ABSTRACTS

A Proposito de um Caso de Manifestação Aguda da Lepra (a Case showing the Acute Leprosy). R. N. MIRANDA, *Revista Brazil de Leprologia*, **29**, 1, March 1961, pp. 3–12, 9 figs. in colour.

The author describes a case of lepromatous leprosy showing acute phenomena, namely fever, adenopathies, and leucocytosis, and erythematous and necrotic patterns of skin lesions The secretion of the skin lesions showed globi of *M. leprae* sited intracellularly in neutrophils. There was an acute episode lasting 30 days. Then the patient got well but entered a chronic diffuse lepromatous leprosy and new acute outbreaks. The author names this state "Acute Leprosy Manifestations," with 5 clinical varieties, nodular, polymorphous, suppurative, dimorphous, and necrotic. The present patient belongs to the necrotic class. He thinks these conditions result from a break in quiescent relations between *M. leprae* and the histiocytic cell, resulting in the liberation and dissemination of bacilli (either *in situ* or in the blood) or a new different reaction of the human body (shown by acute lesions, leucocytosis, and neutrophilia) which attempts to destroy the bacilli in the tissues and the disseminated bacilli. The author draws attention to the finding of the bacilli phagocytised by neutrophils, which he thinks to be the main event in acute leprosy manifestations. It points to a destruction of the disseminated bacilli, which is especially attained when there is pus in the lesions.

Viragem Lepromínica Após Retestagem em Crianças de 0 *a* 4 *Anos* (Lepromin Conversion after Retesting in Children 0–4 Years). L. M. BECHELLI and R. PAULA SOUZA. *Revista Brasil, de Leprologia*, **29**, 1, March 1961, pp. 13–18.

In 1953 and 1955 the authors studied with Ferraz and Quagliato the action of BCG in school children 5–9 and 10–14 years of age, who were free of leprosy and without known previous exposure. They found that retesting by lepromin converted from negative to positive in a proportion of cases practically similar to results obtained by oral BCG. In the present study, 46 children were retested for the Mitsuda reaction, of whom 12 were under 1 year old, 26 from 1–2 years of age, and 8 from 2–4 years. In the first test, of 46 children 0–4 years, 27 were negative, and 8 became positive 1+ and 11 positive 2 plus. The test was repeated after 40 days in the 46 children; in 41 children 14 were negative, 21 positive 1 plus, 5 positive 2 plus, and 1 positive 3 plus.

Lepromatosos em Tratamento Sulfonico (Lepromatous under Sulphone Treatment). R. QUAGLIATO, E. BERQUÓ, and W. LESER. *Revista Brasil de Leprologia*, **29**. 1, Mar. 1961, pp. 19–30.

The authors studied lepromatous patients discharged from sanatoria with the disease arrested, registered in the Campinas dispensary, negative on the occasion of their registry and up to 6 months afterwards kept under observation on a regime of sulphone treatment. It was found that the patient registered 1949–1952, expressed as cumulative coefficient of reactivation during $3\frac{1}{2}$ to $6\frac{1}{2}$ years of observation, were significantly more frequent than those registered from 1953–1959. In this latter period 1953–59 the cumulative coefficient of reactivation ran from 0.7% in the first half year to 26.4% at $6\frac{1}{2}$ years. It was noted that reactivated patients soon became negative again.

Alguns Aspectos da Nutricão em Face de Profilaxia e Tratamento da Lepra (Some Aspects of Nutrition Affecting Prophylaxis and Treatment of Leprosy). D. N. DA CUNHA, *Revista Brasil de Leprologia*, **29**, 1, Mar. 1961, pp. 31–33.

The author states that the leprosy bacillus is of limited pathogenicity but malnutrition of the body is a relevant factor in the beginning, clinical form, and later evolution of leprosy, whether human or experimental. Numerous papers show this, from the well-know studies of BADGER and SEBRELL with rats, wherein the incubation time for murine leprosy was shortened by Vitamin-B--deficient diets. Also, COLLIER inoculated leprotic material in monkeys on a diet containing sapotoxins and the disease rapidly appeared. Also AKROYD-KRISHNAN in Madras studied areas of high prevalence of leprosy, and found very varied nutritional deficiencies in families with a high incidence of leprosy among their members, these deficiencies being protein, mineral, vitamin, and even caloric in almost all cases investigated. On the other hand, simple improvement in the conditions of living had a decisive influence towards decrease of the leprosy infection, which attests the markedly social character of this disease, analogous to tuberculosis and chronic infections. It seems that the body resistance and the state of immunity of any infection are primarily and intimately bound up with the nutritional state of the individual, chiefly during childhood and in some cases even prenatal. Evidently other factors, such as heredity fatigue, intoxications, and nervous tension and anxiety, are responsible, but have less influence than the state of bodily nutrition. In the case of leprosy this method of approach is important in prophylaxis and treatment, and these factors can impede or retard the initial signs, can modify their evolution or accelerate their clinical cure. In leprosy the study of these influences on the peripheral nerves and skin has peculiar interest.

LUITHLEN supports this point of view. He says (1) nutrition can increase or diminish the reactivity of the skin against inflammation; (2) acid diets make the skin hypersensitive to external irritation and alkaline diets diminish the cutaneous reactivity; (3) skin reactivity is increased by an excess in the tissues of potassium and sodium, and is diminished by a greater concentration of magnesium and calcium. MAYER and SULZBERGER also confirm the influence of acid and alkaline diets, noting that guinea pigs can easily be sensitized to necarsphenamine in the summer, but not in the winter. When the guinea pigs had the winter diet during the summer, of dry forage, they attained a hyper-reactivity not only to necarsphenamine but also to other drugs.

In the prophylaxis of leprosy there should be given a balanced diet, normal in all respect for a higher protein value with the idea of favouring antibody development. In the ACKROYD-KRISHNAN investigation in Madras in 1937 there was a low protein level in the diets of the families studied, and the 42 g. per diem that each had was mostly vegetarian in origin. (It should be 1.5 g./kilo/day).

During the treatment of leprosy, diets poor in sodium and potassium and rich in calcium and magnesium, favour better internal conditions, diminish the incidence of reactional forms and the skin sensitivity, are more favourable to oedematous states. Acidity or alkalinity of the diets should be carefully studied. A slight disequilibrium in favour of alkalinity is beneficial. Because of numerous studies on the influence of certain vitamins on the skin and nerves, such as those of BADGER and SEBRELL, already cited, the diets prescribed in human leprosy, particularly in the predominantly neural forms, should possess a high level of vitamins B^1, A, and E. The evolution and duration of reactional phases in leprosy can be greatly influenced by a diet poor in salt, rich in glycines, proteins, and vitamins, with a light alkaline predominance, poor in fats and fried foods in general, a diet on the whole similar to that used in hepatic conditions. The proteins of milk and its derivatives are beneficial. RAMBO thinks that the diet in leprosy should be made up on the following basic principles:—(a) a variation in the dietary elements, to avoid monotony; (b) need for leaf vegatables; (c) raw foods, vegetables or citrus fruits, to maintain Vitamins A and C; (d) milk and its derivatives, as the chief source of protein and minerals; (e) a partial substitution of rice by oats, as a cereal rich in the B complex. Beer can also be used as a source of B complex.

Estudo Anãtomo-Clínico de 18 *Casos de Lepra Dimorfa* (*Study of* 18 *cases of Dimorphous Leprosy*). A. M. ALONSO and R. D. AZULAY, (paper delivered at the Symposium on Dimorphous Leprosy in Rio de Janeiro, Mar. 1960, under the auspices of the Brazilian Association of Leprology). *Arquivos Mineiros de*

Leprologia, **20,** 3, July 1960, pp. 303–313. Original in Portuguese. 5 photographs.

The authors conclude that the manner in which we combine the characteristic aspects of the 2 polar forms of leprosy in considering possible borderline leprosy varies greatly whether the point of view is clinical or histopathological. Both the typical cellular and tissue features of the polar types can co-exist entirely or partially in the same single lesion, and sometimes it is necessary to make several biopsies to confirm the case. Sometimes the clinical approach is without any evidence to cause one to suspect dimorphous cases, and the histopathological result from the laboratory is a complete surprise. At other times clinically it is possible to recognise a dimorphous case but this is not confirmed from the laboratory, with its limited microscopic field of examination. Though smears from the mucosa and lesions can be bacteriologically negative, the histology should always show the presence of acid-alcohol-fast germs in the lesions. The results of the lepromin test are variable, and can be positive, negative, or oscillating. From their experience the authors give in general a grave prognosis to such cases, but all their patients responded to modern therapy. A tuberculoid reactional eruption supervening in lepromatous cases under treatment must be related to this dimorphous kind of leprosy: the same applies to leprominic positivization which arises in patients considered lepromatous and negative bacteriologically after clinical improvement.

Consideracões Sõbre Casos Dimorfos (*Discussion of Dimorphous Cases*). A. C. PEREIRA (paper delivered at the Symposium on Dimorphous Leprosy in Rio de Janeiro, Mar. 1960, under the auspices of the Brazilian Association of Leprology). *Arquivos Mineiros de Leprologia*, **20,** 3, July 1960, 00. 231–331. Original in Portuguese. 12 photos.

The author concludes that the dimorphous group described by WADE and other authors is an intermediary clinical form of leprosy, and was studied in its transition from reactional tuberculoid towards lepromatous. Before the advent of the specific treatment the dimorphous form was also met with in the regression of lepromatous to tuberculoid. The dimorphous cases of greater diagnostic interest are those which show clinical symptoms of the lepromatous form with positive bacteriology and negative lepromin test, and these reveal a greater gravity. The leprologist working in the great centres in general has no other means but the clinical in order to classify his cases. Dimorphous cases need the help of histopathology in order to be diagnosed, and in fact do not give great anxiety when they are put under adequate treatment. The specific treatment of leprosy has modified our ideas as to what to do for the dimorphous case. The

author uses BCG to help to increase the resistance of the dimorphous case, chiefly in those who have weakly positive lepromin test, and thus obtains 33% of conversion or reinforcement of the reaction.

Considerõces Sôbre Casos Dimorphous (*Discussion of Dimorphous Cases*). I. R. VIEIRA (paper delivered at the Symphosium on Dimorphous Leprosy in Rio de Janeiro, Mar. 1960, under the Auspices of the Brazilian Association of Leprology). *Arquivos Mineiros de Leprologia*, **20,** 3: July 1960, pp. 342–353. Original in Portuguese.

The author thinks that lepromatous cases do not have the supervention of dimorphous aspects, nor do they have mutation phenomena. Central lepromatous cases decline in the direction of the indeterminate group passing through a stage which we can call pseudo-tuberculoid because of a transitory similarity with tuberculoid. In the reactional tuberculoid cases, chiefly those of an enduring permanent nature are those which we can define as borderline or dimorphouse and as part of a group of that nature.

Contribuicão a Estudo Clínico da Lepra Dimorfa (*Contribution to the Clinical Study of Dimorphous Leprosy*). NELSON SOUZA CAMPOS paper delivered at the Symposium on Dimorphous Leprosy in de Janeiro, Mar. 1960). *Arquivos Mineiros de Leprologia*, **20**, 3: July 1960, pp. 354–366. Original in Portuguese.

The author thinks from his study of the matter, with other colleagues, that the present group called dimorphous or borderline should be suppressed, re-establishing the true idea of the polar type. A new group should be created, joining the tuberculoid-in-reaction variety with the present dimorphous or borderline group. To denominate the new group to be created, author should remember the existing expressions (dimorphous, bipolar, interpolar, transitional, limitrophic, limitant). The expression "borderline" should be rejected, as being strange in our language. The expression "interpolar" which so far has had a less generalized use, should be favourably considered. He suggests that for greater precision the cases of the new group might be further designated as Xt (X being the general name of the new group) for cases belonging to present reactional tuberculoid leprosy, with positive Mitsuda reaction, and XI for cases of the present dimorphous group and the tuberculoid reactional variety with negative Mitsuda.

Contribuição ao Estudo Histopatológico da Lepra Dimorfa (*Contribution to the Histopathological Study of Dimorphous Leprosy*). P. RATH DE SOUZA. (paper delivered at the Symposium on Dimorphous Leprosy, Rio de Janeiro, Mar. 1960). *Arquivos Mineiros de* **20,** 3: July 1960; pp. 367–375. Original in Portuguese.

He has studied many cases and found a very mixed and variable cellular and tissue pathology. Along with NELSON SOUZA CAMPOS he makes the proposal of suppressing the present group of dimorphous or borderline and excluding the reactional tuberculoid variety, and creating a new group which fuses the tuberculoid reactional variety in the present dimorphous or borderline group, etc, (as in the paper by NELSON SOUZA CAMPOS.

Borderline Group under the Clinical Viewpoint. F. E. RABELLO, (paper delivered at the Symposium on Dimorphous Leprosy, Rio de Janeiro Mar. 1960). *Arquivos Mineiros de Leprologia*, **20**, 3, July 1960, pp.412–429. Original in English.

The author points out that the architecture of both polar forms may occur in the same patient, either both pictures standing together in the same skin area or in different places, or the lesions occur in different periods during the evolution of the disease. As to whether the tuberculoid and lepromatous granulomata keep their fundamental characteristics, the opinions of various authors are as follows: AZULAY does not report alterations in any structural aspect. So he found Virchow cells in 14 of 18 cases, well-defined tuberculoid granulomata in 11, and there were gigantocytes in 5 of these. The infiltrates were nodular, of focal or diffuse aspect, and the inflammatory reactions around the granulomata were scanty, of a few plasmocytes and lymphocytes. The tuberculoid part could be torpid or reactional. The diffusion of the cellular exudate varied, sometimes mainly lepromatous, sometimes mainly tuberculoid. Unna's band was present in 15 cases. The exudative process reached the epidermal limits in 6 cases. The mixed structure of T plus L in the same slide was noted in 11 cases. Diagnosis was made by examination of several slides of the same case. In all cases bacilli were positive, though in some cases only by histological examination. Y. R. VIERA thinks the existence of bipolar structure is doubtful. He never saw an undoubted lepromatous structure alongside the tuberculoid one. More often there is an encroachment or mild mixture of opposite structures. P. RATH DE SOUZA says that the bipolar granuloma are formed by cells of two types, epithelioid-like cells and histiocytes with vesicular nucleus and un-vacuolated cytoplasm, very similar to macrophages found in active lepromatous lesions. There is evident disagreement in these 3 reports, perhaps because of different conditions and circumstances. Interesting features also reported are (1) lipid researches by AZULAY, (2) the fibrous stroma granulomata reported by VIERA (3) the variations in length of bacilli noted by P. RATH DE SOUZA. Research on these lines should be developed, and agreement will finally be reached. The present disagreement on borderline leprosy may be only transient.

Lepra Borderline. J. GAY PRIETO, (paper delivered at the symposium on dimorphous leprosy held in Rio de Janeiro, Mar. 1960, under the auspices of the Brazilian Association of Leprology). *Arquivos Mineiros de Leprologia*, 20th year, No. 3, July 1960, pp.444–456. Original in Spanish.

This paper is a systematic study of all the opinions about Borderline leprosy. It concludes that borderline leprosy consists of a group of cases which are intermediary between the tuberculoid type and the lepromatous type, which exceptionally can evolve towards the lepromatous type. Borderline forms an unstable group, generally not malignant, which responds well to treatment, and its clinical manifestations are influenced well by the treatment made up of sulphone therapy, thiosemicarbazones, and corticosteroids. It contains 2 varieties, one nearer to the tuberculoid type, which is the reactional tuberculoid form or variety TR, the other nearer to the lepromatous type, with which some cases have a singular similarity, which is the genuine borderline variety or form.

Clinically it is characterized by elevated lesions of nodules, plaques, or bands or reddish or vinous colour, sometimes grayish, which sometimes resemble the tuberculoid reaction, and at other times especially in some regions like the lobules of the ears they resemble the lepromatous type. The general state can be affected. Almost always the bacilloscopy is positive, and is more intensely so as the case is nearer to the lepromatous type. The lepromin test is apt to be negative; in some cases it is positive although never intensely so. The so-called macular dimorphous form is neither a group nor a type. It is a habitual stage of evolution of indeterminate cases towards the lepromatous type, and exceptionally the designation has been given to cases of abrupt transformation of indeterminate into tuberculoid cases.

Simposio de Lepra Borderline (Symposium on Borderline Leprosy) O. SERRA: *Arquivos Mineiros de Leprologia* **20,** 3, July 1960 pp.456-460.

The author discusses several points raised by other authors, and gives his scheme of Borderline and its place in the classification of leprosy. For him the Tuberculoid polar form contains tuberculoid in reaction (Tr.), which is lepromin-positive. In between is the perilepromatous group (PL), including lepromin-negative pseudo-tuberculoids. On the right of that, verging towards the polar form L, is perilepromatous in reaction (pLr). The polar form L also contains leprotic reaction (Lr).

Lepra Borderline: Grupo Perilepromatouso, Satelite do Tipo L. (Borderline Leprosy: the Perilepromatous Group, Satellite of the

L Type). A. ROTBERG. *Arquivos Mineiros de Leprologia*, **20**, 3, July 1960 pp.463–469.

The author thinks that the lepromatous and tuberculoid types are very firm and stable and there has been no convincing evidence of transformation of one to the other, and there is no place for transitional, borderline, or dimorphous cases. These words only signify transition from L type to another usually anergic and often bacillary group called reactional tuberculoid. But only resistant lepromin-positive cases should be labelled "tuberculoid", The lepromin-negative or lepromin-doubtful often bacillary pseudo-tuberculoid cases (as well as their advanced stage, which is borderline) should be located within the L pole as a satellite group which may be called "perilepromatous".

Symposium on Leprosy 30 *Jan.* 1961 *at School of Tropical Medicine, Calcutta.* Bulletin of the Calcutta School of Tropical Medicine **9,** 2; April 1961, pp.69–79.

Dr. R. N. CHAUDURI gave the address of welcome at this symposium and said that leprosy research, control, and relief cannot be separated from each other, and that the introduction of the sulphone drugs has encouraged experiments with mass treatment and selective segregation. He recounted the origin and develpoment of the Calcutta School, and the work of MUIR and LOWE, and the great work of the School in training doctors. Lt. Gen. D. N. CHAKRAVARTI gave the inaugural address and mentioned that there are 300,000 leprosy patients in the State of West Bengal, and the rather poor response from the medical profession to come forward for antileprosy work. Dr. S. N. CHATTERJEE spoke on the Classification of Leprosy and gave a good historical review of its development and of the preferences of Indian leprologists on this subject. The Indian Classification divides leprosy into Non-lepromatous, Intermediate, and Lepromatous, and uses the terms maculo-anaesthetic, tuberculoid, polyneuritic, borderline, indeterminate, and of course lepromatous. Dr. S. GHOSH described clinical features and reaction in leprosy. Dr. S. KUNDU discussed helpfully the diagnosis and described the laboratory tests. Dr. S. P. BASU described radiological bone findings and angiography, and Dr. P. C. SEN GUPTA the pathological changes, including the histology, and the histology of reaction. Major E. J. SOMERSET described ocular lesions and their management, and Dr. P. N. KHOOSHOO the working of the national leprosy control scheme which runs on Five Year Plans and Control Centres and Subsidiary Centres and makes use of paramedical workers. Dr. N. MUKERJEE dealt with therapy and mentioned the standard drugs and many others under trial. Immunity in leprosy was dealt with by Dr. D. C. LAHIRI and experimental leprosy by Dr. N. C. DEY. The important

subject of surgery in leprosy was dealt with by Lt. Col. N. C. CHATTERJEE, and plastic surgery in leprosy by Dr. M. M. MUKHERJEE, and plastic surgery in leprosy by Dr. M. M. MUKHERJEE. Physiotherapy was discussed by Dr. S. K. SARKAR. The whole symposium report deserves to be studied carefully in the original, as it gives an encouraging impression of the high standard of present-day work by Indian leprologists and surgeons.

History of Antileprosy Legislation in South America in the Colonial Period. H. C. DE SOUZA-ARAUJO. Revista Brasileira de Medicina, **18,** 2; Feb. 1961.

For *Argentina* the author recounts that since 1596 there were imported African negroes who were subjects of leprosy, as well as white European immigrants. In 1778 the population of Buenos Aires was 37,130 of whom 30,196 were of Spanish and negro origin, mostly the latter, and there were 6,934 natives, Indians and mestizos and mulattoes. In 1778 control measures were established against endemic diseases, including leprosy, and leprosaria were set up in Santa Fe, Cordoba, Salta, and Tucuman. In Santa Fe in 1793 the total population of 2000 contained 14 leprosy patients who were begging from door to door, and 6 deaths from leprosy were registered. In *Brazil* leprosy was imported by European immigrants and especially by African slaves, about 50,000 of them from 1500 to 1591. In 1798 the total population was $3\frac{1}{4}$ million, of whom about 60% were Africans. Rio in 1741, Bahia in 1787, and Recife in 1789 had the first leprosaria, and most of the inmates were negroes or mulattoes. The first antileprosy law for Brazil was drawn up by a committee of 3 Lisbon doctors in 1741. The rules considered leprosy a contagious disease of greater or less degree according to its clinical type, and segregation of all confirmed cases was recommended without distinction. Leprosaria were established, with separation of sex and social classes. A health officer was given full authority to enforce the law. There was compulsory notification of cases, and special diagnostic examinations were given to distinguish leprosy from other diseases. In *Paraguay* leprosy was known from the beginning, and the natives were free of the disease. In *Peru* in 1563 there were a few cases in Lima, but the disease did not become prevalent. In *Venezuela* the first case of leprosy was reported in 1626, the patient being the Governor of the province, and other cases appeared between 1627 and 1640. Caracas leprosarium was founded in 1572. The author's *general conclusions* are (1) there was no pre-columbian leprosy; (2) the disease was introduced into South America by European colonists and African slaves; (3) the Colonial regulations considered leprosy contagious in varying degree; (4) there was compulsory notification and segregation of cases until cure; (5) cases of higher social class were allowed to be treated at home.

Statistical Records for the Medical Services of the Uganda Protectorate issued for 1959 give a *Summary of Leprosy Work*, pp.41–42 in 2 tables.

Details are given from the 4 provinces of Uganda, and for the 4 leprosaria (Buluba, Nyenga, Kumi-Ongino, and Kuluva). These are Mission leprosaria subsidised by Government and contain about 3000 patients. *The all-Uganda totals* given are: (1) The gross intake of patients for 1952–1959 was 71,147 patients; (2) Those diagnosed as leprosy patients were 57,424; (3) The total of cured 1952–1959 was 16,276; (4) Absentees over 1952–58 totalled 19,682, with about 8,000 in 1959; (5) In 1959 there were 31,444 patients attending; (6) There were 85 leprosy treatment villages and 126 other clinics, and the village accommodation for 4,069 patients had an average resident number of 3,320. There is no information given about the incidence of deformities.

(From the WHO Technical Report 16,221 Geneva, 1961 on Rehabilitation in Leprosy "those with physical disability represent one quarter of the total cases", EDITOR).

Incaparina (*low cost vegetable food with adequate protein developed against protein malnutrition.*) Report of the 12th Meeting of the Panamerican Health Organization, Washington, D.C. 1961. Document No. 36, pp. 286–291.

The Annex describes a vegetable food developed by the initiative and scientific work of INCAP for use in Central America in the prevention of protein malnutrition, which will be of great interest also to those in charge of leprosy institutions and leprosy control schemes. There are many parts of the world where protein malnutrition touches closely the therapeutic regimes used for leprosy, and the social background of the disease).

INCAP Vegetable Mixture 9 B contains 29% ground maize, 29% sorghum grain, 38% cottonseed flour, 3% Torula yeast, 1% calcium carbonate, and 4,500 I. U. of added Vitamin A per 100 g. It has a protein content of 27.5% and is similar to milk in protein quality. It can be produced at very low cost, (6.21 cents per pound at maximum production) and in the form of a thin gruel has proved to be highly acceptable in Central America. This formula and other similar ones will be known by the generic name of INCAPARINA. Under the Council of INCAP the public health authorities of any country in South America can be authorized to produce and distribute INCAPARINA, and it will be now feasible to produce for widespread use this low-cost, highly nutritive, and extremely acceptable vegetable mixture and use it in national efforts against protein malnutrition.