels in *L* patients

Duration of treatment (years)	No.	Percentage with elevated levels of FSH	LH
0–4	24	11 (46%)	2 (8.3%)
5–9	16	8 (50%)	4 (25%)
10–14	11	6 (54%)	2 (18%)
15+	15	11 (73%)	(5) (33%)
Total	66	36 (54.5%)	13 (19.7%)

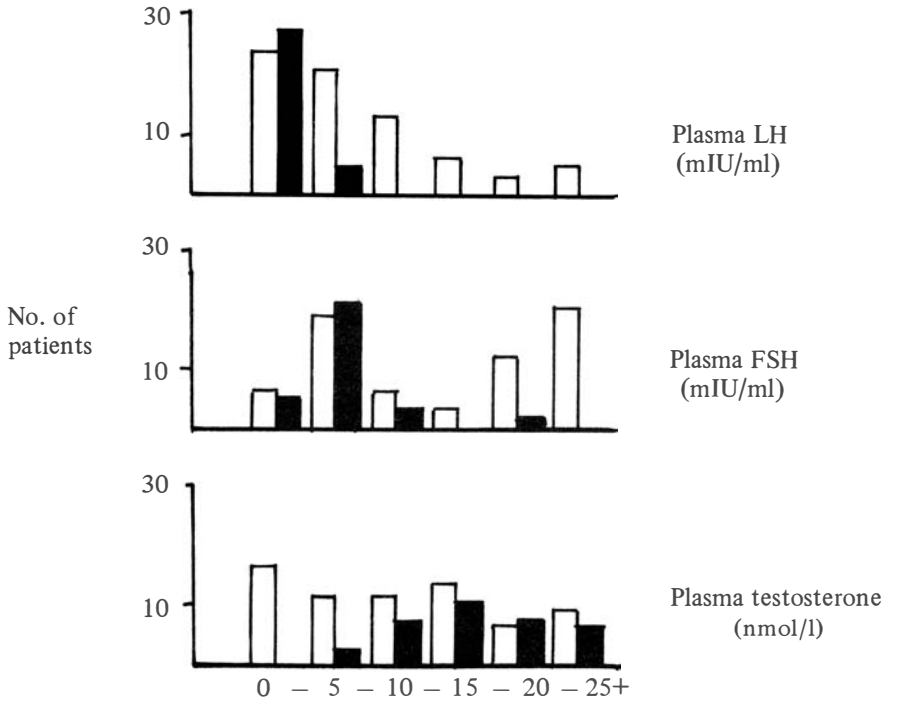


Figure 1. Distribution of Serum FSH, LH and testosterone in male *L* and *T* patients. □, *L* patients; ■, *T* patients.

Among the *L* patients, there was a significant relation between FHS ($r = 0.3199$, $p < 0.02$) and LH ($r = 0.4755$, $p = < 0.01$) with duration of treatment (Table 3).

No positive correlations between gonadotrophin levels and either duration of treatment or age were found in the *T* patients, suggesting that the changes in the *L* patients are not due to increasing age.

Plasma testosterone levels were significantly lower in the *L* patients (Table 2) but there was no correlation with either age or duration of treatment. A significant negative correlation was found between testosterone and both LH ($r = -0.3655$, $t = 3.14$, $p < 0.01$) and FSH ($r = -0.3248$, $t = 2.7474$, $p = < 0.01$) in the *L* patients.

Discussion

The early impact of lepromatous leprosy on the testis, as with many other toxic agents such as heat, irradiation and mumps, is on the seminiferous tubules rather than the interstitial cells of Leydig. This is reflected in elevation of FSH being at a much earlier stage than LH: infertility rather than hypogonadism is

the clinical result of this, though hypogonadism with or without gynaecomastia may occur later. There are few studies of the fertility of lepromatous patients: Kumar *et al.*[10] reported a significantly lower fertility and birth rate among the spouses of lepromatous males in India than in a control group of non-leprous patients. A study of fertility of male leprosy patients is now under way in Papua New Guinea. Morley *et al.*[5] found a significant positive correlation between duration of disease and basal LH, but do not comment on the relationship with FSH: their study population was, however, significantly different, with respect to the age of their patients (14 ± 16.1 years), from the present study, and a direct comparison is therefore not possible.

The normal levels of gonadotrophins in the *T* patients is strong confirmatory evidence that testicular damage does not occur in this group of patients. Job and Macaden[11] described 3 patients with tuberculoid granulomata of the testes during reactional phases: the diffuse damage that occurs in lepromatous leprosy was not seen. Thus clinicians involved in the care of leprosy patients can in practice safely ignore testicular function in tuberculoid (TT and BT) patients.

All the patients, in both the *L* and *T* groups, were being treated with standard doses of Dapsone at the time of the study. The standard regime in use in Papua New Guinea for all forms of leprosy has been Dapsone 200 mg twice a week: this was changed to Dapsone 100 mg daily about 1 year before the study was undertaken. In non-leprous patients Dapsone has not been reported to lead to testicular damage, and the normal levels of FSH and LH in the *T* patients suggest that this treatment was not the cause of the abnormalities. Of greater significance is the apparent failure of treatment to prevent testicular damage. If part at least of the damage is due to immune complex vasculitis, anti-mycobacterial treatment could aggravate the problem.

Throughout the world, including Papua New Guinea, registered leprosy is commoner in males than in females, and lepromatous leprosy is generally agreed to be commoner in males after puberty than before. These facts may reflect a hormonal dependence of *Mycobacterium leprae*, or alternatively may result from a steroid-modulation of immune processes. If so testicular failure may have a beneficial effect on the course of the disease, and replacement hormonal therapy may be harmful. However, so far there is no firm evidence to support this hypothesis, and in its absence it would seem unfair not to use testosterone therapy. As far as we are aware, there have been no combined histological and hormonal studies of patients with lepromatous leprosy. Dass *et al.*[4] measured plasma testosterone in lepromatous patients, in some of whom testicular biopsies were performed but no correlation between the two was attempted. If testicular damage occurs at an early stage of the disease biopsies from a large number of lepromatous males at various stages of their disease would need to be obtained to study the natural history of progression.

The history of onset of leprosy in Papua New Guinea is frequently chronologically imprecise, and in this paper duration of treatment refers to the time

lapse since the patient first presented with clinical symtoms, taking no account of the regularity of treatment. Approximately one-half of the *L* patients seen within 3 years of starting treatment had significantly elevated FSH levels: and if the regression line of FSH against duration of treatment is extrapolated backwards, it crosses the upper limit of normal 4 years before treatment is started. It would thus seem that in many patients testicular damage has been gradually occurring for several years before the actual presentation for treatment.

In much of the world, the stigma of leprosy is a considerable social burden. Sterility and impotence will aggravate an already disastrous situation, particularly since many patients, and probably even more physicians, are incapable of rational discussion of human sexuality. A greater emphasis on this aspect of leprosy is urgently required to properly treat the disease in all its aspects.

References

[1] Grabstald H, Swann LL. Genito-urinary lesions in leprosy with special reference to the problem of atrophy in the testes. *J. Amer Med Assoc*, 1952, **149**, 1287–92.

[2] Job CK. Gynaecomastia and leprous orchitis: a preliminary study. *Int J Lepr*, 1961, **29**, 423–41.

[3] Martin FIR, Maddocks I, Brown JB. Leprous endocrinopathy. *Lancet*, 1968, **2**, 1320–1.

[4] Dass J, Murugesan K, Laumas KR, Deo MG, Khandari KC, Bhutani LK. Androgenic status of lepromatous leprosy in patients with gynaecomastia. *Int J Lepr*, 1976, **44**, 469–74.

[5] Morley JE, Distiller LA, Sagel J, Kok SH, Kay G, Carr P, Katz M. Hormonal changes associated with testicular atrophy and gynaecomastia in patients with leprosy. *Clin Endoc*, 1977, **6**, 299–303.

[6] Wall JR, Wright DJM. Antibodies against testicular germinal cells in lepromatous leprosy. *Clin exp Imm*, 1974, **17**, 51–9.

[7] Burger HG, Baker HWG, Hudson B, Taft HP. Gonadotrophic and gonadal function in the normal adult male. In Saxena BB, Beling CE, Gandy HM (eds), *Gonadotrophins*. New York: Wiley-Interscience, 1972, pp. 569–92.

[8] Paulsen CA. The testes. In Williams RH (ed), *Textbook of Endocrinology*. WB Saunders Co., Ch. 6.

[9] Ridley, Jopling. Classification of Leprosy according to immunity, a five-group system. *Int J Lepr*, 1966, **34**, 255–73.

[10] Kumar A, Bagghis C, Indrayan A. Impact of lepromatous leprosy on fecundity. *Fert Steril*, 1973, **24**, 324–5.

[11] Job CK, Macaden VP. Leprous orchitis in reactional borderline cases. *Int J Lepr*, 1963, **31**, 273–9.

Lepr Rev (1981) **52**, 127–134

Gonadal function in lepromatous leprosy*

S SHILO,† Y LIVSHIN,‡ J SHESKIN** & I M SPITZ†
†*Department of Endocrinology and Metabolism, Shaare Zedek Medical Center,* ‡*Department of Obstetrics and Gynaecology, Bikkur Cholim Hospital and* **Department of Dermatology, *Hadassah University Hospital and Government Hospital for Hansen's Disease, Jerusalem, Israel*

Received for publication 25 November 1980

Summary Gonadal function has been studied in 14 male patients suffering from lepromatous leprosy. Eleven out of the patients showed reduction in testicular size. Eight had azoospermia and four oligospermia. Gynaecomastia was noted in 12 patients. In only 2 recently discovered cases was this absent.

Twelve of our patients had increased basal and peak FSH responses to LHRH. The LH response to LHRH was heterogeneous. Four patients had normal basal and peak levels; 4 had normal basal levels with an increased response to LHRH; 4 had elevated basal and peak responses and the remaining 2 had elevated basal levels with normal peak responses to LHRH.

Testosterone was normal in all patients while oestradiol 17β and oestrone levels were significantly elevated. There was no correlation between basal and peak gonadotrophins and testosterone, oestradiol 17β, oestrone or any of the clinical parameters.

Introduction

Testicular involvement in lepromatous leprosy is very common with atrophy of the testis being reported in up to 50% of patients.[1–3] In contrast, the ovary is almost never involved. The testicular lesions are associated with direct invasion by acid-fast bacilli and post-mortem studies have demonstrated that this occurs in 90% of male patients. Both seminiferous tubules and Leydig cells

*This paper is dedicated to the memory of Professor Alexis Ilukevich and was supported by a grant to J Sheskin from the Deutsches Aussätzigen Hilfswerk.

Address reprint requests to S Shilo, MD, Department of Endocrinology and Metabolism, Shaare Zedek Medical Center, Box 293, Jerusalem 91 000, Israel.

0305–7518/81/052127 + 08 $01.00

are involved and the later stages are characterized by fibrosis and no outline of the tubules can be detected.[1-2]

Gynaecomastia may be seen in lepromatous leprosy with an incidence ranging from 8.6% to 83%.[3-4] Usually gynaecomastia is associated with testicular atrophy and is seen both with and without concomitant generalized feminization.[4]

High levels of urinary gonadotrophins were reported by Grabstald and Swan.[1] Martin *et al.*[5] confirmed this and also found reduced plasma testosterone and urinary oestrogens in lepromatous patients with gynaecomastia and testicular atrophy. Increased basal luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels with an exaggerated gonadotrophin response to LH-releasing hormone (LHRH) has been described in lepromatous leprosy. In contrast, the response was normal in those with the tuberculoid form of the disease.[3]

In the present study, we have evaluated hypothalmic–pituitary–gonadal relationships in males with lepromatous leprosy and correlated this with the clinical presentation.

Materials and methods

PATIENTS

Fourteen unselected male patients with proven lepromatous leprosy under treatment with dapsone either alone or in combination with Thalidomide were studied. Clinical details are given in Table 1.

EXPERIMENTAL PROTOCOL

The test procedure was performed between 8.00 and 8.30 h after an overnight fast. A needle inserted into an antecubital vein was kept patent by slow administration of normal saline. Three blood samples were drawn during a 30-min equilibration period. All subjects then received 100 μg luteinizing hormone-releasing hormone (LHRH) by rapid i.v. injection. Blood samples were drawn at 10-min intervals following LHRH. Thyrotrophin-releasing hormone (200 μg) and the dopaminergic antagonist, Metoclopramide (10 mg) were given 30 and 60 min after LHRH. Full details of the prolactin, thyrotrophin and gonadotrophin responses will be published separately.[6] The results of the patients were compared to 28 healthy controls aged 20–40 years who received the same protocol. Informed consent for the test procedure was obtained from both patients and controls.

METHODS

Serum LH, FSH, testosterone (T), 17β-oestradiol (E_2) and oestrone (E_1) were determined by previously described methods.[7] LH and FSH levels were

Table 1. Clinical features of patients with lepromatous leprosy

Case No.	Age	Duration of disease	Grade of gynaecomastia	Testis size (cm)		Spermogram		Motility
				Left	Right	Volume (ml)	Count	
1	27	1	0	2 x 3	2.5 x 3		AZ	
2	38	1	0	2.5 x 2	2 x 3	2.5	14×10^6/ml	15%
3	48	1	1	1.5 x 2	1.2 x 2.5	2.8	52×10^6/ml	30%
4	29	9	2	2 x 3	2.5 x 2	2.0	30×10^6/ml	50%
5	35	14	3	1.5 x 1.5	0.5 x 1		AZ	
6	40	15	2	1.5 x 1.5	1.5 x 1.5		AZ	
7	36	15	3	0.5 x 1	0.5 x 1		AZ	
8	40	15	1	1.5 x 2	1.5 x 2		AZ	
9	37	15	2	1.0 x 1.5	1.5 x 1.5		–	
10	43	17	3	2.5 x 1.5	2 x 1.5		AZ	
11	33	18	2	2.5 x 2.5	2.5 x 2.0	1.5	4×10^6/ml	60%
12	35	20	3	2.5 x 2.5	2 x 2.5	2.0	11×10^6/ml	35%
13	56	27	3	1.0 x 1.5	1 x 1.5		AZ	
14	42	30	1	2 x 1.5	2 x 3		AZ	

AZ = azoospermia; 0 = patient without any enlargement of the breast.

expressed by reference to the second International Reference Preparation of human menopausal gonadotrophins. The actual standard used in the assay was the International Reference Preparation of pituitary FSH and LH (69/104), which was kindly provided by the Division of Biological Standards and Control, Hampstead, London, England. Antisera to LH (final dilution 1 : 200,000) and FSH (1 : 400,000), were kindly supplied by the National Pituitary Agency, National Institute of Arthritis, Metabolism and Digestive Diseases, Bethesda, Md. ^{125}I-Labeled LH and FSH were purchased from Cea-Sorin. Intra- and interassay coefficients of variation were as follows: 5.8% and 19.1% (LH) and 4.3% and 6.9% (FSH). E_2, E_1, and T were determined after pooling equal volumes of the three basal samples.

Student's *t*-test was used to compare responses in patients and controls.

Results

CLINICAL FEATURES (TABLE 1)

The age of the patients ranged from 27 to 56 years, the mean being 41.5 years. The duration of the disease process varied from 1 to 30 years. Thirteen out of 14 patients were married, but only 6 had their own offspring. In almost all cases, the children were born during the first years of the disease; only in one case was a child born 14 years after the disease was diagnosed. Libido was reported normal in 12 out of 14 patients. Hair distribution was normal in all cases with the exception of loss of eyelashes and eyebrows – a consequence of the primary disease. No change in sex hair distribution was found.

Gynaecomastia was classified according to Hall[8] into 3 grades and was found in 12 out of 14 cases. The 2 cases without gynaecomastia had the disease for less than 1 year. There was, however, no correlation between the severity of the gynaecomastia and duration of the disease. Thus, in case no. 14, there was only slight gynaecomastia after 30 years of leprosy.

The testes were clearly reduced in size in 11 patients. This became more evident with the increase in duration of the disease. All patients with leprosy for 10 years or longer had small hard testes. Semen analysis was performed successfully in 13 patients. Eight patients showed azoospermia, while in 4, oligospermia was found with sperm counts ranging from 4×10^6/ml to 30×10^6/ml. Case 3 had a count of 52×10^6/ml. No correlation between duration of the disease and the spermogram could be demonstrated.

GONADOTROPHINS (TABLE 2)

Normal values for both LH as well as FSH have been defined as values within 2 s.d. of the mean in the controls. Using these criteria, 4 of the patients had normal basal and peak LH responses to LHRH. Another 4 had normal basal

Table 2. Laboratory findings

Case no.	LH (mIU/ml) Basal	LH (mIU/ml) Peak	FSH (mIU/ml) Basal	FSH (mIU/ml) Peak	Testosterone T(ng/ml)	17β oestradiol E_2 (pg/ml)
1	11.3	58.6	6.6	10.6	12.0	68
2	6.1	200	10.4	19.0	9.5	80
3	3.5	71.6	13.2	24.0	6.0	42
4	5.9	214.8	12.4	29.3	3.2	42
5	42.4	191.4	55.3	83.1	6.0	50
6	77.7	302.7	71.0	120.2	4.7	46
7	46.2	196.6	33.3	44.9	4.5	52
8	28.4	80.0	42.1	71.0	8.0	64
9	49.6	107.8	44.4	61.7	4.2	54
10	10.6	31.6	28.7	43.6	2.9	70
11	4.0	34.6	3.5	5.1	8.0	46
12	5.3	129.1	12.9	22.7	4.3	52
13	24.1	50.0	11.8	191.0	6.5	60
14	7.2	349.4	11.0	41.8	3.9	52
Mean	24.5	118.9	25.6	42.1	5.98	55.6
± s.d.	23.8	90.1	21.5	36.7	2.6	11.3
Controls						
Mean	11.5	57.1	5.9	9.4	5.9	22.1
± s.d.	4.2	13.9	2.1	2.7	2.0	6.9

levels with exaggerated LH responses to LHRH. A further 4 had elevated basal and peak LH responses and the remaining 2 subjects had high basal LH levels, but their response to LHRH was normal.

Twelve out of the 14 patients had high basal levels of FSH with an exaggerated peak response to LHRH. In general, the highest levels of FSH were observed in patients who had lepromatous leprosy longer than 14 years. There was, however, no significant correlation between the basal and peak gonadotrophins and duration of the disease, testicular atrophy, spermogram or gynaecomastia.

STEROIDS (TABLE 2)

As a group, the lepromatous patients demonstrated normal T levels, the mean ± s.d. being 5.9 ± 2.6 ng/ml as compared to 5.9 ± 2.0 ng/ml in the controls. All the patients showed an increase in E_2 levels. The mean in the patients was 55.6 ± 11.3 pg/ml as compared to 22.05 ± 6.9 pg/ml in the controls ($p < 0.01$). Similarly, the patients showed an increase in E_1 levels (92.5 ± 63.0 pg/ml) compared to 52.9 ± 15.5 pg/ml in the controls ($p < 0.001$). No correlation between T, E_2, E_1 or E_2/T and the various clinical parameters could be demonstrated. In addition, there was no correlation between steroid levels and the basal or peak level of gonadotrophins.

Discussion

Although it is well known that the testis is involved in leprosy, there is a paucity of data relating clinical findings with fertility potential and gonadotrophin secretion. In the present study, we found that 71% of our patients had reduction in testicular size. This is a high percentage compared to other studies which report testicular involvement in 10–50% of patients.[1,3] As the duration of the disease process progresses, more of our patients presented with testicular atrophy. Our patients had azoospermia or oligospermia. This however, did not correlate with the duration of the disease process. Azoospermia was observed in some patients with leprosy of 1 year's duration and oligospermia was noted in some after 18 years of the disease. The lack of association between sperm count and duration of disease is probably related to the pathogenesis of the testicular involvement in leprosy. In the initial phase, there is perivascular infiltration by lymphocytes and lepra cells.[1] Later, there is clumping of Leydig cells with tubular destruction. The final stage is characterized by fibrosis of the tubules. This progressive destruction explains the presence of low grade spermatogenesis.

Gynaecomastia was noted in 12 of our patients, only 2 recently discovered cases did not show this phenomenon. This percentage is much higher than that observed by Watson *et al.*,[4] and similar to the incidence reported by Morley *et al.*[3] Gynaecomastia was not related to the duration of the disease.

Twelve of our patients (85.7%) had increased basal and peak FSH responses to LHRH. This is compatible with previous observations.[9–10] In contrast to FSH, the LH response was heterogeneous. Four out of 14 had an LH secretory pattern similar to the controls. This is contrary to Morley *et al.*[3] who noted exaggerated LH as well as FSH responses. However, Dash *et al.*[10] found that the LH response to LHRH in lepromatous leprosy was not different from the controls. Four of our patients had normal basal LH levels with exaggerated responses to LHRH, indicating the presence of increased LH reserve. Two cases, in contrast, showed high basal LHs with a normal response to LHRH.

The normal LH profile in some patients indicates that Leydig cell function may be relatively spared. The high basal levels of FSH and the increased response to LHRH implies damage to the seminiferous tubule. Since all except 2 subjects had increased FSH levels, this is an indication that seminiferous tubule damage is common. This is supported by pathological studies showing invasion by acid-fast bacilli in 90% of cases.[2] Of interest was the high FSH level in 3 patients who had their own children. Conception evidently occurred prior to seminiferous tubule destruction. The one patient who had a child after 18 years of leprosy, had a normal LH and FSH profile. We could not demonstrate a correlation between duration of the disease and either of the gonadotrophins. Morley *et al.*[3] in contrast, did find a correlation with basal LH.

T was normal in all patients, while E_1 and E_2 were elevated. There was no relation to duration of the disease and no correlation with the gonadotrophins. This is contrary to what was found by Martin *et al.*[5] and Morley *et al.*[3] who noted lower T, E_2 and urinary oestrogens in lepromatous leprosy. Dash *et al.*,[9] in contrast, has observed elevated serum E_2. It is possible that the high plasma LH noted in some patients maintains normal T levels. The increased levels of E_2 and E_1 could result from direct testicular secretion, or aromatization from T and androstenedione respectively.[11–14] Alternatively the increased levels may be due to inadequate inactivation of oestrogens occasioned by deranged liver function.[15] Testicular secretion of E_1 accounts for less than 5% of E_1 production in normal man.[12] Thus nearly all the E_1 is accounted for by peripheral conversion of androstenedione and to a lesser extent E_2.[13] Similarly the testis only accounts for one-fourth of E_2 production in normal man.[11] It has been shown that the pathological testis secretes even less E_1 and E_2.[11–12] Hence it is most likely that the elevated E_1 and E_2 levels are due to increased aromatization rather than testicular secretion.

Finally, it should be stressed that all of the patients were receiving Dapsone and a few Thalidomide. It is not known as to whether these compounds contributed to the testicular pathology or the hormonal aberrations which we have observed.

In conclusion, it is apparent that most patients with lepromatous leprosy have increased FSH secretion; in contrast 78% had normal LH values. T was normal, although E_1 and E_2 were increased. We could demonstrate no correlation between gonadotrophins, steroids and the various clinical parameters.

References

1 Grabstald H, Swan LL. Genitourinary lesions in leprosy with special reference to atrophy of testes. *JAMA*, 1952, **149**, 1287.

2 Job CK. Gynaecomastia and leprous orchitis. *Int J Lepr*, 1961, **29**, 423.

3 Morley JE, Distiller LA, Segal J, Kob SH, Kay G, Katz M. Hormonal changes associated with testicular atrophy and gynaecomastia in patients with leprosy. *Clin Endoc*, 1977, **6**, 299.

4 Watson RA, Gangai MP, Skinsnes OK. Genitourinary Leprosy. *Urol Int*, 1974, **29**, 312.

5 Martin FIR, Maddocks I, Brown JB, Hudson B. Leprous endocrinopathy. *Lancet*, 1968, **2**, 1320.

6 Shilo S, Livshin Y, Sheskin J, Spitz IM. Gonadotrophin, prolactin and TSH secretion in lepromatous leprosy. (Submitted for publication.)

7 Spitz IM, LeRoith D, Livshin Y, Zylber-Haran E, Trestian S, Laufer N, Ron M, Palti Z, Schenker J. Exaggerated prolactin response to TRH and metoclopramide in primary testicular failure. *Fert Ster* (in press).

8 Hall PF. Gynaecomastia. In Astwood E (ed.), *Clinical Endocrinology*. New York: Grune & Stratton, 1960, Vol. I, p. 468.

9 Dash RJ, Samuel E, Kaur S, Rastogi D, Rastogi GK. Evaluation of male gonadal function in leprosy. *Horm Metab Res*, 1978, **10**, 362.

[10] Dash RJ, Kumar B, Sialy R, Rastogi GK. LH, FSH, responses to GnRH in lepromatous leprosy. *Horm Metab Res*, 1979, **11**, 413.

[11] Kelch RP, Jenner JR, Weinstein RL, Kaplan SL, Grumbach MM. Estradiol and testosterone secretion by human, simian, and canine testes in males with hypogonadism and in male pseudohermaphrodites with feminizing testes syndrome. *J Clin Invest*, 1972, **51**, 824.

[12] Weinstein RL, Kelch RP, Jenner JR, Kaplan SL, Grumbach MM. Secretion of unconjugated androgens and eostrogens by the normal and abnormal human testis before and after human chorionic gonadotrophin. *J Clin Invest*, 1974, **53**, 1.

[13] Wilson JD, Harrod JH, Goldstein L, Hemsell DL, MacDonald PC. Familial incomplete male pseudohermaphroditism. Type 1. *New Eng J Med*, 1974, **290**, 1097.

[14] Naftolin F, Ryan KF. The metabolism of androgen in central neuro-endocrine tissues. *J Ster Biochem*, 1975, **6**, 999.

[15] Dass, J, Murugesan K, Laumas KR, Deo MG, Kandhari KC, Bhutani LK. Androgenic status of lepromatous leprosy patients with gynaecomastia. *Int J Lepr*, 1976, **44**, 469.

Lepr Rev (1981) **52**, 135–140

Clofazimine (Lamprene or B663) in lepra reactions

F M J H IMKAMP
Westfort Institution, Private Bag X559, Pretoria, 0001, South Africa

Received for publication 2 September 1980

Summary In view of some confusion in the literature as to the value of clofazimine in the treatment of patients with Type 1 reactions, due to cell-mediated immune mechanisms, 10 patients with this type of reaction were treated with this drug. The results were unsatisfactory; clofazimine had to be either stopped or withdrawn in favour of treatment and control of the reactions with prednisolone. Two of these cases are described in detail.

The anti-inflammatory effect of clofazimine, first suggested by Browne,[1–2] has proved to be effective in controlling erythema nosodum leprosum (ENL).[3–5] It is, however, important to distinguish the lepra reactions and to specify accurately where in the spectrum TT–LL the reactions occur.[6] Turk[7] describes two lepra reactions depending on the presence or absence of cell-mediated immune processes, i.e. the lepra reactions depending on the immunity present and those occurring in pure lepromatous patients (LL) where cell-mediated processes are depressed.

The two lepra reactions have also been described by Jopling[8] as lepra reactions Type 1, indicating that the reaction occurs in patients with immunity however slight, and lepra reaction Type 2 which occurs in the pure lepromatous patients (LL) where there is no cell-mediated immunity present. Type 2 reaction is known as erythema nodosum leprosum (ENL).

Using Jopling's terminology to stress the difference of the two lepra reactions indicating the presence or absence of cell-mediated immune processes, it is therefore clear that lepra reactions Type 1 are different clinically, immunologically and histologically from the lepra reactions Type 2. The therapeutic management of the two lepra reactions is also different. For example: most clinicians would stop dapsone therapy at the onset of a Type 1 reaction, but there is doubt regarding the need to do so in Type 2 reaction.[9] Also, though

0305–7518/81/052135 + 06 $01.00

corticosteroids have an important place in the management of both types of reaction, Thalidomide is effective only in Type 2 reaction. There is a large literature on the good effect of clofazimine in Type 2 reactions and since early 1966 the author has found the drug consistently effective, but little is known of its effect in Type 1 reaction.

It is therefore important to find out if clofazimine was equally effective in lepra reactions Type 1, but after treating 10 patients with this type of reaction the use of clofazimine had to be abandoned as it was observed that clofazimine aggravated the reactional state of the patients and had either to be discontinued and prednisolone given or reduced and prednisolone added. Two of these case histories from patients seen and treated in Zambia will now be given in detail with photographs.

Case I

On 12 August 1969 an adult female African patient was admitted for treatment from another leprosarium after being classified a lepromatous case. From the scanty notes we learned that on admission two smears were recorded: left ear negative and right ear positive. Prior to admission she was treated with CIBA 1906, 1.5 g daily, without any improvement. On 24 February 1970 she had a 'reaction'. Treatment with CIBA 1906 continued but the patient's condition worsened. On 23 March 1970 CIBA 1906 was discontinued and after a course of chloroquin she was given clofazimine, 500 mg daily. As the patient's reaction worsened she was transferred to Liteta Leprosarium (Zambia) on 28 May 1970. On examination the patient showed raised hyperpigmented facial skin lesions with definite edges (Figure 1). Her nose was swollen, hyperpigmented due to clofazimine treatment, dry and crusty. She had numerous raised lesions, varying in size with good edges, distributed all over her trunk and limbs. The areas between these lesions were healthy skin. Out of 7 smears, 6 were negative and the left ear was 2+, fragmented bacilli only.

The patient was reclassified BT in lepra reaction. A biopsy from a lesion on the right upper arm was reported as BB leprosy and a second opinion from Dr D J Harman, Leprosy Study Centre, London confirmed this classification. No leprosy drugs were prescribed but a course of prednisolone started at a dosage of 3 x 10 mg daily and gradually reduced. On 15 August 1970 the skin lesions were quiescent and flat and treatment with low-dosage dapsone commenced. The patient responded without complications and was discharged on 28 April 1971 on dapsone, 100 mg three times weekly (Figure 2).

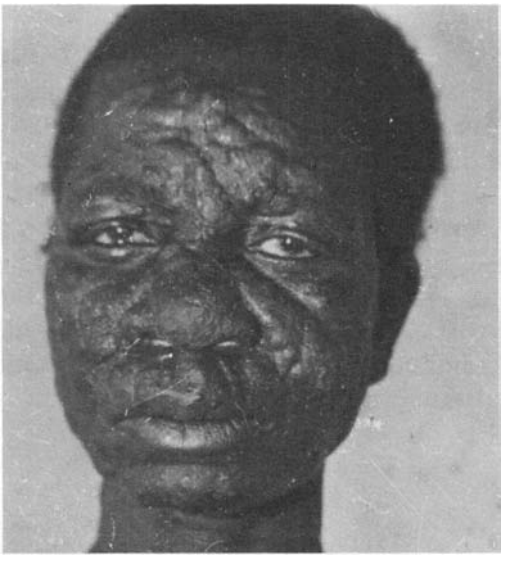


Figure 1. Case I. Condition on admission to Liteta Leprosarium. Worsening of reaction after taking clofazimine.

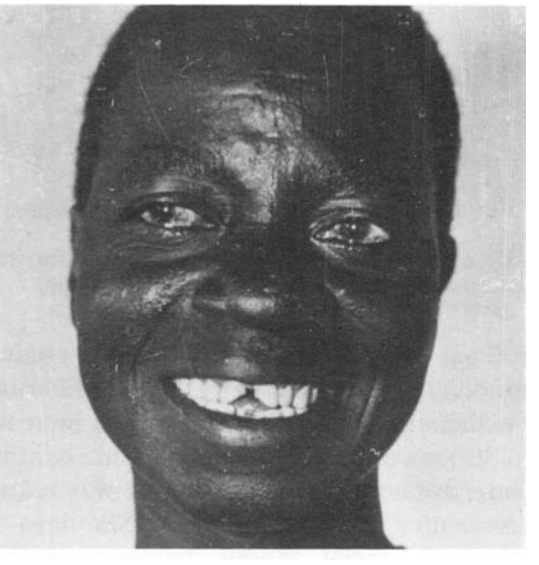


Figure 2. Case I. After discontinuation of clofazimine treatment and replacement by prednisolone and dapsone. Marked improvement prior to discharge from hospital.

Case II

An adult female African patient, aged 35, was admitted to Liteta Leprosarium on 12 September 1968 and examined by an experienced Medical Assistant (at the time the author was away). She was classified as a BB leprosy in reaction. Smears on admission were: left and right ears negative. Right cheek 5+. After a course of Chloroquin and Stibophen she was given prednisolone, 5 mg t.d.s. increasing to 10 mg t.d.s. as from 25 November 1968.

The patient was seen and examined by the author on 4 January 1969. Raised, erythematous, infiltrated skin lesions covered forehead, nose, cheeks and upper lip (Figure 3). In addition she had a few raised, small lesions on the right and left breasts and one large raised lesion on the right shoulder, of which the edges were irregular and sloping.

Figure 3. Case II. Raised, erythematous infiltrated lesion on forehead, nose, cheeks and upper lip at time of treatment with clofazimine with prednisolone.

Clofazimine, 100 mg daily, was prescribed and prednisolone reduced to 20 mg daily and gradually to 7.5 mg daily. On 10 February 1969 the facial lesions were more oedematous and clofazimine was increased to 200 mg daily and a week later to 300 mg daily, while prednisolone continued at 2.5 mg t.d.s.

After a slight improvement the prednisolone was reduced to 2.5 mg daily with clofazimine remaining at 300 mg daily. Six days later the condition of the patient worsened, facial lesions became more erythematous and oedematous and after another 2 weeks it was considered necessary to discontinue clofazimine completely (Figure 4). Prednisolone, 20 mg daily, finally

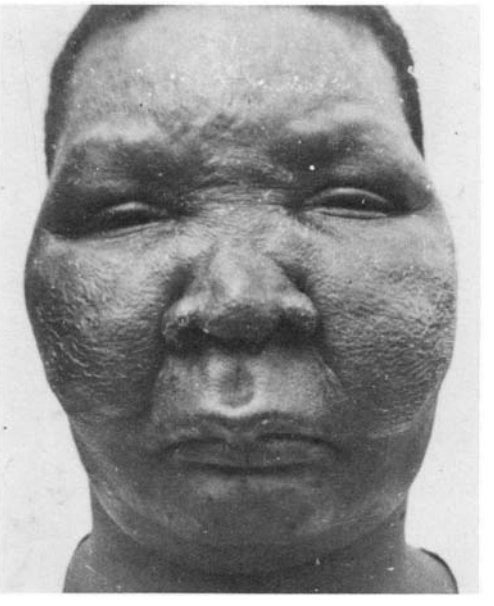


Figure 4. Case II. Worsening of reaction (see text).

controlled the reaction. Eight weeks later while the patient was still on 17.5 mg prednisolone daily, clofazimine at 100 mg *weekly* was resumed with no reaction occurring. After a further 7 weeks clofazimine was stopped and the patient given low-dosage dapsone. Prednisolone was discontinued after 2 more weeks and the reactional state was quiescent. The patient continued to improve without reacting to a gradual increase of dapsone and was finally discharged on 30 July 1971 to an out-patients' clinic (Figure 5).

Her facial lesions were inactive, the skin smooth and the hyperpigmentation caused by clofazimine was fading. Skin smears were negative.

Summary

It is important not to use the terminology 'lepra reaction' without specifying in which part of the TT–LL spectrum the reaction occurs. Jopling's terminology of reactions (Type I and Type 2) is greatly to be recommended. Clofazimine appears not to be effective in lepra reaction 1.

Acknowledgement

I wish to thank Dr D J Harman of the Leprosy Study Centre, London for his report on the biopsy on Case I.

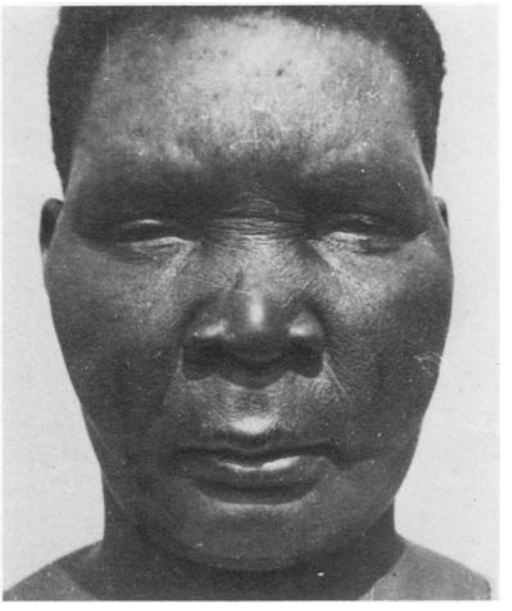


Figure 5. Case II. Marked improvement after discontinuation of clofazimine and maintenance on prednisolone (see text). Patient on dapsone only prior to discharge.

References

1 Browne SG. B663 – possible anti-inflammatory action in lepromatous leprosy. *Lepr Rev*, 1965, **36**, 9.

2 Browne SG. B663 (Geigy) – further observations on its suspected anti-inflammatory action. *Lepr Rev*, 1966, **37**, 41.

3 Imkamp FMJH. A treatment of cortico-steroid-dependent lepromatous patients in persistent erythema nodosum leprosum. A clinical evaluation of G30320 (B663). *Lepr Rev*, 1968, **39**, 119.

4 Jopling WH. *Management of Lepra Reactions* (9th Int. Leprosy Congress, London, 1968).

5 Morgan J. Management of steroid dependency with Clofazimine (Lamprene or B663, Geigy). *Lepr Rev*, 1970, **41**, 229.

6 Ridley DS, Jopling WH. Classification of leprosy according to immunity. A five-group system. *Int J Lepr*, 1966, **34**, 255.

7 Turk JL. Cell-mediated immunological processes in leprosy. *Lepr Rev*, 1970, **41**, 207.

8 Jopling WH. *Handbook of Leprosy*, 1971, London: Heinemann.

9 Pearson JMH *et al.* The effect of stopping Dapsone treatment for two months and then restarting it in full dosage in patients with moderately severe erythema nodosum leprosum.

Lepr Rev (1981) **52**, 141–149

An assessment of the usefulness and acceptability of eye shields under field conditions

NITTIN VERMA
Christian Medical College & Hospital, Vellore, and Schieffelin Leprosy Research & Training Centre, Karigiri, North Arcot District, India

Received for publication 13 September 1980

Summary Fifteen patients with varying degrees of lagophthalmos and neuroparalytic keratitis were fitted with eye shields made in the field, and an assessment at 1 and 2 weeks has shown that there is a definite improvement in the eye condition and that the community's acceptance of such a procedure is good. For introduction into the present leprosy control programmes, an evaluation of the paramedical workers has shown that they require only minimal additional training.

Introduction

Neuroparalytic keratitis and keratitis with lagophthalmos resulting from paralysis of the fifth and seventh cranial nerves respectively, commonly lead onto subequatorial or total blindness. In view of the fact that these lesions are common in leprosy (1% in South India) and easily preventable by timely tarsorraphy or a temporalis transfer, this constitutes one aspect of leprosy rehabilitation which has been grossly neglected, and thus needs immediate and early action. The risk of irreversible eye damage increases with each passing day that the patient stays away from hospital, due to numerous socio-economic reasons prevalent in his own surroundings. It is for this interval (that is between diagnosis and treatment) that we require a simple measure, which, in addition to affording protection to the eyes, is cheap and easily constructed in the field, on the spot, by the paramedical worker (PMW) in the absence of a doctor, who is the point of first contact between the patient and the hospital.

The above is merely to emphasize the need for a study of the acceptability and usefulness under field conditions of the eye shield so as to introduce it on a large scale in the present leprosy control programmes.

0305–7518/81/052141 + 09 $01.00

Preventive opthalmology still does not figure predominantly in the country's community health programmes, and least of all in the field of leprosy. Its introduction is urgently needed since the rehabilitation of the Hansen's patient is made much simpler and far more rewarding if this aspect of his well being is not neglected.

Objectives

1. To evaluate the effect of wearing an eye shield under field conditions.
2. To evaluate the acceptability of the shield by patients, with special reference to the social problems that it could create.

Materials and methods

The study was conducted in two regions of North Arcot District, South India (Gudiyatham and Vellore) under the auspices of the Schieffelin Leprosy Research and Training Centre, Karigiri and Rural Health Centre, Bagayam. Fifteen patients were studied – 14 out-patients and 1 in-patient. The distance on closing the eyelid varied from 2 to 15 mm, thus eliminating any discrimination with regard to the degree of lagophthalmos. The patients had varying degrees of epiphora, conjuctival injection, circum-orbital itching and dimness of vision. Three weekly visits were made along with the PMW to the patients' homes and an assessment of the shield and the eye was made at intervals of 7 days. They were then asked to come to hospital for definitive treatment of their ocular condition.

A *pro forma*, made after pre-testing with 4 PMWs and 1 doctor, was filled in. A provision was made for the assessment of:

1. The condition of the eye evaluated subjectively and objectively before and after shield application.
2. Changes in the condition of the shield after use.
3. Social problems which the patient might encounter during the course of the 2 weeks.
4. The condition of the skin after prolonged plaster application.

A grading for all the eye symptoms and signs was prepared and used to compare the changes in the eye before and after shield application.

The *pro forma* was filled in by the PMW after prior instruction and was further counter-checked by the doctor present. The mistakes made were noted and this, combined with a simple test given to 27 PMWs later, has been used to assess their attitudes towards (and knowledge of) eye complications in leprosy.

Patients with bilateral lagophthalmos had a shield fitted into one eye only, so that the other could act as a control.

CONSTRUCTION AND APPLICATION OF THE SHIELD

Materials required:

1. Adhesive plaster 3″ x 6″.
2. Plane glass (circular or square in shape as in spectacles).
3. Razor blade.
4. Spirit, tincture benzoin and cotton wool for local preparation of the skin.

The adhesive plaster is laid on a clean non-sticky surface. A hole 10 mm less in diameter than the size of the piece of glass to be used is cut out. The cleaned glass is then placed on the sticky surface of the plaster and fixed on to it by small strips of plaster applied to its edges. The contour of the shield is fashioned according to individual requirements so as to exclude beards, nose rings, etc. Prior to shield application the skin is cleaned with spirit and then painted with tincture benzoin for better adhesion. If the shield is reasonably air-tight it becomes misty from inside, an observation that serves as a quick guide as to whether it has been applied correctly. The mist clears almost immediately. Each shield takes about 15 min to make at first, and costs very little.

Results

The population studied consisted of 15 patients of whom 12 (80%) were men and 3 (20%) were women, residing in towns (5 people – 33.3%) and villages (10 – 66.7%). Four of them had already undergone previous surgical treatment (3 had undergone tarsorraphy and 1 a temporalis transfer); 4 of them had corneal opacities of varying depth and shapes; only 5 (33.3%) had corneal anaesthesis accompanying the lagophthalmos. The distribution of the type of Hansen's disease is shown in Tables 1 and 2.

Table 1

Lepromatous leproma	10
Borderline leproma	2
Borderline tuberculoid	1
Tuberculoid	2

Table 2

Duration of disease:	
Under 5 years	9 (60%)
6–10 years	3 (20%)
Over 11 years	3 (20%)

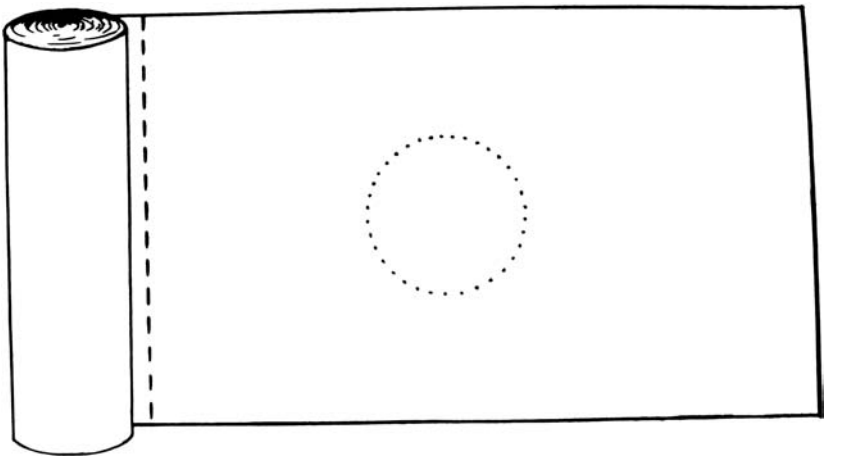


Figure 1

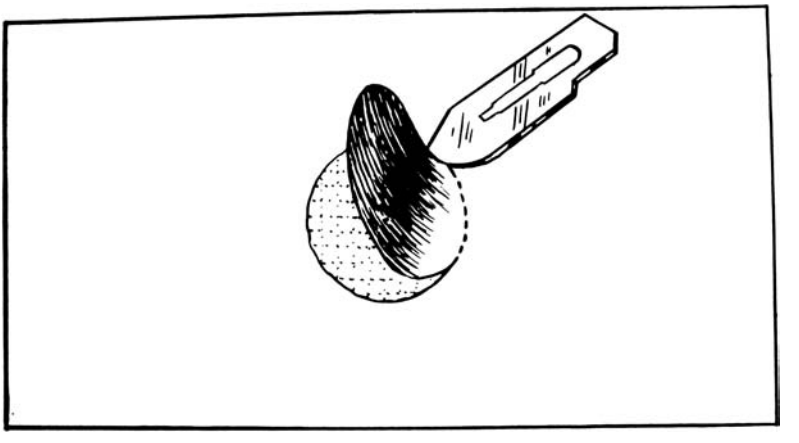


Figure 2

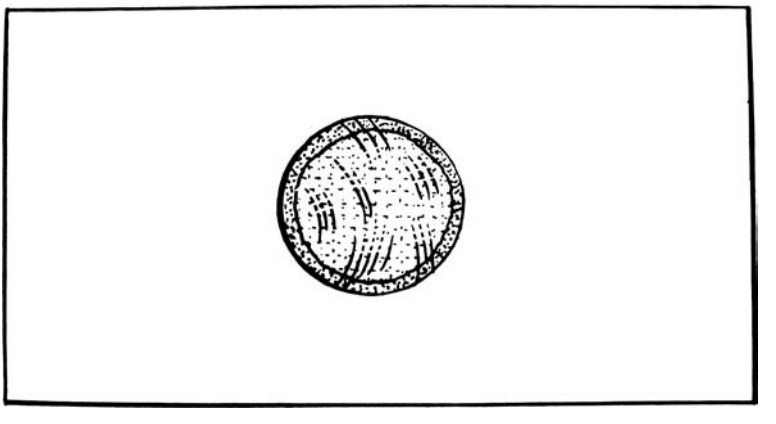


Figure 3

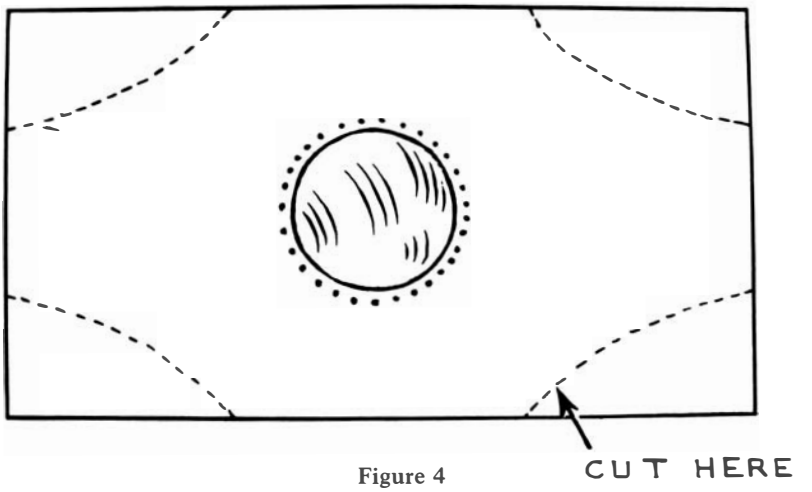


Figure 4

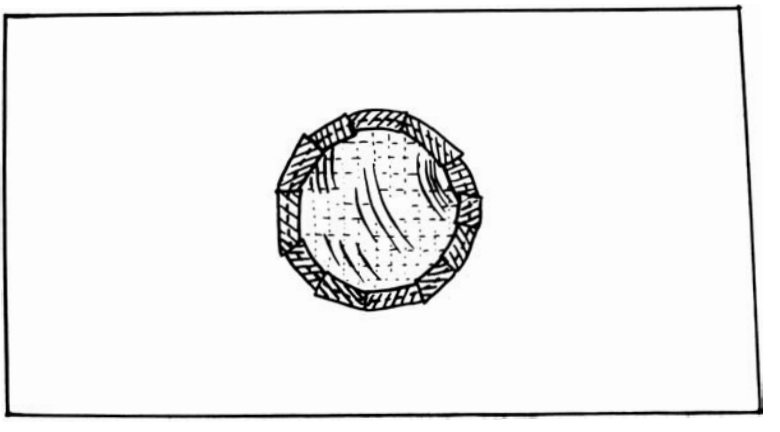


Figure 5

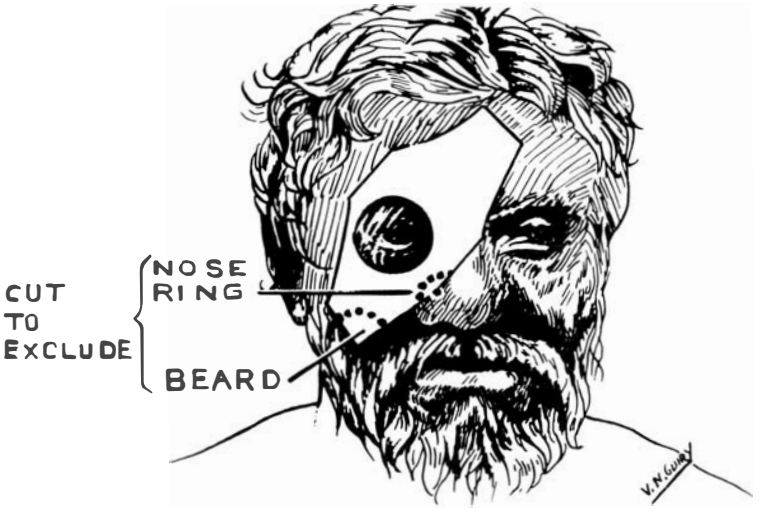


Figure 6

None of the patients were blind. All the patients were on regular treatment. Of the 15 patients originally selected, 3 were initially unco-operative, 1 of them removed the shield soon after application, 1 could not be traced, and 1 continued wearing the shield for the full period of 2 weeks. In 8 (61.5%) patients epiphora was an aggravating factor in addition to the lagophthalmos, 6 (75%) of the patients had ectropion; 2 (25%) of the patients had foreign bodies in the eye. These were removed before applying the shield. Five of the 15 patients had bilateral disease. The results presented in Table 3 are those of the 13 patients who could be followed up.

Table 3. Eye changes after shield application (2 weeks later)

	Initial		Marked improvement		Mild improvement		No changes	
	No.	%	No.	%	No.	%	No.	%
Watering	12	92.3	9	75.0	2	16.7	1	8.3
Itching*	10	76.9	9	90.0	1	10.8	–	–
Redness†	13	100.0	10	76.9	2	15.38	1	7.69

*Itching developed in 1 patient after wearing the shield.
†Redness appeared in 1 patient.

Figure 7. Initial condition of the eye.

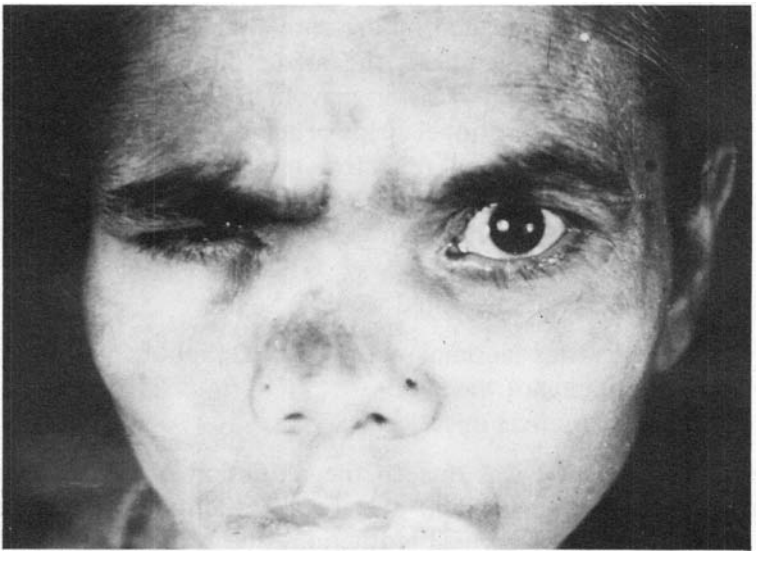


Figure 8. After 2 weeks of shield application.

The complications noted are shown in Table 4. The assessment of the PMWs showed that though all were aware of lagophthalmos and could recognize it, they were unable to recognize:

1. Cataract.
2. Scleritis.
3. Iridocyclitis.
4. Ectropion.

Twenty-six (96.3%) of the 27 were able to recognize the parts of the external eye correctly.

Table 4

Complications	No.	%	Patient complained of the symptom
Discharge collected in the chamber	2	15.39	Yes
Allergic manifestations	2	15.39	No
Dimness of vision	7	53.09	Yes (3), no (4)

Discussion

The study demonstrated the adequate functioning of the shield. During the 15 days that the 13 patients wore it, there were only five instances of the shield

coming off. This was due to sweating, carelessness, displacement during sleeping and in 1 patient it followed a drinking spree. This difficulty can be offset by using spirit to thoroughly clean the skin, followed by tincture benzoin application. The use of porous plaster may help further. The shield can also be reinforced by applying additional strips of plaster.

There was symptomatic and objective improvement in all the patients. In the series there was only 1 patient who had no change in the amount of epiphora and conjunctival injection. In this case the shield had become unstuck on the nasal side.

The major beneficial effects noted were:

1. A decrease in the reflex lacrimal secretion and epiphora.
2. Decrease in the itching of the eye.
3. A decrease in conjunctival injection.

The improvement noticed was due to the reduction of irritation to the eye from dust and other agencies, and also to a reduction of the desquamation of the superficial layers of corneal epithelium due to dessication. In addition, since the cornea is kept moist, the degree of reflex lacrimation is drastically reduced. The complications arising from application of the shield are very minimal and can be ignored. The shield can further be adapted for patients with photophobia by using a dark glass.

The major problem anticipated was an adverse reaction by the community to the shield. It was very surprising to note that the interaction between the patient and the community was in fact favourable. Some patients made excuses such as the shield was meant to treat a foreign body in the eye, others said it was a preparation for an operation. Some of the villagers were very happy since something was at last being done to help the patients and improve their condition. 'What does it matter how it looks as long as Abdul is benefiting from it', said one.

None of the patients was made fun of or rebuked in any way. An initial unco-operative patient removed the shield on going home as he strongly felt that it would interfere with his job in the local cinema. This is in contrast to a villager who was unco-operative but continued wearing the shield. On being asked the reason, he said that it was because of the comfort that he obtained from its usage. Hence it appears that the shield is not a source of ridicule and rejection of the patient by the community, nor does it add to the stigma of leprosy.

Finally, an assessment of the PMWs has shown that there is a need for teaching them the basic technique of evaluation of the eye as well as the importance of the major lesions in leprosy.

It must be emphasized that this is a temporary measure meant to provide relief and afford protection to the eye, rather than to act as a cure.

Such a procedure, simple yet so useful, does not require any special effort, since it can be incorporated smoothly into the existing leprosy control

programme without requiring any special equipment. The materials required to make the shield, apart from the glasses, are part of a normal village clinic equipment and hence the introduction of the shield could be started as soon as the PMWs have been trained.

Acknowledgements

I wish to express my heartfelt thanks to Dr E P Fritschi, Superintendent and Consultant Surgeon, Schieffelin Leprosy Research and Training Centre, Karigiri, for all the help and guidance that he has so willingly given me. I am also very grateful to Dr K Jesudasan, Dr Suriyawanshi and the Departments of Epidemiology and Opthalmology, Karigiri, as well as the staff of the Rural Health Centre of the Christian Medical College Hospital, Vellore, South India, especially Dr V Benjamin, Dr A Joseph and Dr J P Muliyil for the encouragement and advice that they have given me. Last but not least, Mr Mohan for helping me prepare the manuscript.

References

1 Agarwal LP. *Diseases of the Eye*, 2nd ed.
2 Brand Margaret. *Watch those Eyes*, 2nd ed. (rev.). London: The Leprosy Mission.
3 Duke Elder, Sir Stewart. *System of Opthalmology*, Vol. XIII, Pt I, *Diseases of the Eyelids.* Henry Kimpton, 1975.
4 Fritschi EP. *Reconstructive Surgery in Leprosy.*
5 Kelly ED. *Physical Therapy in Leprosy for Paramedical Workers*, 2nd ed. American Leprosy Missions, 1980.
6 WHO. *Workshop of Reconstructive Surgery and Prosthetics in Leprosy, 1978 (December).* Christian Medical College Hospital, Vellore, South India.

Lepr Rev (1981) **52**, 151–154

Leprosy and Curieuse Island

C R GRAINGER
Grimsby Health District, Queen Street, Grimsby DN31 1QQ

Received for publication 20 November 1980

An outline of the historical development of leprosy as a public health problem in the Seychelles[1] indicated the part played by the different leprosaria which were set up in the hope of treating and preventing the spread of leprosy, and in the earlier days the spread of leprosy in Mauritius and its other dependencies. The principal leprosarium during this period was situated on the island of Curieuse. The history of this island and its connections with leprosy is of interest and illustrates the changing importance and the attitudes to the disease in the past.

The island itself is about 3,500 m long and 2,000 m wide and reaches a maximum height of 172 m. It was named Curieuse after the ship which first discovered it in 1768.[2] It has had a continuing use for agricultural purposes and at some time in the past the bay on the east coast was walled off from the sea in an attempt to rear and keep green turtles. This island is coincidentally the natural home of the unique plant the coco-de-mer which is a large tree bearing double coconuts that were reputed to have aphrodisiac properties and were therefore of enormous commercial value in the past.

As the number of detected cases in the islands increased the need was felt for a place of isolation. Curieuse was not the first island used or suggested as a leprosarium in the area, as Providence Island had been in use for some time and Isle Platte and Denis Island were suggested before Curieuse was proposed as a suitable place.

The Commissioner in Seychelles wrote to the government of Mauritius on 21 October 1828:

> 'What I recommend is that Government should endeavour to regain possession of the island of Curieuse by offering an equivalent to its present proprietor thus ensuring the preservation of the coco-de-mer trees that are still to be found there in their pristine beauty, secondly an eligible spot upon which the sickly crews of vessels in quarantine might be landed and

0305–7518/81/052151 + 04 $01.00

> lastly a desirable place of residence for the leprous and other diseased blacks scattered over the various islands of this archipelago, Agalega and Diego Garcia.'

When one considers the speed of travel and communication at that time, commendably rapid action must have been taken as a result of this suggestion for in September 1829 a Mister William Ray offered his services as Superintendent for Curieuse, and the Commissioner in Seychelles was already having to bring to the attention of the authorities in Mauritius the difficulties of running a leprosarium for the whole territory in a letter dated 30 September 1829:

> 'Sir,
>
> As you merely mention in one of your letters having put on board the "Isette" provisions for one month I am left in doubt whether or not Government expects that the quantity of rice necessary for the subsistence of the lepers may be supplied at this Dependency and under this feeling consider it proper to acquaint you that grain of every kind is extremely scarce at the Seychelles and that it will be necessary to send from Mauritius such supplies of rice as the lepers stationed on Isle Curieuse may require.'

In July 1830 HMS *Jaseuse* visited Curieuse and the report of the doctor to the commanding officer describes the settlement as having 40 clean and commodious huts each having a small well-arranged garden stocked with vegetables and a good-sized airy hospital. There were 50 male patients, 12 women and 2 children in the leprosarium at that time.

More cases were arriving from the other islands all the time. Some of the cases were obviously extremely ill, including 4 who came in the brig *Sans Pareil* from Mauritius in August 1830. One patient, Clary Claire, died before she could embark and a further patient was too ill to travel. A patient reputed to have leprosy came from Diego Garcia in October of that year with small ulcers about the face and nose as well as having a destroyed soft palate.

Ray, the first medical superintendent of Curieuse, died in July 1833 and was replaced by Mister Boswood, late surgeon of the *Harriet*. In 1835 Doctor Robertson was appointed to the post. It was shortly after his appointment that trouble erupted, when a man with an axe attacked a women with whom he had previously lived. She had left him because of his violent temper. As a result of this incident Doctor Robertson arranged for two men from Praslin to build a prison on Curieuse for 30 dollars. Doctor Robertson was drowned in an accident on 30 June 1846 when returning to La Dique after a visit to Curieuse. Although few people now know the origins of the name, the pass in the reef where the accident happened is still known as Passe Robertson.

Curieuse does not seem to have been used much as a quarantine station, but in 1836 the New Bedford whaler *Ansley Gibbs* was quarantined there because

of a case of smallpox on board. The maximum number of leprosy patients in the camp was 100 in 1838, but by 1851 the number of cases had dropped to 38, although there were at that time 50 people resident in the camp. This reduction in the number of cases prompted the Commissioner to write to the Government on 24 April 1851:

> 'While upon the subject of Curieuse I would beg to suggest it as a matter worthy of being submitted to the consideration of His Excellency the Governor whether initiating measures might not very soon be taken for giving up the Leper Establishment altogether. I believe it has never been settled beyond a doubt that the disease is neither contagious nor infectious.
>
> A much greater boon that is greater than the leper hospital and that is most imperiously required would be the establishment of a general hospital for all kinds of diseases at Port Victoria which after the first outlay might be maintained by the Government at a no greater expense than it now incurs for the leper settlement.'

It was decided in 1855 that the Mauritian Government would cease to use Curieuse for leprosy patients from Mauritius and the island would in future be used for cases of Seychelles origins only and also as a pauper camp.

The leprosy settlement was on one side of the island and the pauper camp some distance away. The numbers in both camps were never high and by 1890 the number of leprosy patients was as low as 4. The doctor continued to visit the camps weekly but the hospital on Curieuse was no longer in use so patients were treated in their own huts or, if more seriously ill, were transferred to the hospital on Mahe.

In spite of the enacting of an ordinance which allowed compulsory segregation of patients under certain circumstances, the numbers in the settlement remained low, so that all were eventually transferred from Curieuse to a smaller island some little distance away.

In 1900 during the Boer War it was proposed to use Curieuse as a prisoner-of-war camp but the idea was never implemented. The island reverted to agricultural use whilst the patients made a number of moves between 1900 and 1937 when the need for more space arose and other sites proved unsuitable. In 1934 it was proposed to once again build a camp on Curieuse at an estimated cost of 6,675 rupees. The transfer of patients did not take place until 1937, however.

The settlement remained under the immediate supervision of the superintendent whilst the doctor visited one day each week and was assisted on those occasions by a sanitary inspector. The numbers had been slowly increasing over a number of years as by 1947 there were 40 individuals segregated in the camp. The settlement itself was divided into three parts, the central dispensary, hospital, recreation room and shop divided the male part of the camp from the female part.

Quite ambitious works were undertaken by the people in the settlement as part of their occupational and recreational therapy and included building and construction work with the objective of making the community self sufficient. A start was made on clearing and draining land for the production of crops as well as pigs and cattle being reared to feed the patients and any surplus being used to create revenue for the settlement. Although official reports were positive and hopeful the inmates were not above creating mischief if the occasion arose. Much to the consternation of the authorities there were a number of occasions when sanitary inspectors were chased or even stoned by the irate patients who did not passively accept the regular injections that were prescribed for them. The last straw, however, was when the inmates carried out the unauthorized slaughter of the Government's cow. This sort of behaviour mirrors the indiscipline often found in other leprosaria where custodial care was practised.

In spite of these setbacks, the regular use of Dapsone either by tablet or injection was improving the condition of the patients, so that by 1965 many of the cases were cured or burnt out. Some of the patients were old and in need of care and attention and others had been in the settlement so long that they had no home to be discharged to, so on 2 March 1965 19 of them were transferred to a new camp at Anse Louis. The history of Curieuse had come full circle as the island reverted to being a place for the preservation of the coco-de-mer, as originally proposed in 1828.

Acknowledgements

I wish to thank Mr Macgaw for his help and permission to examine the records in the National Archives, and not to forget Mr C Hoareau the last Sanitary Inspector to be chased on Curieuse.

References

1 Grainger CR. Leprosy in the Seychelles. *Lepr Rev*, 1980, **51**, 43–9.
2 Bradley JT. *History of Seychelles*, 2 vols. Mahe, Seychelles, 1940.

Lepr Rev (1981) 52, 155–159

Leprosy in Indonesia

MARWALI HARAHAP
Department of Dermato-venereology, School of Medicine, University of North Sumatra, Rumah Sakit Dr Pirngadi, Medan, Indonesia

Received for publication 5 January 1981

Summary The leprosy control programme in Indonesia is discussed. The epidemiological situation of leprosy is assessed from the statistics of the registered cases and through comparison of several leprosy surveys.

In a certain province leprosy showed a marked decline, while in other provinces the prevalences are still high.

We need to recognize the limitations of the present measures employed to control leprosy, and to accept that if we want to control the point of eradication, the only hope lies in immunization.

Introduction

Indonesia is a country of more than 3,000 islands stretching along the equator with 140 million inhabitants – 72,000,000 women and 68,000,000 men. The age distribution indicates a population with 2.4% of growth. It is not equally distributed, the most densely populated area is Java with a population density of 565 people per square kilometre, while in the outer islands the density is 1.8–35 per square kilometre.

Although poverty is widespread, expanding building programmes and rising standards of living bear witness to economic advances.

Major health problems

The major health problems in the present stage of economic development in Indonesia do not differ much from those of other developing countries.

Communicable disease is a major part of the total health problem, followed by the problem caused by malnutrition.

0305–7518/81/052155 + 05 $01.00

Communicable disease control is the first priority programme within the framework of the general health programme in Indonesia. At present, the 5 major national programmes in communicable disease control are: malaria, improvement of rural sanitation, basic immunization, tuberculosis control and epidemic containment programme. At a lesser extent follow second priority programmes: the leprosy control programme, filariasis, soil transmitted helminths and rabies control.[1]

Leprosy control programme

It was the late Dr Sitanala who, since 1928, pioneered leprosy control by giving treatment to patients outside the leprosaria and colonies. In early 1935 a survey was carried out among the population in Bali and Lombok. It was found that the prevalence rate was 1‰. With the assistance of the WHO and UNICEF leprosy control has been started since 1950, but a systematic leprosy control started in 1956 in several areas in Java and Bali and gradually extended to the other provinces.[2]

There are now 39 leprosy hospitals in Indonesia. All of them are financed by the government, except a few leprosy hospitals/leprosy villages which obtained support from foreign foundations.[3]

The prevalence of leprosy in Indonesia is relatively high, and scattered throughout the many islands with unequal distribution of the cases in each location. A national leprosy control programme within the framework of the communicable disease control programme is being constrained by the limited resources and manpower. Therefore, for the sake of efficiency, the leprosy control programme in Indonesia has been integrated in the health centre system. At present there are more than 3,500 health centres throughout the country.[4]

The present national policy[1] of leprosy control consists of:

1. Case finding through the health centres available in the country.
2. Treatment by the health centres and hospitals on ambulatory basis.
3. Health education.
4. Training of leprosy workers and professional staff.

Epidemiology of leprosy

Data reported by the provincial health authorities to the Ministry of Health show that the highest prevalence of leprosy is in the eastern part of Indonesia with the highest prevalence in Irian Jaya (7.7 per 1,000 population). The total number of leprosy cases registered by the health services throughout Indonesia

Table 1. Leprosy situation in Indonesia (as of 30 June 1977)

No.	Province	Population (in thousands)	Number of cases to be treated				Regularity of Treatment (%)	Prevalence rate (‰)
			I	*T*	*L/B*	Total		
1	D.I. Aceh	2,499	468	1,361	1,290	3,119	72.42	1.24
2	North Sumatera	7,996	202	1,710	1,395	3,307	73.83	0.41
3	West Sumatera	3,249	261	536	526	1,323	62.33	0.40
4	Riau	1,999	53	139	243	435	58.62	0.21
5	Jambi	1,249	26	27	58	111	91.89	0.08
6	South Sumatera	3,998	95	926	491	1,512	61.11	0.37
7	Bengkulu	750	61	282	93	436	61.68	0.58
8	Lampung	3,249	2	63	33	98	89.79	0.03
9	D.K.I. Jakarta Raya	6,458	97	1,061	551	1,709	56.93	0.25
10	West Java	24,185	596	5,167	1,999	7,762	78.82	0.32
11	Central Java	24,992	54	4,663	1,067	5,784	47.06	0.23
12	D.I. Yogyakarta	2,419	39	125	71	235	43.40	0.09
13	East Java	29,022	2,234	14,137	8,234	24,605	68.64	0.84
14	Bali	2,431	33	446	578	1,057	87.79	0.44
15	West Nusa Tenggara	2,532	300	913	887	2,100	83.29	0.82
16	East Nusa Tenggara	2,734	2,707	7,110	2,526	12,343	67.13	4.51
17	West Kalimantan	2,383	169	622	470	1,261	71.46	0.52
18	Central Kalimantan	827	52	246	112	410	69.59	0.49
19	South Kalimantan	2,000	447	1,531	1,463	3,441	71.46	1.72
20	East Kalimantan	869	120	466	545	1,131	82.31	1.30
21	North Sulawesi	2,014	292	1,232	1,082	2,606	75.05	1.29
22	Central Sulawesi	1,108	247	646	537	1,430	44.61	1.29
23	South Sulawesi	6,142	379	9,553	6,728	16,660	52.44	2.53
24	South East Sulawesi	806	652	1,151	677	2,430	75.96	3.07
25	Maluku	1,317	209	2,870	2,203	5,282	79.52	4.01
26	Irian Jaya	1,087	11	6,358	1,811	8,180	17.20	7.52
	Total	138,325	9,809	63,341	35,670	108,817	64.13	0.78

Source: Louhenapessy.[5]

are 108,817. The *L* proportion is 33.16%. Male and female ratio among the cases is 2.3 : 1.0. The child proportion is 13.4%.[5] Only 64.13% of the cases are having regular treatment.[1] (See Table 1.)

Between 1975 and 1977, random surveys have been carried out by Dr B Zuiderhoek, WHO leprologist, in the eastern part of Indonesia, in the provinces of South Sulawesi, Maluku and Bali. The results throw a new light on the local leprosy situation.[6] (See Figure 1.)

Figure 1. Indonesia (scale 1 : 30,000,000): (1) Jakarta, (2) Bali, (3) South Sulawesi, (4) Maluku.

In the province of Bali, 3 out of 8 regencies were surveyed among the 903,000 population (37% of the total population of the island of Bali). New cases have been found in the survey: 9 active *T*, 1 active *L* (adult patients). The prevalence is 0.8 per thousand while in 1957 it was 2.4 per thousand. It is concluded that after a successful campaign of 20 years, leprosy is not a problem any more in this island.[6]

In the Maluku province, 9 out of 20 sub-districts were surveyed among 12,960 population. Registered cases found in the survey were 19 *T*, 14 *L*. Newly detected cases: 1 *I*, 74 *T* and 6 *L*. The total is 1 *I*, 93 *T* and 20 *L*. Before the survey started the prevalence was 2.6 per thousand, and after the survey 9.1 per thousand.

The result showed that although leprosy is found spread over the whole area, it occurs in pockets.

In the South Sulawesi province, 10 regencies showed registered cases (6 *T*, 18 *L*) and newly detected cases (2 *I*, 232 *T* and 33 *L*). In the South Sulawesi province with 3.5 million population, the known prevalence was 1–2 per thousand.

Statistically analysed, the true prevalence appears to be at least 10 per thousand, most probably higher.

Treatment

Ninety per cent of the leprosy patients were treated with Dapsone. Thiambutosine is used when the patient is allergic to Dapsone. Clofamizine (Lamprene) is used only for leprosy reaction. Rifampicin is not used in the leprosy control programme, except in hospitals and private practice. Chloroquin tablet 100 mg, 3× a day for 3 days, is given for light leprosy reaction.

Data on resistance to Dapsone is not well-documented because of the lack of laboratory facilities.

In view of the therapeutic difficulties in leprosy, together with the limited number of drugs available and the practical problems of distributing them regularly and for long enough to a significant percentage of those with the disease, many will consider that the facts call for even further impetus towards the development of a vaccine.[7]

References

1 Bahrawi W. The role of voluntary organizations in leprosy control with special reference to the Indonesian situation. *The First International Workshop on Leprosy Control in Asia.* Jakarta, *Nov. 28–Dec. 1, 1977.* Sasakawa Memorial Health Foundation, p. 15.

2 Louhenapessy AA. Community participation in assisting leprosy control in Indonesia. *The Second International Workshop on Leprosy Control in Asia.* Katmandu, Nepal, *Oct. 11–14, 1979.* Sasakawa Memorial Health Foundation, pp. 53–60.

3 Louhenapessy AA. *Review penyakit kusta di Indonesia. Rapat Konsultasi Kusta Nasional. Ciloto. Jan. 14–16, 1980* (mimeograph).

4 Siwabessy GA. Inauguration Address. *The First International Workshop on Leprosy Control in Asia.* Jakarta, *Nov. 28–Dec. 1, 1977.* Sasakawa Memorial Health Foundation, p. 15.

5 Louhenapessy AA. Leprosy control in Indonesia. *The First International Workshop on Leprosy Control in Asia.* Jakarta, *Nov. 28–Dec. 1, 1977.* Sasakawa Memorial Health Foundation, pp. 24–39.

6 Zuiderhoek B. The result of random surveys in South Sulawesi, Maluku and Bali provinces and its consequences for the leprosy control campaign in Indonesia. *The First International Workshop on Leprosy Control in Asia.* Jakarta, *Nov. 28–Dec. 1, 1977.* Sasakawa Memorial Health Foundation, pp. 72–81.

7 McDougall AC. Leprosy control in Ethiopia. Editorial. *Lepr Rev*, 1979, **50**, 181–182.

Lepr Rev (1981) **52**, 161–170

Basic nerve function assessment in leprosy patients

W BRANDSMA
Physiotherapist, All Africa Leprosy and Rehabilitation Training Centre, PO Box 165, Addis Ababa, Ethiopia

Received for publication 31 March 1980

Summary Nerve function assessment is important in the prevention of deformities in leprosy patients. Simple nerve function tests are presented which will make it easy for the leprosy worker to make records of nerve damage and will enable him to evaluate changes in nerve function.

Introduction

Deformities and disabilities in leprosy patients are due to nerve damage. Without this complication leprosy would not be the debilitating disease that it is for many of its sufferers. The following factors are often responsible for permanent nerve damage in leprosy patients:

1. Delay in reporting for diagnosis and anti-leprosy treatment.
2. An irregular or interrupted treatment.
3. Inadequacy of medical treatment, e.g. drug resistance, chronic ENL and release from control when the patient should have continued treatment.
4. Undetected early 'neuritis'.

Many patients first report for treatment when nerves are already irreversibly damaged. This is mainly due to the fact that in those countries where leprosy is endemic, basic health service facilities are not well developed and knowledge about the disease is deficient.

However, all too often patients in a leprosy control programme develop unrecognized nerve damage because nerve function assessments are not performed regularly.

Early recognition of decreasing nerve function and properly instituted medical treatment (corticosteroids) will usually prevent permanent nerve damage in most leprosy patients.

The medical officer or field worker may have told the patient to report any

0305–7518/81/052161 + 10 $01.00

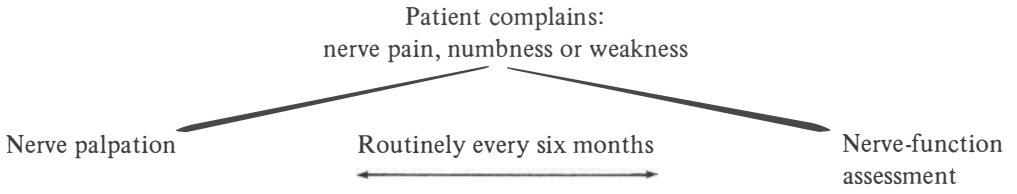


Figure 1. Nerve function should be assessed whenever the patient complains about pain, numbness or weakness and every six months to detect early nerve function loss.

nerve discomfort and weakness or numbness. He may also have established a routine of regular nerve palpation. But if nerve functions are not tested many patients may develop skin dryness, anaesthesia and paralysis unnoticed (Figure 1). It is generally accepted that nerve damage may sometimes occur without the patient being aware of it and in the absence of the classical signs of nerve inflammation. In our experience the basic nerve function assessment described here requires no more than 2–4 min.

When should nerve functions be assessed?

1. All new patients. A baseline is needed against which possible future changes can be assessed.
2. All patients with 'neuritis'. In the All Africa Leprosy and Rehabilitation Training Centre (ALERT) the assessments are usually repeated every 2–3 weeks to monitor the effect of the treatment.
3. Every time the patient reports nerve pain, weakness or numbness.
4. Routinely every 6 months for all patients to detect early nerve damage.

Muscle testing and sensory testing are reliable tests in the assessment and evaluation of nerve functions.

Muscle testing

The grading of muscle power is adapted from the grading system recommended by the Medical Research Council.[1]

Grade 5. Full range of movement of the joint on which the muscle or muscle group is acting. Normal resistance can be given.
Grade 4. Full range of movement but less than normal resistance.
Grade 3. Full range of movement but no resistance.
Grade 2. Partial range of movement with no resistance.
Grade 1. Perceptable contraction of the muscle(s) not resulting in joint movement.
Grade 0. Complete paralysis.

It is not enough to ask the patient to perform certain movements in order to assess the motor functions of a nerve. The examiner should always test for muscle resistance in patients with normal joint movements. This is particularly important as weakness, indicating early nerve damage, cannot be seen in most patients and should therefore be tested. The leprosy worker will soon get the feeling of what muscle power can be regarded as normal for the movements described below when he performs the tests on a number of people without nerve damage. The examiner should first demonstrate the correct movement for the patient and when the patient is able to perform the demonstrated movement resistance should be applied.

Test 1. *Facial nerve* (Figure 3)
The patient is asked to close his eyes tightly. The examiner tries to pull the eyelids apart when the patient is able to close his eyes. Weakness if present, will then be revealed. (Full range of movement = complete eye closure.)

Test 2. *Ulnar nerve* (Figure 4)
The patient is asked to move his little finger straight out and a little up (abduction). Resistance is applied at the base of the finger if this movement can be completed. With the supporting hand the examiner will be able to feel the contraction of the little-finger muscles.

Test 3. *Median nerve* (Figure 5)
The patient moves his thumb away from the palm of the hand at right angles (abduction). Resistance to this movement is applied perpendicular to the palm of the hand at the base of the thumb.

Test 4. *Lateral popliteal nerve*, deep branch (Figure 6)
The patient is asked to lift his foot (dorsiflexion). Resistance is applied by trying to push the foot down.

Testing these four movements routinely would be sufficient to detect the beginning of nerve damage to the motor fibres in most patients. These four tests are also mentioned in a paper by Ross and Pearson[2] but the importance of giving resistance to the movements is not emphasized. For the leprosy field worker it will be sufficient to use three grades of muscle strength only as illustrated in Figure 7. He should refer the patient that had normal muscle strength on a previous examination. For more detail, especially in the follow-up of patients with neuritis, one could in addition test the following movements:

Test 5. *Ulnar* (*Median*). Abduction of the index finger (Figure 8).
The patient is asked to move his index finger away from the long finger, while the index finger is slightly bent at the knuckle joint. Resistance is applied at the base of the finger after completion of this movement. The muscle responsible for this movement is very superficial and the contraction of this muscle can therefore be felt.

ALERT

PHYSIOTHERAPY DEPARTMENT (MUSCLE and SENSORY TESTING CHART)

Name: H. M. No: LFO 34

MUSCLE TESTING

Right								Left						
11-3 80	28/2 80	11-2 80	28-1 80	15-1 80	24/10 77	29/6 77	Date	29/6 77	24/10 77	15-1 80	28-1 80	11-2 80	28-2 80	11-3 80
							FACIAL							
4^+	4	4	4	4	5	5^-	Eyeclosure	5^-	5	4	3	3	4	4^+
							ULNAR							
2^+	2	0	0	0	5	3^+	Abd. 5th finger	2^+	5	2	?	2	3^-	3^-
							ULNAR/MEDIAN							
5^-	4	3	2	1	5	5	Abd. index finger	4^-	5	2	2	2^+	3^+	3
							MEDIAN							
5	5	5	3	3	5	5	Abduction thumb	5	5	0	2	4	5	5
5	5	5	3^+	3	5	5	Opposition thumb	5	5	0	2^+	3^+	4	5
							RADIAL							
5	5	5	5	5	5	5	Wrist extension	5	5	5	5	5	5	5

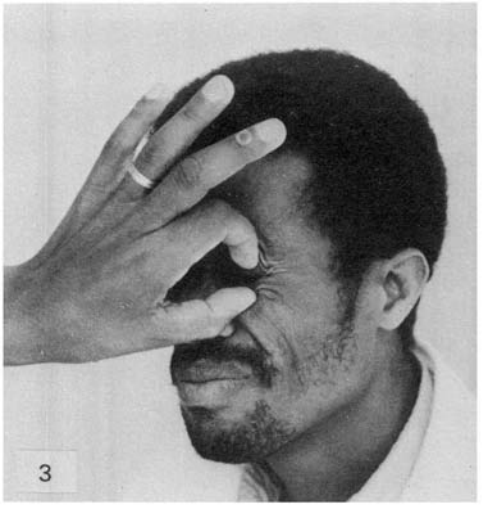
LAT. POPL.

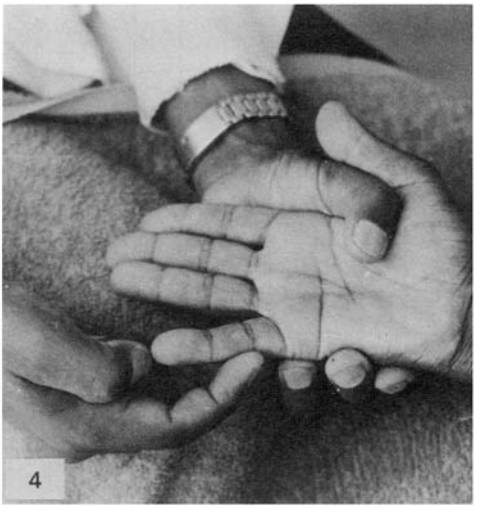
5	5	5	4	3^+	5	5	Dorsiflex. foot	5	5	4	4	5	5	5
5	5	5	5	5	5	5	Eversion foot	5	5	4	4	5	5	5
SZ	MK	MK	MK	SZ	SZ	LMH	Sign.	LMH	SZ	SZ	MK	MK	MK	SZ

First assessment weakness/paralysis in red. Follow-up assessments only deterioration in red!!

Comments:

Figure 2. Nerve-function assessment form as used in the All Africa Leprosy and Rehabilitation Training Centre (front page is shown). Outlines of hands and feet are printed on the backpage for sensory evaluation.
This patient reported for diagnosis and treatment on 29 June 1977 with bilateral ulnar weakness of two-months duration.
The patient was admitted to the hospital for treatment with corticosteroids. He recovered sensation and muscle strength. See muscle assessment of 24 October 1977. (Intermediate assessments are not shown.)
The patient then went home to the countryside for further treatment in his local clinic. He stopped his treatment and returned back to the hospital in January 1980 with new nerve damage of four-months duration.
Again the patient was admitted for treatment with corticosteroids.
The patient is recovering well as shown by serial muscle testings.





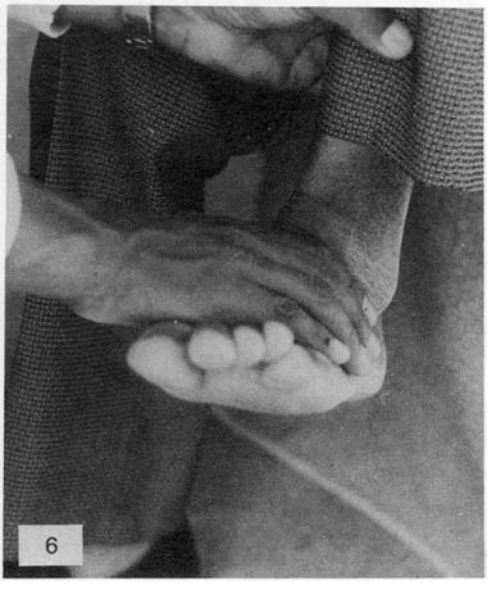


Figure 3. Test to detect early loss of motor function of the facial nerve.
Figure 4. Motor function test for ulnar nerve.
Figure 5. Motor function test for median nerve.
Figure 6. Motor function test for lateral popliteal nerve (deep branch).

Test 6. *Median*. Opposition of the thumb (Figure 9).
The patient is asked to move the thumb away from the palm of the hand with the pulp of the thumb facing the other fingers. Resistance is applied on the inside of the thumb at right angles to the resistance given in Test 3 by trying to push the thumb back.

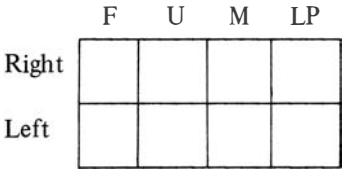


Figure 7. Testing of only the four basic movements (Tests 1–4).
This 'block' could be stamped or printed in the patient's record. A stamp can also be made with an outline of hands and feet for sensory testing. Simple grading: N, normal; W, weakness; P, paralysis.

Test 7. *Radial nerve.* Wrist extension (Figure 10).
The patient moves his clenched fist up. The examiner applies resistance trying to push the wrist down. Radial nerve (motor function) damage is not very common in Ethiopia and rarely occurs without associated ulnar nerve and median nerve damage.

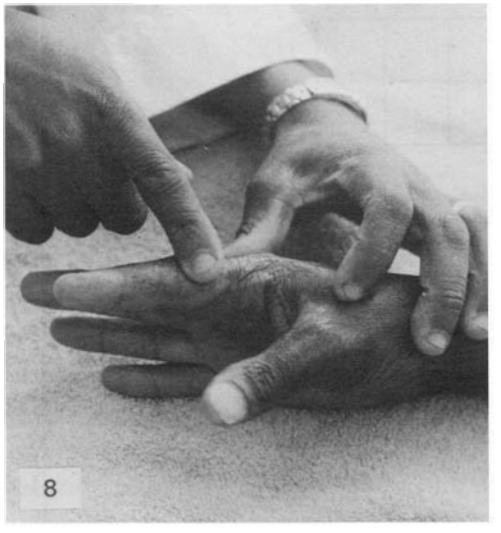
Test 8. *Lateral popliteal nerve*, superficial branch (Figure 11).
The patient moves his feet outwards (eversion) and at the end of this movement the examiner applies the resistance to the outside of the foot trying to move the foot inwards. Isolated weakness of the muscles that move the foot out is rare. When this movement is weak then there is also usually weakness of the muscles that lift the foot.

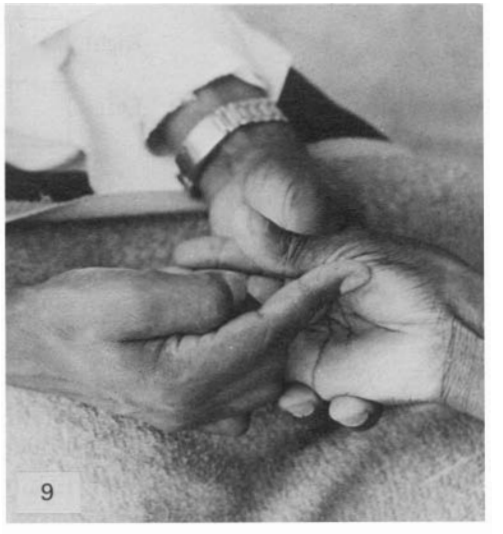
Sensory testing

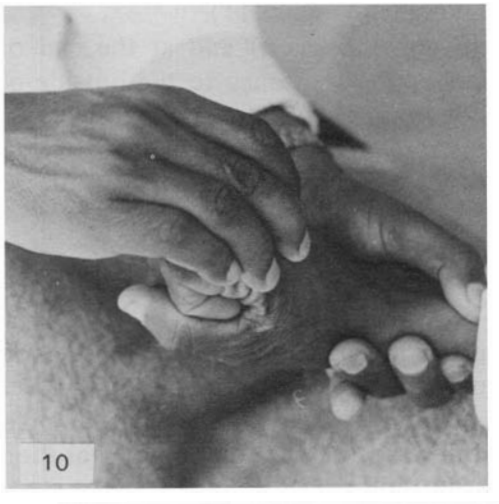
In many leprosy control projects and leprosy hospitals in Africa it is common practice to test sensation of the palm of the hands and the sole of the feet with a ball-point pen. The stimulus is applied to the skin gently and mild pressure is given just denting the skin. A more accurate evaluation of sensation is possible when the stimulus to the skin is not influenced so much by the force of application.

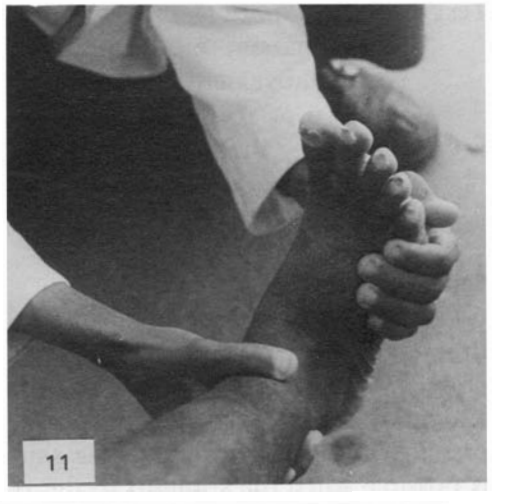
Naafs and Dagne[3] described a method of sensory testing in leprosy patients using standard monofilament nylon threads. The nylon thread is fixed to a handle leaving 1 inch of the thread free. The stimulus is then applied to the skin until the thread just bends, thus giving a standard pressure (Figure 12). Quantification of changes in sensory function is possible by using a set of nylon threads of different diameters. The stimuli are applied a certain number of times in circumscribed areas, e.g. the ball of the thumb for the median nerve. The number of stimuli felt are then noted (Figure 13).

Corneal sensation should be tested when there is no blink or insufficient eye closure. A wisp of clean cotton wool is rolled to a point and the margin of the cornea is tested for sensation.









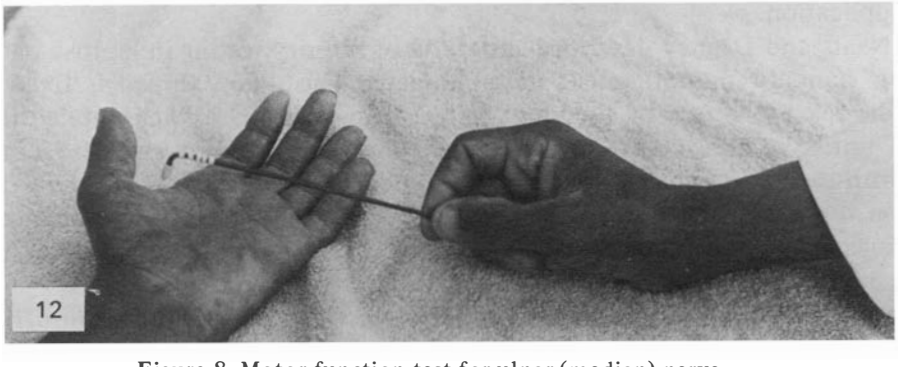


Figure 8. Motor function test for ulnar (median) nerve.
Figure 9. Motor function test for median nerve.
Figure 10. Motor function test for radial nerve.
Figure 11. Motor function test for lateral popliteal nerve (superficial branch).
Figure 12. Sensory testing using 'bristles'.

	No. of bristles				
	2	3	4	5	6
Right ulnar	0	1	1	1	1
R. median	3	5	5	5	5
Left ulnar					0
L. median	1	2	2	4	4

Figure 13. Sensory testing using nylon threads. Thenar and hypothenar area were tested five times each with every bristle. The number in the boxes indicate the number of times that the bristle was felt. Sensory loss of both ulnar nerves is shown. Almost normal sensation in right median distributed area, but diminished sensation in left median area. This is the same patient as in Figure 2, tested on 11 March 1980.

Discussion

Disability grading of the leprosy patients is done in most leprosy control projects. However, it would benefit the leprosy control programme and the patients more if regular nerve function assessments were also done. Disability grading is not a substitute for nerve function assessment. Using standard WHO grading the hand of a leprosy patient may be graded 1 on the first examination because of loss of sensation due to ulnar nerve damage. On a follow-up visit the patient may have also developed median loss of sensation but his disability will still be graded 1. Furthermore, weakness is not given a grade on the disability scale. It is not necessary to assess all the muscles that are supplied by the nerves that can become damaged in leprosy. The pathology is within the nerve and it would be very unlikely if the motor fibres of only one of the muscles, innervated by that nerve, were damaged. All muscles innervated by one nerve will be more or less weak to the same extent. The testing of one or two movements will therefore give sufficient information about nerve damage and changes in nerve function.

The Voluntary Muscle Test as described by Goodwin[4] has the following disadvantages:

1. Difficult latin names would have to be taught to all the leprosy workers.
2. The test is time consuming as many muscles have to be assessed.
3. Electromyographic examinations by Forrest and Basmajian[5] showed that most of the intrinsic muscles of the hand cannot be tested in isolation.

When we test intrinsic muscles of the hand, muscle groups rather than individual muscles are tested.

Thumb abduction might sometimes be weak in an isolated ulnar palsy due to the innervation pattern of the muscle flexor pollicis brevis and the abduction component of this muscle. Lumbrical muscles also cannot easily be tested in

isolation. Both lumbricals and interossei stabilize the fingers and will prevent the fingers from clawing.

Here again, the ability to take and maintain the lumbrical position against resistance in an isolated ulnar palsy will reveal weakness in the index and long fingers. Most patients with an ulnar palsy only will eventually develop four-finger clawing. We cannot always conclude that there is median nerve damage when muscle testing reveals slight weakness of thumb abduction and weakness or even complete loss of lumbrical position in the index and long fingers.

More muscles would have to be tested for the irregular types of nerve damage, e.g. high median nerve damage. An experienced assessor could give more detail to the grading by adding a $\frac{1}{2}$ or + or − sign to the whole number (Figure 2). The muscle tests presented here are easy to perform and should be known by all leprosy workers.

Acknowledgements

I would like to thank Miss J M Watson MCPT for introducing me to the assessment of nerve function in leprosy patients.

References

[1] Medical Research Council War Memorandum. *Aids to the Investigation of Peripheral Nerve Injuries.* (Memo. no. 7), 2nd ed. London: HMSO, 1962.

[2] Ross WF, Pearson JMH. The recognition and management of nerve damage under field conditions. *Lepr Rev*, 1975, **46**, 231–4.

[3] Naafs B, Tamru Dagne. Sensory testing: a sensitive method in the follow up of nerve involvement. *Int J Lepr*, 1977, **45**, 364–8.

[4] Goodwin CS. The use of the voluntary muscle test in leprosy neuritis. *Lepr Rev*, 1968, **39**, 209–16.

[5] Forrest WJ, Basmajian JV. Functions of human thenar and hypothenar muscles. *J Bone Joint Surg*, 1965, **47A**, 1585–94.

Lepr Rev (1981) **52**, 171–173

Leprosy and syphilis: a case report

J K NSIBAMBI
Training Unit, All Africa Leprosy and Rehabilitation Training Centre (ALERT), PO Box 165, Addis Ababa, Ethiopia

Received for publication 1 October 1980

This is a report on a leprosy patient who was referred from a provincial leprosy clinic to a leprosy hospital because of skin lesions that could not be diagnosed at the clinic. The patient was found to be suffering from secondary syphilis as well as leprosy. Comments are made on the findings that could be common to both leprosy and secondary syphilis.

A 22-year-old unmarried man was referred to All Africa Leprosy and Rehabilitation Training Centre (ALERT), Addis Ababa, because of skin lesions on the scrotum (Figure 1). At the referring clinic, a diagnosis of leprosy and a classification of 'lepromatous' had been made. The patient was on DDS and had received treatment for 3 months.

History taken at ALERT revealed that the patient had noticed skin patches of leprosy for 1 year. Three months prior to coming to us he had observed lesions on the scrotum. His parents were dead and he was living with his uncle. There was no history of contact with leprosy. He admitted to sexual intercourse before developing the scrotal lesions.

On examination the patient was found to have a thickened right ear and papulo-squamous lesions around the nose and between the lower lip and the chin (Figure 2). There were ill-defined hypopigmented macular lesions on the face, on the upper arms and on the buttocks. Sensation was intact in these lesions. Erythematous lesions were seen on the palms. The cervical and inguinal lymph nodes were enlarged. Of the nerves, the great auricular, the ulnar and the radial cutaneous were bilaterally enlarged and they were not tender.

On the scrotum and in the anal area foul-smelling lesions of condylomata lata were observed. Sensory testing revealed anaesthesia of the hands and feet.

Laboratory investigation: the bacillary index (BI) was 3.0 (3+, 2+, 2+, 5+, 3+, 2+), morphological index (MI) was 0, and VDRL reaction ++++ (the

0305–7518/81/052171 + 03 $01.00

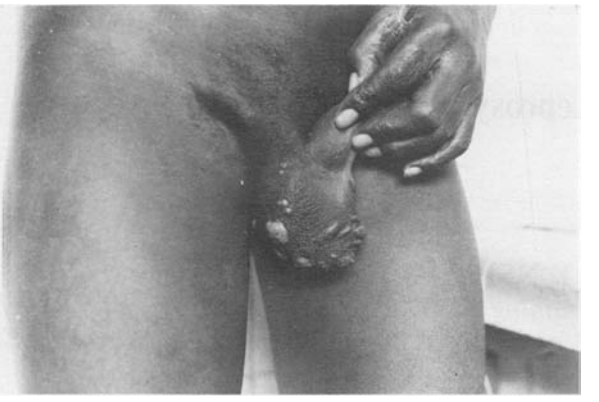


Figure 1

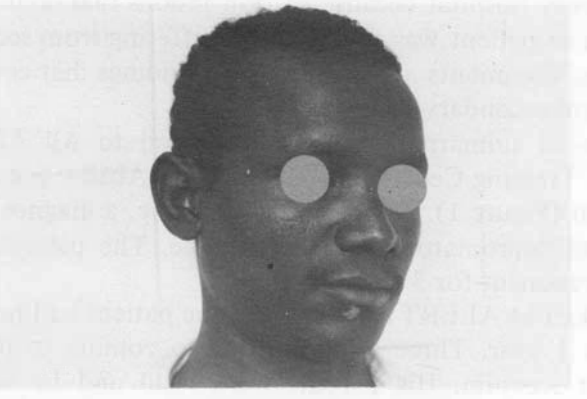


Figure 2

strongest recorded in our laboratory). Dark ground illumination examination and other serological tests for syphilis could not be done at our hospital.

A diagnosis of BL leprosy and secondary syphilis was made.

Treatment included a course of procaine penicillin and DDS and later a course of Rifampicin with DDS.

The patient showed very remarkable improvement on penicillin, all syphilitic lesions disappearing after about 3 weeks.

Comments

Syphilis, and secondary syphilis in particular, has been known to be a great imitator of other diseases. The following manifestations can be found in secondary syphilis:

(a) *Leucoderma*, i.e. hypopigmentation of the skin. This could be confused with hypopigmentation in tuberculoid, borderline or lepromatous leprosy.
(b) *Annular lesions* could be confused with similar lesions in tuberculoid or borderline leprosy.
(c) *Papular lesions* can look like papules of lepromatous leprosy or BL.
(d) *Joint pains with fever and malaise.* These are also common manifestations in leprosy patients especially during reactions.
(e) *Uveitis.* Occurs in lepromatous leprosy as well.
(f) *Hoarseness of the voice.* Also found in cases of lepromatous leprosy.
(g) *Lymph node enlargement.* Is a common finding in leprosy especially during reaction.
(h) *Positive reagin serological tests for syphilis.* Lepromatous leprosy is one of the causes of biological false positive tests for syphilis.

Leprosy patients may be exposed to syphilis just like other people in any community and we should be alert to the possibility of confusing syphilitic manifestations with those of leprosy.

Acknowledgements

I would like to thank Dr Tom Kollstrom of the Armauer Hansen Research Institute for taking the photographs and the staff of ALERT who carried out the laboratory tests.

Lepr Rev (1981) **52**, 175–179

Leprosy and the Community

INTERNATIONAL YEAR OF DISABLED PERSONS, 1981

The following is an extract from WHO Press Release of 5 January 1981:

> In the world today, there are about 450 million disabled persons despite the fact that 50% of disabilities can be prevented.
>
> This sad situation becomes even more dramatic when we realize that at present nearly 90% of all the resources to help the disabled are expended in the industrialized countries, while 80% of the world's disabled live in the developing countries.
>
> The International Year of Disabled Persons, 1981, will be a year of hope for millions of the disabled. They look to the coming year as the beginning of a period in which there will be a true awakening to the need for their full participation and integration within society, and to a further development of rehabilitation care, especially for those who now have little or no access to the help they require. For the one person in ten in our world who suffers from a physical or mental impairment, this is an occasion on which to expect that society will adopt new and more positive attitudes towards its disabled citizens.
>
> But 1981 can also be a year of great significance for those untouched by disablement – as we all live in the shadow of a temporary or permanent disability. We have within our grasp the technical and financial means to prevent many of the situations which daily add to the heavy toll of impairment. This is particularly true for children. The number of disabled children now totals about 140 million, 80% living in developing countries. Let 1981 be the year for aggressive international and national actions to reduce the magnitude of preventable disabilities.
>
> Emphasis on prevention will be placed on actions by and within the community, through the process of primary health care. Programmes to reduce the impact of existing disabilities also must be fully integrated within existing services. Many rehabilitation procedures for the physically and mentally disabled can be carried out by families and others in the community. WHO and UNICEF will strongly support such 'health by the people' approaches. Family and community-centred support and assistance is equally important in those industrialized societies where many specialized services are available.

PRIMARY HEALTH CARE SYMPOSIUM No. 3, THERAPEUTIC ASPECTS: THE DEPARTMENT OF INTERNATIONAL COMMUNITY HEALTH, LIVERPOOL SCHOOL OF TROPICAL MEDICINE, PEMBROKE PLACE, LIVERPOOL, L3 5QA: 13–16 April 1982

Dr David Stevenson of the above department has kindly submitted the following information on this symposium, originally planned for this year but now to be held in 1982:

> This international symposium, to cover therapeutic aspects of primary health care, was planned for 27 September to 1 October 1981. A number of prospective sponsors

0305–7518/81/052175 + 05 $01.00

for participants have regretted that their funds are fully committed for 1981. One international organization has indicated that support would be likely if the symposium could be held in 1982.

In view of this, it has been agreed to hold the symposium in April 1982. It is expected that participants will arrive on Tuesday 13 April 1982 (Easter Sunday is 11 April 1982). Accommodation has been booked in a well-appointed University Residence, for the expected number of participants from outside Liverpool, for the nights of 13, 14 and 15 April. Meetings will be held on 14, 15 and 16 of April with departure on the afternoon or evening of Friday 16 April. Meals and meetings will be either in the Halls of Residence or in the School of Tropical Medicine, and provision will be made for drug company displays.

The cost of accommodation and meals for each participant for the duration of the symposium is estimated at about £60.00.

It is expected that proceedings will be published. Speakers will be asked to provide texts of their contributions.

Subjects which it is hoped to cover include:

How appropriate lists of drugs to cover the principal needs of health care units and practitioners in developing (and developed) countries may be devised.

How supplies of listed drugs can be maintained at prices which can be afforded by the countries and communities concerned.

The places of imported and locally made drugs, intravenous fluids, dressings and medical equipment, under varying circumstances.

The benefits and dangers of improvisation and local initiative.

Co-operation and conflict between different systems of therapy in one community or health care system – Allopathic (Western), Ayurvedic, Homeopathic, Traditional.

Many expressions of interest and of intention to attend the symposium have been received. It is hoped that the later date will not deter these, and that more offers of papers and suggestions of speakers or participants will be received.

Any inconvenience from the change of date is deeply regretted – the intention is to provide a better symposium.

David Stevenson, MD
Department of International Community Health
Liverpool School of Tropical Medicine
Pembroke Place, Liverpool L3 5QA England

January 1981

GERMAN LEPROSY RELIEF ASSOCIATION: REPORT FOR THE YEAR 1979

We congratulate DAHW and Dr Horst Frank on the production of this excellent report (it is in German), outlining the extensive work of this organization during 1979. On page 7 it is remarkable to note the sustained increase in spending from 1957 to 1979; from a tiny figure at the outset, it is now in the region of 27 million DM. Pages 20, 21 and 22 give maps of the extent of the work in Africa, Latin America and Asia. We congratulate our colleagues in Germany on achievements during 1979 and look forward to receiving further news of activities in the report for 1980.

SALUBRITAS: THE AMERICAN PUBLIC HEALTH ASSOCIATION, WASHINGTON, DC

David Morley in TALC, London, has also kindly directed information on this newsletter for health information exchange to our office. To quote from volume 4, July 1980:

> SALUBRITAS is published quarterly in English, French and Spanish by the International Health Programs of the American Public Health Association (APHA) and the World Federation of Public Health Associations (WFPHA). SALUBRITAS is funded by the United States Agency for International Development (USAID) and distributed free upon request to individuals and organizations delivering public health services in developing countries.
>
> APHA is a non-governmental, professional society. Through its International Health Programs, it provides professional and technical assistance. WFPHA, a federation of national public health associations, works closely with the World Health Organization (WHO).
>
> The views and interpretations in SALUBRITAS should not be attributed to the United States Agency for International Development (USAID), APHA, WFPHA, or to any individual action on their behalf.
>
> Articles from SALUBRITAS may be quoted or reprinted without permission, provided that credit is given to SALUBRITAS and that a copy of the publication is sent to the editor.
>
> Editor: Ina Lee Selden
> *American Public Health Association*
> *International Health Programs*
> *Director: Susi Kessler, MD*
> *1015 Fifteenth Street, NW*
> *Washington, DC 20005, USA*
> *(202) 789-5679*

This issue also contains an article of particular interest concerning the use of simple flip charts for use at village level, based on black and white photographs 40 x 46 cm, together with lesson books, all based on observations made in the area of work. The article ends with a note that a small booklet is being produced by TALC in London (30 Guilford Street, London WC1N 1EH) on the construction of flip charts for this type of work. Contributions to SALUBRITAS are welcomed; the sum of $25 is paid for an article and $10 for suggestions on appropriate technology, or other material.

WORLD NEIGHBORS; OVERSEAS DEVELOPMENT MATERIALS; 5116 NORTH PORTLAND AVENUE, OKLAHOMA CITY, OKLA 73112, USA

TALC in London have drawn our attention to yet another organization which is obviously very active in the field of 'Materials for Person-to-Person Education in Health, Family Planning, Agriculture and Community Development'. Their price list and order form (address above) gives detailed information on filmstrips, booklets, books, flipcharts and newsletters. The slant is perhaps mainly community and village development together with agriculture, but it is apparent, if only from the illustrations, that this agency has expertise in the field of communication which may be of considerable value to those attempting to produce similar health learning materials for diseases, including leprosy. Sharon Dowell, the Publications Correspondent of World Neighbors, also included a copy of their quarterly newsletter *Soundings from Around the World* which gives up-to-date information on more recent publications in the field of basic health and community services. Two of these, both training manuals for village health promoters, will be reviewed in detail in a subsequent

number of *Leprosy Review*, dealing with leprosy and primary health care; they have been produced by Project Concern International, PO Box 81122, San Diego, California 92138, USA. (The extent and quality of the material described in this one communication, from one agency, unknown to many people in the UK and elsewhere, prompts one to ask if a much more determined and systematic effort should not be made by those working in leprosy to centralize and sift all such publications, for use by appropriate personnel in the field.)

EUROPEAN SOCIETY OF MYCOBACTERIOLOGISTS. NEWSLETTER No. 1, July 1980

Dr P A Jenkins of the Public Health Laboratory, Mycobacterium Reference Unit, University Hospital of South Wales, Heath Park, Cardiff CF4 4XW, has kindly written with news of the inaugural meeting of the Society, in June 1980. The ESM has so far been more concerned with laboratories which offer a clinical service, but those whose interest is research orientated are more than welcome to participate. A recent publicity statement ran as follows:

> The inaugural meeting of the European Society of Mycobacteriologists (ESM) was held at the Forschungsinstitut, Borstel on 5 June 1980. Representatives from Belgium, Denmark, Finland, France, Germany, Italy, The Netherlands, Sweden and the United Kingdom were present. Papers were presented by Dr Herman Kolbel on 'Potential growth cycles in mycobacteria – a new concept' and Dr John Grange on 'Research activities in mycobacteriology in progress'. The afternoon session was devoted to a round table discussion of the primary objectives of the Society. Suggestions for a variety of co-operative studies were made including one on drug sensitivity tests; the evaluation of culture media; storage of culture collection data; documentation of unusual mycobacterial infections; and, most important, an attempt to produce a manual of public health diagnostic methods for mycobacterial infections. The latter would incorporate the wide variety of techniques in use for the primary culture and subsequent identification and sensitivity testing of mycobacteria.
>
> The next meeting of the Society will be in Florence in June 1981 and is open to anyone interested in problems associated with mycobacteria. Details are available from Dr H David, Chef de Laboratoire, Institut Pasteur de Paris, 25 rue du Docteur Roux, 75015 Paris, France. A newsletter will be produced at intervals and is available from Dr P A Jenkins at the above address. A list of reference facilities was produced by Dr J E M Whitehead, Public Health Laboratory Service, 61 Colindale Avenue, London NW9 5EQ., following the first meeting of the Heads of European Public Health Microbiology Services, and the following appear under the heading 'Tuberculosis: other mycobacteria':
>
> Professor V Bonifas, Université de Lausanne, Institut de Microbiologie, 44 rue du Bugnon, CH-1000 Lausanne, Switzerland. (Tel. 021 22 33 91.)
>
> K Bunch-Christensen and A Ladefoged, BCG Department, Statens Seruminstitut, Amager Boulevard 80, DK2300 – Copenhagen S, Denmark. (Tel. 01 952817.)
>
> Dr Hugo David, Chef de Laboratoire, Institut Pasteur de Paris, 25 rue du Docteur Roux, 75015 Paris, France. (Tel. Paris 541 5266, Ext. 799.)
>
> Dr H W B Engel, National Institute of Public Health, PO Box 1, Bilthoven, The Netherlands. (Tel. 030 789111.)
>
> Dr P A Jenkins, Mycobacterium Reference Unit, Public Health Laboratory, University Hospital of Wales, Heath Park, Cardiff CF4 4XW, United Kingdom. (Tel. 0222 755944, Ext. 2049.)

Dr M Laidlaw, Director, Mycobacteria Reference Laboratory, Mearnskirk Hospital, Newton Mearns, Glasgow, United Kingdom. (Tel. 041 639 2251.)

Mogens Magnusson, Tuberculin Department, Statens Seruminstitut, Amager Boulevard 80, DK 2300 Copenhagen S, Denmark. (Tel. 01 952817, Ext. 2388.)

Additions to the list or comments about it should be addressed to Dr J Chr Siim, Statens Seruminstitut, Amager Boulevard 80, DK2300 Copenhagen S, Denmark.

LEPRA PRIZE ESSAY COMPETITION FOR REGISTERED MEDICAL STUDENTS IN THE UNITED KINGDOM, 1981: either '*THE* IN VITRO *CULTURE OF THE LEPROSY BACILLUS*' or '*LEPROSY AND PRIMARY HEALTH CARE*'

Since 1972, first in Oxford, then in Birmingham and Edinburgh, LEPRA, the British Leprosy Relief Association, has annually offered prize money of £100 for essays on various aspects of the leprosy problem. In 1977, it was decided to extend the offer to all universities with a medical faculty in the United Kingdom. The response has been encouraging, and the competition is therefore being continued in 1981 with the above alternative titles.

Entry requires neither clinical experience nor original work; essays will be most welcome from students in the junior years of medical study.

Candidates will be expected to summarise published work on either of the above subjects and to review it critically, recording their own comments in the form of a discussion or summary. No credit will be given for the mere reproduction of material which has already been published in books or journals; students should concentrate more on constructive comments and critical appraisal of what has so far been accomplished in the study of these two aspects of leprosy. Each candidate should choose only one of the above subjects.

References should be included as in scientific journals.

Entries should be typed on A4 paper, double-spaced with good margins, and should be of not more than 10,000 words. However length is not important and in previous years prizes have been awarded for essays of only 3,000 words.

The closing date is 31st December 1981. Entries should be submitted to Dr Colin McDougall, MD., F.R.C.P., Briscoe 1 Ward, The Slade Hospital, Headington, Oxford OX3 7JH, and must include the candidate's full name, home address, college (if relevant), university and year of study.

The sum of up to £100 will be awarded at the sole discretion of the judges, either to one candidate, or divided amongst several. If entries are not of sufficient merit, no prize will be awarded, but if there are several of high standard, LEPRA may extend the available prize money accordingly.

LEPRA PRIZE ESSAY COMPETITION, 1980

The winner of the 1980 LEPRA prize essay competition was Miss Janet Price of the Sheffield Medical School, who received a prize of £100 for her entry on '*BCG vaccination in tuberculosis and leprosy*'. We congratulate this medical student on a manuscript of exceptional quality, which is soon to be submitted for possible publication in the medical press.

LEPRA PRIZE ESSAY COMPETITION FOR REGISTERED MEDICAL STUDENTS IN ST JOHN'S MEDICAL COLLEGE, BANGALORE, INDIA, 1981: '*LEPROSY CONTROL IN SOUTH INDIA*'

The conditions of this competition are similar to those for the United Kingdom given above but with its own subject '*Leprosy Control in South India*'.

For complete details please write to The Dean, St John's Medical College, Bangalore, India.

Lepr Rev (1981) **52**, 180–184

Field Workers' Forum

THE USE OF BICYCLES IN OUT-PATIENT TREATMENT IN MALAŴI

J H ELDON, *LEPRA Control Project, Blantyre, Malaŵi.* Received for publication 22 October 1980

When the LEPRA Control Project started in Malaŵi in 1965, an area of the country with a high population density was chosen. The area embraced four principal towns with Blantyre/Limbe, the commercial centre, on the western perimeter. The intention of the Project was to demonstrate that an efficient domiciliary out-patient treatment service could be established using four-wheeled vehicles. Planning envisaged the vehicle, working on a weekly programme, visiting the same point on the same day at the same time every week with unfailing regularity. By this regularity the confidence of the patient was cultivated and the possibility of regular attendance assured. However, by the very nature of an ideal circuit, there was a gap in the middle of the circuit where a patient could not be reached and to overcome this a Clinic Attendant was employed. A bicycle was carried on the vehicle and, at an arranged point, the Clinic Attendant was put down, together with his bicycle, when he would cycle across the diameter of the circuit, treating patients en route, to be picked up at the other side of the circuit. In addition it soon became apparent that there were areas in the Project which could not be covered by four-wheeled vehicles, particularly in the rainy season, and here again Clinic Attendants were used. In this case they operated on their own and were allotted circuits which they covered independently. Weekly circuits were again used and the Clinic Attendants would cycle up to 30 miles during the day.

With the rapid increase in the cost of vehicles, fuel and spare parts the efficient distribution of out-patient treatment became prohibitive and, if efficient control were to be achieved throughout Malaŵi an alternative method of tablet distribution had to be found. The experience gained in the original Project area in the use of Clinic Attendants riding bicycles taking treatment to the patients suggested that this method could be used throughout Malaŵi. Supervision of the Clinic Attendants could be by Leprosy Control Assistants using motor-cycles, preferably of the trail type, which are economical to run, easily manhandled and relatively cheap to buy. Initially, as area by area was opened up, Land Rovers with 'LEPRA' painted on the sides were used to make the people aware of our work whilst attracting patients. Once patients were under treatment and confidence gained the Land Rover circuits were analysed and broken down into bicycle circuits. At the same time Clinic Attendants were appointed and trained in their duties.

Ideally the Clinic Attendant should be chosen from the area in which he would be expected to work. A person with some standing in the locality, with some education and neatness in writing is required. His role is the dispensing of tablets and simple dressings to those patients who have been diagnosed and registered by the Clinic Attendant's immediate supervisor, in Malaŵi, a Leprosy Control Assistant, plus the accurate recording of treatment given. Additional qualities sought in the Clinic Attendant are devotion to duty, a willingness to obey instructions, a certain degree of stamina. They have no specific training in the

0305–7518/81/052180 + 05 $01.00

recognition of leprosy, its complications and treatment, although they are made aware of the necessity to refer any complaints made by the patient to the Leprosy Control Assistant. Supervision of the Clinic Attendant is undertaken by a Leprosy Control Assistant who should have not more than four Clinic Attendants under his control; three is a much more convenient number for this allows the supervisor to have 1 week during the lunar calendar for non-specific duties such as compilation of records, visits to persistent absentees, propaganda work and school surveys.

The appointment of a Clinic Attendant who lives in the area overcomes the necessity for him to find accommodation and he should come from roughly the centre of the area in which he will operate. In Malawi we would not expect the Clinic Attendant to cycle more than 30 miles in a day and his case load should not exceed 400 patients during the week (there are some dealing with up to 600 but it is hoped that this number will reduce through discharges and reorganization of circuits). In order to allow time for bicycle maintenance, collection of drugs and the completing of returns, circuits should be arranged for the 5 days, Monday to Friday, leaving Saturday free for these tasks. When planning bicycle circuits it is important to have the days' runs going near concentrations of patients but the need to travel on motorable roads is not there. In most cases in Malawi where a bicycle can go a motorcycle can also. It is well to study proposed circuits on the map as well as on the ground; the writer was guilty of planning a circuit which rose 1,000 ft in 5 miles, much to the consternation of the Clinic Attendant, and more so to the writer when he accompanied the Clinic Attendant on this journey. After that the circuit was reversed. In some cases it is not necessary for the Clinic Attendants to return to his base at night, for it may be more convenient for him to stay out one night and complete the circuit the next day. So much depends on the terrain, distance and the case load.

The employment of somebody from the area means he is familiar with all the villages to be covered and, if a man of good repute, he will be known to most of the patients he will treat. He will also rapidly know where the patients live making the task of contacting absentees that much easier. We have found in Malawi that an excellent rapport develops between patient and Clinic Attendant thus ensuring a high percentage rate of attendance.

As in most countries the cost of providing bicycles for this type of work has increased considerably but not as much as the cost of four-wheeled vehicles. In Malawi the current cost of a bicycle is in the region of K170 (US $180). This would be of a standard type bicycle with 26 or 28 inch wheels. If a wheel taking a $1\frac{3}{4}$ inch tyre is available, such as is fitted to a messenger-type machine, these are to be preferred for they make riding on sandy tracks that much more comfortable. Carriers on the rear of the bicycles have been avoided for they have been used to carry heavy personal loads thus causing damage to the bicycle. In this connection it has been found, in recent years, that the quality of tubing used in the construction of the bicycles has deteriorated and frequent fractures of the frame, at the handlebar stock, and of the front forks, at the neck of the forks, have occurred.

There should be at least one spare bicycle for every three in daily use thus ensuring continuity of treatment should a breakdown occur.

Each Clinic Attendant is instructed to maintain his bicycle and is issued with a tin of lubricating oil to enable him to oil the moving parts. Inevitably repairs are necessary, however, from time to time and this work has to be undertaken at the Project Headquarters, or if the Clinic Attendant is far away, by the local bicycle repairer. During 1979 one Project spent K800 (US $820) on spares for bicycles used by the twelve Clinic Attendants in that Project. Major items supplied were new frames, handlebars, chains, pedals, saddles, front forks, wheel hubs and tyres and tubes. It is important that Project centres carry a comprehensive stock of the more frequently called for spares and the list above, together with spokes, ball-bearings for wheel hubs and pedal spindle, cotter pins and brake rubbers are essential.

There are disadvantages to this method of taking treatment to the patient, such as the inability of the expert staff to see the patient weekly, the difficulty of conveying the patient to a hospital should hospitalization be necessary, only a limited quantity of drugs being readily available; but these are far outweighed by the advantages. The Clinic Attendant is far more accessible to the patient who, by the case load given to him, is able to see patients far more easily than where the case load is high and the feeling of 'the need to get on' is present. He is able to establish, by this ready contact, a much more personal relationship. The Leprosy Control Assistant, on his motor-cycle, is able to devote more time to the examination of suspected leprosy cases (referred to him by the Clinic Attendant) on his monthly supervisory visit and the review of patients, in addition to the routine checks on the Clinic Attendant's work. Routine slit skin smears can easily be taken every 6 months too. The cost of the service is so much smaller than when using four-wheeled transport; approximately 3.5 tambala (4 US cents) per patient per week, that it must, of necessity, be considered seriously as the method of choice when out-patient control is discussed. Supervision is also relatively cheap for the two-stroke trail type motor-cycles used in Malaŵi are economical in petrol consumption, 80–100 m.p.g., and if standardization of the motor-cycle in use is achieved, spare parts can easily be carried at the Project centres. Again to ensure continuity of supervision, one spare motor-cycle for two in use is a desirable ratio to achieve.

By adopting this method of taking treatment to the patient a high attendance rate is achieved. In Malaŵi, where all the Projects employ Clinic Attendants at the ground level with a pyramid of Leprosy Control Assistants supervising them, who are, in turn supervised by the Project Centre, the attendance rate is over 70%. In addition, when modifications or alternatives to circuits are needed these can be made with the minimum of disturbance to the other adjacent work.

[This valuable contribution from John Eldon could profitably be read in conjunction with a publication in the *International Journal of Health Services* (1978) 8/4 (633–51) entitled 'Alternative forms of transport and their use in the health services of developing countries', by Gish, O and Walker, G of the London School of Hygiene and Tropical Medicine. *Editor.*]

THE CARE OF THE EYE

By Margaret Brand, Chief, Ophthalmology Department, USPHS Hospital, Carville, Louisiana 70721, USA

This 15 page reprint, A4 format, has been issued by *The Star* from Carville and is written by an expert with great experience of leprosy in the eye. Typical of the high standard of educational and instructional material which is presented by *The Star*, this one is full of practical advice, clearly illustrated with diagrams, and provided with an excellent glossary of all the relevant terms. The main subject headings are:

Common problems in Hansen's Disease

Damage to structures neighbouring the eyeball

madarosis; trichiasis; the dry eye; the tear drainage system; dacryocyctitis; and its management.

Eyelid muscles

anatomy of; paralysis of; signs and symptoms of orbicularis weakness; evaluation; and management; management of weakness coincidental with corneal hypesthesia.

Infiltrative lesions of the eyeball

pathology; corneal lesions and pearls; pannus; iris lesions; nodules; examination of eye, technique; management of infiltration.

Inflammation

pathology; course and prognosis; clinical symptoms and signs; differential diagnosis of 'red eye' table; and additional comments on the table; management of inflammation; general measures; mydriatic agents; anti-inflammatory agents; steroids; adverse effects of steroids; cataracts.

Glaucoma secondary to inflammation

estimation of intraocular pressure; management of abnormal pressure in acute inflammation; management of condition secondary to blocked pupil; management of condition secondary to anterior angle block.

Summary

Glossary of terms

A particularly valuable table entitled 'The Red Eye' is 'a guide to locating the site of the problem' – and a reminder that not all conjuctivitis, especially in leprosy endemic areas, is due to leprosy; in many parts of the world, it should in fact be kept in mind that conditions other than leprosy may account for a high percentage of all ocular problems– ophthalmological problems. A careful study of Margaret Brand's monograph will contribute enormously to diagnostic accuracy and the correct management of patients with eye involvement.

WHO FORM FOR RECORDING DISABILITIES FROM LEPROSY

Prompted by the 'Editorial' in this number, together with the original article by Nittin Verma on the eye and the above monograph, we reproduce below, with acknowledgements to WHO and the *Guide to Leprosy Control* (1980), the standard 'Form for Recording Disabilities from Leprosy'.

FORM FOR RECORDING DISABILITIES FROM LEPROSY

Grades	Hand			Foot			Eye			
	Sign	L	R	Sign	L	R	Sign	L	R	Involvement of larynx
Grade 1	Insensitivity			Insensivity			Conjunctivitis			☐ Yes ☐ No
Grade 2	Ulcers and injuries			Trophic ulcer			Lagophthalmos			
	Mobile claw hand			Clawed toes			Iritis or keratitis			Collapse of nose
	Slight absorption			Foot drop			Blurring of vision			☐ Yes ☐ No
				Slight absorption						
Grade 3	Wrist drop			Contracture			Severe loss of vision			
	Stiff joints									Facial paralysis
	Severe absorption			Severe absorption			Blindness			☐ Yes ☐ No
Maximum grade										

Reproduced with acknowledgements to the World Health Organization. This form appears on page 81 of their *Guide to Leprosy Control* (1980).

Readers will see that an entire column is devoted to the eye, with disabilities ranging from conjunctivitis to blindness. In view of the influence which such eye disabilities may have on the disability rate for the country as a whole, taken with the fact that clinical assessment of the eye is more difficult (and less practiced) than for the hand and foot, we would welcome letters and contributions from field workers who have actually used this form and paid attention to the eyes. Is it possible that there is an area of confusion between leprosy and other (unrelated) conditions causing conjuctivitis, iritis, keratitis, blurring of vision, loss of vision and blindness? How disabling, in practice, and in a leprosy endemic area, is blindness in *one* eye only?

Lepr Rev (1981) **52**, 185–192

News and Notes

THE EYE IN LEPROSY (ABSTRACTS FROM THE LITERATURE), SECOND EDITION, 1933–1960 LEONARD WOOD MEMORIAL (AMERICAN LEPROSY FOUNDATION), SCIENTIFIC RESEARCH PROGRAM, 1832 M STREET, NW, WASHINGTON 6, DC, USA

This interesting collection of references, two decades out of date but still valuable, has been on the shelves of the Editorial office, first in London and now in Oxford, for some years. In view of the articles on the eye in leprosy in this number of *Leprosy Review*, it is interesting to turn the pages once again and to read the excellent summaries. The introduction, dated 15 July, 1960, from the Leonard Wood Memorial office in Washington reads as follows:

> *The Eye in Leprosy (Abstracts from the Literature)* was originally prepared in June 1954, and has been revised as part of the Scientific Research Program of the Leonard Wood Memorial. The abstracts have been collected and arranged by the Medical Director, Dr James A Doull, assisted by Miss Delta Derrom, Miss Pamela Millett and Mrs Patricia Scorgie.
>
> The abstracts are intended to be a guide to the publications from which they are derived. No matter how able a reviewer may be, it is impossible in most instances to present satisfactorily an author's method and findings. It is hoped, therefore, that the circulation of the collection will lead to careful study of valuable original papers and to further research on one of the most distressing complications of leprosy.
>
> Among the points of interest and importance that have been emphasized in discussions with the Medical Director are: (1) the peculiar geographic (climatic or racial?), differences in incidence and severity of lepromatous eye lesions; (2) the need for controlled studies of the value of the sulfones and/or cortisone in treating these lesions; and (3) the urgent need for ophthalmological training of leprologists. Earlier and more accurate diagnosis should be the primary goal.
>
> The collection has been revised primarily for use by the participants in the Scientific Meeting on Rehabilitation in Leprosy, scheduled to be held at Vellore, India, 21–29 November 1960, under the sponsorship of the World Health Organization, the International Society for the Welfare of Cripples and the Memorial.
>
> C I Crowther
> *President*

To those with a special interest in leprosy in the eye this collection may be of interest, and also difficult to obtain. We are prepared to loan it to *bona fide* applicants, preferably in the UK; an application for its lodgement with a medical or scientific library could be considered by the Editorial Board.

Editor

0305–7518/81/052185 + 08 $01.00

RECOMMENDED SAFETY REQUIREMENTS FOR THE PREPARATION OF LEPROMIN: A WHO MEMORANDUM

We are grateful to WHO for permission to reprint the following summary from the *Bulletin of the World Health Organization,* **57** (6), 921–3 (1979):

> The need for standardizing the preparation of Lepromin and establishing safety requirements for it was recognized by the Scientific Working Group on the Immunology of Leprosy (IMMLEP) and its Steering Committee in 1978. It has now recommended the preparation of standard integral (Mitsuda-type) Lepromin and, in collaboration with the WHO Biologicals unit, has drafted requirements for its preparation and testing. These direct that the source material should be *Mycobacterium leprae* from biopsy specimens of skin obtained from human (lepromatous) tissues or from the tissues of armadillos infected with *M. leprae*. The procedures to be followed for processing and testing the source material and for the preparation of Lepromin from it are described. Requirements are laid down for the safety testing and labelling of the final product. In future, IMMLEP will consider supporting only those projects involving the use of Lepromin prepared in accordance with these regulations.

1980: THE YEAR OF LEPROSY GLOBAL RELIEF

At the occasion of '1980: The Year of Leprosy Global Relief', created by Mr Ryoichi Sasakawa, a session on 'Health and Peace' was organized in Tokyo on 25 September 1980, under the joint sponsorship of the Sasakawa Memorial Health Foundation, Leprosy Relief Conference, and the Japan Science Society. Guest speakers included Dr Halfdan Mahler, Director-General of WHO, and Professor M F Lechat, President of ILA. In his address, the Director-General of WHO outlined the place of leprosy as an essential component of primary health care in the context of 'Health for all in the Year 2000'. Dr Mahler stressed that:

> . . . one of the essential components of primary health care is the prevention and control of locally endemic diseases. Leprosy is one such disease that is endemic in many countries of the developing world. However, it is only one of many; it can only be effectively controlled together with the control of other diseases and with the improvement of economic and social conditions. The economic and social implications of leprosy are far-reaching and profound. Think of the terrible burden it places on communities. Not only are those afflicted by the disease incapable of exploiting their potential economic energy; they are also nearly always unable to derive the social satisfaction of being able to realize their latent intellectual, cultural and spiritual talents.

Dr Lechat in his talk 'The Way towards Eradication of Hansen's Disease' reviewed the scientific and human approach to the social and health aspects of leprosy, considering leprosy as part and parcel of total community development:

> Control of the disease, cure of patients, protection of future generations, these cannot be dissociated from history, cultural patterns, economics, aspiration of the people, and political choices. Whatever the amount of dedication, science, and human solidarity, Hansen's disease will only be overpowered if this means a better life for the people. We must try to integrate our control effort with the development of primary health care, in so far as it is possible, practicable, and does not jeopardize prospects of success. In countries where control is well organized while general health care is rudimentary;

specialized services for Hansen's disease should serve as a model and be used as a spearhead to develop basic health services for the population in general. In such conditions, the motto should be: 'through leprosy to better health care for all'. Time is not with us. Control of this disease, if not its eradication, requires urgent and concerted efforts if the achievements of the last thirty years are not to be lost.

This meeting, which was attended by a large audience, was one more illustration of the considerable efforts developed by the Sasakawa Memorial Health Foundation to help leprosy patients and promote leprosy control and research.

SIXTH SCIENTIFIC MEETING OF ADELF, November 1980

The Sixth Scientific Meeting of ADELF, the French-speaking Association of Epidemiologists ('Association des Epidémiologistes de Langue Française') took place at the Institut Pasteur in Paris, 24–25 November 1980. The general topic selected for the presentation and discussion was the 'Evaluation of Preventive Intervention in Public Health'. Leprosy was taken as a key example of the application of epidemiological methods for the development and evaluation of preventive measures; Dr Sansarricq, Chief, Leprosy Unit, WHO, presented the problem encountered, the design of vaccine trials for the leprosy vaccine presently being developed in the context of the IMMLEP project, which is part of the UNDP/World Bank/WHO Special Program for Research and Training in Tropical Diseases.

SIXTH INTERNATIONAL CONFERENCE ON GLOBAL IMPACTS OF APPLIED MICROBIOLOGY, August–September 1980

The Sixth International Conference on the Global Impacts of Applied Microbiology (GIAM VI), was held at the University of Lagos, 30 August to 7 September 1980. A special session was dedicated to leprosy, under the Chairmanship of Professor M F Lechat. Presentations were made on *M. bovis* (I Alhaji); Leprosy Cases in India (S Goyle and V Virmani); Epidemiology (M F Lechat); Recent Problems in Chemotherapy (L Levy); Primary prevention (S K Nordeen); N Cardiosis (O U Osoaybakadna and A N U Njoku Obi); Leprosy Control in Nigeria (E R Pfaltzgraff); Problems and Trends in Leprosy Research (H Sansarricq); and Cell-mediated immunity (S N C Wemambu).

CONSENSUS CONFERENCE ON CLASSIFICATION OF LEPROSY, November 1980

The consensus conference on 'Classification of Leprosy' was jointly organized by the Indian Association of Leprologists (IAL) and the Acworth Leprosy Hospital Society for Research, Rehabilitation and Education in Leprosy (RRE Society), Wadala, Bombay-400 031, India on 29 November 1980 during its 10th year celebration.

The confusion arising principally out of different nomenclatures used by the existing Indian system of classification was attempted to be cleared at this conference and evolve a simple classification based on clinical and bacteriological features, uniformly acceptable to paramedical workers, medical officers and scientists. This was organized by Dr R Ganapati, Director of the Bombay Leprosy Project and Vice-President of the RRE Society; Dr P Kapoor, President of the IAL was the President of the conference.

Comments received from 36 expert members of the IAL over a working paper prepared

by Dr K V Desikan, Director, Central JALMA Institute for Leprosy, Agra, were discussed and a give-group system including a place for early (indeterminate) lesions was arrived at. This system would be nearer the international classification.

The features of indeterminate leprosy as well as other types of leprosy were discussed. However, this modified Indian classification needs to be accepted by the general body of the IAL.

Among the prominent leprologists who participated in the session were Dr C G S Iyer, Dr B R Chatterjee, Dr K C Das, Dr M S Nilakanta Rao, Dr R Ganapati, Dr D S Choudhary, Dr M Christian, Dr C J Chacko, Dr A J Selvapandian, Dr H Srinivasan and Dr K K Koticha.

FILM. A NEW PRODUCTION BY THE KATHARINA KASPER LEPROSY CONTROL SCHEME, BANGALORE, INDIA: 'LEPROSY – THE MISUNDERSTOOD DISEASE'

Dr D S Choudhary has supplied the following description:

> The above colour film has been produced by the Katharina Kasper Leprosy Control Scheme, Bangalore. The film was screened for the delegates of the recent Workshop held on Leprosy at Bangalore from 2–8 October 2980 by WHO (Regional office, New Delhi) in collaboration with the Directorate General of Health Services and Indian Council of Medical Research.
>
> The film has been directed by Mr T S Ranga. It runs for 21 mins. The medical expertise in the production of this film has been provided by Dr M S Nilakanta Rao (who has also written the script), Dr E Vomstein and Mr H Meermeier. At present the film is available in three languages: English, Kannada and Tamil. Production in other languages, e.g. Telegu, Urdu, Hindi and Marathi are also contemplated.
>
> Essentially the film is meant for the lay people who, even those who are otherwise well informed, often do not have factual information about leprosy. Many, in fact, labour under wrong ideas about the disease and the problem it poses. Extreme fear and out-moded notions of incurability of leprosy and the spread of the disease retard unfortunately the popular participation of the Community in leprosy work, which is a *sine qua non* to achieve success in leprosy control. The value of the film happily lies in this respect. It will promote public education about leprosy in a rational and scientific manner and this in turn will contribute to increased community involvement in our leprosy control activities in various fields.

For details of cost and posting, apply to Project Officer, Katharina Kasper Leprosy Control Scheme, 38/4 Davis Road, Bangalore 560 005, India.

ACWORTH LEPROSY HOSPITAL SOCIETY FOR RESEARCH, REHABILITATION AND EDUCATION IN LEPROSY, WADALA, BOMBAY 400 031. PROCEEDINGS OF THE 'IXth WORKSHOP ON LEPROSY' (Xth ANNIVERSARY CELEBRATION OF RRE SOCIETY)

The IXth workshop on leprosy was conducted by the Acworth Leprosy Hospital Society for Research, Rehabilitation and Education in Leprosy (RRE Society) on 9 June 1980 at the Hospital premises under the Chairmanship of Dr K D Sharma, Director, Haffkine Institute, Bombay. This function also marked the 10th year of the inception of the society.

Mr S S Naik, Hon. Secretary of the Society, summarized the highlights of the decade of the activities and achievements which included: (1) the series of epidemiological investigations based on surveys of schools and slums in the city of Bombay; (2) studies on rehabilitation of leprosy patients and the establishment of a sheltered carpentry workshop for infectious patients; and (3) health education involving programmes directed towards the

medical profession and students. Dr V V Dongre, Jt. Hon. Secretary, gave an outline of future activities to be undertaken by the society.

The RRE Society presented the following papers:

1. Integration of leprosy into general health service in an urban area – a feasibility study. (Sponsored by the Indian Council of Medical Research.) Revankar CR, Jha SS, Dongre VV and R Ganapati.
2. Prevalence of leprosy in slums in Bombay including a Leprosy Colony. Ganapati R, Revankar CR and Khot Sunanda M.

CHINA. THE CHINESE MEDICAL JOURNAL, 1980

The following items are extracted from the 'News and Notes' section of recent issues of this journal, one of many we receive on an exchange basis with *Leprosy Review*. The address is *Chinese Medical Journal*, 42 Dongsi Xidajie, Beijing, People's Republic of China.

First National Epidemiology Conference

The First National Epidemiology Conference was held during July 1980 in Harbin. One hundred and ninety-five representatives from various parts of the country attended. Of the over 450 papers received, 33 were read at plenary sessions and 131 at smaller discussion groups.

Experience was exchanged on research in infectious disease epidemiology, e.g., epidemiologic characteristics and control of hepatitis, dysentery, typhoid fever and leptospirosis, serologic diagnosis and preparation of vaccine. Shanghai and Beijing representatives reported on disease monitoring during recent years and detection of influenza, encephalitis B and epidemic meningitis, demonstrating new techniques in epidemiologic study.

Epidemiologic research in non-infectious diseases as cancer, cardiovascular diseases, diabetes mellitus, psychiatry, Keshan disease, Kaschin-Beck's disease and endemic goiter was also covered. *The Atlas of Chinese Cancer Mortality* edited by the Cancer Institute of the Chinese Academy of Medical Sciences depicts the cancer distribution in the country, providing scientific grounds for cancer control.

At the conference, the Chinese Epidemiology Society was set up with Professor Su Delong as chairman.

Eleven Chinese Medical Research Institutes Appointed WHO Collaboration Centres

Eleven Chinese research institutes have been designated centres for collaboration with the World Health Organization (WHO), under an agreement between WHO and China's Ministry of Health.

These institutes and the fields in which they will collaborate with WHO are: Institute of Medical Biology, Chinese Academy of Medical Sciences (CAMS), enteroviruses; Institute of Virology, CAMS, viral diseases; Institute of Parasitic Diseases, CAMS, malaria, schistosomiasis and filariasis; Beijing Institute of Tropical Medicine, paragonimiasis, clonorchiasis and Leishmaniasis; Shanghai Institute of Entomology, Chinese Academy of Sciences, insect toxicology and physiology; Cancer Institute, CAMS; Shanghai Cancer Institute, Cancer Research Institute, Zhongshan Medical College; Institute of Cardiovascular Diseases, CAMS; Institute of Cardiovascular Diseases, Shanghai First Medical College and Institute of Cardiovascular Diseases, Guangdong province, cardiovascular diseases.

Higher Medical Education in China

China now has 113 medical colleges with more than 126,000 students and 2,731 post-graduates, it was revealed at a recent National Working Conference on Higher Medical Education.

Medical colleges made up one-sixth of China's institutions of higher learning and their students, one-tenth the annual college enrolment. They have a teaching staff of 30,000 including 1,400 professors and 9,600 lecturers.

The country had only about 50 medical colleges at the time of liberation in 1949 and 92 in 1965.

Border and other regions inhabited by minority nationalities, where modern medical facilities were almost non-existent before liberation, now boast 17 medical colleges.

Most of China's medical colleges give 4–6 years' schooling. They have trained more than 380,000 senior medical workers during the past 30 years. A considerable number of them now work in county hospitals all over the country.

Medical colleges integrate teaching with research. Notable are the research in acupuncture anaesthesia and urology at Beijing Medical College; research in intrauterine fetal development and physiology and pharmacology at the Shanghai First Medical College; treatment of burns and hand surgery at the Shanghai Second Medical College and research in clinical endocrinology at Tianjin Medical College.

The country has established 24 institutes to train practitioners of traditional Chinese medicine as well as Uygur (uighur), Mongolian and Tibetan medicine with a total enrolment of 20,000 and a small number of post-graduates.

Preventive medicine departments in medical colleges take in 1,500 students annually.

One hundred medical teachers are sent for advanced studies abroad yearly.

A 10 year (1981–1990) programme for the development of higher medical education was discussed at the conference.

ILEP. INFORMATION ON SIDE-EFFECTS FROM CLOFAZIMINE (LAMPRENE; B663)

Dr Harold Wheate, Secretary to the Medical Commission has passed the following notice for insertion in this number of the journal:

> The Medical Commission of ILEP would be grateful to receive information concerning the frequency and severity of the side effects of the drug Clofazimine (Lamprene). It is generally agreed that severe gastro-intestinal disturbances are encountered only when large doses (300 mg per day or more) are given for periods exceeding 3 months. It is not yet certain whether minor disturbances are dose-related or time-related or both. May we ask readers who are interested to send information concerning:
> 1. Numbers of cases treated with Lamprene during the past 12 months.
> 2. Number and nature of gastro-intestinal side effects and the dose and duration of therapy in each case.
> 3. Number and nature of any other side effects of note and the dose and duration of therapy in each case.

Although an attempt has already been made to obtain similar information through a questionnaire devised by CIBA–GEIGY, the response was far from satisfactory. *All those with experience of this important drug are earnestly requested to send in information as requested above.*

ILEP. SPECIAL MEETING OF THE MEDICAL COMMISSION ON DRUG THERAPY FOR LEPROSY, ROME, 9 December 1980

Members of the Medical Commission had previously been invited to submit their own views in writing on suitable regimens of drug therapy for use in the field. These were circulated and discussed in detail, with the following main conclusions:

1. In many programmes priority needs to be given to improving the quality of service, particularly the laboratory control – skin smears and tests for patient compliance. This is particularly necessary in integrated programmes.
2. Patient compliance is of great importance. It is unlikely that if a patient takes one drug (Dapsone) irregularly, he will take two or three drugs more regularly.
3. The relative merits of each of the drugs available and their advantages and disadvantages were appraised. There are difficulties and dangers in the use of the newer drugs and it is important to emphasize how to avoid these. Advice on what *not* to do is a matter of priority, especially as concerns Rifampicin which should be described only by a medical practitioner, should never be given as monotherapy and should never be given unsupervised for self-treatment at home. It is already being given, indiscriminately in some areas, with potentially much harm.

From discussion of these points, it was concluded that the Commission was too large a group to distil, from the very many alternatives which had been suggested, practical recommendations which will take all the factors of importance into account. A sub-group was therefore appointed to formulate these recommendations. Their preliminary document is currently (February 1981) under assessment.

ILEP. 35th MEETING OF THE MEDICAL COMMISION, ROME 10 December 1980

The Commission covered a wide range of subjects during its all-day session in Rome. These included: new ILEP guidelines; ILEP strategy; Clofazimine (tolerability); research projects; career structure for leprosy workers; the International Leprosy Congress, New Delhi, 1983; training; the writing of a technical guide for slit skin smear examination by direct microscopy; the vaccine for leprosy; International Year of the Disabled; an ILEP centre for the production of *Mycobacterium leprae*; conference on Leprosy in Europe (see below); and the 35th World Health Assembly.

The variety and depth of the matters discussed underlined the desire of members of the Medical Commission to identify those matters in leprosy which are of prime importance, especially to leprosy control and to the management of the patient in the field. Their attempts to do this, and to produce advice and information of practical value are already benefiting from the appointment of Dr Harold Wheate as Secretary to the Medical Commission whose office is at the ILEP headquarters, 234 Blythe Road, London W14 0HJ. (Telephone 01-602-6925.)

EDITORIAL NOTE

Special Number of Leprosy Review, **52**, *1981: 'Leprosy and Primary Health Care'*

Subject to the receipt of an adequate number of original contributions, it is planned to devote Number 4, **52** (1981) to the subject of 'Leprosy and Primary Health Care'. Invitations

have already been sent out to experts in various parts of the world asking for suitable contributions and we would also welcome letters or comments on any aspect of this important subject for possible publication. These should be sent to Oxford as soon as possible, since we are now actively planning the content of this special number.

Vancouver style

We apologize for having failed to establish this decisively in 1980, but take this opportunity to emphasize that it will be used in all original articles and editorials from now on. Authors are asked to follow this format, which can easily be seen in any issue of the *Lancet*, *British Medical Journal* or the *New England Journal of Medicine*.

Items for printing in 'Leprosy and the Community', 'News and Notes' and other sections of the journal can usually be included in the next number with virtually no delay if they are in the Vancouver style and written in a brief form.

Editor

Lepr Rev (1981) **52**, 193–196

Book Reviews

Damien, the Leper Priest, by Anne E Neimark. William Morrow & Company, Inc., New York, 160 pp. $7.95.

This is the story of Father Damien, told in colourful prose and written for the young people of today. The saga of the Belgian priest who devoted the last years of his life to the neglected sufferers from leprosy in Hawaii a century ago, certainly bears re-telling. His life – and more especially his death, thanks largely to Robert Louis Stevenson – awoke the conscience of the world to the victims of leprosy. Contracting the disease himself, Damien demonstrated that it was possible to *catch* leprosy, and this was only a few brief years after the (London) Royal College of Physicians had pronounced in a weighty report that leprosy was probably an hereditary disease – and Father Damien's Belgian ancestry was impeccably free from leprosy.

The book is definitely not written for critical leprologists, but factual blemishes like 'itchy spots on the skin' (p. 51), and 'leprosy . . . a fatal infection' (p. 51) should have been detected and removed; and historical inaccuracies (e.g. leprosy occurring 2000 BC in India and China, and 1500 BC in Japan (p. 52), and Moses advising 'oil and tree sap' (p. 56) as treatment, might have been avoided; and the statement (on p. 59) that 'a quarter of Europe's people had been attacked by leprosy' is surely an exaggeration. The unknown 'mysterious causes of leprosy' (p. 91) had been earlier discovered and 'labelled *Mycobacterium leprae*' (p. 82). It was news to the reviewer that leprosy could 'degenerate the tissues of the body' (p. 51), and that earlobes could be so enlarged that they 'hung to the shoulders' (p. 52).

However, these professional cavils will probably pale into insignificance in the eyes of those who are offended by the recurrence of the banned word 'leper' in the title and throughout the book. The word has today a false ring about it, as dated and objectionable as the word 'native' (p. 29). Has *The Star* been fighting a lone and losing battle over the years, in the country of its birth?

Apart from these criticisms, the story unfolds with the maximum of evocative and gruesome descriptions of the horrors of the untreated disease. One's admiration for the figure who found Molokai a cesspool of vice and filth and left it a garden is enhanced by the retelling of this heroic and challenging tale.

S G BROWNE

A Guide to Leprosy Control. WHO publication, Geneva, 1980, 97 pp. Sw. fr. 15.

The fact that there were 3,599,949 registered cases of leprosy in the world at the end of 1976 does not reflect the magnitude of the leprosy problem as the actual number of cases is estimated to be at least three times greater. Furthermore, only three-quarters of all registered cases are receiving treatment. Clinical disease appears only in a small proportion of infected persons as the majority have subclinical infection; therefore leprosy is a disease with a high infectiousness and a low pathogenicity. Disabilities are the main cause of prejudice against the disease, affecting about 50% of registered cases, and in addition there may be 2 million unknown partially handicapped patients, thus illustrating the social relevance of leprosy control, for no other disease

0305–7518/81/052193 + 04 $01.00

arouses such adverse reactions in the community and causes so much distress to patients and their families.

Although the mode of transmission has not been established with certainty, airborne spread is probably the most important, but other modes of transmission cannot be ruled out. All patients should be treated, even though some non-lepromatous skin lesions may disappear without treatment, but every effort must be made to establish certainty of diagnosis. Details are given of the physical signs of leprosy, neural and dermal, but in discussing palpable nerve thickening no mention is made of the diagnostic importance of hardness or of surface irregularity. Further help in diagnosis can be obtained from the histamine test, the indelible pencil test (in hot countries), skin smears, nasal smears, and in some cases skin biopsy may be necessary.

Two systems of classifying leprosy are described in detail, the Madrid classification of 1953 and the Ridley–Jopling classification of 1962 and 1966. Dapsone is given priority in chemotherapy, and in order to minimize the emergence of resistant strains of *Mycobacterium leprae* it is advised that full dosage should be used throughout treatment and without interruption. In multibacillary forms of leprosy initial treatment should be with two drugs, after which Dapsone can be used alone; one method is to add Clofazimine in dosage of 100 mg daily, or 3 times a week, for the first 4–6 months, and another is to add Rifampicin 300–600 mg per day for a minimum of 2 weeks. Other possible combinations must be considered in relation to toxicity, effectiveness, cost and availability. At follow-up examinations of multibacillary cases it is important to take skin smears every 6–12 months in order to observe the reappearance of solid-staining bacilli as bacteriological relapse due to secondary resistance precedes clinical relapse. Secondary resistance to Dapsone is likely to develop in 5–20 years after beginning treatment, and under field conditions it can be confirmed if there is lack of improvement after giving regular and supervised treatment for 3–6 months. Alternative lines of treatment are described for such cases, Defaulting is the main obstacle to the effectiveness of leprosy control, and the even greater importance of guarding against defaulting on combined treatment is stressed. Advice is given regarding the assessment of clinical inactivity and the duration of treatment in the various types of leprosy.

As regards complications of leprosy and their management, the following are described: reactional states, hand deformity, neuropathic bone and joint damage, planter ulcer, foot drop, claw toe, infected vascular lesions and eye complications.

The basis of leprosy control is case-finding and effective therapy (secondary prevention), for BCG vaccination is not a specific prophylactic measure and it may be a long time before a specific vaccine is available (primary prevention), and other aspects include heath education, protection of household contacts, prevention of disabilities, rehabilitation, and social assistance to patients and their families. Important adjuncts to leprosy control are a rising standard of living and education, and more effective drugs. Priorities in leprosy control are effective treatment and follow-up, especially for multibacillary cases, and surveillance of contacts. Treatment should be on an out-patient basis, beginning with mobile units until health clinics can be established, and compliance with a tablet regimen may be helped by giving an injection of Acedapsone 225 mg every 75 days (the reviewer would prefer 3 months as Acedapsone is effective over this period and lunar months are readily comprehended). In-patient care should be carried out in a general hospital, should be short-term and restricted to special cases. Leprosy villages are not advocated.

The general objective of leprosy control should be to cover the whole country and to combine leprosy services with other health services, and detailed advice is given regarding the planning and running of a control programme, including a description of the set of forms for recording data prepared by WHO and the International Federation of Anti-Leprosy Associations (OMSLEP).

In a series of Annexes details are given of the preparation of standard integral (Mitsuda type) lepromin, techniques in clinical and bacteriological examinations, schemes of treatment for patients harbouring Dapsone-resistant bacilli, adverse reactions to Rifampicin, control of Dapsone intake by urine testing, and the OMSLEP system of recording disabilities. At the end of the book there are 57 references and an Index.

This is a comprehensive and authoritative publication which deserves to be widely circulated and studied.

W H JOPLING

Leprosy in Tropical Australia, by J C Hargrave and E R Jones. North Territory Medical Service, Darwin, 1980. Unpriced.

This well produced and profusely illustrated book of 59 pages sub-titled 'A short guide for field staff in the diagnosis, treatment and management of leprosy', is an enlarged and up-to-date edition of an earlier book published in 1970 entitled *Leprosy in Northern Territory Aborigines*. The various sections include descriptions of 4 types of leprosy (indeterminate, tuberculoid, lepromatous and borderline), the management of paralysed and insensitive hands and feet, the causation and treatment of plantar ulcers, eye damage, reactional states, the taking of smears and biopsies, and the basic principles of leprosy control. On the subject of chemotherapy pride of place is given to Dapsone and Acedapsone, and side effects of Dapsone are fully discussed. Other drugs described are Clofazimine and Rifampicin. Compelling reasons are given for treating patients on an out-patient basis and reserving short-term hospital admission for the management of complications.

All photographs are in colour and are of the highest quality; there are 15 showing the various types of leprosy, with additional illustrations of muscle paralysis and ulceration of hands and feet. Finally there are 17 pictures of skin conditions and limb deformities which could be confused with leprosy.

The reviewer was particularly interested to learn three things about leprosy as it affects Australian Aborigines: there is no stigma associated with the disease (although it is known as the 'Big Sickness'), blindness is rare, and indeterminate macules most commonly affect the face. The authors are to be congratulated on this excellent publication which deserves a wide readership.

W H JOPLING

The Application of Advances in Neurosciences for the Control of Neurological Disorders. Report of a WHO Study Group. (World Health Organization Technical Report Series, 1978, No. 629, ISBN 92 4 1206292.) Sw. fr. 9. (French and Spanish editions in preparation).

The following is extracted from a recent WHO announcement:

'In the last few decades considerable progress has been made towards achieving an understanding of organic infections of the nervous system, largely through the use of new techniques and the application of knowledge derived from the basic sciences. Unfortunately, few of the developing countries possess the facilities, personnel or equipment required for effective neuropathological investigation, prevention and treatment. The high prevalence in the Third World of a variety of diseases that cause disorders of the nervous system thus presents a formidable challenge.

'It was with the specific objective of trying to ameliorate this situation that WHO convened a Study Group to discuss the application of recent advances in the neurosciences for the control of diseases with neurological sequelae, some of which afflict millions of people yearly in Africa, Asia and Latin America.

The report of the Study Group, which has just been published, is concerned essentially with the following diseases: epilepsy and other convulsive disorders; cerebrovascular diseases, with special reference to

stroke; malnutrition and nutritional neuropathies (e.g. tropical ataxic neuropathy); cerebral malaria; trypanosomiasis; cysticercosis of the nervous system; leprosy, bacterial meningitides; viral infections such as kuru and other infectious disorders of the nervous system; and parkinsonism. Each disease is dealt with in a separate section, which examines etiology, epidemiology, neuropathology, therapeutic approaches (including pharmacokinetics), measures for prevention and control and, in some cases, the social implications for those afflicted.'

Office Techniques for Diagnosing Skin Disease, by William H Eaglstein and David M Pariser. Selected photography by Carroll H Weiss. Year Book Publishers, Inc., Chicago and London, November 1978.

This is a hardback of 194 pages, including a good index, by authors from the University of Miami Medical School and the Eastern Virginia Medical School, USA. The book was originally written to describe techniques which could be performed almost on the spot, in or near the consulting room, thus minimizing the usual delays in submitting material for routine laboratory examination. 'It is not often', reads one sentence in the Preface, 'that clinicians can confirm their diagnoses with techniques that they control.'

A wide range of diagnostic procedures are described for fungal, infective and parasitic diseases, many of them of perhaps prime interest to the clinical dermatologist, although many are relevant to tropical medicine. This book is noteworthy in that pages 83–185 are devoted to illustrations, many of them in colour, of the relevant techniques. Some of those on slit-skin smear techniques in leprosy, especially Fig. 10-3 and Fig. 10-4, showing incision and scraping, are unfortunately below the general standard. (To our knowledge, a really good series of absolutely clear photographs illustrating this simple but important procedure has yet to be published.)

Unusual Presentation of Extragenital Cutaneous *Schitosomiasos mansoni*, by W K Jayck, R V Lawande and S S Tulpule. *British Journal of Dermatology* (1980) **103**, 205.

'An African patient with a hypopigmented plaque on the face, suggesting clinically tuberculoid leprosy or sarcoidosis, is described. Histology revealed palisading granulomas surrounding ova of *Schistosoma mansoni*.'

This interesting single lesion, which was not anaesthetic, is well described and illustrated in this report by two plates showing the histological changes, including many ova within the granuloma. In the discussion, the authors point out that only 10 cases of extragenital and extra-anal cutaneous schistosomiasis have been reported and they conclude that in endemic areas this condition must be considered in the differential diagnosis of cutaneous granulomas, along with sarcoidosis, granuloma multiforme and tuberculoid leprosy.

Health Education Index and Guide to Voluntary Agencies, 1980, compiled and edited by Brian Edsall. Published by B Edsall and Co. Ltd, 36 Eccleston Square, London SW1V 1PF. £15.

This is a paperback of 362 pages, measuring 18 by 23 cm, with no less than 500 sources, classifying over 9,000 different items on health education material. It also includes an exhaustive listing of the names, addresses and telephone numbers of specialist voluntary and professional organizations likely to be helpful in this subject, and the availability of speakers on various subjects. The price is considerable, but the information extensive and extremely well presented; it covers books, pamphlets, films, film strips, slides, flannelgraphs, lecture notes, loops, overhead transparencies, posters, tapes and tape casettes, video casettes and wallcharts.

A C McDOUGALL

Lepr Rev (1981) **52**, 197–200

Abstracts

3. **Sociological Studies on Leprosy from the University of Barcelona.** Dr Alicia Kaufmann of the University of Barcelona, Facultad de Ciencias Economicas y Empresiarles, Departmento de Sociologia, Spain, has kindly drawn attention to two publications which may not have received adequate circulation:

1. 'Study of the Perception of Health in General and Leprosy in Particular in Buenos Aires (Argentine Republic) by Alicia Kaufmann (sociologist), Maria Baliña (psychologist) and Luis M. Baliña (doctor).

This was published in *Revista de Leprologia de Fontilles*, September–December, 1975, Vol. X, No. 3, and is an interesting contribution to the sociology of this disease. The objectives were to determine (1) the degree of knowledge the community had about illness in general and (2) the specific knowledge the community had about leprosy.

2. 'The Leprosy Patient and His Integration into Society: Some Psycho-social Aspects of the Problem', by Alicia Kaufmann and Carmen Sotorrio.

This was published in *Boletin de estudios y documentacion del SEREM*, No. 13, April 1979, Madrid. This paper includes a review of the pattern and extent of leprosy in Spain.

For those who do not have access to these publications, it may be possible to obtain typescripts from Dr Kaufmann at the above address, and one set is available from the Editorial Office in Oxford.

4. MILLAN J **Leprosy control in Guadeloupe.** 1980 *Médecine Tropicale* **40** (4), 433–8, 441–5.

Part I. The special features of leprosy control in the West Indian islands collectively called Guadeloupe is that the medical personnel responsible for the organization are microbiologists with epidemiological leanings.

When the individual leprosy sufferer has been diagnosed (apparently after self-presentation – the precise mode is not indicated), he is provided with a record card containing all relevant social and medical information, including the report of the slit-smear examination, nasal mucus, biopsy and lepromin test. He may choose to be treated either at the Pasteur Institute or at a dispensary or hospital.

The diagnosing doctor notifies the administration (without revealing the patient's identity if the latter so desires), and close watch is kept on the developing epidemiological situation as disclosed by the accumulated notifications. Regular examination of contacts is instituted, and social security grants are made where necessary. Close liaison is maintained with a hospital in Paris when immigrants from Guadeloupe suffering from leprosy seek employment in metropolitan France.

Certain tensions are apparent between doctors in private practice and those in charge of the leprosy control programme, but on the whole co-operation is satisfactory, providing as it does for a reliable laboratory service and expert advice on treatment and contact examination, and the resources of the social services.

0305–7518/81/052197 + 04 $01.00

Part II. In view of the threat of widespread sulphone resistance consequent on prolonged monotherapy with dapsone, since 1975 multi-drug therapy for multibacillary forms of leprosy has been advocated. The two aims of the programme, based on the treatment of tuberculosis, have been the rapid reduction of infectivity and ambulatory treatment wherever possible.

The details of treatment follow accepted patterns; for tuberculoid and near-tuberculoid leprosy, monotherapy with dapsone for a period of 5 years is advised; for multi-bacillary leprosy, a multi-drug regimen consisting of rifampicin and dapsone (and frequently, ethionamide as well) is followed for 2 years, and then dapsone is continued for life.

In cases of clinically suspected sulphone resistance, dapsone is replaced by clofazimine; but dapsone is reintroduced sometimes subsequently if clofazimine proves unacceptable for some reason. (The reasoning for this unusual recommendation is not given.)

Corticosteroids are prescribed (a) at the beginning of treatment of patients with tuberculoid leprosy where nerve damage is already present; and (b) in patients developing mild forms of reaction. Reversal reactions, very common in all forms of borderline leprosy, are treated with clofazimine.

Admission to hospital is allowed for certain categories of patients, and surgery of peripheral nerves is practised where indicated.

Domiciliary treatment is controlled by a nurse who gives a month's supply of tablets to individual patients. She is responsible for the early diagnosis of the reactional state.

Overall supervision is provided by the anti-leprosy service, which is responsible for the 6-monthly examination of all patients. After a suitable period of treatment, patients are placed in the category 'under observation without treatment' for 2 years, a period that may be lengthened in cases of nerve damage or its sequelae.

An epidemiological enquiry is instituted after notification, so that any antecedent details of interest and particulars of household contacts may be elicited and followed up by regular examination.

As a rule, chimioprophylaxis is not advocated, nor is lepromin testing done on contacts.

S G Browne

5. NEGASSI K, CLOSS O, HARBOE M. (1979) **Cross-reactions between serum proteins and water soluble liver tissue antigens of the nine-banded armadillo (*Dasypus novemcinctus Linn*) and man.** *Clin exp Immunology* **38**, 135–47.

This elegant paper demonstrates by crossed immunoelectrophoresis (CIE) that there are at least 12 antigenic determinants in common between liver tissue of the nine-banded armadillo and human serum proteins. Nine of the cross-reacting substances were identified by modifications of the method using monospecific rabbit antisera for human serum proteins. In addition, by a modification of CIE to show separation of cathodic moities the authors demonstrated 12 cross-reacting substances between human and armadillo liver homogenates.

The authors also mention their previously unpublished observation of the presence of armadillo antigen in *Mycobacterium leprae* preparations from several sources. They review the experimental evidence for breakdown of tolerance to self antigens and suggest that the microgram quantities of armadillo antigen that might contaminate the 'dirtiest' preparations that have been used for skin testing in man are within the danger limits. Finally, the authors suggest that a form of CIE could be adapted for monitoring *M. leprae* preparations for contamination with armadillo antigen and that such preparations should meet certain standards before they are used.

That there should be common antigens shared between armadillos and humans is hardly surprising. After all, we are only animals within the same class and separated by only a few tens of millions of years; nothing in evolutionary terms. Neither is it conceivable that there are so many different antigenic determinants that a few of

the thousands expressed by any one mammalian species should not be shared by another. Thus the findings of Negassi and his colleagues were to be expected.

The warning given in the final paragraph of their discussion should not be taken too lightly. After all, it is probable that armadillo protein bound to the cell wall of mycobacteria harvested from the tissues of armadillo may be exceptionally immunogenic. (This was shown to be the case for bovine serum albumin or egg albumin readily coated onto BCG by Crum and McGregor, 1976.) In view of this, repeated use of armadillo-grown *M. leprae* products in an individual would be unwise and there can be little doubt that the number of bacilli included in any would-be vaccine should be kept to a minimum. One must be wary of accepting that many doses of armadillo lepromin have been given with complete safety. Autoimmune sequelae could ensue months after the injection and, in underdeveloped parts of the world at least, such complications or even deaths might not be related to a past skin test.

J L Stanford

6. MILLAN J (1980) Le dépistage de la lépre dans un secteur de la Guadéloupe [French West Indies]. **[Leprosy case-finding in an area in Guadeloupe.]** *Médecine Tropicale,* **40**, 161–8.

This article, summarizing as it does the results of case-finding activities in a delimited area of Guadeloupe (French West Indies) over a period of six years (1973–79), not only provides precise figures of cases of leprosy detected, but also gives indications of wider application. While the anti-leprosy service has remained the lynchpin of case-seeking and case-finding during the period under review, the number of practising doctors has increased from 190 to 307, two-thirds of the latter being general practitioners.

The most productive methods of case detection are the systematic and regular screening of the entire population of school-children every 2 years by a competent team accustomed to rapid examination of the entire exposed skin. The organization of the visits of the team is accepted by staff and children, and apparently causes minimal disruption. Such active case-finding reveals early leprosy and thus is of importance in forestalling infections among contacts as well as instituting treatment before nerve damage has occurred. On the other hand, the 'passive' detection by practitioners reveals an established deformity rate of 12.8% among those diagnosed after self-presentation.

Of the 275 cases detected during the period under review, 113 were found among school-children; 40 of the 275 were domestic infections – a figure indicating the importance of regular examination of contacts of known cases, and the high effectiveness of the examination of this group. A disquieting feature of the survey is the unchanged level of patients detected with multibacillary forms of leprosy, despite the notable reduction by four-fifths of the total number of patients diagnosed during the 6 years.

The author sees in the statistics he quotes a justification for the preservation of a leprosy case-finding service as providing a real contribution to leprosy control and a source of continuing education for doctors who rarely see (or recognize) cases of leprosy occurring in their practice.

S G Browne

7. BOVET JL (1980) Traintement chirurgical de la paralysie faciale chez le lépreux en Iran. **[Surgical treatment of facial paralysis in leprosy sufferers in Iran.]** *Médecine Tropicale* **40**, 185–8.

The high incidence of facial nerve damage in Iran (12%) suggests that the preparalytic stage of leprosy is undetected and untreated.

The author gives an account of the main surgical procedures adopted to remedy the results of facial nerve damage, emphasizing the frequency of unilateral upper facial palsy accompanied by sensory loss

consequent on concomitant damage to the ophthalmic division of the trigeminal nerve. The overriding aim of surgical intervention is to prevent blindness from exposure keratitis.

He favours temporalis transfer as giving good results in the hands of visiting surgical teams from France, Switzerland and India, and has reservations about the usefulness of the silicon thread insertion technique.

Unfortunately the author does not present a critical follow-up of the results obtained, nor does he indicate the place of physiotherapy and re-education in the prevention of recurrence of the condition that surgery may claim to relieve. The questionable long-term results of the surgical treatment of the sad cosmetic and functional aftermath of upper and lower facial palsy, emphasize the importance of vigorous case-finding and adequate treatment of all cases of leprosy, particularly those in which the facial nerve is at risk, from neighbouring tuberculoid lesions, acute inflammation or exposure to severe cold.

S G Browne

8. **Skin Biopsy.** *The British Medical Journal*, 24 and 31 May 1980; sections 1 and 2.

Dr Allan Highet and Dr Robert Champion from the Department of Dermatology in Addenbrooke's Hospital, Cambridge, UK have contributed two most valuable sections on skin biopsy to the *BMJ* under the heading 'Procedures in Practice'. The first deals with local anaesthesia, the excision of the biopsy by scalpel and punch, and other techniques which may be used in dermatology – curettage, epidermal (shave) biopsy, needle biopsy and skin surface biopsy. The second describes the choice of lesion, body site and the orientation of the incision. There is an important warning about the dangers of using vasoconstrictors (adrenaline or felypressin) in the local anaesthetic in the fingers, toes, ears or penis – where intense vasospasm may result in tissue necrosis. Full details are given, under fixation, for material which is to be examined by immunofluorescence. The authors conclude with a plea for the submission of adequate clinical information, a diagram of the body site from which the biopsy was taken, and correct labelling. Both sections are well illustrated.

A C McDougall

®Lamprene Geigy

Antileprosy drug with anti-inflammatory[1] properties

effective in the prevention[2] and treatment[3] of lepra (ENL) reactions

indicated as a part of combined therapy for the prevention and treatment of dapsone resistance in lepromatous and borderline leprosy[4].

1. Browne, S.G.: Lepr. Rev. *37*, 141 (1966)
2. Azulay et al.: Lepr. Rev. *46* (Suppl.) 99 (1975)
3. Schulz, E.J.: Lepr. Rev. *42*, 178 (1972)
4. Yawalkar, S.J., & Vischer, W.A.: Lepr. Rev. *50*, 135 (1979)

011 LAM 7479 e

CONTENTS

Printed in Great Britain at the Alden Press
Oxford, London and Northampton