Leprosy Review.

The Scientific and Research Quarterly of the British Leprosy Association

LEPRA

Editorial Board

(Editorial Offices: 57A Wimpole Street, London, W1M 7DF)

Dr. S. G. Browne, O.B.E. (Chairman)
57A Wimpole Street
London, W1M 7DF

Dr. R. J. W. Rees (Vice-Chairman)
National Institute for Medical Research
Mill Hill, London, N.W.7

Dr. G. Ellard
Royal Postgraduate Medical School
Hammersmith Hospital
Ducane Road, London, W.12

Dr. W. H. Jopling
Hospital for Tropical Diseases
St. Pancras Way, London, N.W.1

Dr. D. S. Ridley
Hospital for Tropical Diseases
St. Pancras Way, London, N.W.1

Copyright © British Leprosy Relief Association

Volume 44: 4 issues, March–December 1973; £4.00 + £0.30 postage inland and abroad.

Subscription orders should be addressed to Academic Press Inc. (London) Limited, 24/28 Oval Road, London NW1 7DX, except those originating in Canada, the U.S.A., and Central and South America, which should be sent to Academic Press Inc., 111 Fifth Avenue, New York, New York 10003. $9.60 + $0.75 postage abroad

British Leprosy Relief Association
Registered Offices: 50 Fitzroy Street, London, WIP 6AL
Editorial

HOW DO LEPROSY BACILLI LEAVE THE BODY?

This apparently simple question, which puzzled clinicians before Hansen raised it again at the Second International Leprosy Congress in Bergen in 1909, is today being asked in the field and in the laboratory. In this issue of Leprosy Review McDougall and Rees remind us of some half-forgotten evidence that the discharge from proximal ulcers in the skin of patients suffering from active lepromatous leprosy—perhaps in a state of tissue hyper-sensitivity, like the erythema nodosum necrotisans of the Lucio phenomenon—may contain vast numbers of morphologically normal bacilli. And Pedley, in the immediately preceding issue (Leprosy Review 44(1), 33), after further painstaking investigations, suggested that the principal portal of exit of *Mycobacterium leprae* is the nasal mucosa of patients suffering from lepromatous or near-lepromatous leprosy. He had earlier demonstrated that the only skin surfaces on which acid-fast organisms could ordinarily be found in leprosy sufferers were those near the nose or those likely to be contaminated by inspissated nasal secretion transferred from the perinasal skin.

Many workers over the years have found, by standard procedures or by concentration techniques, acid-fast organisms on the surface of the skin and in the dermis of subjects in contact with leprosy patients. The identification of the organisms and the significance of the findings are both matters of discussion and dispute. (This is quite apart from the occasional presence of an acid-fast organism that the patient histopathologist may find deposited from tap-water on the surface of a section.)

The subject is of far more than merely academic interest.

Knowledge of the common sites of exit of viable leprosy bacilli will resolve many thorny questions of transmission. It will determine attitudes to contagion and prevention; and it will provide another important piece for the jigsaw puzzle of the spread of leprosy in the world, its persistence in some foci, and its disappearance from others.

Fashions in leprosy are as transient and inexplicable as fashions sartorial or tonsorial. Time was when “prolonged and intimate contact” with a leprosy patient was held to explain all. But there were curious and well-authenticated exceptions; and in any case, no convincing evidence was forthcoming that *Mycobacterium leprae* is at all commonly seen in histological sections traversing the epidermis. The bacilli may be present in enormous numbers in the dermis, but they are rarely present in the sub-epidermal clear zone. In tuberculoid leprosy they may be found in the ulcerating edges of an acutely inflamed lesion. Bacilli are present in greater numbers, it is true, in the epithelial cells lining the hair follicles and in the acinar cells of sweat glands and within their lumina. However, Pedley failed to find many bacilli on hairy and sweaty skin.

In the Far East the genito-urinary tract has for many centuries been associated in popular belief with the transmission of leprosy, but *Mycobacterium leprae* are but
infrequently found in the glomeruli or cellular lining of the kidney tubules, and studies of *Myco. leprae* in urine and semen are rare. Similarly, while acid-fast organisms are found in the intestinal wall, investigations of the intestinal contents have not been enlightening. The milk of lactating females contains enormous numbers of *Myco.leprae* when the cells lining the galactophorous ducts, replete with organisms, burst and evacuate their bacillary contents into the milk stream, to be imbibed by the suckling infant. Their subsequent fate is unknown, but calls for investigation.

The discharge from neuropathic ulcerations of the extremities has for long been considered by the laity in many lands to be highly contagious, but systematic examination of both the exudate and the wall and floor of the ulcers usually fails to reveal any *Myco. leprae*, even degenerate forms of the organism.

The mucosa of the upper respiratory tract, however, provides a more convincing picture. Ulcerations of the lepromatous granulation tissue found in the soft palate, uvula and naso-pharynx discharge acid-fast organisms in abundance; they may be found in scrapings or in the saliva. The mucosa of the trachea and larynx may be heavily infected, but curiously, not the trachea or the lungs. It is the nasal mucosa *par excellence*, however, that is severely and generally invaded in lepromatous and near-lepromatous leprosy, and it is the abundant mucoid discharge from the hyperaemic nasal mucosa that is probably the vehicle for the exit of the vast majority of *Myco. leprae* leaving the body of the infected host.

Slight and transient nasal obstruction is frequently adduced as an early symptom of leprosy, and epistaxis may on occasion be the presenting sign. Now, after Pedley's convincing demonstration, examination of the nasal mucus will be a necessary prelude to any discussion of the infectiousness of a patient suffering from leprosy, and direct rhinoscopy of all patients with lepromatous and near-lepromatous leprosy may provide evidence, hitherto lacking, of a possible portal of entry, as well as an accepted site of exit of *Myco. leprae*. It is most unusual for the nasal mucus or the nasal mucosa to contain acid-fast organisms before they appear in the dermis in lepromatous leprosy, but systematic examination may one day conceivably reveal the site of inoculation of organisms deposited by droplet infection on the vulnerable and hospitable mucosa.

These findings and these possibilities should stimulate clinicians. It is well known that the stratified epithelium of the vestibule is rarely the site of lepromatous infiltration, although a tuberculoid lesion in the vicinity may encroach on this area. Also, the mucous membrane covering the anterior aspects of the inferior and middle turbinates, as well as the septum, may be hyperaemic and oedematous, and perhaps show small yellowish elevations of lepromatous tissue set in a thick velvety mucosa, or areas of frank ulceration. The mucosa itself, as shown by microscopical examination, is thickened and infolded; its surface area is greatly increased, and the superficial cells discharge *Myco. leprae* in enormous numbers, a high proportion of which may be morphologically normal. It is evident that, consistent with a lengthy generation time, *Myco. leprae* must be present in the cells of the mucosa in numbers many times greater (possibly 15 times greater) than the daily discharge of several hundred million bacilli.

The nasal mucus used not to be examined systematically, though in patients with lepromatous leprosy it was known to contain acid-fast organisms. These were suspected to be harmless saprophytes unless they were present in obvious *globi*. Despite the attested value of smears obtained from the nasal mucosa in patients with lepromatous leprosy, the popularity of the procedure waned somewhat. As
evidence of potential infectivity, however, and as providing evidence of bacterial relapse and persistence of morphologically normal bacilli, the nasal mucosa may be a more sensitive tissue than the dermis. Now that positive means of identifying *Mycobacterium leprae* are available, intracellular acid-fast bacilli aggregated in globi—whether appearing in scrapes from the septal mucosa, or in histological sections, or in the mucoid nasal discharge—may be assumed to be *Mycobacterium leprae*. And examination of the discharge from the nasal cavity entails no discomfort to the patient.

The epidemiological interest of these findings will not be overlooked. The important question of the duration of viability of organisms discharged from the nasal mucosa (and present in, for instance, house dust) may be answered by recourse to the established mouse-footpad technique, and the possibility of infective fomites will need to be investigated anew. When culture techniques are developed, further outstanding questions will be more readily answered, as in the case of other diseases transmitted by droplet infection. In this context, "prolonged and intimate contact" comes to mean propinquity of such a nature as to be within the range of infected particles either floating aerially or deposited on some fomites. On the practical side, the hygienic disposal of the nasal discharges assumes a new importance; and staff in contact with leprosy patients need to be warned to take precautions against droplet infection.

The clinician-cum-pathologist will be intrigued by these stimulating observations, and will be drawn into further inquiry and speculation. The pathology of direct bacillary invasion of the nasal cartilages, the specific bony erosion of the nasal spine, the virtual sparing of the tracheal mucosa and that of the nasal duct and conjunctival sac, the importance of temperature and oxygen availability, the existence of healthy carriers and inapparent infections—all are aspects that immediately come to mind.

The experimental microbiologist will be examining his immunologically deficient mice and his armadillos for signs of spontaneous infection of the nasal mucosa. Just as nasal washings from untreated cases of lepromatous (and borderline) leprosy have provided material for animal experimentation, so animal infections may shed light on the pathogenesis of leprosy lesions of the upper respiratory tract in human beings, and the portal of entry of the organisms.

From droplet infection to the penetration of deposited organisms through a protective epithelial barrier is a very big step. It may be that some inapparent clinical infections, or some tell-tale immunological modification of lymphocytes, may register a transient exposure to *Mycobacterium leprae* in the inhaled air. It is easier to demonstrate the flooding of the circulation by thousands of *Mycobacterium leprae* derived from endothelial cells lining the smaller arterioles and literally bursting with organisms, than to show convincingly the genesis of an implantation lesion in the respiratory tract or elsewhere.

Once again, as in other portions of the growing edge of leprosy research, co-operation between the observant clinician, the epidemiologist, and the laboratory research worker is needed. The question "How does *Mycobacterium leprae* leave the body?" is now in the process of being answered. How it gets in is quite another matter.
News and Notes

THE LASKER AWARDS, 1972

Among the recipients of the 1972 Albert and Mary Lasker Awards for distinguished international service to the handicapped was Mrs Kamala Nimbkar of India. The awards were presented at the opening session of the Twelfth World Congress of Rehabilitation International held in Sydney, Australia, in August, 1972.

Leprosy Review adds its congratulations to Mrs Nimbkar and its appreciation of her work not only among leprosy sufferers in Bombay, through the influence of her editorship of Rehabilitation in Asia, but also in a much wider area of human need.

POSTGRADUATE COURSES AT A L E R T

The following postgraduate courses and seminars for doctors are planned by A L E R T:

Seminar on Neurology and Ophthalmology in Leprosy, 27-31 August, 1973

The purpose of this seminar is to give participants an opportunity to examine cases with neurological and ophthalmic problems and to discuss them with our own staff. The sessions on neurology will be led by Dr John Pearson of the MRC Research Team, and on ophthalmology by Dr Margaret Brand.

Clinical Leprosy, 1-27 October, 1973

The post-graduate course in clinical leprosy is intended to meet the needs of those doctors with little or no experience in leprosy, but who will responsible for the medical care of leprosy patients either as a part of general medical or dermatological practice or in specialized institutions. The course will be run at a level high enough to be of value to teachers in medical schools. In addition to clinical aspects, relevant data concerning basic science, anatomy, immunology and histopathology will be presented, and the course will conclude with studies in epidemiology and leprosy control. The course will be run by A L E R T staff, including Drs John Pearson, Harold Wheate, Ernest Fritschi, Dr Cap, and Dr Felton Ross, with the assistance of Professor Michel Lechat and Médecin-Général Languillon.

Management of Medical Care Programmes, 30 October-4 November, 1973

The purpose of this course is to enable the participants to apply modern management principles to their medical care programme. The courses will be directed by Dr E. J. Cummins, with the assistance of staff from the Centre for Ethiopian Management, and A L E R T. The subjects covered will include: (1) Explanation of management principles. (2) Identification of the skills needed
to ensure success. (3) Application of the principles of skills to specific medical situations.

Further details about these courses and opportunities for in-service training and other courses offered by A L E R T for para-medical workers, may be obtained from:

The Director of Training,
Dr W. Felton Ross, A L E R T,
P.O. Box 165,
Addis Ababa,
Ethiopia.

TEACHING COURSES AT FONTILLES

Dr José Terencio de las Aguas is again organizing teaching courses in leprosy at the Fontilles Leprosarium in Spain. A course for paramedical workers to be held from 12-22 September, 1973, will be followed, from 1-27 October, by a course for health auxiliaries. Full particulars may be obtained from Dr J. T. de las Aguas at Fontilles, Alicante, Spain.

The courses will be given by the staff of Fontilles, assisted by outside lecturers. They are sponsored by the Order of Malta.

W.H.O.—FELLOWSHIP MEDICAL OFFICER

The World Health Organization Regional Office for Europe announces the appointment of Dr Romeo Manrique-de-Lana of Mexico as the new medical officer with special responsibility for handling the placement of W.H.O. Fellows from all parts of the world. In 1972, no fewer than 2773 Fellows were helped in this way by the European office. The address of Dr Manrique-de-Lana is:

W.H.O. Regional Office for Europe,
8 Scherfigsvej,
D K–2100 Copenhagen,
Denmark.

THE SYMBOL OF ACCESS

Disabled leprosy patients will share the appreciation and gratitude expressed to the Council of Europe that it has recently recommended the use of the International Symbol of Access (Fig. 1) to its constituent governments. The
Committee of Ministers of the Council comprised official representatives from Belgium, France, the Federal Republic of Germany, Italy, Luxembourg, the Netherlands and the United Kingdom.

While the great majority of leprosy patients in the world may never have the opportunity of propelling their wheeled chair (if they have one) up ramps and into shopping precincts, hotels, and theatres, the news that some countries of the Western World now recognize that such categories of the disabled deserved special consideration will be welcomed in many quarters.

**COMPREHENSIVE RURAL HEALTH PROJECT IN JAMKHED, INDIA**

Dr and Mrs (Dr) R. S. Arole have embarked upon a comprehensive rural health project in Maharashtra Sta. work in similar situations. After a period of five years spent in the accepted curative hospital environment, Dr Arole became disillusioned with the minimal impact his medical activities had apparently had on the health of the community that his hospital served. He and his wife therefore studied public health at the Johns Hopkins University in Baltimore and returned to India to put their vision into practice. From the outset they determined to bring health care and preventive medicine to the whole community, and saw that the care of sufferers from leprosy would be a major part of their work in reducing morbidity and restoring human dignity.

By enlisting the co-operation of village heads and community leaders in their programme of health care, they were able to combat the prejudice that kept the leprosy sufferers outside the life of the villages. There are now no separate clinics for leprosy patients: all are welcomed at the central hospital. In a period of 18 months, 460 such patients have been placed on treatment, many of them having very early leprosy diagnosed by the survey teams that were engaged on an enquiry into all causes of ill-health. Dr Arole admits that the strongest and most vocal objection to the treating of leprosy patients at the same clinics as those suffering from tuberculosis or other diseases and conditions came, not from the community, but from his own nurses. Patience and perseverance are considered to be the prime needs of a programme of education in all aspects of health care.

**DR WAYNE MEYERS TO HAWAII**

Dr Wayne Meyers, who for the past 9 years has been in charge of the leprosy programme of the Institut Médical Evangélique in Kimpese, Republic of Zaire, has been appointed Professor of Pathology at the Medical School, Hawaii. Here he will work in close co-operation with Dr Olaf Skinsnes in the American Leprosy Missions' Atelier; he will be supported by this body.

**NEWS FROM BRAZIL**

A widely representative national body has been formed in Rio de Janeiro under the title of “National Committee for Leprosy Control”. Government and religious agencies will unite in a programme of public education and enlightenment, and will encourage community participation in leprosy control and rehabilitation. The Committee will work towards integration of leprosy control into public health
services, and will hope to campaign for the revision of all discriminatory legislation. The number of registered leprosy patients in Brazil is stated to be nearly 124,000; the present estimate is much higher.

NEWS FROM GREECE

On 15 December, 1972, new buildings in the Santa Barbara Leprosarium in Athens were inaugurated by the Greek Government. In addition to dormitories and wards, there is a spacious restaurant where ambulant patients may obtain their meals.

NEWS FROM THE L E P R A PROJECT IN MALAWI

The L E P R A Control Project based on Blantyre continues to report progress. The flood of new patients has become a trickle, though careful examinations are still bringing to light further cases of leprosy, both through the skin clinics at Blantyre and Zomba and the activities of the mobile teams.

At the end of 1972, no fewer than 12,400 leprosy patients has been registered in the Control Area, and 1919 patients had been discharged “disease arrested”. The regularity of attendance in December 1972, was 49%

The modest hospital admirably fulfills its function as a centre to which patients in need of hospital care for a limited period can be brought. Being in close proximity to the Queen Elizabeth Hospital and adjacent to an orthopaedic workshop, its wards are always full of patients requiring minor surgery or intensive care during reactional episodes.

NEWS FROM TRINIDAD

In the days of Dr Ernest Muir, the veteran leprologist, all persons suffering from leprosy in Trinidad and Tobago were directed towards the leprosarium at Chacachacare. Now, they may obtain treatment as out-patients if they wish, thanks to a new emphasis on domiciliary care. As a result of the new policy and the new attitude encouraged by the Ministry of Health and the American Leprosy Missions, Inc., patients are presenting themselves more readily and earlier for diagnosis and treatment. The Trinidad and Tobago Leprosy Relief Association is actively assisting in this programme, working in close co-operation with the Leprosy Control Unit and supplementing its activities in social support for the islands’ leprosy sufferers.

REFERENCE LIBRARY FOR LEPROSY

Plans are being prepared for the creation of a Centre in Germany that will act as a clearing house or reference library on leprosy. Professor K. F. Schaller will be responsible, as Director, for the organization of the Centre, which is being financed generously by the German Leprosy Relief Association (Deutsches Aussätzigen Hilfswerk) with an annual grant of about 30,000 D.M.

The Centre will act as an abstracting service, covering some 2700 periodicals in many languages, and will be able to furnish references appearing in the literature, to answer questions on any aspect of leprosy, and even to offer disinterested advice to enquirers on research projects. The Centre will draw upon the
bibliographical resources of the Ernst-Rodenwaldt Institute, which has a scientific library and research laboratories in the same complex, with 30 research workers and 150 technical assistants. The special services to be made available to leprosy workers are listed as follows: (a) the present position of research in leprosy; (b) epidemiology; (c) prevention, treatment, and rehabilitation; (d) all related branches of science that deal with the study of tissues and of *Myco. leprae*; (e) the present state of the world-wide leprosy campaign; (f) a collection of photographs and films relating to all aspects of leprosy. The Centre will thus be in a unique position to supply information on all aspects of research and treatment/control programmes.

All enquiries should be addressed to:

Professor Dr K. F. Schaller,
Ernst-Rodenwaldt Institute,
Viktoriastrasse 11-13,
Koblenz,
Germany.

It is hoped that the Centre will be in full operation in 18 months' time. The annual costs are expected to remain at the remarkably low level indicated by reason of the fact that no expense is incurred for furnishing, rent, or the Director's salary.

**CHANGE OF NAME**

The International Federation of Physical Medicine will be known henceforth as the International Federation of Physical Medicine and Rehabilitation. The change of name indicates the increasingly strong and practical links between these two activities in many countries. The Honorary Secretary is:

Dr A. P. M. van Gestel,
Rehabilitation Centre, Eindhoven,
96 Kempensebaan,
Eindhoven,
Holland,

from whom a list of the representatives in the various countries may be obtained.
The Hansen Centenary

1. Hansen, Bergen and Leprosy*

Nearly 700 years ago, the first St Jørgen Hospital was built in Bergen; its purpose was to house those afflicted by leprosy. The disease was then rife in Western Norway, having been brought from Britain (according to some) by the returning Vikings. At that time, leprosy was probably very prevalent in Ireland and also in England. The Crusaders also brought back leprosy with them from the lands of the Eastern Mediterranean. Bergen and its neighbourhood became leprosy in Norway. According to some accounts, the prevalence reached a record of 1 in 50 suffering from the disease.

The situation became alarming, and it was decided in 1838 to build two more leprosy hospitals in Bergen, in addition to one at Molde and one at Trondheim. More was happening, however, in the world of leprosy than building hospitals for the segregation of leprosy patients and the encouragement of indigent patients to cease "boarding out" and to enter hospital. In that same year, 1838, Dr Daniellsen began his life's work for leprosy in Bergen, and only nine years later published, with the collaboration of Boeck, his monumental clinical work on leprosy, *Om spedalsked*, which was to revolutionize medical thinking about the disease.

On 20 July, 1841, the unnoticed birth of G. H. Armauer Hansen into a humble Norwegian home also marked the end of an era and the beginning of a new one. Twenty-seven years later, after working as a doctor in the Lofoten Islands and studying in Austria, young Hansen returned to Bergen and began work at the St Jørgen Hospital. It was here that he pursued his hard course of observation and study, the sympathetic interrogation of leprosy patients and their relatives, and the painstaking pathological examinations that led him to his discovery of *Mycobacterium leprae* and his identification of the bacillus as a necessary and unique factor in the causation of leprosy.

Bergen as a city was no longer ashamed of its leprosy problem. It became proud that one of its young sons, a scientist only 31 years of age, was unravelling some of the tangled skeins of transmission and infectivity in his little laboratory in the St Jørgen’s Hospital. Bergen became the Mecca of investigative scientists, of bacteriologists, and of laboratory workers using the new techniques and the new aniline stains being made available.

In 1909 the Second International Leprosy Congress was held in Bergen under the presidency of Hansen himself. Internationally renowned scientists paid tribute to him. His researches have a decidedly modern ring about them, as he skilfully presided over the Congress and helped to word the resolutions passed: leprosy is contagious, they said, and the portal of entry is most likely the nose. Man alone is

*Notes of an address given by Dr S. G. Browne in the Håkonshallen, Bergen, Norway, on the occasion of the Centenary commemorative celebrations, on 28 February, 1973.
the victim of this disease; leprosy is not hereditary, though there were some awkward facts that still did not seem to conform completely to the theory of contagion; and—one thing they all agreed on—leprosy is most difficult, if not impossible, to cure.

King Haakon travelled from (the then) Christiania to attend this Second Congress. Railways, museums and tramcars gave free passes to the participants. The whole town of Bergen was beflagged and gay with bunting for the occasion. Why should Bergen thus be favoured to take to its heart not only Hansen himself, but the whole Congress? Part of the answer lies with Hansen, and what he had done. He was a more significant pioneer, a deeper thinker, than most of his contemporaries realized. He learned from the past, but was not enslaved by any predetermined theory or way of looking at a socio-clinical entity. He learned from many workers in many lands, and tasted of the cosmopolitanism of true medicine. He had made for himself a sound basis of pathological observations. Thus, he submitted his findings to the severe and necessary arbitrament of observable fact and scientific demonstration.

He did not shrink from espousing an unpopular cause, or from facing criticisms alike from those who were bound by tradition and those whose feelings were swayed by emotionalism. He listened to ordinary folk—leprosy’s victims—with an open and enquiring mind, trying to elucidate the pattern of spread of the disease. He was, in the days when the term was unknown, already an epidemiologist at heart. He never forgot the terrible end-results of progressive leprosy, and the social consequences of deformity and blindness and peripheral ulcerations. The fascination of the disease overcame his early revulsion and spurred him into renewed intense study of pathology, of pathogenesis, and of microbiology when these branches of science were still in their infancy. He profited from researches in related fields—in staining techniques and microscopical investigations of all kinds.

He was, moreover, understandably cautious in his oral and published statements, not claiming too much and not going beyond logical deductions of his observed findings. Some would suggest that he was a trifle too diffident, and could have claimed more than he did.

He epitomized much that remains enigmatic in leprosy investigation. He and his father-in-law, Danielssen, studied the same material, and came to diametrically opposed conclusions. The Royal College of Physicians of London had categorically stated in 1869 that leprosy was a hereditary disease, and then shortly afterwards Father Damien contracted it in Molokai—and he had an impeccable history of Belgian peasantry going back for many years. That is Hansen—a man with a vision, with an infinite capacity for hard work, and with a persistently enquiring mind.

Today, Hansen’s example challenges young Norwegian scientists to reach out, both geographically and academically, to explore new areas of beckoning research, to employ all the new investigative techniques and tools as they venture into the realms of immunology, electron microscopy, biochemistry and the rest. Not only in Bergen, in the Gade Institute, but away out in Addis Ababa in the Armauer Hansen Research Institute there is good work waiting to be done in the pursuit of knowledge, the relief of suffering, and the enhancement of human happiness.

Hansen thus challenges today’s youth in Norway and elsewhere.
2. Commemorating Hansen

On 28 February, 1873, G. H. Armauer Hansen concluded that the small brown staff-like bodies he had been seeing frequently during the previous four years in material obtained from nodular lesions in patients suffering from certain kinds of leprosy, must be causally connected with the disease. It is true that the anthrax bacillus had been recognized a quarter of a century earlier, and the spirillum of recurrent fever had been identified before Hansen published his findings, yet to him belongs the honour of demonstrating the bacillary cause of a specifically human disease, leprosy.

One hundred years later, in the Botanic Gardens in Bergen, Norway, a group of scientists, leprologists, national and civic dignitaries and members of the Hansen family gathered around the bust of G. H. Armauer Hansen to commemorate this epoch-making discovery. Professor Th. M. Vogelsang, the Norwegian medical historian, spoke of Hansen the man, the scientist and the epidemiologist, before laying a wreath at the foot of the pedestal bearing the bust. Dr S. G. Browne, representing the International Leprosy Association—the inheritor of the scientific legacy and the social concern of Hansen—referred to the repercussions of Hansen’s discovery on the world of leprosy sufferers. Hansen’s seminal influence was to be seen today in such institutions as the Royal Society of Tropical Medicine and Hygiene, London, and in the increasing emphasis placed by such voluntary agencies as The Leprosy Mission, LEPR A, and the Member-Organizations of ELEP on the treatment and control of leprosy. Dr Browne then placed a wreath from the International Leprosy Association at the foot of Hansen’s memorial bust.

Soon after this ceremony, held in the open air in a Bergen cradled in snow-clad hills, a representative audience filled the historic Håkonshallen (rebuilt after being totally destroyed in World War II), where His Majesty King Olav V, Royal Patron of the Tenth International Leprosy Congress to be held in Bergen in August, 1973, graced the commemorative celebrations. After a prize-winning male-voice choir had given a superb rendering of a lament and tribute in Norwegian specially written for the occasion of Hansen’s funeral service on 17 February, 1912, and sung to an air by Edvard Grieg, Professor Erik Waaler welcomed King Olav, together with representatives of the City of Bergen, the diplomatic corps, and the worlds of art, science, and learning. The Mayor of Bergen (Professor Ole Myrroll) paid a fitting tribute to the discovery by one of Bergen’s most illustrious sons of the bacterial cause of leprosy, and Dr S. G. Browne followed with a speech that assessed the significance of Bergen in the national and international history of leprosy. Professor Morten Harboe gave a masterly disquisition based on a study of the relevant original texts—including all Hansen’s early scientific papers—and emphasized the quality of the scientific observations that led to, and stemmed from, the identification of Mycobacterium leprae as the causative agent of leprosy.

At a luncheon given by the City of Bergen, H.M. King Olav—the guest of honour—spoke of Armauer Hansen as a great son of Bergen and citizen of Norway. Professor Erik Waaler presented the King with a gold and a silver medal specially struck for the centenary commemorations. One side depicts Armauer Hansen and his microscope, and the obverse shows St Jørgen’s Leprosy Hospital, Bergen. (The medals are now on sale.) Special stamps were also issued on 28 February to mark the occasion.
The Second International Leprosy Congress was held in Bergen in 1909, Hansen himself presiding. The Tenth Congress will provide a fitting forum for presenting and discussing aspects of advances in leprosy research that reflect not only the perennial problems debated in 1909—such as transmission and treatment and the influence of hereditary factors—but also the tremendous progress made of recent years by the exploitation of modern investigative techniques.
Ulcerating Lepromatous Leprosy in a Patient with Dapsone-resistant *Mycobacterium leprae*

A. C. McDougall
*Cochrane Annexe, Slade Hospital, Headington, Oxford*

and

R. J. W. Rees
*The National Institute for Medical Research, London NW7 1AA*

While under treatment with dapsone for lepromatous leprosy, a 50-year-old male Greek Cypriot presented with multiple widespread lesions of a bizarre type, many of them freely ulcerating.

Clinically and histologically, peripheral nerves were little affected, but biopsy samples of skin and ulcers contained enormous numbers of leprosy bacilli. Mouse footpad inoculation of the bacilli from skin homogenates showed resistance to all three levels of dapsone tested, namely 0.01, 0.001 and 0.0001% in the diet of mice.

From two of the larger ulcers bacilliferous discharges were collected in occlusive dressings over a 24-h period. Attention is drawn to the very large number of bacilli counted in this discharge. Excluding the nose, the shedding of dapsone-resistant bacilli into the environment from body ulcers in this case could have been in excess of 20 millions daily.

**Case Report**

Mr C. Z., a Greek Cypriot aged 50, gave a history of leprosy treated in Cyprus for 3 years before his first registration as suffering from lepromatous leprosy in London in 1965. At that time he presented with scattered deep ulcers on the legs and trunk, varying in size from one to several centimetres in diameter, together with a number of infiltrated plaques and nodules. He responded to dapsone, 100 mg twice weekly, and did well until April, 1970, when it was thought that the plaques and nodules had increased. A single skin biopsy at that time failed to reveal any acid-fast bacilli, but was reported as consistent histologically with lepromatous leprosy; routine slit-skin smears revealed only small numbers of degenerate bacilli. The patient continued on the same dose of dapsone, but became irregular during 1971, missing a total of about 5 months' treatment in that year. In November, he presented in Oxford with a remarkable crop of intra- and sub-cutaneous nodules on the arms, legs and trunk (but not on the face or ears), varying from 0.5 to 2 cm in diameter. One or two isolated lesions on the

*Received for publication 12 January, 1973.*
trunk were strongly suggestive of histoid leprosy (Wade, 1963; Rodriguez, 1969). Several lesions on the arms and legs (but not those on the trunk) were ulcerating freely (Figs 1 and 2); some had a raised, rolled edge, and others had a surrounding zone of dilated skin vessels. Lesions were neither painful nor tender. The skin between the lesions showed areas of atrophy and sub- and intra-cutaneous induration, suggesting previous inflammation. Lymph nodes in drainage areas were, surprisingly, either normal in size, or impalpable. Peripheral nerves were generally unaffected, only the terminal radials and superficial peroneals showing any definite enlargement; this was bilaterally symmetrical. There was no nerve tenderness in any site, and the patient had no evidence of anaesthetic damage to hands, feet or eyes, despite the fact that he was working as a machine operator in a screw factory. Response to pin-prick was normal throughout, but to cotton wool it was marginally defective in patchy areas on both hands and feet. He had a controlled atrial fibrillation, impalpable apex beat, normal heart sounds and blood pressure, slight pitting oedema of both ankles, and hepatomegaly (3 finger-breathths below the right costal margin). The thyroid gland was not enlarged, and there was neither tremor nor exophthalmos. Other systems gave results within normal limits, and the patient's general appearance and state of nutrition were excellent. His past history was without significant illness, but in October, 1970, he had been admitted to hospital in London with cardiac failure and atrial fibrillation, thought to be due to thyrotoxicosis.

Fig. 1. Left arm.
Investigations

Skin smears from 6 different ulcerating areas showed enormous numbers of acid-fast bacilli; the bacillary index (BI) was 6+ and the morphological index (MI) 28%. Six slit-skin smears taken from between these ulcers, and from skin that appeared to be as normal as possible, gave a BI of only 2, with an MI of 3.

Biopsies were taken from (1) skin nodules, to include neighbouring and apparently normal skin, (2) skin ulcers, to include the edge, and (3) the left radial nerve at the wrist.

The histopathology was as follows.

1. SKIN NODULES

These showed enormous concentrations of bacilli, often to the exclusion of almost everything else in the field, with a high percentage of solid-staining forms. The infiltrate was of histiocytes and fibroblasts; there was an increase of small vessels, some with endothelial swelling. Lymphocytes were not numerous, but plasma cells were conspicuous in some fields. Nerve filaments were not identified in these sections; they may have been obscured by infiltrate.

2. SKIN ULCERS

Here the bacillary concentration and the infiltrate were similar, but merged into an open and partially necrotic zone, where polymorphonuclear leucocytes

![Knee region.](image)

Ulc erating lesions, together with intra- and sub-cutaneous nodules on presentation in November, 1971. The patient had 7 similar open ulcers, and the excretion from them could have been in excess of 20 million dapsone-resistant bacilli per 24 h.
were common, and bacilli (solid and non-solid) could be seen "flowing out" to the surface.

3. RADIAL NERVE

At the wrist: this showed a quiet, old-looking, lepromatous histopathology with occasional non-solid bacilli lying in foamy macrophages and a scanty infiltrate of lymphocytes and plasma cells. There was minimal endoneurial collagenization. Histologically the picture resembled that seen between nodules or ulcers in the skin.

BACILLARY COUNTS

Homogenization of skin gave a total yield of \(5.5 \times 10^8\) organisms per g of tissue; the MI was 9. Using the thin plastic covers from proprietary adhesive dressings, collections were made of the discharge from two skin ulcers over a period of 24 h. These together yielded \(7.5 \times 10^6\) bacilli, with an MI of 20. Nose-blow material (mainly mucus), collected over 24 h, yielded \(4.1 \times 10^5\) bacilli; MI 18.

DAPSONe SENSITIVITY

Bacilli from the skin, inoculated into mouse footpads, showed complete resistance to the three levels of dapsone used in the test, namely 0.01, 0.001 and 0.0001% in the diet of the mice; the highest level is equivalent to a dose of 100 mg of dapsone daily for man.

OTHER INVESTIGATIONS

These included: haemoglobin, 15.8 g%; leucocytes, 6900; erythrocyte sedimentation rate (ESR) 31; no LE cells were found; immunoglobulins IgG 2170, IgA 725, IgM 250 mg per 100 ml; the Wassermann reaction and Kahn and Reiter's PCFT were all negative as was also the latex screening test (RA); the urine protein level was 78 mg per 100 ml, in a volume of 630 ml; electrolyte, bilirubin, and alkaline phosphatase values were all normal. A chest radiograph with barium swallow showed cardiac enlargement and a prominent left atrium and appendage, the appearances suggesting mitral-valve disease. No lung lesion was seen; the ECG showed atrial fibrillation, but no other change. At the time of the patient's admission to hospital in October, 1970, with heart failure, his ECG showed no evidence of infarction; the P.B.I. was then found to be 8.1, with T3 (Sephadex) 7.2, and T4 8.2.

Progress

The patient's clinical deterioration while being treated with dapsone, 10 years after the initial diagnosis, together with the highly positive skin smears and a histopathology of straightforward ulcerating lepromatous leprosy, suggested there was resistance to dapsone, and this drug was therefore replaced by rifampicin, 600 mg daily. All ulcers healed completely in a matter of days, and many of the intra- and sub-cutaneous nodules became smaller. He was treated throughout as an out-patient; checks of the urine usually, but not invariably, showed the reddish colouring suggestive of regular rifampicin intake. In July, 1972, biopsies from skin and scrotum showed marked histological improvement, and homogenization of the latter yielded \(4.1 \times 10^6\) bacilli per g of tissue, MI 3 (1/33). At the time of
writing (December, 1972) the patient is extremely well, possibly better than he has ever been since first diagnosis, and there are no clinical signs of lepromatous activity. At no time has he shown any suggestion of *erythema nodosum leprosum* (ENL) or other manifestation of Type-2 reaction.

**Discussion**

The patient presented himself in November, 1971, for routine checking; he was not seeking advice, and had no general complaints. At this stage, a number of experienced observers were reluctant to admit that his lesions were all ascribable to leprosy, resistant or otherwise, and the possibility of syphilis, tuberculosis, exotic tropical disease, or malignancy was discussed.

An ulcerating form of *erythema nodosum leprosum* was also considered, but skin and ulcer smears, together with the histopathology, soon revealed that this was not the explanation, and that the picture was simply one of ulcerating lepromatous leprosy. In the material obtained by biopsy no histoid features were found, but this may well have been due to the selection of the site for biopsy. The clinical and histopathological "escape", or sparing, of nerves is difficult to understand; in both respects, they seem to have been minimally affected, in contrast to the gross involvement of skin, sometimes only a few millimetres distant. Lymph nodes, even in nearby drainage areas, were also unaffected clinically. In view of his previous experience of skin ulceration in 1965, together with a possible relapse in 1970, and his admission to hospital later that year with cardiac failure, consideration was given to the possibility that thyrotoxicosis might have had a recurring, adverse effect on the lepromatous leprosy. The laboratory findings support this diagnosis; and certainly no other cause has been found for his atrial fibrillation. However, there are no references to this conjunction in the literature, although the converse has attracted more attention, with studies such as those on thyroid and anti-thyroid substances in murine leprosy (Jai-Kyoung Koh *et al.*, 1969), and methimazole in lepromatous leprosy (Levy *et al.*, 1967; Browne and Hogerzeil, 1962; Rojas, 1963). It may be worth recalling that an early publication on thalidomide (Murdoch and Campbell, 1958), now known to be dramatically successful in certain lepromatous reactions, described this drug as having significant anti-thyroid activity.

Of perhaps major interest in this case were the counts of bacilli excreted from skin ulcers over a period of 24 h. The collection of $7.5 \times 10^6$ bacilli from two ulcers in 24 h implies that from the 7 open lesions, over 20 million dapsone-resistant organisms per day might well have emerged. This exit route for bacilli into the environment in lepromatous leprosy is often underestimated or overlooked, nasal discharges in lepromatous leprosy usually being of much greater importance. In this case, a 24 h collection of nose-blow material yielded only $4.1 \times 10^5$ bacilli—a rather low figure in comparison with the yields obtained from several other patients examined in this unit during the past year, where figures of the order of $3.4 \times 10^8$ bacilli per 24 h have been recorded.

**Acknowledgements**

This work was supported by grants to A. C. McDougall from the Medical Research Council and the British Leprosy Relief Association (LEPRA).
References


Chiropody and Leprosy*

MONIKA MECKLENBURG

Leprosy Service, Northern Province, Sierra Leone

The idea of employing a chiropodist in leprosy schemes is presented. After a detailed description of a chiropodist's knowledge and of his or her possible rôle in leprosy, the reasons for employing chiropodists in leprosy are stated.

Introduction

The reason for this article is to present the idea of employing a chiropodist in leprosy schemes to deal with the major part of the foot problems in leprosy. This idea has been largely realized in the leprosy project based at Makeni, Northern Province of Sierra Leone, West Africa. It serves some 4000 out-patients in a district of about 3000 square miles (18,000 km²). There are 2 Mobile Units, each consisting of 2 leprosy assistants and a driver with a Land-Rover. The teams visit the 120 out-stations fortnightly. They give the anti-leprosy treatment, they do some health education and dressing work, examine and register new cases, follow up irregularly-attending patients, and bring patients with complications to the one hospital of 30 beds. This hospital is run mainly by a qualified nurse and a doctor on part-time contract. A full-time doctor is responsible for the organization and management of the whole leprosy project in the area, for all medical problems, and for the education and further training of the leprosy assistants.

The above figures show that our leprosy service is a "regular" one. Consequently our experience with a chiropodist can be regarded as not only of general interest, but of principal evidence. In the following section Mrs Valerie Lal, our chiropodist, will herself describe a chiropodist's training and work and discuss the possible rôle of a chiropodist in leprosy. In the conclusion, I will attempt an evaluation of the idea: "Chiropodists' employment in leprosy". In considering the advantages and the disadvantages of employing a chiropodist in leprosy, the following arguments might be advanced:

Pro

(1) There is no profession with such an intensive education in foot-care.
(2) About 10% of all leprosy patients suffer from foot-ulcers. Doctors and leprosy assistants are usually already overloaded with work, even excluding foot-complications. Therefore this large amount of time-consuming work could ideally be passed over to a chiropodist.

* Received for publication 27 November, 1972.
(3) Individual, multi-factorial foot care speeds up the healing time of the ulcers.
(4) An individual ulcer service combined with health education will satisfy the patient. The fact that his ulcers are healing quickly can often help the patient to overcome his fatalistic attitude towards his disease. Moreover, as the patient is not cut off from his normal environment for so long, the problem of re-socialization is reduced.

Contra

(1) "Doctors always know best".
(2) Chiropodists do not learn very much about leprosy during their training.
(3) Mass-treatment of foot-ulcers (soaking, oiling and dressing) can be done by an attendant with no training apart from that which makes him competent in his own job.
(4) A chiropodist will overstress the importance of ulcer complications in leprosy. Thus, a disproportionate amount of time, money, energy and hospital beds will be absorbed by the chiropody department.

Conclusion

As in our project, in many leprosy schemes the time and energy of the medical personnel will not balance with the amount of work to be done. The result is neglect of "subordinate" problems, such as health education and individual ulcer care. But this neglect is like a boomerang: without health education and good ulcer care it is difficult or often even impossible to get good co-operation from the patients. And, without the patients' co-operation, leprosy will become like the task of Sisyphus, hard effort leading to nothing, as the patients do not see any reason for coming since there is no result of treatment.

Thus, it would be a good decision for many leprosy schemes to employ a chiropodist. The huge amount of work and time involved in ulcer treatment, and health education regarding good foot care could confidently be handed over to him or her.

I conclude by providing addresses of societies in different countries able to supply further information regarding the employment of a state registered chiropodist:

The Society of Chiropodists, 8 Wimpole Street, London W1M 8BX, England
The Australian Chiropody Ass., 446 Elisabeth Street, Sydney, N.S.W., Australia

American Podiatry Association, 20 Chevy Chase Circle, N.W.,
Washington D.C. 20015, USA
The Australian Chiropody Ass., 446 Elisabeth Street, Sydney, N.S.W., Australia

Canadian Podiatry Association, 3017 Bathurst Street,
Toronto, Ontario, Canada

New Zealand Society of Chiropodists
P.O. Box 387,
Christchurch, New Zealand.
What is a Chiropodist?

VALERIE LAL*

Leprosy Service, Northern Province, Sierra Leone

In order to become a State Registered Chiropodist in the United Kingdom it is now necessary to have undergone a 3-year full-time course of training. Some chiropodists have been admitted to the Register without this training, but their training and/or experience had to have taken place prior to 1963 and they had to satisfy the Chiropodists' Board of their competence, usually by means of a practical and oral test.

In Britain the subjects studied during the 3-year recognized course of training are: the basic sciences, anatomy, physiology, elementary pathology and bacteriology, surgery and medicine in relation to disorders of the lower limb, dermatology, regional surgery, podology, (study of the foot in health and disease), therapeutics, practical chiropody, appliance making, and footwear.

Other countries offering similar training and qualifications are New Zealand and Australia. France has a 2-year training period, to which a 3rd optional year is expected to be added shortly. The USA has a 4-year training scheme leading to a doctorate of surgical chiropody, or podiatry as it is known in the United States.

Many countries have chiropodial societies, but their members receive very little, or no, formal training. In Germany a well-known company which manufactures various foot appliances also offers a 6-week training scheme in chiropody which is available in several countries. Of course very little medical knowledge is obtained in this time, especially as the course is concerned mainly with sales promotion!

The Chiropodists' Rôle in Leprosy

This includes the following:

(a) RECOGNITION AND TREATMENT OF MINOR INJURIES BEFORE THEY BECOME ULCERS

Most important are the recognition of the pre-ulcer state and subsequent advice, and also supervision to ensure that the patient has complete rest.

Fungus infections, wounds, "jiggers", heel cracks, blisters, and interdigital fissuring are all conditions that may be found in leprosy patients, and these should be treated before they become infected and lead to formation of an ulcer. Such minor lesions on the anaesthetic foot may easily provide entry for bacteria, and if undetected and untreated will "track down" to the deeper tissues.

(b) SPECIALIZED AND INDIVIDUAL TREATMENT OF ULCERS

Mass treatment of plantar ulcers in leprosaria tends to give inadequate results.

* Member of the Chiropodial Society, State Registered Chiropodist.
In my opinion, based on experience, regular soaking (especially daily soaking) of ulcers deters healing. Soggy wounds do not heal.

The in-patient probably needs to wash his feet when he first attends the chiropodist. After this the foot should be kept dry until healing has occurred. Out-patients generally have to wash their feet at every visit, although if the ulcer has been well dressed on the previous visit and kept dry this may not be necessary. Feet without ulcers should be soaked daily and a little oil applied to aid water retention, and help defective sweat glands.

Recognition of the type of ulcer presented and its effect on underlying tissues is important, as is an assessment of the patient’s general disabilities and deformities, and how these in turn affect body-weight distribution. His general health must be taken into consideration, as well as the condition of local tissues immediately surrounding the ulcer and in the lower limbs. If the blood supply to the extremities is good and no infection is present, then quick and good healing can be expected. Poor quality skin with loss of elasticity will need a longer time for healing, as will also previously scarred and deformed feet.

Correct trimming of an ulcer is extremely important in promoting healing in the minimum amount of time. This not only requires the removal of dead and fibrous tissue surrounding the ulcer, but also cutting the callus right down so that it blends into the healthy skin around it. Any other callus on the sole of the foot should be trimmed to leave the plantar surface smooth and even. A certain degree of suppleness may also be obtained in the skin of some feet when trimming is correctly done.

Heel fissures can sometimes be completely cut away, as they are often found to be only "callus deep", but if they are not trimmed they will usually become deeper and eventually open the epidermis. Flaps of dead epidermis, perhaps resulting from a superficial scratch, should be cut away as they provide a lodging place for dirt and bacteria. In unshod races a certain amount of callus formation is functional, so that drastic reduction of callus is to be avoided, especially in the case of out-patients. Patients presenting with osteomyelitis or any other deep-seated infection are referred to the doctor for systemic treatment. Bony sequestra are removed and one of the new enzyme drugs is preferred for local application. We have had very good results with these drugs.

The correct use of medicaments speeds the healing process. Generally speaking, dry ulcers situated in hard anhidrotic skin will respond well to a cream-based medicament, whereas a soggy ulcer will respond more quickly to a powder-based drug. Ulcers should be measured when first seen, and thereafter at each subsequent visit. If little or no progress has been made, then the medicament being used should be changed, and any other suspected underlying cause investigated.

(c) AFTER-CARE AND HEALTH EDUCATION

When an ulcer has healed the covering skin is usually fairly weak and thin, so that the patient may still require protective dressings in addition to his special sandals. Incidentally, daily soaking at this stage may also re-open the wound.

After being discharged the patient should be able to visit the chiropodist occasionally for checking of his feet, for advice, and for any trimming of the callus that may be necessary. Frequently a hard core of callus forms in the centre of an old plantar ulcer scar. This may fall out of its own accord, but usually it gets pushed deeper into the foot and acts like a stone, penetrating deep into the tissues.
and causing the ulcer to reopen. This callus can be easily removed and a softening agent such as a cream containing vitamins A and D may be applied to improve the quality of the skin.

Health education is given to the patients throughout their treatment and thereafter. They are helped to understand the nature of their disease and its effects. In this way we hope to get their co-operation. They are advised on how to care for their own feet at home in their villages, and made to realize that the responsibility is now mainly theirs.

(d) P.O.P. AND THE KARIGIRI BOOT

The application of plaster-of-Paris casts, back-slabs, splints, and the Karigiri boot are well within the capabilities of the chiropodist.

(e) SHOEMAKING

During their training chiropodists learn to make various foot appliances and simple footwear from different materials. Although the training in this field and the skill and experience acquired cannot be compared to that of an orthopaedic shoemaker, the type of sandal suitable for leprosy patients i.e. those made of microcellular rubber or of Plastazote, is within our capabilities. Many chiropodists, especially the men, enjoy this aspect of the work and seek to further their skills and knowledge after training. With the current shortage of shoemakers perhaps a chiropodist might be considered for this work.

(f) SUPERVISION OF ATTENDANTS AND TRAINING

A chiropodist employed in a leprosarium will have more time than the doctor for training attendants in, e.g., ulcer care and all the subsidiary education needed to bring about an understanding of the treatment. Furthermore, attendants will work alongside the chiropodist and be encouraged in their work by seeing the results achieved by giving individual treatment to each patient. Too many attendants lose interest in ulcer care simply because they have been told to use the same course of treatment for every patient, a practice which results in a “hit or miss” affair.

(g) ADMINISTRATION

Correct record keeping and terminology as applied to foot-care is an important part of ascertaining results, reviewing treatment, and conducting surveys.

Acknowledgement

The authors wish to thank the Society of Chiropodists, London, for their suggestions and information given in preparing this paper.
Leprosy and "A Disease Called Leprosy"*

T. A. STRINGER

United Kingdom Activities Officer, LEPRA, London W.1.

Despite arguments put forward in Leprosy Review (1972) 43, 69-105, there is a case for retaining the substance of current terminology related to leprosy, particularly because of its value to fund-raising. In any event, the public will not co-operate in the adoption of new terminology, but find a vital rôle for the linguistic associations of leprosy. Acknowledging the nature of general knowledge about the "disease called leprosy", we have opportunities of educating the public in both endemic and non-endemic countries about the true nature of the disease. Identifying the nature of semantic confusion involved in the Christian motivation towards leprosy sufferers, we may be able to influence the public reaction to leprosy by correcting the old terminology in religious texts, and by referring to the defined disease as "actual leprosy".

In Leprosy Review (1972) 43, 69-105, various aspects of leprosy are examined in a range of articles from the superficial to the scientific. Taken as a whole, these articles reveal extraordinary conflicts within the world of leprosy workers and suggest, between the lines, that a continuous close proximity to the disease may not be conducive to a factual assessment of its rôle in the public sphere.

We are dealing with a disease which occurs in a number of countries, of different cultures, traditions and languages. Moreover, we have to take into account that the traditions associated with leprosy carry the local history into the present in each country. In such circumstances, generalizations are foolhardy, and subjective conclusions dangerous, particularly if they invite world-wide participation in costly exercises of verbal manipulation which are not by any means guaranteed to solve local problems and which would create problems elsewhere.

It is perhaps a little to one side of the purpose of Leprosy Review to suggest that those engaged on raising funds for anti-leprosy work may have a case to present in this area of controversy as to public reaction to the disease, but since it is acknowledged that voluntary agencies have a rôle in field work, it seems only fair to consider some aspects of the methods which enable them to fulfil this rôle, and indeed, some of the problems which they face, in arousing public attention for a forgotten, unfashionable and undramatic cause.

Fund-raising for leprosy in the United Kingdom is competitive, with some 77,000 charities vying for a share of public goodwill and generosity. The vast majority of these are small, local efforts, to serve a local purpose. Appeals for world-scale humanitarian purposes invite an instinctive resistance, justified by the authority of the quotable, if questionable, aphorism “Charity begins at home”. To go beyond this barrier, an “overseas” charity must involve the potential donor

* Received for publication January, 1973.
in thought and in feelings of obligation which pre-suppose some philosophical standpoint, however undefined. The clarity of appeals is their greatest chance of success, and since the public is ill-informed about leprosy, nonetheless adhering to traditional beliefs about a “disease called leprosy”, details of which have never been assimilated, the evocation of a reaction to the word “leprosy” is an essential factor in stating the case. “Leprosy” is related to Biblical and mediaeval traditions, and is thought by many to have “died out” centuries ago. Appeals must, therefore, correct these misunderstandings before they can hope to present true current information. The process is one of relying on the existence of misunderstanding, and making the correction reveal the case for concern and generosity. It is a process of associating the traditional “disease called leprosy” with the actual currently defined disease to provide for the dissociation of fiction from fact. To attempt to do this without a total reliance on the abundant misuse of the word “leprosy” would be a complex and vain exercise and would, moreover, lose the interest and imagination of the public, who are saturated with information, most of which is irrelevant to any of their needs or interests. Because of all the factors which frustrate the efforts of field workers, the word “leprosy” invites curiosity and attention, and provides for a strategy in gaining support. This seems a legitimate and harmless process. In a similar way, pictorial representation of leprosy sufferers provides immediate information about the nature of the disease, but, stressing the unaesthetic aspects of it, seems calculated to reinforce its stigma.

Fund-raisers are, then, faced with a considerable dilemma. Do their efforts to provide funds for treatment and research perform a disservice to their medical colleagues, prolonging traditions, the redundancy of which is eagerly awaited in the field? It is a dilemma which cannot be resolved if the answer is affirmative, for a loss of income must affect the anti-leprosy effort adversely. And so it is legitimate to refer the quandary back to those who use the funds and ask if they would be prepared to do without such financial assistance as can be offered in favour of obtaining a new identity for the disease?

We take it for granted that the more money we can provide, the greater will be the momentum of treatment and research programmes, and from this increased momentum, eventual conquest will result. Even against the background of implicit disapproval, we have to continue to rely on the dynamic of an appeal for participation in a world-wide campaign to overcome an ancient enemy, which, when understood, is far less fearsome than its legends suggest. The pursuit of these endeavours is conducted on the basis of evidence of public misunderstanding, and of the need and opportunity to correct it, arising out of just the kind of hysterical clumsiness embodied in the Brazilian headlines referred to (Rotberg, 1972). “Hippies espalham a lepra” has its United Kingdom equivalent “Immigrants bring in leprosy”, but such exaggerations invite correction from authoritative bodies, and serve to further the education of the reading public. Journalistic irresponsibility is very much here to stay all over the world and may be relied upon to perpetuate the damaging myths of leprosy, as well as other diseases and conditions. Rather than shrug our shoulders at the profundity of malevolence or ignorance of this kind, we take advantage of the occasion to put a factual case to a public whose interest has, however sensationally, been aroused. Such opportunities are infrequent, and it is certainly more difficult to get correct information printed for its own sake, since there exists a paradoxical conviction among newspaper editors which denies that there is any public interest in the disease.
Such a stance as that of journalists assumes the existence of some degree of public stigma of leprosy, and it is in this connection that the paper of Gussow and Tracy (1972) is of particular interest. Granted that all circumstances in the United States do not apply elsewhere, the fact that the presumption of stigma by anti-leprosy workers is not borne out by scientific investigations suggests the need for the provision of as much evidence about other countries before conclusions can be reached, and certainly before educative programmes are drawn up. The paper is an invitation to us all to reassess our assumptions of positive public attitudes, and a warning that we may be wide of the mark in the particular area of assuming universal hostility towards leprosy and the leprosy sufferer. The suspicion arises that it may be our own familiarity with leprosy and its traditional associations which makes us ascribe traditional reactions to the public which is, in fact, indifferent, and which does not replace out-dated reactions with any attitude towards actual leprosy, so much as maintains a familiarity with a "disease called leprosy," which is of curiosity and historical interest. The strength of appeals based on this assumption may begin to emerge, and equally the opportunity of supplanting myth with fact, emphasizing that the exaggeration in the myth has no application in consideration of the actual disease. So, we may be able to achieve a possible objective, of defining actual leprosy as a "mildly contagious condition, effectively and cheaply checked by prompt and regular treatment" only because, in doing so, we can deny its high contagiousness and incurability, both of which are assumed by the public.

These considerations of the strategy applied to fund-raising in non-endemic countries may seem irrelevant to the problems faced by anti-leprosy workers in endemic countries. But certain aspects of the situation appear to be universal, not least among these the lack of tangible evidence of the degree or nature of stigma. Even accepting that the stigma was intensely felt in all endemic countries, should not the same strategy apply as that which seems to work among indifferent and unthreatened people elsewhere?

The populations of endemic countries divide themselves into the suffering and the non-suffering, and the latter group has two definitions. They are at once potential victims and potential repositories of attitudes towards actual victims. Resistance to educative measures which aim at protecting them from a hazard has a number of counterparts in many societies. The dangers of drug-taking, smoking, alcoholism and road accidents all seem to fall into a category of items about which the public would rather not think at all, but the significant difference between leprosy and these other hazards is that the resistance to knowledge is based in this case on false assumptions rather than on facts chosen to be ignored. Programmes of propaganda based on the slogan "Leprosy is NOT . . ." therefore have an immediate advantage over measures in other areas which have to create information from the outset. The same applies to dealing with non-suffering endemic populations in respect of their attitude towards leprosy sufferers. Again, the essential ingredient of such campaigns is the acknowledgment of existing public misunderstanding and the conception of this as an opportunity for correction, and this necessitates an identification of the disease which is misunderstood, by the name by which it is misunderstood.

Inevitably, this leads us to further consideration of the absorbing paper of Rotberg (1972) and to the debate on nomenclature. One approaches such a paper with profound humility, and with acknowledgment that the deeply felt plea is for help with a local problem. This in turn invites a desire to agree with the
recommendations, but, despite the wide catalogue of arguments rebutted, the paper omits a vital consideration. What happens to the terminology of leprosy after the successful adoption of a substitute vocabulary?

We have to accept that traditional misunderstandings about leprosy are embodied in the cultures of both currently endemic and non-endemic countries. These misunderstandings have given connotations to a range of terms whose medical usage is lost on the public at large, but which form valuable instruments of communication, particularly in the descriptive area (Skinsnes and Elvove, 1970). Such usages will not die out, since they are based on casually acquired misinformation and the correction of terminology in restricted circles will not cause so much as a ripple on the mainstream of public usage. This is evidenced by the headline which includes the word “leprosos” despite efforts, which we realize have been considerable, for the exclusion of the word, and in the United Kingdom, even among informed supporters of anti-leprosy work, as well as among politicians and footballers, the word “leper” perhaps only used for its conciseness, and dead though it may be in medical circles, will not conveniently lie down in public. It is perhaps worth saying in parentheses that the suggested portmanteau “Hanseniasis—formerly leprosy” just as any suggested alternative single word, suffers in a comparison with the methods used in currency changes because of the lack of opportunity for the public at large to exercise with the new term, which is vital to any process of linguistic change.

Accepting that we are few indeed to attempt to take on the entire world population in this question of linguistic misuse, might we not do well to consider some of the factors in the creation of the problem to see if there may be other influences which might have a more profound effect in obviating the need for public re-education as to the true nature of leprosy?

The paper from Brazil already cited listed a number of influences which persist in propounding the legends of leprosy as opposed to its actual nature and referred among these to religious instruction. It is surely here that all the problems begin and to this same point that they all return. Certainly the Christian motivation in anti-leprosy work has effectively covered the endemic world, so that even cultures where other traditions are in play have been made aware of the special significance given to succouring leprosy victims by the Christian Church.

It is agreed that leprosy as referred to in the Bible is nowhere necessarily—and in some places certainly not—leprosy as currently clinically defined (Browne, 1970). The attention that Christianity has paid to Biblically defined “lepers” has reflected the vagueness of the original terminology over the ages. It is only after 1873, at a time when it became possible to define leprosy by reference to its presumed causative agent, that it became “the disease caused by Mycobacterium leprae”, and Christian, or any other motivation, could be related to this specific disease. That the disease did not change because of the definition is self-evident, as it is that the disease was the subject of investigation and concern beforehand, but the particular concern for “lepers” reached a crossroads at a point in time when “lepers” became only those who suffered from the specific defined disease. That these people were at best a portion of those for whom the original concern was urged is also agreed (Browne, 1970). It is the most extraordinary and paradoxical coincidence that one modern manifestation of concern originates in 1874 with the foundation of The Leprosy Mission, whose contribution towards the anti-leprosy campaign has been properly acclaimed.

Once leprosy sufferers could be defined in the modern sense, the nature of the
Christian motivation towards them embodied all the Biblical connotations of the disease called leprosy into their identity, at a time when, if ever, their identity might have changed. Further, the evangelization of leprosy sufferers added another contribution to their list of isolating qualifications. It has, through the desire to evangelize and through misconception, been in the interest of Christians to see modern leprosy sufferers as "Biblical lepers", and we have only to consider the rôle that missionary doctors have played and continue to play in effective field work to accept that this interest will continue, and will go on inviting the same confusion as to the similarity between those who suffered from the disease called leprosy and those who are today's leprosy sufferers.

It seems redundant to observe that the terminology of leprosy should have been varied at the time of Hansen's discovery, but such a shift would have emphasized the medical factors in the leprosy sufferers' identity. As it is, a hundred years of reinforcing the semantic confusion has taken us beyond the point where verbal manipulation stands any chance of success, and where it would not deny us the vestigial consciousness of the disease's existence in non-endemic, fund-providing communities. The cart is now truly before the horse, and we might do well to think of ways of isolating the associations of the "disease called leprosy" from those of actual leprosy, as a means of avoiding reliance on public co-operation which will certainly not be forthcoming.

In the first place, it should be possible to impress appropriate religious authorities with the desirability of eschewing reference to leprosy in reproductions of religious texts. This would ensure that future generations will not associate out-worn connotations with the actual disease. Such measures as have already been taken in this direction (Browne, 1970) leave enough to be desired to deny their value altogether. Secondly, reference to "actual leprosy" whenever appropriate in publications of all kinds would effectively dissociate "the disease called leprosy" from the disease as defined. The epithet carries a strong sense of "current" in Romance and non-Romance languages.

If there is a further contribution that anti-leprosy workers in all areas of the subject can accomplish "at a stroke" it is through a determination not to perpetuate leprosy myths by exaggerating the significance of uninformed statements. Incidents of the kind quoted by Pedley (1972) generalize by implication from insignificant episodes, and create myths of public reaction among leprosy workers that outweigh the myths of leprosy among the public.

References
Rothenberg, A. (1972). The serious Latin American problems caused by the complex "Leprosy, the Word, the Disease" and an appeal for World co-operation. _Lepr. Rev._ 43, 96.
The Effect of Stopping Dapsone Treatment for Two Months and then Restarting it in Full Dosage in Patients with Moderately Severe *Erythema Nodosum Leprosum*

J. M. H. PEARSON†

Physician, Leprosy Research Unit,
National Leprosy Control Centre,
Sungei Buloh, Malaysia‡

and

H. S. HELMY

Medical Officer, National Leprosy Control Centre, Sungei Buloh, Malaysia

Nine patients suffering from persistent, moderately severe *erythema nodosum leprosum* (ENL) were included in this study. During a period of 8 or 10 weeks when anti-leprosy treatment with dapsone was replaced by a placebo capsule, there was a fall in the severity of the ENL which just attained statistical significance. When dapsone treatment was restarted in full dosage (100 mg daily), using a capsule identical to the placebo, there was no effect on the ENL for the first 2 weeks, and a slight (but statistically insignificant) increase in its severity thereafter.

*Erythema nodosum leprosum* (ENL) was first recognized, and indeed named, long before the development of chemotherapy against leprosy (Murata, 1912). However, with the introduction of effective anti-leprosy drugs, ENL became a more common and much more serious problem in the management of patients with lepromatous leprosy. The sulphone drugs, and particularly dapsone (4',4 diaminodiphenyl sulphone, DDS) were the first such drugs to be used; at first the increased incidence of ENL was considered to be a complication caused by the drug itself, and the practice of discontinuing dapsone treatment in the presence of ENL was introduced.

It has, however, since become apparent that treatment with any effective anti-leprosy drug is liable to be associated with ENL; and moreover the

---

* Received for publication 18 January, 1973.
† Requests for reprints of this paper should be addressed to Dr J. M. H. Pearson at the National Institute for Medical Research, Mill Hill, London N.W.7, England.
‡ Present address: Armauer Hansen Research Institute, P.O. Box, 1005, Addis Ababa, Ethiopia.
mechanism of ENL, long suspected to be "allergic", is now better understood, and appears to be a manifestation of the toxic effect of antibody/antigen complexes (Wemambu et al., 1969). Thus the relationship between the anti-leprosy drug and ENL is not a direct one, but indirect, due to the effect of the drug in causing the death of the leprosy bacilli. Subsequent autolysis and disintegration of the bacilli with the release of antigenic material is probably not in any way affected by anti-leprosy treatment.

Despite a strong general opinion that ENL becomes more severe when treatment with dapsone in high dosage is employed, and less severe when this treatment is discontinued, there has been no adequate trial of the effect of these manoeuvres on the severity or frequency of episodes of ENL. Recently, however, methods have been developed at the Leprosy Research Unit, Sungei Buloh, Malaysia, for testing the effect of drugs against ENL, and the technique has been applied to evaluate the action of thalidomide (Pearson and Vedagiri, 1969; Waters, 1971) and clofazimine (Helmy, Pearson and Waters, 1972). It has been shown to be sufficiently sensitive to detect the beneficial effect of clofazimine in a dosage of 300 mg daily within the first 3 or 4 days of starting treatment. In this paper we describe a trial in which we used the method to assess the effect of discontinuing and then re-introducing dapsone treatment in a group of patients with active, fairly continuous, and moderately severe ENL.

Patients and Methods

The patients were selected from among those attending the Research Unit; some were in hospital wards, but the majority were living in quarters within the hospital grounds and attending Research-Unit wards for treatment. All the patients had been suffering from ENL for at least 6 months prior to the study, and it was known therefore that their reaction was fairly continuous and did not vary greatly in severity from week to week. One patient required prednisolone during most of the trial period, but in the others the reaction was usually controlled satisfactorily by analgesics and antimonials (stibophen), though 3 patients had to have prednisolone for periods of several weeks during the trial.

The trial followed the general pattern of our previous studies, i.e. standard anti-ENL treatment was employed in addition to the trial treatment, and the benefit of the trial drug was measured by its sparing effect on the requirement of the standard drugs.

DESIGN OF THE TRIAL

The trial was divided into 4 periods as follows.

Period 1—Initial control period (weeks 1-4); during this time all patients continued on their normal anti-leprosy treatment.

Period 2—Placebo period (weeks 5-12 or 5-14); all patients received one placebo capsule daily.

Period 3—Dapsone period (weeks 13-16 or 15-16); in this period patients received every day a capsule, identical in appearance to the placebo but containing dapsone, 100 mg.

Period 4—Final control period (weeks 17-20); the patients reverted to their usual treatment, as in the initial control period.

Patients received the dapsone capsule for either 2 or 4 weeks according to a random distribution plan, the key to which was not known till the trial was
completed and results tabulated. The trial was thus a blind study of the effect of stopping anti-leprosy treatment, and a double-blind trial of the effect of restarting dapsone in full dosage.

During the period of the trial all the patients attended the Research Unit daily (except Sundays) at about 2 p.m. Here their temperature was recorded, and during weeks 5-16 a capsule was issued and seen to be swallowed.

All patients were seen by a doctor twice a week and the severity of ENL, presence of nerve pain or tenderness, and treatment received since the previous visits were recorded. Every 2 weeks and sometimes more frequently, the urine was checked for dapsone content. White blood-cell counts were performed approximately every 2 weeks; but for technical reasons the timing was irregular and the results, which do not affect the findings, are therefore not included.

METHODS OF SCORING

On each occasion when the patient was examined the clinical findings (i.e. the number of ENL lesions, “activity”, and extent of nerve involvement) were recorded. The treatment received during the period since the previous assessment was also noted, as were also the maximum temperature and the results of the W.B.C. count if performed.

Of these assessments of the severity of ENL, only the temperature and W.B.C. count are objective; the most important parameters (i.e. the number and “activity” of lesions and the presence of nerve pain and tenderness) can only be subjective judgements. However, with experience, assessments and treatment become more consistent and thus it is possible to evolve a reproducible “scoring system” (see Table 1) allotting numerical values to the clinical findings.

While effective anti-reaction treatment is being administered the reaction is decreased, and the “clinical score” alone is not a true index of the severity of the ENL; it is necessary therefore to allot additional “points” for anti-ENL treatment. Moreover, if the score is to represent the true severity of the ENL, the total score (for clinical severity plus treatment) should amount to roughly the figure that the clinical score would have reached in the absence of anti-ENL treatment. The system shown in Table 1 was designed to achieve this balance.

Results

A total of 14 patients were admitted to this study, but 2 were withdrawn during the period of the trial—1 patient on account of haematemesis, and the other because she was found to be pregnant. A further 3 patients completed the trial but were not included in the analysis—in 2 cases because tests for the presence of dapsone in the urine indicated that it was being surreptitiously taken during the period of placebo treatment; in the 3rd case the tests suggested that in the initial control period the patient was not taking the full dosage of dapsone prescribed.

Clinical details of the 9 patients finally included in the study are given in Table 2. The diagnosis both of lepromatous leprosy and of ENL was confirmed histologically, in the latter case just before or during the course of the trial. The period of anti-leprosy treatment varied between 2 and 5 years, except that one patient who had been treated with thiambutosine for 11 years developed resistance to this drug, and had received dapsone for 9 months before the start of the trial. The period during which the patients had been suffering from ENL
Table 1: Clinical grading of ENL and scores allotted for different parameters on each twice-weekly examination

<table>
<thead>
<tr>
<th>Score</th>
<th>ENL Lesions</th>
<th>Nerve involvement</th>
<th>Maximum temp. recorded</th>
<th>Anti ENL treatment</th>
<th>Total white blood cell count</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
<td>None</td>
<td>Below 99°</td>
<td>Nil</td>
<td>0-9900</td>
</tr>
<tr>
<td>1</td>
<td>Few</td>
<td>Indolent, 1 tender</td>
<td>99 to 99°</td>
<td>1-4 cc, 25 IU, 0-19 mg</td>
<td>1 injection, 10,000-13,900</td>
</tr>
<tr>
<td>2</td>
<td>Moderate number</td>
<td>Moderately active</td>
<td>More than 1 tender</td>
<td>5-8 cc, 50 IU, 20-29 mg</td>
<td>2 injections, 14,000-17,900</td>
</tr>
<tr>
<td>3</td>
<td>Many</td>
<td>Active, a few ulcerating or almost ulcerating</td>
<td>1 painful</td>
<td>-</td>
<td>75 IU, 30-39 mg, 3 injections, 18,000 or more</td>
</tr>
<tr>
<td>4</td>
<td>Many</td>
<td>Active, many ulcerating</td>
<td>More than 1 painful</td>
<td>-</td>
<td>100 IU, 40-49 mg, 4 injections</td>
</tr>
</tbody>
</table>

a Other nerves may or may not be tender.
b The score increases with further increase of dosage: i.e. 70-79 mg = 7.
c This consists of perineurial injection of hydrocortisone acetate, 25 mg in 2% procaine with added hyalase.
DAPSONE TREATMENT

TABLE 2
Details of trial cases.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Duration of treatment (years)</th>
<th>Dosage of DDS (mg twice weekly)</th>
<th>Duration of ENL (years)</th>
<th>Clinical assessment* at finish of trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>16241</td>
<td>2</td>
<td>100</td>
<td>1½</td>
<td>Improved</td>
</tr>
<tr>
<td>16011</td>
<td>5</td>
<td>100</td>
<td>4½</td>
<td>Improved</td>
</tr>
<tr>
<td>15657</td>
<td>4</td>
<td>100</td>
<td>2</td>
<td>Improved</td>
</tr>
<tr>
<td>16206</td>
<td>2½</td>
<td>100</td>
<td>2</td>
<td>Worse</td>
</tr>
<tr>
<td>49125</td>
<td>3½</td>
<td>100</td>
<td>3½</td>
<td>No change</td>
</tr>
<tr>
<td>13399</td>
<td>11</td>
<td>100</td>
<td>½</td>
<td>Worse</td>
</tr>
<tr>
<td>15631</td>
<td>3</td>
<td>50</td>
<td>3</td>
<td>No change</td>
</tr>
<tr>
<td>16359</td>
<td>5</td>
<td>100</td>
<td>2</td>
<td>Worse</td>
</tr>
<tr>
<td>16030</td>
<td>4½</td>
<td>100</td>
<td>4½</td>
<td>Not assessed</td>
</tr>
</tbody>
</table>

*This is an assessment of the underlying leprosy condition, and not of the ENL.

ranged from 6 months to 4½ years; but none had received treatment with clofazimine or thalidomide during the 3-months' period before the trial started. All the patients were receiving dapsone initially—the dosage is shown in Table 2.

Because the study included a 2-month period during which these patients were receiving no anti-leprosy treatment, the state of the leprosy was assessed by an independent clinical assessor at the start and finish of the trial. These findings are shown in Table 2; 3 patients were considered improved, 2 unchanged, and 3 worse. (One case was not assessed.)

The total scores for all patients by all criteria are shown in Table 3; they are divided into 9 periods of 2 weeks each. These periods consist of the first 4 weeks initial control period, and the first 8 weeks of placebo treatment. At this point 4 patients restarted dapsone, the remaining 5 continuing on the placebo for a further 2 weeks. Thus the scores for the first 2 weeks on dapsone were in 4 cases those of weeks 13 and 14, and in the other 5 cases of weeks 15 and 16; in 5 cases the final 2 weeks on placebo have been omitted from this Table, and in 4 cases the second 2 weeks on dapsone, 100 mg daily. The final control period, when the

TABLE 3
Total scores of all patients throughout the trial period

<table>
<thead>
<tr>
<th>Weeks</th>
<th>Initial control period</th>
<th>Placebo period</th>
<th>1st 2 weeks on DDSa</th>
<th>Final control period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-2 3-4 5-6 7-8 9-10</td>
<td>11-12</td>
<td>13-14/15-16</td>
<td>17-18 19-20</td>
</tr>
<tr>
<td>ENL</td>
<td>104 79 77 70 45 62</td>
<td>44 107 121</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nerves</td>
<td>10 3 15 9 3 5</td>
<td>6 25 15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>20 8 13 16 18</td>
<td>11 13 21 13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steroids</td>
<td>42 25 32 23 22</td>
<td>36 25 13 13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stibophen</td>
<td>32 18 25 18 14</td>
<td>13 14 20 16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>208 133 162 137 102 127 102</td>
<td>186 178</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a In 4 cases, weeks 13 and 14; in 5 cases, weeks 15 and 16.
dosage of dapsone was the same as in the initial control period, was in all cases weeks 17 to 20.

Comparing the scores for the initial and final control periods, it will be seen that although there is a wide range of totals (133 to 208), the average fortnightly scores for the initial and final control periods are very close to each other (171 and 182 respectively). It seems likely, therefore, that there was no serious alteration in the severity of the ENL during the 20-week period of the trial.

The total scores during the period of placebo treatment also show a wide range (102 to 162): the average is 132, which is 25% below the scores for the initial and final control periods. If the figures for weeks 5 and 6, when the patients could still not have excreted all their dapsone, are excluded, the average is 122 (a fall of 32%).

The period of 2 weeks when dapsone was reintroduced, in a dosage of 100 mg daily, was not marked clinically by a recrudescence of the ENL, and the total scores for this period of 2 weeks remained in the same range as those during the placebo period. This absence of acute flare-up of ENL is confirmed by Table 4, in which the scores for the last week of placebo treatment and the first week of dapsone treatment are shown.

The fortnightly scores for the last 4 weeks of placebo and the first 6 weeks of re-starting dapsone therapy are shown in Table 5. They confirm that there was a

<table>
<thead>
<tr>
<th>TABLE 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total scores during the last week of placebo treatment and the first week of DDS treatment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Score during last week of placebo</th>
<th>Score during first week of DDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity of ENL</td>
<td>27</td>
</tr>
<tr>
<td>Nerve involvement</td>
<td>0</td>
</tr>
<tr>
<td>Maximum temperature</td>
<td>6</td>
</tr>
<tr>
<td>Dosage of steroids</td>
<td>7</td>
</tr>
<tr>
<td>Dosage of stibophen</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>The effect of restarting DDS on the severity of ENL</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Placebo period</th>
<th>Period on DDS treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Next to last 2 weeks</td>
<td>Last 2 weeks</td>
</tr>
<tr>
<td>ENL</td>
<td>52</td>
</tr>
<tr>
<td>Nerves</td>
<td>5</td>
</tr>
<tr>
<td>Temperature</td>
<td>17</td>
</tr>
<tr>
<td>Steroids</td>
<td>36</td>
</tr>
<tr>
<td>Stibophen</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>124</td>
</tr>
</tbody>
</table>
delay of some 2 weeks after re-starting dapsone before the scores increased. This delay is still seen if the 2 groups of patients are tabulated separately (Table 6), though in one group the score showed some increase in the initial 2 weeks of dapsone. The total scores were higher in fortights 9 and 10, though the dapsone dosage was only about one-quarter of that received in fortnight 8.

**TABLE 6**

_Total scores during trial period_

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of cases</th>
<th>Fortnight of trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases restarting DDS</td>
<td></td>
<td>1 2 3 4 5 6 7 8 9 10</td>
</tr>
<tr>
<td>in fortnight 7</td>
<td>4</td>
<td>110 66 69 27 22 25 42 50 63 65</td>
</tr>
<tr>
<td>Cases restarting DDS</td>
<td>5</td>
<td>96 67 93 109 80 102 74 63 113 108</td>
</tr>
<tr>
<td>in fortnight 8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Discussion**

Despite the small number of patients included in this trial, two negative findings are quite clear: (1) there was not a dramatic fall in the severity of ENL when dapsone was discontinued; and (2) there was not an acute exacerbation of ENL when dapsone was restarted in maximal dosage after 2 months of treatment with a placebo.

Both of these findings are in agreement with current understanding of the bacteriostatic chemotherapeutic action of dapsone (Bushby, 1964) and of the pathogenesis of ENL (Wemambu et al., 1969). The only reservation that might be made is that these patients were a selected group, with a particular severity of ENL. The selection was deliberate; they were chosen as being those most likely to demonstrate minor changes in the severity of ENL (Pearson and Vedagiri, 1969). It is possible, though unlikely, that different selection would alter the results.

Positive findings are harder to evaluate. The figures give the impression that there was a mild reduction in the severity of the ENL during the placebo period and an increase on restarting dapsone, with a lag period in each instance of 2 weeks or so. This impression is not contradicted by study of the figures for individual cases. The number of patients included in this study is small for statistical analysis, but the fall in weeks 9 to 12 just attains statistical significance (North, 1972). It was, however, much less evident in the group of patients restarting treatment in week 15 (Table 6).

The scoring system cannot be directly related to the severity of the ENL, i.e. a fall of 25% in the score should not be read as equivalent to a fall of 25% in the severity of the ENL. It is clear that trials on a much larger scale will be required if it is considered desirable to demonstrate accurately the degree of improvement that occurs when dapsone treatment is discontinued for 2 or 3 months. This study, however, demonstrates incontrovertibly that ENL is not abolished by this manoeuvre, and suggests that it is only marginally affected. Further, it gives no information on the effect of lowering the dosage of dapsone during reactions. *Myco. leprae* is highly sensitive to dapsone (Rees, 1967) and a dosage as low as 1 mg daily is chemotherapeutically effective (Waters, Rees and Ellard, 1968). Such low dosages, however, may predispose to the emergence of dapsone...
resistance and should be avoided in the management of lepromatous leprosy; the effect in alleviating the severity of ENL is unlikely to be great enough to be of practical value.

This small scale trial makes it clear that no great improvement in ENL can be expected even if dapsone treatment is totally discontinued for 2 or 3 months. Similarly, it is clear that initiating anti-leprosy treatment in full dosage when ENL is present does not cause a violent flare-up of the reaction. Both these findings have immediate application to the management of ENL, and could considerably simplify the problems of management of ENL for the paramedical workers who usually have responsibility for such cases.

Acknowledgements

This trial was designed in collaboration with Dr R. J. W. Rees of the National Institute for Medical Research, London. We are grateful to Dr M. K. Bhojwani, Director, National Leprosy Control Centre, Sungai Buloh, Malaysia, for the clinical assessments of patients in the trial and for advice during the preparation of this paper; to Dr D. S. Ridley, Hospital for Tropical Diseases, London, for the biopsy reports; to the Malayan Pharmaceutical Factory Sdn. Bhd., for the generous gift of dapsone and placebo capsules used in the study; and to our staff and patients for their support and co-operation. One of us (H. S. H.) wishes to thank the Malaysian Ministry of Health for permission to publish. The Leprosy Research Unit, Sungai Buloh, is jointly sponsored by the Malaysian Ministry of Health and the (British) Medical Research Council.

References


The Effects of Cocktail Anaesthesia on Blood Pressure*

A. GRACE WARREN  
Medical Superintendent, Hay Ling Chau Leprosarium, Hong Kong

and

PHYLLIS M. TAYLOR  
Previously at Hay Ling Chau  
Physician, S.I.R.S. Karigiri, South India

In leprosy centres, the absence of a second trained doctor to give anaesthetics often limits the surgeon wishing to operate. "Cocktail anaesthesia", as described here, has proved to be useful in this and other situations, and is often preferred to general anaesthesia by those accustomed to administering it. Many different drugs have been used for this purpose. In this paper a method that has been found effective in Chinese patients is described, with details of observations that indicate its safety and adaptability.

Introduction

In leprosy patients, surgery is required most often for deformities of the hands, feet or face. Since trained staff are not always available, a method of anaesthesia that does not require constant skilled supervision is an advantage. Local anaesthesia and regional block have their advocates, but these methods may not in all cases render bearable the discomfort that results from the pressure of the tourniquet. If restlessness should occur at the critical time when the tendons are being sutured towards the end of the operation, then the consequences may be serious.

For the past 7 years, one of us (A. G. W.) has been using a type of lytic “Cocktail”, modified from one previously used at Karigiri. With this form of anaesthesia, the patient sleeps deeply throughout the operation and retains no memory of the procedure, but keeps his normal protective reflexes. He usually awakens some 3 or 4 hours later, with no nausea, vomiting, headache or any of the other side-effects that may follow general anaesthesia. Because questions have been raised concerning the fall in blood pressure that occurs, a study was undertaken on the effects of Cocktail Anaesthesia on blood pressure.

* Received for publication 26 January, 1973.
Materials and Methods

1. THE COCKTAIL (as given at Hay Ling Chau)

   (a) Premedication

   1. The night before the operation:
      amyllobarbitone (100 to 200 mg); with chlorpromazine (Largactil) (50 to 100 mg) if the patient is apprehensive or is known to have been uncooperative.
      On the morning of operation day a light meal is given at least 4 h before the time fixed for operation.
   2. Two hours before operation:
      amyllobarbitone (200 mg) and phenergan (25 mg) by mouth.
   3. One hour before operation:
      morphia (10 mg) and hyoscine (0.4 mg) by intramuscular injection.

   (b) Preparation of the Cocktail

      Between 20 and 30 minutes before beginning the actual operation, the Cocktail itself is prepared; this consists of: pethedine (100 mg), chlorpromazine (50 mg) and saline to 20 ml. (Note: distilled water or glucose may be used, though the latter may give a cloudy solution.)

   (c) Administration of the Cocktail

      In Chinese patients weighing about 50 kg, 8 to 10 ml of the mixture, administered intravenously, usually gives an analgesia of sufficient duration to enable the surgeon to perform such operations as foot-drop correction, lumbrical replacement, or triple arthrodesis of the ankle. In the elderly or the young, a smaller dose of morphia may be given as premedication and a smaller volume of the Cocktail. In an elderly subject, 2 or 3 ml of the Cocktail is often sufficient for foot-drop correction. The maximum depth of sedation is attained in 20 to 30 min; if sedation is not adequate, a further dose of 2 to 5 ml may be given, and repeated as required.

   (d) Post-operative care

      After the operation the patient is returned to the ward; his head should be turned to one side to help maintain the airway. Close supervision is not required, since the patient is rarely restless, and he will answer to his name in a few hours. If not roused, he may pass from post-operative haziness into normal sleep. There is no nausea, vomiting, or headache, and the appetite is unimpaired. Urinary retention is almost unknown.

2. PATIENTS

      In this series all patients chosen for operation who would normally have been given “Cocktail Anaesthesia” were at first included; even those patients known to be, or to have been, addicted to “hard” drugs. However, those whose operation was of less than one hour’s duration are excluded from this report, since insufficient data are available. The 77 patients included, comprise 69 men and 8 women, their ages ranging from 16 to 73 years.

3. SCHEDULE OF OBSERVATIONS

      Blood pressure, pulse rate, respiration rate were recorded: (a) the day before surgery, (b) before sedation, (c) before anaesthesia, and then (d) every 10 min
after the intravenous injection of the Cocktail until the patient was returned to the ward.

**Results and Discussion**

1. **BLOOD PRESSURE**

The maximum fall in systolic blood pressure, as compared with the recording taken the day before operation, is shown in Fig. 1. The patient who had a fall of 70 mm Hg systolic had an initial blood pressure of 170/90. While he was under sedation his blood pressure was 100/70. This apprehensive patient has a very labile blood pressure. In the majority of patients the fall in systolic blood pressure was not more than 30 mm (60 cases out of 77).

![Graph showing maximum fall of systolic blood pressure](image)

Fig. 1. Maximum fall of systolic blood pressure, after the intravenous injection, as compared with the reading taken the day before operation.

Figure 2 shows that the maximum fall in blood pressure occurred most commonly within 30 min of the intravenous injection. Although 2 patients had a low blood pressure and a pulse pressure of less than 20 mm Hg, as shown in Fig. 3, they presented no other evidence of shock. Both had a pre-operative blood pressure of 100/70 mm Hg.

2. **PULSE**

The pulse rate is often raised before the operation, but usually settles after the premedication, and may slow more after the intravenous medication is given. In fact, it was noted that although the blood pressure fell markedly in a few patients, the pulse showed no compensatory rise (Fig. 4).

3. **RESPIRATION**

Patients who had rapid shallow respirations at the beginning of the operation usually breathed more slowly and more deeply within 15 min. In no case did the
Fig. 2. Scattergram to show the relation between the time taken for the maximum fall in blood pressure to occur and the size of that fall.
Fig. 3. Distribution of the pulse pressure correlated with the minimum diastolic pressure.

Fig. 4. Scattergram to show the relation between the minimum systolic blood pressure and the pulse rate.
respiratory rate fall below 10 per min, nor did any patients become cyanosed. We consider that the respiratory rate is a good guide to the safety of giving a supplementary dose of cocktail should this become necessary. This should not be given if the rate falls to below 10 per min; rather, oxygen should be given until the rate has returned to normal. Since tone of the jaw muscles is maintained, no airway or jaw support is usually required; it is found that simple posturing is adequate to maintain an adequate airway. In a few patients undergoing operations on the head or neck, a rubber airway was well tolerated.

4. HYDRATION EFFECTS

We do not routinely give intravenous fluids during or after operation. All patients are admitted to hospital for at least 36 h before operation; adequate hydration can therefore be achieved in this period, and fluids are given by mouth as soon as the patient is able to take them. Dehydration has rarely been serious during or after the operation, even during the humid heat of summer. However, it is not advisable to use this type of anaesthesia for patients who are already suffering from oligaemic shock or in whom marked blood loss is expected.

5. ENVIRONMENTAL EFFECTS

Most of the patients in this series were operated on in summer and in an air-conditioned theatre. This probably saved them from the consequences of reflex vasodilatation which may occur in a non-air-conditioned theatre: this effect is accentuated by the heat conserved by coverings and also that of the theatre lamp.

6. OTHER EFFECTS

Because the reflexes are maintained, external stimuli may cause rapid fluctuations in pulse and blood pressure. This was noticed when the patient complained of discomfort because of the pressure of the tourniquet, or during the more painful procedures of the operation, or in being moved to the ward. Patients frequently become restless after about 1 h, but once the tourniquet is released they soon become quiet and relaxed. If the tourniquet has to be reapplied, patients usually tolerate the pressure for half to three-quarters of an hour before needing further intravenous medication. This may indeed be given, but the patient may lapse into a prolonged heavy sleep.

Comments

1. USES

Cocktail Anaesthesia can be used in many situations where there is a staff shortage and yet surgery needs to be performed. Supplementation with local anaesthesia makes it suitable for procedures such as thyroidectomy and simple abdominal procedures, where full muscle relaxation is not required. It is ideal for minor operations on the hand or foot, such as curettage of abscesses or sinuses.

2. DISADVANTAGES

The interval between the injection and the attainment of the required level of analgesia is variable. Since patients retain deep reflexes, spasmodic muscle movements may occur if nerves are stimulated. This does not however indicate that the patient feels pain.
References


A Developing Country, Leprosy Control, and the Severely Disabled*

THOMAS F. FRIST
Calle de la Paloma, 10, Bajo 1, Madrid 5, Spain

Patients who are severely disabled by reason of past leprosy are frequently neglected by government planners and communities, when emphasis is increasingly laid on medical care and preventive measures. Even in the presence of economic stringency, efforts should be made to help these patients.

Introduction

Although leprosy policy planners in Tanzania emphasize the medical aspects of the disease, they are also interested in helping the patient to deal with his disease-linked, non-medical problems. On the basis of this concern, the writer undertook a study of severely deformed patients in the four major leprosy settlements† of a central region of the country to see if their difficulties warranted special external assistance. The following is a synopsis of the findings, and various suggestions emerging from this investigation.

The Four Settlements in an Historical Setting

Large-scale care of leprosy patients in Singida region probably began in the early 1900's near the government posts of Kilimatinde and Mkalamá to which patients often came seeking medical help. Some officials did what they could for the patients, but worsening conditions in the settlements soon caused the government to turn to nearby missions. Around 1930, the Church Missionary Society (CMS) agreed to supervise the leprosy work in the Nunge settlement near Kilimatinde, and in 1947 the Augustana Lutherans at Mkalamá and the Lutheran dispensary at Tintigulu were giving the only available treatment for leprosy. In time, the Nunge and Mkalamá settlements developed into leprosaria, each treating around 1000 patients. At both places the growing of food presented problems, since the land at Mkalamá was rocky, and that at Nunge was too dry. Only at Tintigulu was the land good for agriculture. The situations at Nunge and Mkalamá finally deteriorated to such a degree that their sponsors began to look for more fertile areas in which to relocate. In 1959 both leprosaria did move, the Lutherans to Iambi in the Singida region, and the CMS to a neighbouring region. Former patients at Nunge, Mkalamá, and Tintigulu who no longer required medical attention, remained. The severely deformed patients left in these 3 settlements,

* Received for publication 29 January, 1973.
† "Leprosy settlements" will be used here to signify locales with very high densities of patients with active or "burnt out" leprosy who are largely dependent on their own resources.
together with those in a 4th that has since developed outside lambi leprosarium itself, form the subject of the present paper.

**Methodology**

The writer studied especially the severely deformed patients in the 4 settlements on the assumption that these would also be the most severely disabled, and therefore the most in need of external assistance. A sample was attempted of those who “looked” severely deformed or disabled. Such a subjective criterion for selection must be explained. A survey of patients attending re-opened leprosy clinics in the settlements revealed that at least 22% of the 300 to 400 patients in each of the settlements were “severely disabled” (World Health Organization, 1970: *Field Classification of Leprosy Disabilities*). It was, however, apparent that most of them were not in reality so disabled that they could not pursue their usual work. Therefore only those who seemed to be obviously and severely disabled (the blind, and patients with several paralysed or absorbed fingers or toes) were interviewed; about 130 such patients were eventually located through clinic surveys, local clinic chiefs, community leaders, and fellow patients.

The patient’s disabilities were assessed according to the “Tanzanian Workman’s Compensation Chart” and 80 questions (dealing with the interviewee’s background) were put to each patient. The interviews were conducted in Swahili or in a local dialect.

**Results**

**GENERAL CHARACTERISTICS**

Of the 129 patients interviewed, 59% were male: men were more likely to have lepromatous leprosy, to be better educated, to speak some language other than their tribal dialect, and to have been employed at some time. The deformed patients were generally older than the average leprosy patient in the settlements. Only 4% were aged under 30 while 29% were over 60 years of age. About one-fourth of Singida’s population (458,000) are reported to be Christian; 67% of the patients studied gave this as their religion.

**Medical Information**

Of the 129 patients 31% had lepromatous leprosy; 95% learned that they had leprosy from someone other than a doctor or clinic worker; 52% had close relatives suffering from leprosy, and 57% had tried “native” treatment before seeking medical advice. After beginning treatment with chaulmoogra oil or sulphones, 84% had been admitted, at least once, to a leprosy institution. Of these, 16% had spent less than 2 years as in-patients, and 15% more than 15 years, while 53% had been discharged from in-patient care more than 10 years previously.

Only 46% of the patients wore footwear or gloves to protect themselves, although 80% admitted having been instructed to do so; two-thirds of them had persistent ulceration of the feet or hands. The same proportion were taking sulphone treatment, and 15% had absconded.
ECONOMIC AND SOCIAL PROBLEMS

The severely deformed patients faced many economic and social problems related to their disease and resulting deformities; 44% were 100% disabled, and only 3% had under 24% disability. Significant differences (at the 5% level) existed between the groups in their ability to contribute to the support of their households; with increasing disability the patient's contribution decreased. About the same proportion of patients in each category had resorted to begging.

Most of the patients could not count upon the help of their families. They had either lost contact with them, or had married another leprosy patient with problems similar to their own; 70% had remained in the settlements near the institution after discharge. The fear of rejection was advanced by one-third (32%) as a reason for not returning. Of those who did go home 27% soon returned to the settlement because they had been repudiated by their families or communities, while of those who had been married at the time of diagnosis 71% were later divorced; 80% of these divorced patients afterwards married other leprosy patients.

Severe disabilities, lack of support from able family members, and poor land were important factors in the economic stress of patients. 66% had no money, poultry, or livestock, and not enough food to eat. The poorest area was Nunge, where 86% were in this category.

PATIENT'S ATTITUDE TOWARDS CONDITIONS

Most (80%) of the patients were basically unhappy, and 50% attributed their unhappiness to "leprosy". 25% thought that their leprosy had been caused by "witchcraft" and 87% imagined, on learning the diagnosis, that their lives would be ruined. Nearly all (91%) thought that if they had never contracted the disease they would be more respected, and 79% thought that they would be wealthier. Yet only 11% felt that they were now abused by their neighbours because of their leprosy.

ABILITIES OF THE SEVERELY DEFORMED

Despite their severe deformities most of the patients wanted to, and did in fact, contribute to the support of their households. Only 27% said that they could do nothing but beg. 62% of the severely disabled farmed occasionally, 40% cut wood, 39% cooked, and 15% reported that they carried heavy loads. While 94% desired government assistance, only 8% thought that their lot would be improved if the government took complete care of them.

The patients had, in general, low levels of skill. Only 8% had ever been employed in jobs other than farming; 86% had received no formal education, and 70% spoke only their tribal language.

Comments and Suggestions

Although most of the severely deformed patients in the settlement areas need help from outside, the government assistance is limited by lack of resources. The government has proposed two plans for assisting the settlement populations: (1) to remove all destitute and friendless patients to centres for the destitute, where they would receive food, clothes and shelter; (2) to encourage other settlement patients and their families to form ujamaa villages (collective farms);
for those who do so the government promised assistance in the form of dispensaries, schools, water supply, agricultural aid and marketing facilities.

The question may, however, be asked: do these proposals meet the felt-needs of the patients? The centre for the destitute would draw the patient away from his community—a genuine fear. Moreover, most patients did not consider that they needed such complete care. 53% had already joined *ujamaas* in the early stages, and 23% indicated that they would join if they could. The remainder did not want to join because they feared the consequent ostracism.

The government was therefore advised to create *ujamaas* rather than centres for the severely disabled. While *ujamaas* should be organized in other settlements, it was suggested that these should not be exclusively for leprosy patients and their families. Nor should they all be concentrated on agriculture, which may be economically insecure. Severely disabled patients should be encouraged to join the communities, and there be assigned useful but less physically demanding jobs, provided with special tools, or subsidized directly by the government through the *ujamaa* administration so that their presence would not jeopardize the success of the *ujamaa*. Helping the patient in his own community is not only more in accord with the wishes of the patients themselves and also more within the goals of leprosy-control philosophy, but it should prove less expensive than providing for the patient in a centre for the destitute. Nevertheless, such latter centres may be necessary in some instances; but patients should be sent to them only after all efforts have failed to help them in their own communities. Since only 30% of the patients knew any other than their own tribal language, removal to a distant centre would entail an added linguistic hardship.
A correction would seem in order for a misrepresentation appearing on page 104, point (8), in the presentation by A. Rotberg, "Leprosy, the Word, the Disease," [Lepr. Rev. (1972) 43, 96-105].

In Hawaii from 1947 to 1969, the term "Hansen's Disease" was the legal term for leprosy. Cooperation in the use of this term during that period seems to have been as good as could reasonably be expected. Certainly the leprosy service and those connected with it made a distinct practice of using the term "Hansen's Disease" and the habit still prevails to a remarkable extent.

Dr Rotberg's sweeping contention that "there is not the slightest resemblance between the social, racial, cultural, religious and epidemiological conditions of Brazil and the State" is a bit exaggerated. However, his point that the situation in Brazil is different in many respects is perhaps well taken. Indeed, this is one reason that one wonders at the effort to change world-wide practice in order to achieve a social and cultural change in Brazil.

The official return to the use of the term "leprosy" in Hawaii was not in any way due to "the existence of a determined opposition by at least one influential author" (Skinsnes, 1966), and Dr Rotberg clearly did not make a reasonable "search of the literature" on this point, as he implies. The pamphlet referred to was sent to him by me, though not requested. In the scores of columns written in the local papers about leprosy since I became resident in Hawaii, there is not a word of "determined opposition" or even casual comment by me on the question of disease terminology. There is no writing by me in any Hawaiian publication on this matter.

The changes respecting the control of leprosy in Hawaii, including the use of the word "leprosy" resulted from long hearings, careful consideration, full discussion and deliberate recommendations made by an appointed "blue-ribbon" Citizens Committee composed, with one exception, of long-time residents of Hawaii, including leprosy patients. I appeared once before this committee as an "expert witness" regarding liberalization of rules of leprosy control and treatment and assuredly did not make any "determined" or other sort of plea regarding terminology. Nor was I then a member of that committee, having become a resident of this state only the year before the committee began its work, i.e. in August, 1967. Altogether 12 expert witnesses or committee members were available. My pamphlet, to which Dr Rotberg gives reference, had at that time no influential circulation in Hawaii that I am aware of, though it was circulated to the committee. The committee concluded, with respect to the matter of terminology: "Finally, the substitution of the term 'Hansen's Disease' in place of the word 'leprosy' only intensifies the problem it is supposed to eliminate—a centuries-old fear of the disease. The solution to this problem is proper education of the medical and lay community, not the substitution of an allegedly innocuous euphemistic term. If 'leprosy' is such a bad word that it should not be used, it automatically follows that the disease is unspeakably dread." The opinion was
that the 22 years’ experience in changing the disease name had done no significant good. No one crusaded for the change.

Dr Rotberg would have been more equitable had he made reference to my three publications relating to the problem of leprosy opprobrium which appeared in *Leprosy Review* (1964, 35, 21-35, 106-122, 175-181), on which studies the pamphlet he refers to were based. The pamphlet, incidentally, carries these references, and reprints were sent to him together with the pamphlet. The pamphlet is not likely to be available to many of your readers and the milieu of Dr Rotberg’s presentation leaves the impression that that writing is unsympathetic to the suffering brought by leprosy and is a “determined opposition” to the forces of good. The study referred to, on the contrary, was a long and extensively researched attempt to reach a rational rather than an emotional basis of understanding for the opprobrium of leprosy. The effort was subsequently reinforced by a later study (*Int. J. Lepr.* 1970, 38, 294-307) in which we noted and documented the fact that there now begins to appear in popular English language literature the use of the term “Hansen’s Disease” in exactly the same context of opprobrium that we all decry when associated with the term “leprosy.”

I did concur with the thinking and action of the Citizens’ Committee in this matter, though not having been responsible for it. The action was of interest to me in that it gave “experimental” support to my evaluation of the problem.

OLAF K. SKINNSNES

*Leprosy Atelier*

Leahi Hospital,
3675 Kilauea Avenue,
Honolulu, Hawaii 96816

26 March, 1973
Abstracts


The authors isolated in pure culture 27 fast-growing and 17 slow-growing mycobacteria from vegetation found in marshy areas in Uganda. Although none of the isolates had the cultural characteristics of *M. ulcerans*, the presence of these organisms on 49 out of 322 samples of grasses and sedges investigated suggests that the epidemiological indications that Buruli-ulcer infections could be implanted intradermally from such a source, may eventually be microbiologically confirmed.

*S. G. Browne*


The authors treated 4 leprosy patients with weekly intravenous infusions of leucocytes obtained from a pint of citrated blood (from healthy donors having had no contact with leprosy) and suspended in 150 ml of normal saline. The infusions were given very slowly. Three of the patients had lepromatous, and one tuberculous leprosy; all were intolerant of drug treatment.

All the patients responded rapidly to the infusions. Full case-notes are provided, together with clinical and histopathological photographs showing the appearance of lymphocytes and multiple small epithelioid cell follicles in the lymph nodes, and the rapid disappearance of *Myco. leprae*. After treatment, all the patients were able to tolerate standard drug therapy.

*S. G. Browne*

The following abstracts are reprinted, with permission, from *Trop. Dis. Bull.* 1973, 70.


In this investigation, carried out in San Francisco, USA, leprosy bacilli recovered from skin biopsy specimens on 11 patients with lepromatous leprosy who had not responded to sulphone therapy were tested for susceptibility to dapsone in the mouse footpad. The organisms from 5 patients were found fully susceptible, and it was later confirmed that these patients had defaulted on treatment. The organisms from the other 6 patients were resistant to dapsone, and 5 of these patients were treated with clofazimine (Lamprene; B663) in a dosage of 100 to 200 mg daily. The patients with susceptible bacilli were given 50 mg of dapsone daily, and in both groups the infectivity of the organisms for the mouse footpad was tested at intervals during treatment. It was found that bacterial killing began at the start of treatment with dapsone, but only after 50 days in those treated with clofazimine. Once started, however, the rate of bacterial killing was the same in both groups of patients.

*W. H. Jopling*
Dapsone (DDS) is converted in man to monoacetyldapsone (MADDS) by the same enzyme system that acetylates isoniazid, but MADDS is rapidly deacetylated in man. The half-life of dapsone in the body, unlike that of isoniazid, seems to be unrelated to the speed at which it is acetylated. A study was made of 6 patients with leprosy who had received 1 mg dapsone daily and responded well initially, and of 23 patients from whom dapsone-resistant Mycobacterium leprae were isolated. The acetylator phenotype of each patient was judged on the capacity to acetylate isoniazid. Two of the 6 patients in the first group were found to be rapid acetylators but they "responded to treatment during the first 4.5 months as satisfactorily as the slow acetylators". In the second group 6 were slow and 17 were rapid acetylators. The authors conclude that "the rate of acetylation of DDS is likely to be without prognostic effect in the treatment of leprosy".

C. S. Goodwin


225 mg acedapsone (DADDS) was injected every 77 days into each of 10 patients with untreated lepromatous leprosy, and material from biopsy specimens taken "at intervals" was inoculated into the footpads of mice. Biopsy specimens from 14 patients receiving dapsone "orally in a dosage rising over a period of 4 weeks to 50 mg daily" were similarly processed. "Infectivity for mice was not detectable after 100 days" in 3 patients receiving acedapsone, and in 12 patients receiving dapsone. Of the other patients receiving acedapsone, this effect was found in 3 patients before 200 days, and in 3 others before 300 days. The clinical response in the 2 treatment groups "appeared to be the same". Four of the patients developed erythema nodosum lepromatous although their dosage of acedapsone was equivalent to only 2.2 mg dapsone daily. The isolates of Mycobacterium leprae showed no resistance to dapsone, and no explanation can be found for the results obtained with the specimens from patients receiving acedapsone.

C. S. Goodwin


600 mg rifampicin (Rifampin) daily was given to 5 patients with untreated lepromatous leprosy. Material from biopsy specimens lost its infectivity to mice before the first specimen was taken, after 7 days' treatment in 4 patients and 14 days in 1 patient. In mice one dose of rifampicin 10-40 mg/kg "produced a bactericidal-type effect". The authors state that patients are being given rifampicin 1500 mg once every 11 weeks with acedapsone.

C. S. Goodwin


Bechelli and Guinto (Trop. Dis. Bull. 1971, 68, abstr. 1014), in discussing the implications of experimental infections with Mycobacterium leprae in the mouse footpad, said that "final proof" of the relationship between the morphological index and contagiousness could come only from a controlled study of children exposed to infection. Worth [ibid., 1969, 66, abstr. 1365] and Worth and Wong [ibid., 1972, 69, abstr. 2203] reported on such a study in Hong Kong and on the situation at follow-up after 3 years. In the present communication, the author analyses and discusses these papers in relation to the observations by Bechelli and Guinto. He concludes that "the Hong Kong study and follow-up come very close to meeting the criteria
proposed by Bechelli and Guinto for ‘final proof’. It is very desirable, however, that these observations should be repeated in another population to obtain confirmation”. Mention is also made of the important study by Shepard et al. (Trop. Dis. Bull. 1969, 66, abstr. 2158) on biopsy specimens from patients with leprosy, in which it was shown that Myco. leprae lost their ability to multiply shortly after the start of therapy, the morphological index falling at about the same time. The closely reasoned argument should be read in the original.

F. I. C. Apted


The skin temperature of 12 patients with active lepromatous leprosy at Carville, Louisiana, was studied, and biopsy specimens of skin were taken from the hairy scalp, the forearm and the axilla. The Biopsy Index of Ridley (Trop. Dis. Bull. 1958, 55, 525) was estimated. The temperatures of the scalp ranged from 35.2 to 37.3°C, of the axilla from 34.0 to 37.1°C, and of the forearm from 32.2 to 37.3°C. The Biopsy Index in the 3 areas was 0.17, 0.09, and 0.81, and the area with the lowest temperature apparently had the highest Biopsy Index. The author concludes that “the scalp and axilla were significantly warmer and had significantly less bacilli than the cooler forearm”.

C. S. Goodwin


Clofazimine in oil, 50 mg/kg daily, was given to rats, and control rats were fed with oil only. Widespread arthritis was induced by one injection of heat-killed Mycobacterium tuberculosis into a footpad. 350 mg clofazimine suppressed the arthritis while 200 mg and 100 mg produced partial suppression. The suppression lasted for 2 weeks. Inflammatory swelling of the footpad was produced by an injection of 0.05 ml Freund’s complete adjuvant. Pre-treatment with clofazimine, 50 mg/kg for 7 days, produced a 40% reduction of paw swelling. Antibody response to sheep erythrocytes and the tuberculin skin test were not suppressed by clofazimine. The authors conclude that “clofazimine exhibits anti-inflammatory (but not immuno-suppressive) activity, and that it should be tested in patients with rheumatoid arthritis”.

C. S. Goodwin
Instructions to Authors

Papers submitted for publication in *Leprosy Review* should be sent to the Chairman of the Editorial Board. The name(s) of the author(s), principal appointments held and the place where the work was done should be clearly indicated below the title of the paper. Degrees and diplomas are not to be included.

It is understood that the paper is offered to *Leprosy Review* alone, that it will be subject to editorial revision, and that its copyright becomes the property of the British Leprosy Relief Association. Papers should be typewritten, in double spacing, on one side of (preferably) quarto paper, with wide margins (4 cm left, and 2 cm right). The top copy and a carbon copy of all papers should be sent.

*Tables* should be typed on separate sheets and numbered in sequence, in arabic numerals; captions should be typed in double spacing.

*Graphs and line drawings* should be in Indian ink on tracing linen (if possible) or plain white board or paper, about twice as large as the probable size of the finished block. They should be numbered in sequence, in arabic numerals. Indicate in the margin of the text where tables and graphs should be inserted.

*Photographs.* A reasonable number of black and white plates will be reproduced. Glossy original photographs (positive prints) should be supplied, and clear indications (number, caption, top side) should be given. Any writing on the back of the photograph should be lightly done in pencil.

*References.* In the text, references are made thus: “Jones (1968) has shown . . .”; or “It has been shown (Smith, 1967; Jones, 1968) that . . .”. If more than 2 authors: “Smith *et al.*” If the same author is cited more than once in a year, then the references should be consecutively indicated thus: “Jones (1968a)”.

In the final list, surnames of authors should be given in alphabetical order, followed by initials, year in parentheses, full title of article, accepted abbreviated name of journal (if in doubt, write the name of the journal in full), volume (underlined), and first page of the article.

*Numbers.* All numbers are to be given in arabic numerals.

*Summary.* A brief summary should be given before the body of the paper.

*Contractions.* All weights, measures, temperatures, etc., should be given in metric units, suitably contracted. Authors may refer to “Symbols, Signs and Abbreviations Recommended for British Scientific Publications”, published by The Royal Society. British (Imperial) equivalents may be added within parentheses. In the case of (body) temperatures, the Fahrenheit equivalents of Celsius (Centigrade) figures should be given within parentheses.

*Proofs* are submitted to authors for immediate return by air.

*Reprints.* Authors receive 50 reprints free. Additional copies may be purchased and a price list/order form is sent to authors on acceptance of their typescript.
CONTENTS

Editorial ........................................ 47

News and Notes .................................. 50

The Hansen Centenary ............................ 55

Ulcerating Lepromatous Leprosy in a Patient with Dapsone-resistant Mycobacterium leprae, by A. V. McDougall and R. J. W. Rees .......................... 59

Chiropody and Leprosy, by Monika Mecklenburg .................. 65

What is a Chiropodist? by Valerie Lal .................. 67

Leprosy and “A Disease Called Leprosy”, by T. A. Stringer .......................... 70

The Effect of Stopping Dapsone Treatment for Two Months and then Restarting it in Full Dosage in Patients with Moderately Severe Erythema Nodosum Leprosum, by J. M. H. Pearson and H. S. Helmy .......................... 75

The Effects of Cocktail Anaesthesia on Blood Pressure, by A. Grace Warren and Phyllis M. Taylor .......................... 83

A Developing Country, Leprosy Control, and the Severely Disabled, by Thomas F. Frist .......................... 90

Letter to the Editor ................................ 94

Abstracts ........................................ 96

Printed in Great Britain by the Whitefriars Press Ltd., London and Tonbridge