Lepr. Rev. (1979) **50,** 264–270

Abstracts

80. CHANG, W. P. **Ethiopian experience in health manpower training.** *Trop. Geogr. Med.*, v. 30, 147.

However great the benefits of modern medicine might be, these are of little use to "the man in the street" when they are not available to him at the moment of need. The problem of availability of such care is universal, even in the highest developed countries but it is—obviously—greatest in the underprivileged countries of the world. Modern medical care is a demanding mistress. Only when material resources are plentiful, a solid infrastructure is available, communications (roads, public and private transportation, telecommunication) are optimal and health manpower is present in adequate numbers of properly trained personnel, can its blessings be reaped.

A tendency towards "maximalization" has until recently been a guiding principle in the affluent parts of the world: an almost malignant proliferation (to speak in Ivan Illitch's terminology) of hospitals and medical institutions, sophisticated diagnostic facilities, huge increases in medical manpower, all needing longer and more expensive training periods before being allowed to become productive, because "health is priceless" and "for health care only the best is good enough, regardless of costs".

The infection with the "maximalization-trend" can be ruinous to the health-care services of underprivileged countries because it creates super-services for the privileged few and leaves the rest out in the cold, as can be illustrated by far too many unhappy examples.

The principle of "minimalization" in which the number of hospital beds, diagnostic facilities, medical workers etc. is just enough to provide proper care within the limits of the national budget is a sounder one, certainly for developing countries.

In the field of health manpower training, the Ethiopian experience as described by Chang is a valuable one that deserves careful consideration by all involved with the training of such manpower for underprivileged countries.

The experience started in 1954 with the establishment of the "Public Health College" in Gondar, a small rural town in the North of Ethiopia, far away from the capital with its university, big hospitals etc. At that time, Ethiopia had no indigenous doctors available and almost no "para-medical" personnel. After extensive surveys, studies and discussions by UNRRA, WHO, and US TCA groups it was decided to train three types of health workers: *Health officers,* receiving 4 years of medical training after 12 years of general education, *community nurses* receiving 3 years of medical training after 10 years of general education and *sanitarians* also receiving 3 years of training after 10 years of general education. Much of the training took place in the rural setting of Gondar and surrounding area, which is very similar to the Ethiopian rural areas in general, the future working area of these health workers. Preventive medicine was emphasized throughout the courses and teams consisting of a health officer, a community nurse and a sanitarian were formed during the training with the intention of staying together to work in the "Health Centres" of which 94 were created between 1954 and 1974. The curriculum was constantly evaluated and has been modified several times during the 20 years of experience described. The "Health teams" trained in Gondar have generally functioned very well and it was clear that the Gondar Health College had managed to maintain a high standard without losing contact with the rural way of life of the Ethiopians. The health teams possessed the rare combination of qualities of being capable health workers and still feeling at home in a rural setting. The main problems came from the Ethiopian ministry of public health of the period, which often broke up efficient teams by transferring one or more of its members, which often could not provide the essential drugs, equipment, means of transportation etc., and which was sometimes in arrears for several months in paying the salaries. A major blow to the "Gondar concept" also was the affiliation of the college to the Haile Selassie I university in 1962, when the health officer's curriculum was modified to meet a science degree of the university and became

"too academic and less service-oriented". Capable health officers got the opportunity to be "upgraded" to fully qualified doctors which resulted in the breaking-up of a number of health teams and a regression of the rural health services, to the benefit of the urban ones.

Unfortunately, the Gondar training programme has been interrupted since 1974, when the new Ethiopian government called all students and teachers from higher educational institution to participate in the "development through cooperation" campaign, which, as far as I know, is still going on. In due time, the Gondar college will probably resume its activities with some modifications and re-adjustments, but with the same basic and—to my mind—sound philosophy.

The problem of treatment and prevention of leprosy in the rural areas of Ethiopia by these health teams is not discussed in Chang's paper. As far as I know, this disease did not receive special emphasis in the curriculum. The health teams certainly had the know-how—though often not the means—to deal with the medical, preventive and sanitarian aspects of this disease but did not reach the stage of case-finding, search for defaulters etc. In some Ethiopian areas another type of health workers existed, the so called "leprosy dressers" who received some months of specialized training in leprosy, were afterwards based in a health centre or clinic and mainly travelled from market to market to meet their "clients" there and make them swallow "the tablets". At present ALERT (the All Africa Leprosy and Rehabilitation Training Centre) at Addis Ababa has a leprosy control section covering the whole of Shoa province, 15,913 patients being treated in 199 treatment centres, of which 111 are general hospitals, health centres or c inics and 88 leprosy treatment centres only. As far as can be made up from the annual report (977). ALERT is run as a separate service with little integration with the general health services f the country.

A. P. Oomen

81. HARBOE, M., CLOSS, O., REES, R. J. W. & WALSH, G. P. **Formation of antibody against *Mycobacterium leprae* antigen 7 in armadillos.** *J. Med. Microbiol.*, v. 11, 525–535.

The armadillo is already a valuable source of large numbers of *M. leprae*, but it could be exploited more efficiently if it were possible to screen newly caught animals for mycobacterial infections picked up in the wild, and also to screen animals infected in the laboratory for the development of progressive disseminated disease. If this could be recognized at an early stage, only these potentially valuable animals would need to be maintained. Professor Harboe and his colleagues have explored the possibility that antibody measurement could solve both of these problems. The authors also point out that a study of the immunology of the infections in armadillos may lead to ways of recognizing the optimum time for harvest, giving the most satisfactory balance between viability and numbers of organisms. No data are presented on this aspect.

They have employed a radioimmunoassay. Leprosy antigen labelled with ^{125}I is incubated with a suitable dilution of the serum to be tested. Then protein-A containing staphylococci are added. The protein-A strongly binds IgG. Thus the IgG in the serum sample binds to the staphylococci, which can be separated from the incubation mixture by centrifugation. If any of the IgG present was specific for the ^{125}I-labelled leprosy antigen, some of the ^{125}I is bound to the staphylococci, and this binding can be assessed in a gamma counter.

The antigen used in this study (leprosy antigen 7) is not specific to *M. leprae,* but is shared by most, perhaps all mycobacteria, and is therefore suitable for screening animals for mycobacterial infection. Most normal armadillos had lower antibody levels than a panel of Norwegian medical students. Two out of 21 animals had higher values, but there was no histological evidence of mycobacterial infection. Seven animals with mycobacterial infections at the time of capture all showed high values. The only exceptions were two animals with *M. ulcerans* infection, in which antibody was very low, but this is a known peculiarity of *M. ulcerans* infection. Several workers have failed to find antibody in mice infected with this organism. Therefore the technique appears reliable for screening newly caught animals.

The test was then applied to sera from armadillos infected 8–12 months earlier with *M. leprae.* There was significant antibody in 14 of the 17 animals with established infection, but there was no correlation with the extent of infection. Two of the three negative animals were amongst the

most heavily infected. However, the correlation was better in a small group of animals tested 2 years after infection with 10^8 *M. leprae* from a single batch of bacilli.

It is clear that detailed screening, before infection, of animals destined to be donors of *M. leprae* for experimental purposes, is essential, and the use of this technique in addition to the established procedures should be encouraged. However, it is difficult to believe that anybody will yet be prepared to discard an *M. leprae*-infected armadillo at an early stage because the antibody level has failed to rise. Such an act of faith may require support from data on armadillo sera.

G. A. W. Rook

In view of the fact that this number of Leprosy Review *carries four separate items on leprosy in Ethiopia, we take the opportunity to reproduce here a review of an important article on leprosy control in Ethiopia, originally published in 1976—*

82. CAP, J. A. & MULATU, B. **La lèpre en Éthiopie: situation actuelle. [Leprosy in Ethiopia.]** *Med. Trop.* v. 36 (1976) 11–15. (In French)

"The estimated number of leprosy sufferers in Ethiopia is between 128,000 and 135,000, of whom about 59,000 are registered. In a population of 24 million, the prevalence rate varies from 0.1 to 7.0 per thousand, or an overall rate of 2.5 per thousand. Most of the registered patients live in the central, hilly areas, but the higher prevalence rate in these districts may be a reflection of such factors as population density, activity of case-finding teams and the provision of more adequate facilities for treatment. Where prevalence is low, treatment is given at general dispensaries (1 for 28,000 persons in some areas; 1 for 220,000 persons in others); a special leprosy service is organized in areas where the prevalence is high, each trained medical auxiliary being responsible for the treatment of leprosy patients from three to five centers."
(*From* Trop. Dis. Bull.)

S. G. Browne

The abstracts which follow are reprinted from the Tropical Diseases Bulletin, *through the courtesy of the Director, Bureau of Tropical Diseases. They are classified according to subject.*

1. MICROBIOLOGY

83. HARADA, K. & KASAI, T. **Two methods of demonstrating leprosy bacilli in smears.** *Int. J. Lepr.,* 1978, v. 46, No. 2, 167–171.

84. KATO, L. **Cholesterol, a factor which is required for growth of mycobacteria from leprous tissues.** *Int. J. Lepr.,* 1978, v. 46, No. 2, 133–143.

2. IMMUNOLOGY, PATHOLOGY

85. RIDLEY, M. J., RIDLEY, D. S. & TURK, J. L. **Surface markers on lymphocytes and cells of the mononuclear phagocyte series in skin sections in leprosy.** *J. Path.,* 1978, v. 125, No. 2, 91–98.

"E, EA and EAC rosetting techniques and Ig fluorescence were used in a study of receptor sites in cryostat sections of lesions through the spectrum of leprosy, and for comparison in some other mycobacterial and granulomatous lesions. Anti-C_3, and trypsin were used as blocking agents.

"Lymphocytes in borderline lepromatous leprosy produced EA adherence and IgG fluorescence indicating B type cells. Lymphocytes in tuberculoid leprosy produced neither E or EA adherence and no fluorescence; these cells were presumed to be T cells.

"EAC and EA adherence was more marked in areas of macrophage infiltration, where there were few lymphocytes, than over the lymphocytes themselves. Two distinct patterns emerged: (i) EA binding together with IgG fluorescence was seen in active lepromatous leprosy and could be localized to the surface of individual macrophages, and (ii) EAC binding together with IgM fluorescence was seen in the granuloma of tuberculoid leprosy and sarcoidosis, but could not be definitely related to cell surface; rather it was diffusely spread over the whole granuloma; EAC adherence was diminished by anti-C_3 serum. Trypsin removed EA binding completely, but only diminished EAC adherence. It is suggested that the EA pattern indicates immunoglobulin receptors on macrophage and lymphocyte surfaces: and that the EAC binding (which is stronger than EA) involves C_3 and IgM receptors at extracellular sites as well as C_3 receptor sites on epithelioid cell surfaces.

"EA and EAC binding were enhanced in borderline tuberculoid leprosy in reaction and erythema nodosum leprosum, suggesting that immunoglobulin and complement receptor sites increase in number with enhanced hypersensitivity."

This is an important paper and merits being read in full.

M. F. R. Waters

86. VENKATESAN, K. & BHARADWAJ, V. P. **Sequential biochemical investigations in lepromatous leprosy.** *Lepr. India,* 1978, v. 50, No. 2, 166–172.

"Sequential biochemical investigations were conducted in cases of lepromatous leprosy in the reactive as well as subsided phases. Low levels of blood sugar and serum cholesterol were indicated in the reactive phase of lepromatous leprosy. Significant increase in thymol turbidity and decrease in A/G ratio were noted in most of the cases of lepromatous leprosy. Enhancement of serum levels of transaminases was observed in the reactive phase of lepromatous leprosy. Serum protein electrophoresis indicated increases in α_2-globulin and r-globulin and decrease in albumin in the reactive as well as subsided phases. The results are discussed in this paper."

87. SAOJI, A., MENE, A. & SHARMA, K. D. **Electrophoresis and immuno-electrophoresis in leprosy.** *Lepr. India,* 1978, v. 50, No. 2, 161–165.

88. CHANDI, S. M. & JOB, C. K. **The early cellular response to *M. leprae:* an ultrastructural study.** *Lepr. India*, 1978, v. 50, No. 3, 345–351.

"The ultrastructural changes that develop in mouse peritoneal macrophages from 10 min up to 14 weeks after exposure to *Mycobacterium leprae* are presented. Phagocytosis occurred by a process of engulfment by cytoplasmic processes and incorporation into a phagosome, into which lysosomal enzymes were subsequently introduced. Electron transparent zones (E.T.Z.) were not observed around phagocytosed bacilli in this study, however discrete droplets of lipid-like material appeared in the cytoplasm of macrophages, between 2 and 4 weeks after ingestion of the micro-organisms. Phagosomes with double limiting membranes were observed in macrophages harvested as early as 40 minutes after exposure to *M. leprae,* contrary to the observations of Evans and Levy (1972)."

[See *Trop. Dis. Bull.,* 1972, v. 69, abstr. 1410.]

89. SHER, R., ANDERSON, R., GLOVER, A. & WADEE, A. A. **Polymorphonuclear cell function in the various polar types of leprosy and erythema nodosum leprosum.** *Infection & Immunity,* 1978, v. 21, No. 3, 959–965.

"Polymorphonuclear leukocyte motility, both *in vivo* and *in vitro*, and reduction of Nitro Blue Tetrazolium was studied in tuberculoid and lepromatous leprosy patients and a group of lepromatous patients with erythema nodosum leprosum (ENL). A profound defect in random migration, chemotaxis, and chemokinesis was found in lepromatous patients with and without complicating ENL, and marked depletion of skin window migration confirmed these *in vitro*

findings. Tuberculoid patients exhibited a mild defect in polymorphonuclear leukocyte motility. Serum inhibitors of normal polymorphonuclear leukocyte chemotaxis were found in all types of leprosy, but sera from lepromatous and ENL patients were most inhibitory. Resting levels of Nitro Blue Tetrazolium reduction were normal in all three groups. Reconstitution of polymorphonuclear leukocyte cells from normal and ENL patients with ENL serum, however, showed increased Nitro Blue Tetrazolium reduction well above the normal range, whereas reconstitution with normal, lepromatous, and tuberculoid sera failed to increase Nitro Blue Tetrazolium reduction above the normal values."

90. STONER, G. L., TOUW, J., BELEHU, A. & NAAFS, B. ***In-vitro* lymphoproliferative response to *Mycobacterium leprae* of HLA-D-identical siblings of lepromatous leprosy patients.** *Lancet,* 1978, Sept. 9, 543–547.

"Lymphoproliferative responses to *Mycobacterium leprae* and P.P.D. were measured in 23 lepromatous and borderline lepromatous leprosy patients and in 27 of their normal siblings. At the same time siblings HLA-D-identical with the patients were identified by the absence of a mixed-lymphocyte reaction. The 7 siblings who were HLA-identical to lepromatous patients responded as well to *M. leprae* as did the 20 HLA-non-identical normal siblings. In contrast, 22 of the 23 lepromatous patients failed to respond to *M. leprae* but responded normally to P.P.D. The specific unresponsiveness of lepromatous patients thus does not result from an HLA-linked genetic defect and the defective cell-mediated immune response to *M. leprae* seems to be acquired, not inherited. Lepromatous patients may be high responders to antigens shared by *M. leprae* and other microorganisms in whom a strong antibody response has blocked the induction of an *M. leprae*-specific cell-mediated immune response."

91. REA, T. H. & TAYLOR, C. R. **Serum and tissue lysozyme in leprosy.** *Infection & Immunity,* 1977, v. 18, No. 3, 847–856.

"Mean serum lysozyme values were found to be elevated in untreated leprosy patients. Statistically significant elevations were present in each of the three major categories of leprosy, tuberculoid, borderline, and lepromatous. Values were particularly high in patients with severe reversal reactions or Lucio's phenomenon. Prolonged sulfone therapy was associated with a fall in serum lysozyme values. With an immunoperoxidase method to localize lysozyme in leprous tissues, two distinct staining patterns were found, granular and saccular. The granular pattern of lysozymal staining was found in epithelioid cells and in giant cells, and the intensity of staining showed a positive correlation with serum lysozyme levels. Conversely, a saccular pattern of lysozymal staining was found in lepromatous histiocytes, but the intensity of staining was unrelated to serum lysozyme levels; the saccular structures contained dense aggregates of *Mycobacterium leprae.* These two patterns of staining probably represent different functional responses of monocyte-derived granuloma cells, whereas the serum levels reflect, to a varying degree, both the absolute number of such cells and the rate of secretory activity of this cell population as a whole."

92. RIDLEY, D. S. **The pathology of leprosy.** *S. E. Asian J. Trop. Med. Publ. Hlth,* 1978, v. 9, No. 2, 205–208.

93. HIRSCHBERG, H. **The role of macrophages in the lymphoproliferative response to *Mycobacterium leprae in vitro*.** *Clin. Exp. Immunol.,* 1978, v. 34, No. 1, 46–51.

"Peripheral blood lymphocytes from patients suffering from lepromatous leprosy do not normally react *in vitro* to stimulation by *Mycobacterium leprae* antigens. In contrast, we found that T cells from non-responding patients in combination with macrophages from responding patients or healthy contacts did respond well to *M. leprae.* Conversely, T cells from responding patients or healthy contacts in combination with macrophages from non-responding patients

failed to respond. It seems, therefore, that the lack of response normally observed in *in vitro* tests using cells from lepromatous leprosy patients is due to a failure of their macrophages to present *M. leprae* antigens in an immunogenic form."

3. CLINICAL

94. NIGAM, P., GOYAL, B. M., MISHRA, D. N. & SAMUEL, K. C. **Reaction in leprosy complicated by filariasis.** *Lepr. India,* 1977, v. 49, No. 3, 344–348

95. FURUTA, M. *et al.* **Frequency of cerebrovascular lesions in leprosaria.** *Jap. J. Lepr.,* 1978, v. 47, No. 2, 61–65.

"Cerebrovascular diseases, especially cerebral haemorrhage, have been thought the most frequent cause of death in Japan. The Ministry of Health and Welfare has reported that cerebrovascular diseases have been the top cause of death in Japan since 1951. Pathologists, however, have not experienced so many autopsy cases who died of cerebrovascular diseases. Then, we investigated the cause of death in two leprosaria and compared it with information from the Ministry of Health and Welfare.

"One hundred and twenty-seven patients died in Komyo-en Leprosarium between 1962, January, and 1971, June. Autopsy was done on 110 cases. The average age was 62.5 years old. Malignant neoplasms (33 cases) were more frequent than cerebrovascular diseases (haemorrhage: 9 cases, softening: 3, microscopic haemorrhage: 11). The major direct cause of death was bronchopneumonia. Investigation of the death certificates in Seisho-en Leprosarium for the years 1967–1976 also revealed that cerebrovascular diseases were not the major cause of death. These results are different from information of the cause of death in Japan published by the Ministry of Health and Welfare. This discrepancy probably comes from inaccurate description of the death certificates and low autopsy rate in this country."

4. THERAPY

96. NOORDEEN, S. K. & NEELAN, P. N. **Extended studies on chemoprophylaxis against leprosy.** *Indian J. Med. Res.,* 1978, v. 67, Apr., 515–527.

"The role of dapsone as a chemoprophylactic was studied among (*a*) 955 household contacts of lepromatous leprosy when the drug was administered once a week in a dose schedule equal to about 1 to 2 mg per kg body weight per week, and (*b*) 2000 household contacts of non-lepromatous leprosy when the drug was administered twice a week in a dose schedule equal to about 4 mg per kg body weight per week. The study was double blind with comparable controls. The contacts received about 90% of the expected treatment. The results showed that chemoprophylaxis with dapsone was effective, the protection received by the contacts of lepromatous cases varying between 37 and 40%, and the protection received by the contacts of non-lepromatous cases being about 35%. The protective value of chemoprophylaxis in preventing occurrence of lepromatous cases could not be studied as no new lepromatous case occurred even in the control group. Although the protection from chemoprophylaxis was moderate, certain subgroups were found to be associated with higher protection. These subgroups, in general, had a higher risk of getting leprosy as observed from the occurrence of disease in the relevant control groups."

6. MISCELLANEOUS

97. BRAND, P. **Insensitive feet. A practical handbook on foot problems in leprosy.** 88 pp. Revised 1977. The Leprosy Mission, 50 Portland Place, London, W1N 3DG. [Free]

After describing the structure of the foot and the protective role of normal sensation, the author

discusses, in turn, paralysis of nerve supply, plantar ulceration, and damage to bones—with special emphasis to tarsal bones. On each of these subjects prevention and treatment are fully covered. The final chapter is devoted to the subject of footwear for insensitive feet.

[The management of insensitive feet is of such importance that all those engaged in treating leprosy should study this clearly written and well illustrated booklet.]

W. H. Jopling