The Star

RADIATING THE LIGHT OF TRUTH
ON HANSEN'S DISEASE

JANUARY - MARCH 2003
VOLUME 62, No. 1
$2.00 PER YEAR

Emanuel Faria, Editor of The STAR and Captain Judith A Bell Krotoski, OTR, CHT, FAOTA

reminisce about significant events that have changed the lives of people living and working in Hansen's Disease. It has been through communication, education and research, with a desire to make life healthier for all, that nourished their integrity of purpose. Another significant event. . . .

(page 14)

Contents

January - March 2003
Vol. 62, No. 1.

Stanley Stein

Founder - Editor, 1941 - 1967

The purpose of **The Star** is to: 1) Promote an educated public opinion of Hansen's disease, 2.) Furnish vocational rehabilitation for interested patients.

Views expressed in **The Star** are those of patients of the Gillis W. Long Hansen's Disease Center at Carville, Louisiana, except in the case of direct quotations or signed articles.

The Star (ISSN: 0049-2116) is now published **quarterly** by patients of the Gillis W. Long Hansen's Disease Center, Carville, Louisiana.

After you have read **The Star**, please pass it on to a friend and if **The Star** reaches you at a library, please place it conveniently for readers.

Editorial Policy On Terminology

The Star stands firm in its opposition to the use of the term "leprosy." We shall never abandon our campaign to secure general acceptance of "Hansen's disease." Nevertheless, the word "Leprosy" does appear in **The Star** under circumstances which we feel are unavoidable, namely: when signed articles are authored by someone who does not agree with us or when material discusses the disease prior to the introduction of the term "Hansen's disease." We dislike the word "leprosy" intensely, but we dislike the practice of censorship even more.

$2.00 Per Year Domestic
$5.00 Per Year Foreign

MOVING SOON?

Please let us know six weeks before you move what your new address will be. Include your old as well as your new address. ✱

" ? NO MORE (read NO, MORE) NERVE DECOMPRESSION SURGERY IN LEPROSY

by Dr Govind N Malaviya*
Central Jalma Institute for Leprosy
Tajganj, Agra (India)

INTRODUCTION

Leprosy is known as primarily the disease of skin and nerves. Neural damage, whether of cutaneous nerves or of peripheral nerve trunks, is the main cause of disabilities, deformities, social ostracism and related problems. Cutaneous nerve endings may or may not regenerate but nerve trunk damage can have devastating problems, being more or less permanent, if not treated properly.

Nerve damage in leprosy can be presenting in one or more different ways in the same patient: chronic, acute and catastrophic. The recovery of sensory-motor functions can occur, depending upon the severity of damage, age of patient, type of disease and, of course, adequate treatment. The adequate treatment of neuritis is the disputed and least defined factor. It is well known that treatment and response vary from patient to patient and treatment has to be tailored, depending upon the needs of individual patients.

The capacity of axons to regenerate has been demonstrated even in patients with long-standing disease and in older age groups. Axons sprouting has been conclusively demonstrated even in severely damaged nerves. This means that young patients, including children with clinical nerve damage, have better chances of recovery.

PRESENT SCENEARIO

The prevalent concept among field workers is to use steroids whenever and wherever one can, in hope that things will settle in due course of time and recovery will occur. Steroids are considered as solutions for all maladies. Drawbacks of such indiscriminate steroid therapy are - steroid side effects and unpredictable results. In some cases the drug may not actually reach proper concentrations at sites of inflammation because of nerve ischemia.

Extent of paralysis is also a factor which determines the outcome of nerve damage. Various series have reported that if paralysis is partial (clinically muscle grade 3 or more in MRC scale), recovery usually occurs if patient is given adequate treatment with corticosteroids. The recovery seems to speed-up if nerve decompression is added to therapy. This probably occurs due to release of compressive forces, thereby improving blood circulation. The anti-inflammatory drugs are able to reach the site of damage in appropriate quantities. Pain relief is an additional benefit. The steroid requirements also come down even though appropriate steroid doses remain to be defined.

When to decompress a diseased nerve trunk?

It is a difficult question to answer. Field workers define the cut-off point as "failure of adequate medical treatment to improve sensory-motor functions." This period usually varies from 16 weeks to 24 weeks. By this time, at least in certain percentage of cases, damage becomes extensive and unlikely to recover even if nerve surgery is attempted. One can always argue that these cases can undergo corrective surgery - tendon transfers, as a palliative measure. In their enthusiasm to treat cases, they tend to forget that palliation is a palliation and cannot match natural recovery. Corrective surgery can bring in gross functions for survival but cannot restore the delicate balance which nature has created in the species.

Can this waiting period for nerve decompression be reduced to 8 weeks or less? By 8 weeks it becomes apparent in most of the cases whether recovery is going to occur or not. If the patient has not stabilized in this time on full adequate doses of steroids, he is unlikely to recover and therefore should be given the benefit of nerve decompression which is a relatively minor procedure in comparison to corrective surgery. Simple external neurolysis along with epineurotomy with one or two incisions, if carefully done, will do immense benefits to the patients who are already suffering. These case do not need hospitalization and can have exercises in the comfort of their homes, thereby reducing the costs of rehabilitation.

*POST BOX 25, GPO, Mall Road, Agra (India) Pin 282 001

Is steroid versus surgery comparison a must?

People comment on results of decompression, saying that it is successful only half the time (50 percent or less cases have full recovery). This concept of recovery may be academically appealing but tends to ignore those cases where the process of damage has been arrested. Arresting the process of nerve damage is also a benefit of surgery because these were "failed cases" who faced the knife. If the two groups are added up, the results reach 75 to 85 percent. Another aspect of recovery is sensory recovery. If protective sensibility is restored, it is going to be very helpful in the long run because it will help in preventing secondary deformities and mutilations. It has been observed that requirements for steroids are reduced if nerve decompression is added to therapy and steroids can be continued for much longer time as immunomodulators.

A need for controlled clinical trial is talked about in forums comparing steroids alone, surgery alone and steroids and surgery together. Where are the patients to form comparable groups? Even when patients were there in larger numbers it was difficult to have properly matched clinical and immunological parameters because nerve damage is such a variable entity in leprosy. Proponents of surgery have said time and again to act together for the benefit of the patient. One is unable to trace the origin of phrase "medicine versus surgery" which has preoccupied the minds of people involved in the care of leprosy patients. It is the combined approach which appears more rewarding than either alone because nerve damage in leprosy is multifactorial and therefore requires a multipronged attack, depending upon the factors participating in nerve damage in a particular case. Published data also support this approach.

What to compress ?

The tunnels, viz cubital, tarsal, carpal and peroneal, are anatomical entities well documented and described. Their boundaries are well-defined - roof, floor and the entry and exit openings. Due to inflammatory swelling of nerves, the pressure in these tunnels is very likely to increase which in turn is compensated by squeezing of the areolar tissue located in between the nerve bundles. After a certain stage, this compensatory mechanism cannot take up the brunt any more and as a consequence capillaries and venules start getting squeezed. In due course of time, edema, both inflammatory and reactionary, leads to further rise in pressure, thereby arresting the blood flow in smaller arterioles.

Studies of Lundborg have demonstrated increased pressures in cubital tunnel during elbow flexion and in carpal tunnel syndromes. These studies have generated at least some proof to show, for the proponents of external compressive forces.

The conventional descriptions about the sites of nerve damage in peripheral nerve trunks affected with leprosy have brought out some interesting facts. Certain sites have been demonstrated where the nerve trunk is specially affected even though leprous neuritis is patchy and histologically lesions can be seen practically in the whole nerve trunk. The ulnar nerve, for example, can be affected in Guyon's canal near wrist or in cubital tunnel near elbow. Likewise, sites for median, radial, common peroneal and posterior tibial nerves have been demonstrated. Studies, even though limited, using nerve root stimulation have shown that most proximal site of nerve trunk compression is much beyond cubital tunnel for ulnar nerve. Surgical release at this site has been shown to improve nerve function. However nerve root stimulation study requires exposure of nerve trunk along its entire length, which may not be a practical thing. Even though the conduction block exists at a higher level, the question remains as to how far to expose.

DISCUSSION

The objectives of early nerve surgery are two-fold - to help in recovery of damaged nerve by relieving compression and improving circulation and arrest the neural damage. Whenever we discuss the results of nerve trunk decompression surgery in leprosy, we: (a) compare the results of operative intervention with that of pharmacological intervention as if two modalities are comparable and competing with each other to gain superiority or professional acceptance over each other, (b) use too ambitious criteria for assessment of outcome of surgery at times, for example, results of posterior tibial nerve decompression have been evaluated in terms of plantar ulcer recurrences even though we know that the ulcer is a multifactorial event, and (c) tend to forget that even if decompression fails to help in complete recovery of nerve function, many a times damage is arrested as evident by muscle VMT grade records which in fact is a big gain. The surgery also brings down the requirements of drugs, preventing side effects.

Let us be sure in our minds that if progress of nerve damage is arrested, protective sensations will be retained. We as normals may not be able to perceive the value by learning it from a person with insensate palm who has regained the ability to feel as to how confident he has become. One has to take off the spectacles which have blinded one for almost past two decades and explore deep in one's heart without bias or prejudice. Benefit of the patient shall be the first and the top-most criterion.

The question remains about availability of surgical facilities and its costs. The cost of surgery is minimal or rather it pays in the long run by saving expenses on drugs, dressings of wounds, cost of reconstructive surgery and the economic costs of social rehabilitation. The decompression procedures can be performed in a general hospital setup and training for the surgeons can be arranged. If there is a will, one can find many interested people who are willing to acquire the skills of nerve decompression. What they need is the right training and right concepts and a desire in field managers to refer patients in time for such surgical intervention.

One can conclude without any bias that nerve decompression is helpful in a number of ways and is a rewarding surgery in a chronic disease like leprosy. One does agree that all cases do not require surgery on the nerves but fails to understand why referral is denied to the deserving cases at the appropriate time. Whether it is due to ignorance or willful ignorance, due to lack of facilities or a consideration deeply rooted in mind that drugs and surgical intervention are two different modalities and cannot be tried together for their idiosyncrasies.

The practice of herding the patients on the pretext of community program and loading them with steroids is unjustified specially when the "workload" has come down due to better leprosy control. If cardiologists are implanting electrodes deep under the skin, dermatologist are performing cutaneous surgery, interventional radiologists are intervening wherever they can, then why not leprologists shed off their cocoon and see the dawn. After all, they are working for the interests of patients and therefore it becomes their moral responsibility to keep their patients informed about the available treatment options.

★

Book Review

Yawalker SJ. Leprosy for Medical Practitioner and Paramedical Workers. Basel: Norvartis Foundation for Sustainable Development. Seventh Edition, 2002. Soft bound, 134 pages.

This handbook on leprosy is now in its seventh edition, being first published in 1986. I have reviewed multiple previous editions of this book in the past. I believe this edition again lives up to its previously respected reputation. It is published by the Novartis foundation (formerly Ciba-Geigy). Novartis in collaboration with WHO presently provides most of the drugs for MDT treatment of leprosy throughout the world.

This book deals with the practical issues of diagnosis, treatment, and the management of complications of leprosy on a level that make it useful for medical practitioners as well as paramedical workers. This book has a remarkable amount of information for a small book. There are chapters on history and epidemiology though diagnosis and treatment, including reactions, followed by discussions of deformities and their management and rehabilitation. Finally there are brief discussions of prevention and control and hopefully the eventual elimination of leprosy. There is an interesting chapter on the history and development of MDT. As in previous editions it has numerous good color photographs and good summary charts of many topics. I would note that the statistics for the US are outdated and should be revised in the future

I would recommend this book to anyone involved in the clinical care of leprosy patients. It will be especially useful as a reference for the nonspecialist who is dealing with leprosy along with a wide range of other medical problems.

Leo J Yoder, MD

★

Critical Issues in Leprosy Elimination

N. P. Shanker Narayan* and G. Ramu**

Introduction

The decade from 1984 to 1994 was a golden period in the long history of prejudice against leprosy and its sufferers. The WHO Study Group on Leprosy (1982) recommended two MDT regiments for the treatment of Paucibacillary (PB) and Multibacillary (MB) leprosy respectively. The main objective was to offset the increasing incidence of dapsone resistance. Dapsone mono therapy was the mainstay of leprosy control till then.

The implementation of MDT in India saw hectic activity on the part of the NLEP; patients were sought after and put on treatment. A well organized network of rural (781) urban (906) leprosy control units and Survey, Education and Treatment centers (573) provided a geographical coverage of 54% and catered to a population of 650 Million. There were at least seven centers, which were engaged in leprosy research; a number of centers provided reconstructive surgery to those who sought treatment. 46 Training centers supplied the manpower requirements. Those of us who had thrown in our lot with that of the leprosy patients experienced an uplifment of our spirits at the favorable turn of events in the fortunes of leprosy control and its sufferers.

The impact of the program on the leprosy situation in the country

The impact of the program according to official sources on the leprosy problem existing at the commencement of program in 1984 and after 10 years of the functioning of the program were as follows; the Prevalence Rate (PR) came down from 5.72/1000 to 1.23/1000. This was mainly due to cleansing of registers and shortened duration of treatment of Paucibacillary leprosy. The duration of treatment for the Multibacillary leprosy was for 24 months or till skin smears became negative whichever was later. Since only cases whose skin smears were positive were included, the number of MB cases Released From Treatment (RFT) formed a lesser proportion than that of the PB patients. In the absence of Incidence Rate (IR), Annual Case Detection Rate (ANCDR), the Child Rate and the Disability Rate may be used as indicators of transmission of the disease in the community. ANCDR fell from 5.83 to 5.48/ 1000. The Child Rate increased from 10.16 to 17.15 %. The proportion of MB cases detected fell from
18 % to 13 %.

However independent sample surveys in North Arcot District (Arokiasamy, et al 1995) and in Chithoor District (Vallishayee, et al 1995) showed 4 to 6 times higher than the PR and significant increases in Child Rate and MB proportion.

The change in the objective of the MDT program

Encouraged by the fall in PR, the World Health Assembly in 1991 resolved (Resolution 44.9) that leprosy should be eliminated as a Public Health Problem by the year 2000 AD. Elimination is defined as a Prevalence Rate of below 1/10000 of the population. It has been assumed that once the PR fell below a certain level, incidence would be reduced; in the long term the chain of transmission would be broken and leprosy would disappear naturally (WHO, 2002).

Change in the regimen

In 1994, the WHO modified the MDT regimen into Fixed Drug Regimen. The duration for PB MDT was fixed as 6 months and that for MB MDT as 24 months.

Fixed Drug Treatment for Paucibacillary leprosy

In a study Katoch, et al (1985) observed that with the PB - WHO MDT in 22 out of 54 patients lesions were active at 9 months. It is supposed that, Rifampicin would kill all the 10^6 *M. leprae* that PB leprosy might contain. The activity still present at RFT would subside in course of time. Very few studies of the follow up of such active cases have been undertaken to verify the assumption. In the follow up of such cases Katoch, et al (1989) observed that the activity subsided in 85 % of the cases in two years. In 15 % the disease worsened. Two patients had late reactions. 13 % Of patients experimented relapse of the disease. Therefore, the assumption about spontaneous subsidence is not entirely correct

Duration of MB-MDT, the relation to persistence of bacilli and relapses

In 1994 the duration of treatment for MB leprosy was fixed as 24 months and in 1998 it was reduced to 12 months. Obviously, this has been done to hasten the process of elimination, by forcing down the PR. In BL/LL leprosy patients with even a moderate BI with a decline of 0.6 log per year the residual bacillary population at RFT at 12 months would be considerable. In the THELEP study Levy (1988) observed that at the end of 12 months the average patients might be shown to have harbored 2.4×10^5 viable organisms. These persisters have been reported in 9 % of cases in the THELEP study and in 16 % by Katoch et al (1989).

These persisters have been correlated with a high relapse rate (Blanc et al, 1993; Jamet et al, 1995; Girdhar et al, 2000). Shetty et al (1997) found 6 out of 26 RFT cases to have viable bacilli in the skin and another 6 patients in the nerve only.

* Consultant, Leprosy Project, Sakthinagar - 638 315, India ** GKNH Hospital Quarters, Coimbatore - 641 037, India

Katoch et al (1991) found that BL/LL patients, who had discontinued treatment after one and a half years of MDT, had a worsening of the disease both clinically and bacteriologically. Therefore if MDT for BL/LL leprosy is stopped after 12 months, a considerable number of patients would have worsening of the disease and would continue to be reservoirs of infection.

Drug Resistance (DR)

With the introduction of MDT it appeared that DR was no longer a problem. In a recent study (Ebenezer et al, 2002) 23 out of 265 biopsies were observed to have drug resistant *M. leprae.* Primary Rifampicin resistance was seen in 17.4 % and secondary Rifampicin resistance in 4.35 %. Interestingly primary Clofazimine resistance was seen in 30 % and secondary Clofazimine resistance in 13 %. One biopsy had resistant organisms to all three drugs. The warning has been sounded against complacency. The treatment with MDT should be adequate so, as not to jeopardize the efficacy of the MDT regimen.

A single dose treatment for single lesion PB leprosy

In 1998 the WHO recommended a single dose of Rifampicin, Ofloxacin and Minocycline (ROM) for single lesion PB cases. According to the NLEP (1994) single lesion cases constituted 63.5 % of cases. A single dose of ROM will thus eliminate a major portion of leprosy on the spot. A multi centric trial (1977) included 1483 patients. At 6 months the cure rate for ROM patients was 49.6 % and for PB - MDT patients 54.7 %. Thus PB - MDT was significantly more efficient than ROM. The patients were followed up for 18 months, which is too short for the study of relapses.

ROM could be indicated in 1: Single hypopigmented lesions of doubtful diagnosis. 2. Single TT lesions with a maximum measurement of 5cm. 3. Single indeterminate lesion less than 5cm. These lesions would have healed in 75 % of cases spontaneously and perhaps ROM would have been helpful. Contra-indicated are lesions in the region of peripheral nerve trucks. Single MB lesions suspected as PB occur in 2 % of cases.

However, in the recent LECs there was only an average of 13 % of patients with single lesions (Rao, 2001). After an initial implementation in India, Brazil and Bangladesh, ROM has been suspended.

To enlarge the scope of on the spot elimination of a large number of patients, another multicentric trial of ROM in patients with 2-3 skin lesion was carried out under WHO aegis (2001). In those patients who had 3 lesions significant difference in favour of PB - MDT was seen. The follow up was only for 18 months. Two patients: one in each of ROM and PB - MDT groups, were positive for AFB in skin smears at RFT. Desikan (2001) commented that the patients who developed type I reaction in the ROM group were obviously cases of reactivation of the disease. If these trials in the anxiety of quick elimination of the disease are put into practice in mass therapy without safety riders and regular follow up, the results could be disastrous. ROM in multiple lesions and large lesions is a violation of the human rights of the patients.

The 12 months MB-MDT might perhaps be indicated in bacteriologically negative BT/BB cases.

Reactions in leprosy

Of considerable physical and mental agony to the patients are episodes in 39 % of MB patients Type I or reversal Reactions (RR) observed during the first 8 months of MDT and after 2 months of RFT.

The so-called delayed RR may be seen up to 2 years after RFT. But most often, histologically in lesions of delayed RR there is an overlap of relapse and acute signs (Ramu and Desikan; 2002). ENL or type II reactions occur in 2 % of BL and 12-14 % of LL patient. The patients with type II reaction is often very ill and besides the inflammatory skin lesions systemic manifestation of neuritis, arthritis, anterior uveitis, myositis, orchitis, lymphadenitis, osteoperiostitis occur calling for expert treatment and compassion. Such patients require hospitalization. In our experience patients who develop reactions with painful symptoms seek the help of other medical centers where they hope to get relief from the excruciating pain particularly of neuritis. Poor patients in the rural areas suffer the pain with silent tears since many of the rural units and PHCs are ill equipped to manage those conditions.

Nerve damage in leprosy

Leprosy would have been a much easier disease to treat but for the nerve involvement and consequent sensory and motor paralysis. Schwann cells of the nerves are a good repository for *M. leprae* during and after MDT (Shetty et al, 1997). This is reflected in the "acquired nerve impairment in 159/396 patients of whom 78 (20 %) developed severe nerve impairments during or after MDT (Brakel and Khwas, 1994). Cases with either primary or secondary neuritic leprosy may relapse with skin lesions, a fourth of them presenting as lepromatous leprosy (Ramu, 1992).

Acute painful neuritis is an important component of reactions, 98/119 reactional patients had neuritis; 68 patients suffered from multiple neuritis (Ramu and Desikan, 2002). Acute painful neuritis, if not properly treated with adequate steroids, has a propensity to develop paralysis.

Contrary to the view that acute neuritis in RR is a DTH reaction, in an immunohistological study of 30 nerve biopsies, number of cells bearing IgG and IgM antibodies were seen in

6 BT cases with neuritis (Ramanathan et al, 1987). This brings out the discrepancy observed by Srinivasn et al (1982) between the lesions in the skin and nerve on histological examination and the high bacillary population in the nerve as compared to the skin. Therefore, steroid alone as advocated is not adequate but should be supplemented with chemotherapy in cases who have completed the MDT schedule.

Disabilities

Solomon et al (1998) have reported incidence rates at RFT of sensory impairment of 45.17/1000 in PB and 128.89/1000 in MB patients and motor impairment in 10,84 of PB and 28.81 in MB patients who had no nerve impairment at registration. Brakel and Khwas (1994) reported severe nerve impairment in 20 % of patients during or after MDT. The prevalence rate of disabilities in Gudiyatham was 92.37/10000 of which only 7,11 % were under treatment. In Sakthi Nagar the PR was 45.31 of whom 1.75 % were under treatment. Only grade II disabilities are recorded because they are obvious. Grade I disabilities and eye involvement go unnoticed. 30 % Of leprosy patients have leprosy related eye problems at one time or the other (Rajan,1990).

MLEC campaigns

Crash programs for detection of cases have been carried out employing a large force of health staff and community volunteers between 1998 and 2000. Fifty thousand new cases were detected in the first campaign. 13 % Had single lesions. One third were MB cases. Deformities were below 5 %. In the second MLEC in the northern states 180000 cases were detected. 38 % had MB leprosy. Partially treated figured from 8 % to 82 % (Rao, 2001).

Missing detections vs detected cases were 38 years vs 3 in Bombay; 60 vs 21 in Wardha (Revankar et al 1997) and 84.3 vs 16.4 in another area (Ramanathan et al, 1988). The objective of the MDT program is to interrupt transmissions. Incidence of leprosy among contacts of leprosy patients is a good indicator. Vijayakumaran et al (1995) observed an incidence of 11/1000 among contacts in the first year of MDT, which declined to 2.5/1000 in the fifth year. The annual case detection rate in the project area was 1.2/1000. Therefore the incidence among contacts could be due to non-household sources. Despite a declining PR, transmission continues to occur as shown by the case detection rate, the child rate, the disability rate and the figures in the various sample surveys.

Compliance

While the monthly doses of rifampicin and clofazimine are administered under direct supervision, 20-30 % of the patients do not take the daily self-administered doses of clofazimine and dapsone (Balakrishnan et al, 1986). In the special action project, the entire quantity of drugs for the PB and MB regimens are supplied in blister packs (WHO, 1998). In the absence of repeated health instructions by health workers the defaulting rate is likely to be considerable. Patients who experience reactions and other complications would lose their confidence in the treatment. The lack of confidence spreads to other patients.

The woes of integration

In 1997, NLEP was integrated with the general health services in Tamilnadu. The time given for integration was one month. The leprosy control units were hastily disbanded. The records of patients were not given to PHCs. The general health staff were not properly trained. With the other medical and health tasks taking most of the time, leprosy is a low priority task. The medical officers who are not trained and the discipline of leprosy being given very little importance in the medical colleges, they are averse to examine patients. They are at a loss when it comes to treating reactions and neuritis. Voluntary agencies were asked to wind up their work in the rural areas thus closing an avenue where the patients could receive succor.

The results of this ill-conceived and ill-planned policy are seen in the form of highly bacillated patients seeking treatment in centers where they are not neglected e.g.. in SHLC Kumbakonam 128 cases with BI of over 3 including 11 patients with histoid nodules were treated (Rajan,2002).

Compromises on certain basic aspects of leprosy by the WHO in its bid to eliminate leprosy in black and white

A "case of leprosy" was defined as a person showing clinical signs of leprosy with or without bacteriological confirmation and requiring chemotherapy. For operational purposes those who have or had leprosy will fall into one of the following three categories: 1. Those requiring or receiving therapy 2. Those who have completed chemotherapy and are under surveillance and 3. Those released from surveillance but in need of care or assistance because of disabilities (WHO, 1980). Bacteriological examination is very important and highly relevant to leprosy control (WHO,1988).

1. The duration of therapy is being shortened without any information on the 12 months MDT being available. It is arbitrary to assume that patients who have completed a period of treatment but who are still clinically and bacteriologically active are no longer cases. Due attention needs to be given to the studies by Katoch et al (1989, 1991).

2. No other laboratory examination is as specific as the skin smear bacterioscopy for AFB when positive. Skin smear positive patients are highly infectious and therefore skin smears should be taken wherever a clinical diagnosis of

MB leprosy is made. Skin smears are highly important in relapsed lesions. Instead of improving the standard of this simple procedure, to discontinue it is less than scientific.

3. **R**eactions, neuritis and disabilities occur during MDT or on the heels of RFT should be deemed as problems of the activity of the disease and the leprosy control agencies should not wash off their hands and remove the patients from the register. The staff should be trained to detect eye involvement. The elimination program should not neglect human suffering.

4. The Who (1994) has ruled out the necessity for annual surveillance and patients have to be taught to recognize the signs of relapse. It is only when the disease has fairly advanced that the patients seek help but to do so in some other center where medical attention would be available because the health workers in the leprosy centers have lost contact with the RFT patients.

The partially treated and relapsed patients are potent reservoirs of infection. With proper treatment and care of the feet and eyes a large number of individuals disabled due to leprosy could be minimized. The WHO should revise its strategy in favor of the patients.

U-MDT regimen for all leprosy patients

The MB-MDT regimen is to be administered to both PB and MB patients. The duration of treatment is 6 months. According to the new classification for operational purposes cases with less than skin patches are classified as PB and patients with more than five patches are classified as MB (WHO,2000). The WHO, TAG in its fourth meeting has proposed a study of three drugs namely Rifampicin, Clofazimine and Dapsone in the treatment schedule given in blister packs lasting for four weeks to be administered six times in nine months both for PB and MB patients. Initial trial would include 2500 PB and 2500 MB patients. An expected level of minimum 95 % efficacy over a period of 5 years is the objective.

The lacunae (purposely incorporated) are: 1. The investigation will not include skin smears for AFB. 2. Ridley-Jobling classification will be kept out. 3. No mention of charting of skin lesion is made. 4. Assessment of sensory and motor function is not mentioned. 5. Method of clinical assessment of the progress under MDT is not mentioned.

U-MDT for PB cases

However, a trial by Katoch et al (1999) has shown that this regimen brings about a subsidence of lesions in all but 7.5 % of cases, which also regresses in two years. As such another study involving 2500 cases is superfluous and unwarranted.

U-MDT for MB

Cases according to the new categorization will include a large spectrum from BT-LL. New cases which are to be included according to the protocol will include less than 5 % of BL-LL cases. Even in sulfone era there were very few amongst new cases. The prevalence data used to include a cumulative number of these cases over several years. Nevertheless, they are very important from their ripple like infectivity. It has been referred to before in detail how short duration treatment amounts to: 1. Partial treatment. 2. Large population of persisting organism. 3. Consequently a high relapse rate. 4. Bacillary population in the nerves leads to nerve impairment. 5. One of the factors in the causation of Reversal Reaction is AFB positivity (Ramu and Desikan, 2002). 6. In the reporting form eye involvement is not mentioned; 30 % of cases develop eye ocular problems. 7. 5 Years is too short for the study of relapses. 8. Reactions occurring after 6 months of MDT put on steroids for certain duration or time would require chemotherapy due to immunosuppressive effects of steroids. It will vitiate the experiments. 9. Grade II deformity occurs due to the worsening of grade I disability. Therefore the reporting form should contain information on grade I disability. 10. The entire exercise is to interrupt transmission. Therefore the most infectious class of patients i.e BL-LL patients should be assessed separately; BI should be given great importance. If the BI remains the same it is beyond scientific temperament to stop treatment. The patients would be still infective and there will be bacterial proliferation without treatment.

Ethical considerations

Ethical clearance is to protect the investigator. The most important consideration should be the rights of the patients to receive adequate treatment including the management of complications.

Informed consent of the patients

The leprosy patient is a most receptive individual and is grateful for any treatment offered to him, eg. below knee amputations and prosthesis was suggested and accepted as the best method of management of any foot ulcers by several patients in a home for destitutes, liver biopsies of a large number of contacts were performed to prove a hypothesis that the liver was the site of primary infection.

The Ethical Committee must have a member from "IDEA" who would ensure that the proposed procedure is in no way detrimental to the patient.

Since the number of BL/LL cases would be very small, the statistical outcome is predetermined. But the new BL/LL cases who would accumulate over the years and 9 to 10 years later will account for the emergence of the pushed out leprosy with large numbers of leprosy disabled in the community.

Adverse Drug Reactions (ADR)

In the protocol on U-MDT ADR is dealt with from a theoretical background. Enough experience has been gathered over the years on Adverse Reactions met with in the treatment of patients with MDT and their management. These are given below.

Dapsone

It is interesting to read (p 26) that complete blood counts should be carried out frequently in patients receiving dapsone. Enormous numbers of patients have been treated throughout the world with dapsone; with normal doses ADR are rarely encountered. This is mentioned because, the most important investigation, namely, skin smears for AFB has been omitted. It is very doubtful if the general health services would be able to undertake complete blood counts and liver function tests for all cases.

It needs to be emphasized that haemolysis and methhamoglobinaemia are dose related. It is suggested that the dose of dapsone not exceed 1.5mg/body weight (Balakrishnan et al 1989). In adults of small build weighing below 50kg, commonly observed among females in Tamil Nadu and Orissa, the dose of dapsone should be the same as for children 10-14 years of age. (i.e. 50mg).

Attention is drawn to a serious hypersensitivity reaction designated as "Dapsone Syndrome" which was rare with dapsone monotherapy but has been reported a little more frequently with MDT. The condition resembles infectious mononucleosis and eosinophilia. Treatment with dapsone should be stopped immediately and the patient put on steroid therapy.

Clofazimine

In addition to the red and black pigmentation, ichthyosis which occurs in 66 % of patients under Clofazimine is considered as adding to the stigma by patients. (Ramu and Iyer, 1976). Reduced levels of vitamin-A have been observed in ichthyotic areas compared to non-ichthyotic area (Bharadwaj et al, 1982); reduced levels of vitamin-A have been also observed in the plasma of some cases under Clofazimine therapy for a long time with associated xerosis of the cornea and night blindness. For ichthyosis a moisturizing ointment with 10 % urea was given for local use and for the ocular involvement vitamin-A 50000 units once a month was administered. For synptoms of acute abdomen caused by accumulation of crystals in the sub-mucous of the intestines and mesenteric glands INH 200 mg a day has been found to relieve the symptoms (Ramu and Iyer, 1976) due to mobilisation of Clofazimine from the tissue by INH (Venkatesan, 1989). However, in paralytic ileus, a serious side effect of Clofazimine therapy INH, does not help.

Rifampicin

Anaphylactic shock is occasionally met with. This occurs within 20 minutes after the oral dose, usually the second dose. This calls for immediate treatment by lying the patient flat on the ground and injecting 0.5 to 1 ml of 1:1000 of adrenaline. Very often the patient recovers. If no improvement is seen and deterioration continues I.V. fluids, antihistamine, aminophylline, O_2 inhalation and assisted respiration are called for after hospitalization (Ramu. 1990).

Rifampicin may induce a cutaneous syndrome with flushing (Redman Syndrome) and/or pruritis with or without rash which includes most of the face and scalp with watering of the eyes. The cutaneous syndrome starts 2-3 hours after the dose. It is self limiting and does not call for stopping the treatment. Antihistaminics relieve the condition. However, pemphigoid lesions induced by Rifampicin call for stopping Rifampicin treatment, hospitalization and steroids as much as 80 mg of prednisolone per day tapered off very slowly.

Flu syndrome occurs an hour after the dose, characterized by fever with chill, body pains and upper respiratory catarrh. Paracetamol 0.5 gm gives relief and may be repeated after 6 hours. Only if flu syndrome is repeated every time for three doses should Rifampicin be stopped.

Abdominal syndrome is characterized by severe abdominal pain, vomiting sometimes hemetamesis, shock and collapse. This is a serious condition necessitating hospitalization, I.V. fluids, antacids, and antispasmodics. Haematemesis requires blood transfusion. In abdominal syndrome erosions in the mucous membrane of the stomach occurs.

Renal Toxicity is potentially fatal. Renal involvement commences with oliguria; when a patient who had a dose of Refampicin complains of oliguria, it is mandatory to examine his urine for albumin, RBCs and casts. On finding these the patient should be kept under observation. The blood urea and creatinine should be estimated. If they are raised above normal range frusemide 40-80 mg may be given with I.V. fluids and enuresis forced. This is followed by recovery acute renal failure portends a fatal outcome. If the above treatment does not result in enuresis haemodialysis would have to be performed (Rajan et al, 1987). Renal toxicity may be associated with hepatic toxicity. While Rifampicin therapy may be associated with a slight rise in the transaminase enzymes, clinical evidence of hepatotoxicity with increase in serum bilirubin calls for stopping Rifampicin altogether. Out of 800 cases on MDT Pemphigns was observed in 1; Abdominal syndrome in 1 and hepatoxicity in 2; oliguria in 2, Acute Renal failure in 3, thrombocytopenic purpura in 1, shock in 1 and Flu syndrome in 6 patients. (Ramu, 1994). This may have been selective because they were observed in patients attending a hospital. Nevertheless, chemotherapy of leprosy needs caution and administration of drugs in blister packs for self-administration

may miss cases who develop toxicity which may prove fatal. In the matter of toxicity statistics has no place. Human life is much more valuable than what numbers indicate.

An example of sustained leprosy control activity

An urban center was handed over to the Voluntary Health Service Leprosy Project, Sakthi Nagar, Erode district in Tamil Nadu bu the government in 1992. The population was 35,198. The PR was 3.49/1000 and ANCDR 2.61/1000. In first year after taking over i.e. 1993 the PR was 3.55/1000 and ANCDR 3.29/1000; over the years there has been a decline in both; in 2002 the PR was 0.10/1000 and ANCDR 0.64/1000. An MLEC conducted on 1, 2/12/2002 by well-trained workers only 4 PB cases out of 264 suspected cases were detected. The treatment schedule and the categorization of patients are according to the recommendations of the WHO 1988. There was only one new MB case in 2002. There is reason to hypothesize that the new cases are those who were incubating the disease. By 2005 it is hoped that not only the PR but also the ANCDR would be under the permissible PR of less than 1/10000.

Conclusion

1. In an attempt at a statistical elimination of leprosy the WHO has progressively reduced the duration of treatment including a one-day schedule. Even in countries which are not considered as endemic anymore eg. Bangladesh and Benin the case detection rate is above 1/10000 (Declercqe, 2001). The disability rate and proportion of MB cases appear to show that the cases are old newly detected cases. The child rate shows that the disease is active in the community.

2. 12 Months or 6 months treatment would leave behind a large number of partially treated cases.

3. Stopping surveillance and suggesting that the patients should be taught to recognize early signs is unrealistic. Relapse is first by bacterial increase and it is difficult for the patients to recognize skin changes (Desikan,1997).

4. It is not difficult to train volunteers from inaccessible areas in the fundamentals of leprosy, its complications and make them responsible for delivering the treatment and arrange for the patients to report to the main center when complications arise rather than handing over the blister oacks for the whole duration.

5. When in another disease i.e. tuberculosis, bacteriological examinations is insisted upon. There is no reason why the bacteriological examinations should be given-up in leprosy.

6. It is high time that the WHO gives up its obsession with quick elimination of leprosy and its statistical obfuscation of the leprosy problem.

7. The duration of treatment for MB patients has to be restored to 24 months; the system of surveillance of two years for PB and five years for MB cases must be followed.

References

01. Arokiasamy J, Gupte MD, Vallishayee, Masood Ahmed TH, Rajendra AG 1995 Sample Survey in North Arcot District, Tamil Nadu 19th IAL Conf, Pune.

02. Balakrishnan S, Kumar A, Rao BR, Petro TP 1986 Implementation of tests for monitoring drug compliance of leprosy patients under multidrug therapy. *Indian J Lepra* **58:** 555-559.

03. Balakkrishnan S, Kartikeyan S, Ramu G 1989 Investigations into the haemolytic effects of dapsone therapy in leprosy patients. *Indian J Lepra* **61:** 10-15.

04. Bharadwaj VP, Sritharan S, Venkatesan K, Ramu G. 1982 Vitamin in A levels of ichthyotic and non-ichthyptic skin and plasma of leprosy patients with and without therapy. *Indian J Med.* **75:** 773-777.

05. Blanc I, Jamet P, Faye O. 1993 Short term Rifampicin regimen for leprosy yield to high risk of relapse 14th Int Leprr Congr Orlando Abst Ch 27 *Inter Lepra* 61 Suppl.

06. Brakel WH Van, Khwas IB. 1994 Nerve damage in leprosy: an epidemiological and clinical study of 356 patients in Western Nepal- Part I *Lepra Rev.* **66:** 204-222

07. Declercqe E, 2001 prevalence valid indicator for monitoring leprosy elimination? *Inter J Lepra* **69:** 111-113.

08. Desikan KV. 1997. The risk of relapse after multidrug therapy in leprosy. *Lep Rev.* **68:** 114-115.

09. Desikan KV. 2001 Correspondence on comparative trial of single dose chemotherapy in Paucibacillary leprosy patients with two to thre lesions. *Indian J Lepr* **73:** 361-367.

10. Ebenezer GJ, Norman G, Joseph GA, Daniel S, Job CK. 2002 drug resistant *Mycobacterium leprae*-results of mouse foot pad studies from a laboratory in South India. *Indian J Lepr* **74:** 301-312.

11. Gridhar BK, Gridhar A, Kumar A. 2000 Relapses in multi bacillary patients effect of length of therapy. *Lepr Rev* **71:** 144-153.

12. Jamet P, Ji B and the Maechoux chemotherapy group. 1995 Relapses after long term follow up of multibacillary patients treated by WHO regimen. *Inter J Lepr* **63:** 195-201.

13. Katoch K, Ramu G, Sengupta 1989. Results of a slightly modified WHO regimen in highly bacilliferous BL/LL patients. *Int J Lepr*. **59:** 451-457.

14. Katoch K, Ramu G, Ramanathan U, Desikan KV. 1987 Comparison of three regimens containing Rifampicin for the treatment of Paucibacillary leprosy patients. *Int J Lepr* **55:** 1-8.

15. Katoch K, Natrajan M, Bagga A, Katoch VM 1990 Follow up of cases treated for varying periods Proc VI Symposium on Leprosy Research, Santa Margherita, Oct, 1990.

16. Katoch K, Natrajan M, Katoch VM, Singh HB, Bhatia AS. 1999 Chemotherapy trial in Paucibacillary leprosy using Clofazimine. *India J Lepr* **73:** 311-334.

17. Levy L 1976 Treatment Failure *Inter J Lepr* **4:** 177-182.

18. Levy L. 1998 Microbial persisters and leprosy in Proc JT Meeting of Indian and THELEP scientists *SLT & RI, Karigiri* pp 11-22.

19. NLEP 1994 Launching of WB assisted NLEP Project Expert Committee on Leprosy.

20. NLEP 1994 Annual Work shop of State Leprosy Officers.

21. Rajan MA, Soundarrjan R, Krisnamurthy V, Ramu G 1987 acute renal failure following Rifampicin *Indian J Lepr*. **62:** 33-38.

22. Rajan MA 1990 Eye in Multi drug Therapy I*ndian J Lepr* **62:** 33-38.

23. Rajan MA 2002 Highly positive and histoid leprosy after integration *Indian J Lepr*. **74**: 64-65 Paper presented at Golden Jubilee of IAL.

24. Ramana V, Devdas TTJ, Abraham T 1998 Crash population service - a tool for demonstrating rapid case detection Ibt National Lepr Congress Abs Co 32

25. Ramu G, Desikan KV 2002 Reaction in Borderline Leprosy *Indian J Lepr* **74:** 115-128.

26. Ramu G , Iyer GGS 1976 Side effects of therapy *Lepr India* **48:** 722-723.

27. Ramu G 1990 Toxic manifestations and side effects of MDT regimen *NLO Bull* **18:** 145-149.

28. Ramu G 1994 The Past, Present and Future of chemotherapy of leprosy *NLO Bull* 37-49.

29. Ramu G, 1992 Clinical features and diagnosis of relapses in leprosy *Indian J Lepr.* **67:** 45-59.

30. Rao CK 2001 MLEC *Bull. Lea* **1**:4-6.

31. Revankar CR, Antonysamy MS, Gandewar KN 1997 Need for intensive leprosy case finding for the elimination of leprosy *Inter J Lepr.* **68**: 177-178.

32 Shetty VP, Suchitra K, Uplekar MW, Anita NH 1997 Higher incidence of viable *M. leprae* within nerves compared to skin among leprosy patients released from treatment *Lepr Rev* **68**:131-137.

33. Single dose multicentric group 1997 Efficacy of single dose multi-drug treatment of single lesion Paucibacillary leprosy *Indian J Lepr* **69:** 121-129.

34. Solomon K, Kurien N, Palani R, Rao PSS 1998 Incidence of nerve damage in leprosy patients treated with MDT *Inter J Lepr* **66:** 451-456.

35. Srinivasan H, Rao KS, Iyer CGS 1982 Discrepancy in the histopathological features of leprosy lesions in the skin and peripheral nerve Lepr Ind **54:** 275-282.

36. Srinivasan H 2000 Disability and Rehabilitation in leprosy: issues and challenges *Indian J Lepr.* **72:** 317-337.

37. Vallishayee P, Gupte MD, Ekambaram V, Reddy P, Arikiasamy G 1995 Sample survey in Chithoor District, Andra Pradesh 19th IAL Conf Pune.

38. Veukatesan K 1989 Clinical pharmacokinetic considerations in the treatment of leprosy patients *Clinical pharmacokinetics* **10:** 505-586.

39. Vijayakumaran P, Jesudasan K, Manimozhi N, John DR 1998 Does MDT arrest transmission of leprosy in household contacts? *Int J Lepr* **66:** 125-129.

40. WHO Study Group 1982 WHO *Tech Rep Series* No. 675.

41. WHO 1988 *Tech Rep Series* 768.

42. WHO Action Programme 1994 *Lep New* **3:** 1-5.

43. WHO 1994 *Tech Rep Serie*s 874.

44. WHO 2000 Global situation *Weekly Epidemio Rev* **75:** 226-231.

45. WHO 2002 The final push, strtegy to eliminate leprosy as a public health problem Q & A 1 Edu.

★

KUSHTAROG NIWARAN SAMITI

Shantivan, Post Nere, Tal. Panvel, Dist. 410206

Anti-leprosy week was observed (January 30th - February 3, 2002) in collaboration with IDBI Bank, Panvel Branch and Hind Kusht Nivaran Sangh, Maharashtra Branch, Mumbai & A.L.H.R.R.E. Society.

1. Awareness march was organized in Panvel and New Panvel. 1000 school students from Bantia High School, K.V. Kanya Vidyalaya, V.K. Vidyalaya and all Municipal Schools participated in the march. 5000 pamphlets were distributed among the public and visitors.

2. Special slide show and exhibition was organized at Panvel and New Panvel.

3. Survey of Panvel slump was conducted during the anti leprosy week. Enumerated population - 14685, examinations - 11875. No. of cases detected, MB 3 and PB 5 = 8.

4. Pre-reconstructive Surgery Examination Camp was organized at Panvel. Dr. Satish Arolkar examined 72 patients and suggested that 38 patients selected for surgery.

5. Financial Assistance was provided to 12 needy leprosy affected persons to start their small business/profession. Shri Vivek Patil, MLA, Shri J.M. Mhatre, President, Panvel Council, Dr. Satish Arolkar, Surgeon, Shri M.K.Bantia, Shri Nare, Nayab-Tahasildar, Shri Uday Thakar & Mrs. Pratibha Kathe were present. Efforts taken by Shri Dhyaneshwar Kholgade and his team were highly appreciated.

Submitted by Uday Thakar, Treasurer

★

Leprosy in Ancient Tamil Literature and Tamil Medicine

K. Kalaiselvi* and G. Ramu**

Introduction

While dealing with the antiquity of leprosy in India, references are made mostly to the descriptions of the disease and its treatment in the anthologies of Aurvedic Medicine compiled by Susruta and Charaka written in Sanskrit (Dharmendra, 1940). The Tamil language is the oldest spoken language in India and has a rich literature, dating from the early centuries of the Christian era (Sastri, 1975). Tamil Nadu, state lies in the south-east of India. It has an area of 130,068 sq. km and had a population of 55.6 million according to the 1991 census. At the beginning of the MDT campaign in 1983, the prevalence of leprosy was 11.8 per 1000 in the state. The New Case Detection Rate as recently as 1992-1993 was 3.2 per 1000. (Ahmed, 1993). With such a state of the disease, it can be visualized that leprosy would have been widespread enough to be mirrored in literature.

Leprosy in Tamil Literature

The earliest epoch in Tamil literature belongs to the Sangam period of the first three or four centuries, A.D. Sangam was a college of poets in the city of Mandurai, the capital of the Pandyan kings. The Sangam literature is grouped in eight anthologies (Sastri,1975). Of interest to us is the forth anthology called 'Pathupattu' or 'Ten Idylls'. Kapilar, a poet of the early Sangam period, in one of his works, 'Kurunjikalai' describes one of his characters, as voiced by the heroine as "an old Brahmin covered over with blemishes and limbs shortened by black leprosy". The adjective black is used to distinguish it from 'white leprosy' i.e. vitilgo (Ramu, 1973).

Another poet of the early Sangam period whose works are included in the 'Ten Idylls' is Nakkirar. He is celebrated both in literature and myth. He was a poet laure in the court of the Pandian king Nedunjelian (about 210 A.D., Sastri,1975). He had the temerity to find fault with a poem penned by Lord Siva himself. Incensed by this misdirected intellectual arrogance, Siva cursed him with leprosy (Jaganathan, 1947). This myth has permeated the population and one of the causes for the superstition that leprosy is caused by God's curse.

The earliest of the sixty-three Saivite saints was Karaikal Ammai or The Mother of Karaikai who lived in 550 A,D. (Sastri, 1975). In one of her verses, she invokes the mercy of Lord Siva to cure one of her hosts of leprosy, which she calls 'the malignant disease. Auvaiyar, a poetess, a contemporary of Kapilar, assigns grades of severity to certain poignant states that afflict human beings. Commencing with dire poverty she ends with the statement that the severest of all is 'the incurable malignant disease' which is understood to be leprosy (Ramu, 1973).

Leprosy in Tamil or Siddha Maedicine

The Age of the Siddhars: Siddha Medicine is so called, because it has its origin from the Siddhars. The Siddhars are well known for their mystic poetry, literature, and alchemy besides medicine. The oldest of them was Agathiyar whose work, 'Agathiyam' is the oldest treaties on Tamil grammar belonging to the early Sangam period (Sastri,1975). His work on Siddha Medicine is known as 'Agathiyar Guna Vadagam' dealing with the properties of drugs. Most of the eighteen Siddhars lived between the second and fifth centuries, A.D.

Terminology

The terms used for leprosy by the Siddhars are 1. Karum Kuttam or Kiratina Kuttam (Black-leprosy). 2. Peru Noi (Big disease). 3. Kurai Noi (Dystrophic

*Dept of Environmental Science PSG College of Arts and Science Coimbatore-641 014 **GKNM Hospital Quarters Coimbatore-641-037

disease). The term 'Thozhu Noi' which is under current use was coined by Appar a Saiviate saint who was a contemporary of Mahendra Pallavan I (590-630 A.D)., (Sastri, 1975). In his devotional ardor in one of his Thevaram songs (psalms), the saint says, "though he may be suffering from festering ulcer with shortening of limb due to Thozhu Noi, I will worship him if he be a devotee of my Lord Siva". Thozhu Noi means worshipful disease.

Causes of leprosy

Yugi Muni, one of the Siddhars assigns the cause of what he calls the incurable form of leprosy to eating rotten fish and eating crabs and snails in excess; close contact with patients, sharing their bed, retribution for sins of a previous life and heredity (Sowrirajan, 1982).

Symptoms and signs

Yugi Muni, classifies Peru Noi or Big disease into 18 types according to the predominant skin lesion encountered. Only some of the eighteen might pertain to leprosy; The others might perhaps be of psoriasis, tertiary stage of syphilis, Norwegian Scabies etc. The skin lesions indicating leprosy are; 1. Nodules 2. Plaques, 3. Thickening of ear lobes 4. Karum Kuttam with thickening of skin 5. Annular lesions 6. Erythematous skin lesions.

In Sarabandrar's Siddha Marthuva Chudar (light of Siddha Medicine) (Sowrirajan, 1982) are mentioned shininess of skin, plaques, nodules, festering sores of fingers and toes leading to their shortening, deformities of hand and feet and sensory loss of skin lesions.

Treatment of leprosy

In Padartha Guna Chinthamani (Materia Medica) the Siddhar Teraiyar mentions that herbal medicines to be effective should be supplemented with salts of heavy metals or sulfur. The drugs mentioned are copper salts, mercurial, gold salts, zinc oxide, lead oxide, arsenic and sulfur.

The herbs used are white Hibiscus and Plumbago zeylancia, Azardica Indica, Centella Asiatica, Ocimum, Calotropis, Oleander and Chaulmoogra (Natarajan, 1944; Anada Kumar, 1944; Narayanasamy, 1999). The root, leaves, stem or bark or seeds were used in the form of thir ash, or powder after drying in the shade or oil from the seeds were used along with one of the heavy metal salts, very commonly of mercury.

Relevance of Siddha Medicine to Leprosy Elimination

Siddha Medicine is widely practiced in Tamil Nadu. In Coimbatore district alone there are about 300 practitioners. There are several medical colleges for Siddha medicine. The textbooks of Siddha medicine carry the outmoded concepts laced with superstition about the cause of leprosy. The signs and symptoms relate to advanced stages of the disease and include those of other diseases also. Because of lack of documentation and follow-up, the progress of the disease under treatment and the toxic effects of drugs have been missed.

In the interest of the patients and the program of elimination, it is imperative that the services of the practitioners and the staffs of the Siddha medical colleges are taken into the fold of the campaign. Illustrated guidelines setting in clear terms established scientific facts should be presented to them. These should include 1. The cause of leprosy is a microorganism called *M. leprae*. Therefore, there is no place for the misconceptions about the causation of the disease traditionally handed down through the ages. 2. The disease can be diagnosed accurately in its early stages and deformities can be prevented. 3. Powerful drugs included in MDT given for a fixed duration can cure the disease in any of its forms. This interrupts transmission. 4. There is no reason, whatsoever, for treating a leprosy patient as different from others e.g. tuberculosis patient. Some of the herbal medicines used in Siddha medicine might be of benefit along with MDT. Chaulmoogra oil which

had been in use for the treatment of leprosy has been found to suppress the multiplication given in the form of its fatty acid Chaulamoogric acid (Levy, 1973). However it is no longer in use as powerful bactericidal drugs have replaced it, included in multidrug therapy (MDT). Ulcerating skin lesions during reactions have been treated with sterile gauze soaked in hydnocarpus oil (Ramanujam and Dharmendra, 1978); it has been observed to promote colagenation in wounds (Oommen et al, 1999) and hence its healing property in ulceration.

Vallarai leaves and stem (Centella asiatica) made into powder after drying in the shade had been used in the treatment of leprosy in Siddha Medicine (Narayanasamy, 1999). It has been found to be promising in clinical studies by Bolteau (1945) in Madagascar; Chaudhary and Ghosh in Kolkatta (1975); and Ali et al in Bangladesh (1986). A study in Kumbakonam (Ramu, 1993) found that patients treated with Centella powder along with MDT showed a regression of clinical signs and BI; in the mouse foot pad inoculation studies, while there was growth of *M. leprae* before treatment in all except one of the 7 patients after three months there was no growth of bacilli obtained from any of the patients. It has also a role in the treatment of neuritis, as it is a known nervine tonic. This calls for studies in other centers for collaboration.

Oil of the seeds of Azadarichta indica (Neem) is an effective agent in clearing the nose and trophic ulcers of maggot infestation instead of chloroform and turpintine mixture, which is an irritant for the tissues.

It needs to be emphasized that most heavy metals and arsenic are toxic to several organs and therefore have no place in therapy of leprosy in which their effect is dubious. However, Zinc sulphate in doses of 200 mg daily is a useful supplement in treating recurrent reactions in lepromatous leprosy. Zinc levels have been found to be significantly low in the serum of these patients and oral zinc helps in restoring the levels and symptomatically improves the reacting condition probably by its role in correcting the immunological perturbation (George et al, 1993).

These observations will tend to show that modern scientific inputs will remove the cobwebs from traditional systems of medicine and bring them in line with modern medicine.

References

1. Ahmed THM. 1993. Trend of Leprosy in Tamil Nadu. Report on the impact of MDT on the trend of leprosy *IAL Madras;* 1993: p21

2. Anand Kumar A. Tamilum Siddha Maruthuvamum. *International Institute of Tamil studies*.1996; 110-114

3. Dharmendra. Leprosy in Ancient Hindu Medicine. *Lepr in India*; 1940; **59:** 35 - 31

4. George J Bhatia VN, Balakrishnan S, Ramu G. Serum Zinc/copper Ratio in subtypes of leprosy and effect of oral Zinc therapy on reactional states. *Inter J Lepr* 1991; **59**: 25 - 31

5. Jaganathan KV, Vazhikatti publ. Alliance Co, Madras 9-15

6. Levy L. The activity of Choulmoogric acid against *M. Leprae. Am. Rev. Resp. Dis* 1973 **111**; 703 - 705

7. Oommen ST, Rao M, Raju CVM. Effect of oil of Hynocarpous on wound healing. *Inter J Lep* 1999; **67**: 154 - 159

8. Narayanasamy V, Kutta Noi Maruthuvam in; Nalavazhvu Siddha vaidyam pb. Narayanasamy, Coimbatore 1999. 295 - 303

9. Natarajan TR Tamilum Maruthuvamum. International Institute of Tamil studies, Madras 1994 95 - 109

10. Ramanujam K, Dharmendra. Management of reactions in leprosy in; *Leprosy Ed*. Dharmendra Kothari Publishing house, 1978; Bombay 532

11. Ramu G. A Thesis for Doctrate of Medicine, APS University. History of leprosy and its spread, 1973

12. Ramu G, The past, present and future of chemotherapy of leprosy. *NLO Bulletin*; 1994: **27**: 37 - 47

13. Sastri Kan, A history of South India. *Oxford University press*; 1975; **7**: 68 - 69, 104 - 139

14. Sowrirajan M. Sarabendra Siddha Maruthuva Chudar, Pub. Saraswati Mahal Library, Thanjavur; 1982: 239 - 244, 380 - 382, 386 - 387, 395 - 396, 451 - 452

★

Captain Judith A Bell Krotoski

In February of 2003 another significant event was added to our history. A long time friend and contributor to The STAR magazine is recognized. Captain Judith A Bell Krotoski, OTR, CHT, FAOTA, better known simply as, 'Judy Bell', retired from the United States Public Health Service as an exemplary Commissioned Officer. Judy Bell's commitment will continue in Hansen's Disease, rehabilitation research, teaching and consulting worldwide after retirement. Her dedication and commitment to the health and welfare of others is consistent with her caring and sharing abilities both professionally and personally. This highly respected therapist acquired many titles: researcher, author, orator, teacher and mentor; while others affectionately referred to her as daughter, mother, wife and friend. Judy Bell Krotoski has one son, John and her husband is WA Krotoski, MD, Ph.D., physician/researcher. Judy Bell helped change a generation of ideas, practices' and most importantly, the hands of patients.

Judy Bell began her career as an Occupational Therapist in the Merchant Marine Hospital in New Orleans, Louisiana. She took a special interest in treatment of the hand and regularly attended the orthopedic clinic of Daniel Riordan, MD, noted hand surgeon and teacher. This led Judy to the Philadelphia Hand Center where she began to study sensibility. In 1978 she began a work study program with a world renowned hand surgeon, Paul Brand, a physician from Great Britain working in Hansen's Disease in India and Carville, Louisiana. Judy Bell immediately began making plans to get an assignment at the Carville facility. Judy was assigned a clinical and research position to work with Hansen's Disease patients who had 50-75% peripheral nerve involvement of their hands and feet. Her research was focused on rehabilitative improvements for the hand, primarily focusing on objective measures, and the monitoring and prevention of peripheral nerve involvement and associated deformities. This mixture of research and clinical treatment in rehabilitation was established by Dr Paul Brand, Chief of Rehabilitation at the USPHS Hospital in Carville, Louisiana. This was the beginning of a revolution of change for patients with insensitivity. At that time, much of hand therapy, hand treatment, and all of rehabilitation needed further documentation, research, and substantiation –. thus, research, writing and teaching were natural outcomes of the hand specialty requiring a combination of clinical practice with clinical research which Judy Bell enthusiastically embraced and was a pioneer leader.

Judy Bell was one of six organizing members that founded a group of specialists which became known as 'The American Society of Hand Therapists'. Judy completed extensive research on sensory testing instruments and the validation of objective testing. She played a major role in developing a screening tool to identify patients in need of treatment, that is now used nationally and internationally, and has become a standard testing procedure for clinical and rehabilitation workers to access nerve involvement. Judy Bell obtained study grants as principal investigator to establish baseline measurements on patients and provide input into international Hansen's disease rehabilitation/prevention efforts. One of the outcomes of these studies was the transfer of knowledge regarding monofilament and other monitoring devices of peripheral nerves in the United States, Brazil, India, Ethiopia, Nepal, China and other countries, and also other related diseases such as diabetes.

Judy Bell's research and clinical practices have contributed to the knowledge base of therapy in general, and to the prevention of disability in Hansen's Disease patients worldwide. Her work has helped to underscore the significance of, and need for therapy research and teaching. Her writing and teaching has led to objective measurements, substantiation of therapy treatment, and meaningful outcomes of therapy which directly affects the daily lives of patients. Judy designed teaching and training materials and contributed to a collaborated National Hansen's Disease Programs (NHDP) website project, (www.bphc.hrsa.gov/nhdp). She independently designed and launched a website to highlight the need for evidence-based practice in therapy, (www.ahtf.org). This website is designed to help make grant information and research skills available to all therapists, to encourage collaboration on research projects, and facilitate

(Contd on 16)

Dr. and Mrs. Judy Bell Krotoski

Dr. and Mrs. Bell

Cover photo by Jerry Simmons and Tanya Thomassie

Photos by Tanya Thomassie

(Judy Bell)

international networking. This site has a list of upcoming meeting and training opportunities worldwide, makes intercommunication of therapists possible, and provides links to fellowship programs and conferences.

Judy Bell has served as Principal Investigator, and published several research projects, some of which includes: "Assessment of Levels of Cutaneous Sensibility" 1978; "Monitoring of Patients, Neural Status During Drug Therapy" pilot study 1984 and approved Hansen's Disease Research 1985; "International Project to Measure Peripheral Nerve Involvement Underlying Disability of the Hand in Hansen's Disease" 1990–1995. Her international contributions helped to launch the development of the International Federation of Societies of Hand Therapists (IFSHT). She attended and presented in four International Hansen's Disease Congresses, including Istanbul, Turkey, where she served as organizer and chair of Plenary Session on peripheral nerves, and gave a post Congress two day Workshop covering objective measures and sensory testing to surgeons and therapists at the Hand Microsurgery and Orthopedic Trauma Hospital, Ismir, Turkey in 2002. Judy Bell was coauthor of the first United States book on hand therapy as correlated with hand surgery. "Rehabilitation of the Hand" (Hunter, Schneider, Mackin, and Bell, C V Mosby) – now in its 5th edition. Over 100 papers, chapters and publications have been written by Judy Bell, and numerous presentations have been made regarding hand therapy, research and patient treatment and education. Research information and materials have been widely disseminated to hand therapists and educational travel have been extended to Japan, India, Brazil, Nepal, Ethiopia, San Salvador, Turkey, Holland, The United Kingdom and Canada; many of these trips at her own expense, in order to exchange information, educate, and train therapists. She has researched monofilament materials for calibration, size, design, and bulk distribution overseas to facilities that cannot afford to have access to test equipment. As part of her research grants obtained through the Hand Foundation and American Leprosy Missions was to investigate 'in country' ways of producing monofilament test kits. This project produced simple inexpensive means for field testing nerve involvement in patients, which helped to identify early nerve damage. Judy has served as an international consultant and has communicated with international treatment programs, therapists, and health care workers for over 20 years. She has evaluated and edited numerous international papers while encouraging therapists to do clinical research and report the results of their efforts to support the team approach, improve the health and preventive goals, and to reach for a healthier society.

She has stated that she finds it hard to leave the daily interaction with patients and staff, but plans to stay in touch with the international community in her continued efforts to encourage more research in the prevention of peripheral nerve involvement that leads to sensory and muscle dysfunction in Hansen's Disease. She believes in the role that the Rehabilitation and Education Branch and rehabilitation research plays in educating professionals and patients about the disease. She sees a need for therapists and rehabilitation researchers to play active roles in addressing the disease until elimination of deformity is not just a dream, but a reality. She plans to continue to work with the Rehabilitation Research Lab as a volunteer, and will continue to support the efforts of The STAR magazine and believes The STAR has chronicled the advancements in Hansen's Disease since it's inception. The STAR remains an invaluable international network to inform all affected by Hansen's Disease of progress and significant events. (www.fortyandeight.org)

It is without doubt that Judy Bell's accomplishments will forever be remembered as significant events in the history of the entire Hansen's Disease community worldwide, and for her contributions to better health for all, through her belief and diligence in the team approach to medicine. So it is with continued respect and affection, we thank you, Judith A Bell Krotoski, for your selflessness, your compassion and your contributions to the science, communication, education and research worldwide in Hansen's Disease. Your dedication and commitment to the health and welfare of others is consistent with the purpose of The STAR, to radiate the light of truth in Hansen's Disease.

★

SOURCES OF HD TREATMENT IN THE UNITED STATES

THE NATIONAL HANSEN'S DISEASE PROGRAMS (NHDP) provides HD care to persons in the United States at 1770 Physicians Park Drive, Baton Rouge, LA 70816 and through the Ambulatory Care Program, which includes the following Outpatient HD Clinics:

NATIONAL AMBULATORY HANSEN'S DISEASE PROGRAM

AREA	FACILITY	ADDRESS	PHYS/NURSE	APPOINTMENT
BOSTON	Lahey Medical Center	41 Mall Rd. Burlington, MA 01805	Samuel Moschella, MD Stephanie Burns, RN	781-744-5670
CALIFORNIA LOS ANGELES	LAC, USCMedical Center Attn: Section of Dermatology Room 8440	1200 North State St. Los Angeles, CA 90033	Thomas Rea, MD Helen Mora, RN	323-226-5240
MARTINEZ	Contra Costa Regional Medical Center Outpatient Clinic	2500 Alhambra Drive Martinez, CA 94553	Sutherland/Saffier, MDs Carol James, RN	925-370-5270 1-800-495-8885 (In state only)
SAN DIEGO	North San Diego Health Center	2440 Grand Avenue San Diego, CA 92109	D. A. Lopez, MD Carmen Rodriguez, RN	858-581-4300
CHICAGO	University of Illinois College of Medicine	Department of Dermatology, (MC 624) 808 S. Wood Street, RM 376 CME Chicago, IL 60612	Carlotta Hill, MD Norma Chelseth, RN	312-996-0734
MIAMI	Jackson Memorial Hospital	Ambulatory Care Center 1611 N.W. 12th Avenue Miami, FL 33136	Anne Burdick, MD Gloria Ingle, RN	305-585-2600
NEW YORK	Bellevue Hospital Center Department of Dermatology Room 17-N-7	462 First Avenue New York, NY 10016	William Levis, MD Aloys Cabrera, RN Louis Iannuzzi, PT, C.Ped	212-562-6096
PHOENIX	Maricopa County Health Dept.	1825 East Roosevelt Street Phoenix, AZ 85006	Ronald Pust, MD Bill Cooper, RN	602-372-6661
PUERTO RICO	University of Puerto Rico Medical School	Department of Dermatology P. O. Box 365067 San Juan, PR 00936-5067	Pablo Almodovar, MD Sonia Santos-Exposito, RN	787-765-7950
SEATTLE	Harborview Medical Center	2 West Clinic - 359930 325 9th Avenue Seattle, WA 98104	James P. Harnisch, MD Rebecca Finch, RN Tom McClure, PT, CH	206-731-5100
TEXAS DALLAS	Texas Dept. of Health	2377 N. Stemmons Freeway, Ste. 5 Dallas, TX 75207-2710	Carolyn Lyde, MD Nancy Bernstein, RN	214-819-2010
HOUSTON	Houston Health & Human Services Dept.	3901 Westheimer, Suite 200 Houston, TX 77027	Terry Williams, MD Doris Gay, RN	713-284-9609
SAN ANTONIO	Texas Center for Infectious Disease	2303 S. E. Military Drive San Antonio, TX 78223	Robert N. Longfield, MD Therese M. Shoffner, RN	210-531-4554
HARLINGEN	South Texas Health Care Center OPCL	1301 Rangerville Road Harlingen, TX 78550	Richard Wing, MD San Juana Thompson, RN	956-423-3420 ext. 351

Other Sources: State of Hawaii Department of Health
3650 Maunalei Ave., Suite 205
Honolulu, HI 96816
Phone: 808-733-9831

Mona Bomgaars, MD
Mike Maruyama, Adm.
Lenette Tam, RN
Fax: 808-733-9836

FOR MORE INFORMATION: Call the NHDP at **1-800-642-2477** or fax: (225) 756-3760
Email: Mickey.Templet@access.gov

The Star
PO BOX 325
POINT CLAIR BR
CARVILLE, LA 70721
carvillestar@aol.com
Phone: 225-642-5559 Fax: 225-642-9764

The expiration date will appear on the upper right corner of your address label. If your subscription expires, please renew as early as possible.

FACTS ABOUT HANSEN'S DISEASE

PRSRT STD
US POSTAGE
PAID
PERMIT #1
CARVILLE, LA.
70721

What is (HD)?

Hansen's disease, erroneously associated with biblical leprosy, is a complex infectious disease which, although recognized for more than two thousand years and found to be caused by a bacterium over a century ago, is not completely understood. Dr Gerhard Armauer Hansen, Norwegian scientist, first discovered the HD bacillus in 1873. Considerable progress has been made during the last 40 years, so that today we can treat the majority of cases without undue difficulty and counteract most of the fears generated by the folklore surrounding this disease.

HD is essentially a disease of the peripheral nerves, but it also affects the skin and sometimes other tissues, notably the eye, the mucosa of the upper respiratory tract, muscles, bones and testes.

There are both localized and disseminated forms of HD. If left untreated, HD causes nerve damage, which can result in loss of muscle control and crippling of hands and feet. Eye involvement can result in blindness.

Where is HD Found

In 1994 the World Health Organization estimated that there were 2.4 million cases of HD worldwide with 1.7 million cases registered on treatment. The estimates for 1985 were 10 – 12 million and 5.4 million respectively. According to these estimates, in 1994, 70% of those who should be on treatment are now being treated. In 1992 there were 690,000 new cases reported and in 1993, 591,000 cases. There are also an estimated 2 – 3 million cases who have completed treatment but who still have residual disabilities who are not included in the above 1994 totals. The largest numbers of Hansen's disease patients continue to be in Southeast Asia and Central Africa with smaller numbers in South and Central America. The largest number of patients in the Western Hemisphere are in Brazil.

In the United States there are approximately 6,500 cases on the registry which includes all cases reported since the registry began and still living. The number of cases with active disease and requiring drug treatment is approximately 600. There are 200 – 250 new cases reported to the registry annually with about 175 of these being new cases diagnosed for the first time. The largest number of cases in the US are in California, Texas, Hawaii, Louisiana, Florida, New York, and Puerto Rico. There are still approximately 150 cases at the Gillis W Long Hansen's Disease Center at Carville, LA, the only institution in the US exclusively devoted to Hansen's disease. The center functions as a referral and consulting center with related research and training activities. Most patients in the US are treated under US Public Health Service grants at clinics in major cities or by private physicians. (See inside back page for listing of clinics.)

How Does HD Spread?

While this aspect of the disease remains a medical mystery, the most commonly accepted theory is that it is transmitted by way of the respiratory tract, and abraded skin. The degree of susceptibility of the person, the extent of exposure, and environmental conditions are among factors probably of great importance in transmission. Most specialists agree that 90% or more of the world's population have a natural immunity to the disease. Persons working with HD contract the disease only rarely. Cases of HD which respond satisfactorily to treatment become noninfectious within a short time.

How is HD Treated?

Although the sulfone drugs, introduced at Carville in 1941, continue to be an important weapon against the Hansen bacillus the rising incidence of sulfone resistant disease necessitates treating all patients with more than one drug. Usually rifampin and sometimes clofazimine or ethionamide are given in addition to dapsone. Treatment rapidly renders the disease noncommunicable by killing nearly all the bacilli and these dead bacilli are then cleared from the body within a variable number of years.

Gillis W Long Center

GET TO KNOW THE FORTY & EIGHT

The Forty & Eight, an honor society of legionnaires created in 1920 and *The Star's* primary funding organization, draws its origin from World War I. Millions of American soldiers in France were transported to the front in narrow French box-cars, called "Voitures," which would only hold 40 men or 8 horses. Remembering the close brotherhood of those box-car days, La Societe des Quarante Hommes et Huit Chevaux (The Society of 40 men and 8 Horses) was formed and local Voitures began organizing as outstanding Legionnaires were invited into membership. Membership is still by invitation only.

Dedicated to the needs of their fellowman, the Forty & Eight raises funds and support not only ***The Star,*** but funds a national nursing scholarship program, various child welfare programs, provides aid to veterans and continues to promote Americanism at both local and national levels.