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The Classification of Leprosy

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INTRODUCTION

This article is an attempt to add to the recent discussions on the classification of leprosy, especially the contribution by Leiker (1966) and to further develop his thesis, seeking clarification of this important problem. Most recent writers, including Leiker (Ridley 1962, Cochrane 1964) have accepted the basic concept of a spectrum of disease in leprosy. However, there is still a great deal of confusion as to the position of specific cases in this spectrum, and more especially is there confusion in the use of terms which are not universally accepted nor uniformly defined.

Frequently, due to erroneous concepts about classification, workers have come to faulty conclusions about many aspects of leprosy. Such conclusions must be in error if based on wrong postulates and concepts of the pattern of disease. A notable example of this is the recent article 'The Onset and Pattern of Deformity' by Mallac (1966). This is an excellent article attempting to refute the old concepts of 'trophic' causes of disability. But in spite of its being a very real contribution to the literature on disability in leprosy, there still remains the implication of the inevitability of deformity in leprosy. The belief that deformity is some inscrutable concomitant of leprosy that may in any patient suddenly arise to curse his future with an irreparably disabling stigma has not been removed. Dr. Mallac fails to show that this is not the case, primarily because he does not fully acknowledge the direct relationship of disability to the classification of leprosy.

There is a fundamental need to understand the classification of leprosy in order to gain a satisfactory concept of the evolution of disease as well as to understand its epidemiology. In fact, when we shall fully understand the concept of disease manifestations as they relate to host resistance there will undoubtedly be a clarification of our understanding concerning the epidemiology of leprosy; at present an area of considerable speculation and conjecture based on inadequate premises and faulty theories. Much more importantly, adequate case handling and treatment are based to a great extent on a complete understanding of classification for, as Leiker explains, there are a number of aspects of treatment and management which are clarified and simplified by accurate classification.

Let us list those factors relating to management:—

1. Infectiousness. The more resistance there is to the *M. leprae* manifested by the individual, the less likely the bacilli will propagate in numbers adequate to cause an infectious state.

2. Complications. There are certain types of leprosy in which complications are never seen. There are others in which they are greatly to be feared. Specific types of complications are related to specific types of disease, thus complications can be anticipated and usually avoided. Certain reactions occur only in tuberculoid leprosy, and others only in lepromatous. The potentiality can be predicted if we clearly understand the position of a patient on the spectrum of disease.

3. Specific therapy. Due to varying responses to treatment there is need to attack differing types of disease with different schemes of treatment.

4. Duration of treatment is directly related to the severity of the disease, and this is adequately depicted in a proper classification.

5. Prognosis of a patient can be more accurately predicted when there is full understanding of the exact placement in the outline of classification.

SOLUTIONS TO THE EXISTING PROBLEMS IN CLASSIFICATION

In the following suggestions there is little, if any, new subject material, but rather an attempt is made to take accepted facts and co-ordinate them into a unity of understanding in order to form a connected and logical whole.

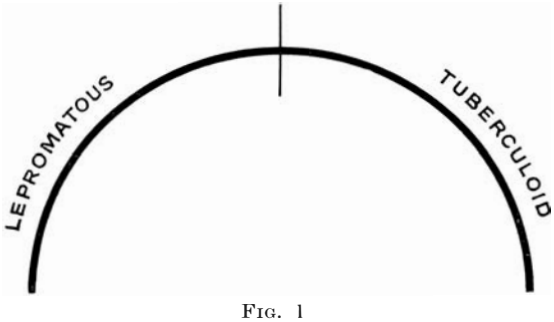

FIG. 1

First, since it can be accepted that there is a spectrum of disease, let us make a diagram to express that as in Fig. 1, with the lepromatous portion to the left and the tuberculoid to the right. It is possible to divide essentially all cases of leprosy into these two categories, but it leaves far too much confusion and would be of relatively little help in clinical management. A still simple classification, but slightly more helpful, includes a third and intermediate group of cases between the lepromatous and the tuberculoid, which it seems would best be called dimorphous (see below), having manifestations of both polar types of disease, Fig. 2.

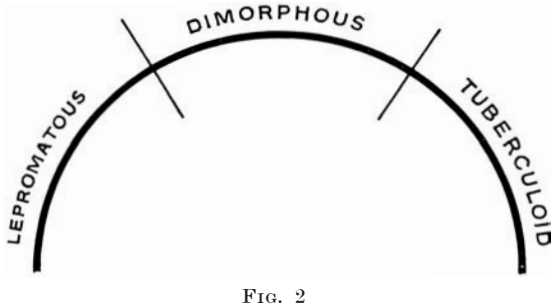

FIG. 2

There are several factors which have at times been implicated as the etiology of the variations in the manifestations of leprosy. The three most commonly discussed are the host, the bacillus and the environment. The last of these is perhaps the easiest to rule out. It is very doubtful if there are variations in leprosy that are due to a geographic differential *per se*. At times it has been suggested that perhaps there are differing strains of *M. leprae*, and this would be a means whereby there could be varying manifestations in different geographic locations. However, there is recent evidence that such is not likely the case (Rees 1965). This work shows that bacilli from different areas of the world all give a similar growth response when inoculated into the footpads of mice. A much more likely reason for such variation in disease pattern lies in the genetic variations of mankind—the host. Thus races and individuals differ in their response to the introduction of *M. leprae* into their bodies. Although we do acknowledge such a difference, yet it should be minimized, since though there are racial differences in the position on the spectrum where the majority of patients may be situated, yet the entire gamut signs and symptoms of leprosy is seen in every race. One generalization can be made here that the severity of the manifestations of disease are in an inverse relationship to the depth of pigmentation of the skin. However, there are always exceptions that do not follow this pattern of the 'average' case, and in a large group of patients of any racial group there are demonstrated all aspects and varieties of the disease.

It is noteworthy that this suggested classification is fully in accord with clinical manifestations of disease, and cases can be categorized on a clinical basis. And it also agrees with the differentiation of patients on a bacteriological, immunological or histological basis. If there is a seeming discrepancy in the findings by any of these diagnostic methods we must look further until every patient can be fitted into the total scheme without disagreement in any aspect. This is all part of a total pattern of disease and must ultimately fit together logically (Ridley 1962).

This classification also has a logical relationship to the evolution of the disease. See Fig. 3. Upon contact the great majority of individuals

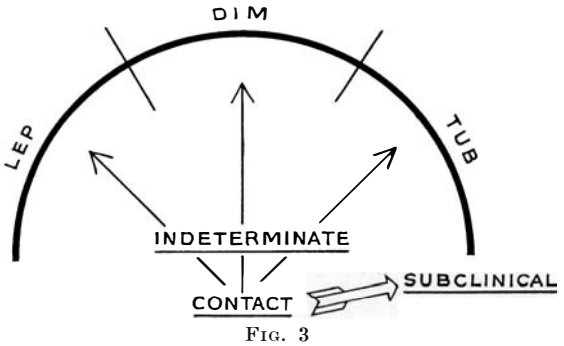

FIG. 3

are either totally resistant to infection or else develop a sub-clinical involvement. What actually happens in instances where an individual does not develop overt infection can at present not be determined. In a relatively small proportion of contacts disease becomes manifest as an indeterminate, insignificant and often overlooked hypo-pigmented and hypo-aesthetic macule. During this stage there is an interaction between the bodily defences and the bacilli which determines the ultimate development of a specific type of disease evolving from the initial indeterminate macule.

The indeterminate macule is *not* a part of the spectrum of leprosy, but is a stage in the development of the disease, as noted in Fig. 3. The presence of disease is not always acknowledged at this stage and quite a few patients are only recognized when the full-blown disease appears. It may well be, as Leiker states (Leiker, 1966), that not all patients go through an indeterminate phase, but this is very difficult to establish with certainty. Again, quite frequently adequate resistance is developed at this stage in the evolution of disease so that it does not progress beyond being indeterminate. This is especially true of patients treated in this early stage. In those not recognized and treated there is an ultimate determination of 'balance of power' between the bacilli and the host, and where resistance is higher moves towards the Tuberculoid side of the spectrum, and with weaker factors of resistance moves toward Lepromatous disease. Indeterminate leprosy as conceived by the South American leprologists (Azulay, 1965) is too large a group of patients including many that should be classed Maculo-anaesthetic, Macular Dimorphous, and Macular Leproma. For purposes of treatment and prognosis it is worthwhile to keep this group restricted to lesions which show vague hypo-pigmentation of a relatively small number of lesions having somewhat indefinite borders, slight hypo-aesthesia or normal sensation, and no bacilli seen by usual means of determination.

The difficulties we have in understanding each other concerning classification will be solved if we have a unified mental picture on which we can base our understanding. For this reason I have attempted to represent diagrammatically the spectrum of leprosy so that we all can more clearly understand just what we are discussing. If all concerned have a common mental image upon which to build further discussions, this skeleton can ultimately be built up into a body of complete understanding. Admittedly there are still areas lacking in clarity, but with these basic concepts perhaps we can work together in filling in the areas of confusion and misunderstanding. I would be the first to recognize that some of this diagram may need alteration to fit the total picture as it is eventually developed.

THE SPECTRUM OF LEPROSY

To visualize adequately the spectrum of leprosy one needs a series of several diagrams which make up a composite and superimposable whole. Fig. 4 depicts the spectrum of the major types of

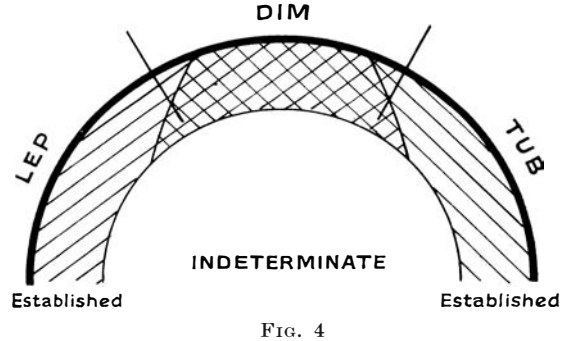

FIG. 4

disease, and the fact that there is a graduation from one type to another. At either end of the spectrum is a group of patients in whom the type of disease is firmly established or fixed, and never alters under any circumstances.

Following common usage as much as possible we will term the large groups of leprosy lesions 'types', e.g., Tuberculoid, Dimorphous and Lepromatous. Since indeterminate lesions are not actually a part of this spectrum of disease, let us use the term more appropriate for a heterogeneous category and call it a 'group' of lesions. For the further division of the three major types let us use the term 'variety'. This follows as closely as possible the terminology used by Cochrane (1964) in his textbook.

At the far right of the spectrum is the established or fixed tuberculoid variety. This is an inoculation type of lesion similar to the 'butcher's tubercle' of tuberculosis (Cochrane, 1964, p. 303). There is an extreme tissue response presumably at the site of inoculation of sufficient number of bacilli to cause a tissue defence reaction. The site of inoculation is infiltrated and surrounded by an intense cellular reaction that walls off and destroys the invading bacilli. This invasion causes the marked infiltration and erythema of this lesion which thus looks much like a *single* reacting Major Tuberculoid lesion. There is so much response to the presence of bacilli that they are very rapidly killed off and removed by the macrophages and thus the lesion disappears rather promptly, whether treated or not.

At the far left hand side of the spectrum is the established leproma. Here there is evidence of a marked lack of bodily resistance. We cannot say an absolute lack of resistance since we as yet have no satisfactory means of making a quantitative determination of this resistance. It is well known that there are in the blood of lepromatous patients large amounts of gamma globulins, but their relationship to the attempt of the body to remove the bacilli is not at present known. The diagnosis of this type of disease is evident only during the clinical course. It is slowly progressive to serious lepromatous involvement, and even with treatment, it shows relatively poor response. However, the lack of resistive powers is evident in that there seldom or never is any evidence of reaction. Thus there is no neuritis nor erythema nodosum leprosum; however, there is very slow but steady improvement under therapy.

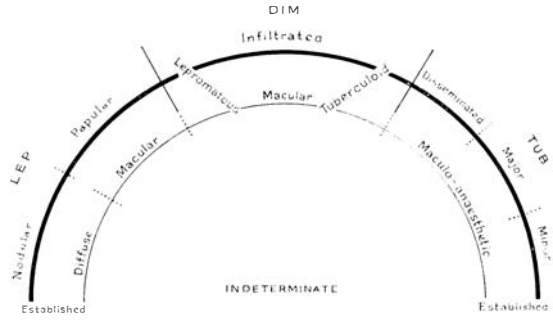

FIG. 5

The most perplexing aspect of this composite diagram is the naming of the various types and varieties of disease. In the accompanying diagrams we have tried to use terms that are most commonly accepted. The lower curving line in the diagram, Fig. 5, represents those lesions which do not incite infiltration as part of the host response, but in some other way alter the characteristics of the skin, such as its colour, the contours of the surface, its appendages (hair, sweat glands) and its tactile sensation. The upper curve represents lesions which produce any infiltration and elevation of the skin of an involved area.

Beginning at the far right side of the diagram are the least severe types of disease. Having already discussed the established tuberculoid, let us consider the other tuberculoid lesions. First there is the Maculoanaesthetic variety, the name describing the character of this lesion. Although this covers a large area in the diagram, it probably does not involve an equivalent number of patients. It is difficult to distinguish from Indeterminate leprosy, and some authorities will call a lesion maculo-anaesthetic whereas others will call it indeterminate, and yet others a tuberculoid macule. Dharmendra (1962), Indian leprologists and others tend to broaden this category to include larger macules with associated neural involvement, which are better considered as part of the Dimorphous type. It would appear preferable to include in maculoanaesthetic only benign patients in which there is never nerve involvement leading to disability. Where there occurs one or a few macules in

which there is unquestioned anaesthesia, no elevation of the surface, and a clear-cut margin they should be placed in this category. As the number of lesions increases there is a shift to the left on the diagram, and an increased potential severity of disease.

In considering the elevated tuberculoid lesions, the first is the Minor Tuberculoid. It occupies the right side of the spectrum just next to the established tuberculoid. It has been clearly defined and accepted universally, and we need not attempt a description. Next to minor tuberculoid should come Major Tuberculoid, although in many patients the differentiation lacks significance for clinical management. Howbeit, it is distinctly a more severe variation in the manifestation of disease. Its characteristics, appearance and location are generally agreed upon, so there is no need to include a description here.

To the left of major tuberculoid is the group of Disseminated Tuberculoid lesions, in which there can be a profusion of lesions which individually appear to be minor or major tuberculoid in appearance, but because of the large number of involved areas, this must be considered in a separate group due to the potentiality for complications, and the need for more prolonged therapy. This is the variety of disease nearest to the Dimorphous, but still in the clearly tuberculoid portion of the spectrum, and therefore the lesions begin to take on the characteristics of dimorphous leprosy. The number of lesions is greater, the edges are less clear-cut, there is less loss of pigmentation, and the tendency for severe neural involvement increases greatly. In this variety the problem of neuritis becomes especially significant although it can be a complication of Major Tuberculoid disease as well, but it is there often limited to single nerves only.

It should be noted that there is no category of reactional tuberculoid in the spectrum, as this is not a true variety of leprosy. When there is an upset of the host-bacillus balance and an increase of defence activity on the part of the host cells there is an increased infiltration and lesions increase in induration. If such lesions were originally on the macular 'line' they simply move centrifugally into that of the infiltrated lesions. 'Transient' is the key word, as the lesions never remain in a category that could be called Reactional, but rather reaction is a temporary phase in the manifestations of disease, and the signs and symptoms will once again revert to those seen prior to the reaction.

Next in degree of severity are the Dimorphous lesions. First of all we should explain the use of this term. The etymology of the word signifies 'two-types' and Dimorphous leprosy encompasses a group of lesions which have some of the characteristics of both polar types of disease. Very frequently the term Borderline is used synonymously, but there is considerable confusion associated with this word since some leprologists use the term for all the cases we categorize as dimorphous, and others use the term only for a restricted portion of the dimorphous spectrum. Since there is this confusion in the usage, and since it is not a suitably descriptive term, I would prefer to omit it altogether.

Dimorphous patients comprise a most significant part of the spectrum of leprosy. If properly conceived this type extends further to the right, and to the left into the tuberculoid and lepromatous portions of the spectrum, and includes a larger proportion of patients than is commonly included in this category. There is good reason thus to broaden this portion of the spectrum in order to include those patients not fully lepromatous, as they respond to treatment more satisfactorily than does true lepromatous leprosy, but yet there is more potential for early and extensive neural damage. This usage is equivalent to Ridley's (1962) term Borderline, which is not used in a restricted sense as it is by the Latin American workers. To include an increased number of patients to the right, e.g., towards tuberculoid leprosy, also brings into the category those patients in whom there is great potential danger of neural destruction. Thus it becomes possible to include in the Dimorphous type the greater majority of patients where nerve involvement is a likely possibility. Thus to lump these cases together in one category

makes for simplification for less skilled workers, and they can understand that caution must be exercised in one category of patients, namely Dimorphous.

As yet we have inadequate terminology for the varieties of patients who are included in dimorphous. It is clumsy to have to say 'Macular Tuberculoid Dimorphous', 'Infiltrated Lepromatous Dimorphous', etc. But the dimorphous type should clearly be split into four distinct varieties, as to whether the lesions are raised or flat, and whether they tend to have predominant tuberculoid or lepromatous characteristics.

The Macular Dimorphous has been recently termed Low Resistant Tuberculoid by Leiker (1964). This terminology, although descriptive, is confusing since it implies that this variety is a part of the tuberculoid spectrum, whereas it is clearly a manifestation of dimorphous disease. Leiker himself suggests this when he states, ' . . . unless a subgroup of borderline (Dimorphous?) leprosy toward the tuberculoid side of the spectrum is created, it is more nearly correct to include these cases in the tuberculoid group'. Such a variety as he mentions is, in fact, that which I propose. He goes on to say that macular dimorphous lesions fit into the low-resistant tuberculoid group. Rather I would say that they are one and the same thing.

Macular dimorphous leprosy is most important because of the high proportion of patients developing severe deformative complications. The skin lesions, although pronounced in most patients for a period of time, are relatively transitory. Dimorphous macules are large hypopigmented patches involving large areas of the skin surface. It probably never occurs as a single macule. The outline of a lesion may be bizarre with an irregular border. The superficial nerve trunks may be markedly involved but usually do not show as great hypertrophy or nodularity as in the infiltrated tuberculoid area of the spectrum. The involvement is manifest, not so much in hypertrophy of the nerves, as by anaesthesia and paralysis. It is from this group that the great majority of mutilated patients with quadrilateral involvement arise. Activity of the disease is relatively transient, but rapid, leaving the patient with severe disability, there being marked neurological deficits in sensory, motor and autonomic nerves. This group is often misdiagnosed as indeterminate. However, indeterminate leprosy *never* causes disability prior to its progression into one of the specific types of disease manifest on the spectrum.

The Dimorphous Infiltrated lesions are those usually called Borderline. There are characteristics of lepromatous disease in that they tend to show more symmetry than any of the lesions thus far discussed. The centre of the lesion is more elevated than the edge, and there is less likelihood that they will be anaesthetic. These lesions readily tend towards the lepromatous pole if there is a decrease of resistance. In these, too, there is always a multiplicity of small lesions. Their small size and large number differentiate them from any of the types heretofore described. The use of the term Borderline for this type of case in a very restricted sense by the South American workers is confusing to those of us who see very many Dimorphous cases. They use the term Borderline only for a very restricted group of lesions somewhere near the very centre of the Dimorphous Infiltrated spectrum (Alonzo 1959, Azulay 1965).

The final category of disease—Lepromatous—is that found to the far left on the spectrum. There are two types of infiltrated lesions, and two that do not show marked infiltration. The non-infiltrated lesions are the Lepromatous Macule, nearer the dimorphous type, and Diffuse Lepromatous leprosy. Individual lepromatous macules may resemble closely the indeterminate macule, but they are distinguished by their multiplicity, symmetry and the presence of bacilli in a smear. There is an area here in which confusion as to the diagnostic category readily occurs. There is similarity in appearance to the dimorphous macule, as well as to the indeterminate macule, which are closely adjacent to the lepromatous macule. These all fade off imperceptibly into each other. Although clinical differentiation may be impossible, a definitive diagnosis should be feasible with evaluation of

other diagnostic means, such as the smear, immunology and histology.

Diffuse Lepromatous leprosy is very difficult to recognize as it causes so little alteration in cutaneous characteristics until at an advanced stage with loss of eyebrows, diffuse infiltration, nasal septal involvement, etc. There may be a slight increase in erythema, and a smooth, glossy surface to the skin, but there are no areas of distinct hypo-pigmentation. It is in this portion of the spectrum that cutaneous endarteritis and arteriolar occlusion occur, producing the Lucio Phenomenon. Although this is a very serious complication in some lighter skinned races, it does occur occasionally as a localized and limited lesion in the darker skinned as well.

It is feasible to divide arbitrarily the infiltrated lepromatous lesions into two varieties, because this division is of some prognostic value. The group nearer dimorphous is named Papular Leproma, with the infiltration manifest chiefly by small lesions, the majority of which are less than 5 mm. in diameter. There may be some larger papules, and some diffuse infiltration, but the most evident lesions are the well-defined papules which almost appear pedunculated, since they are so markedly elevated. The only purpose for distinguishing this variety from the final one is that this type of disease responds to treatment considerably more rapidly than does the Nodular Leproma. There is also a greater tendency for lepromatous reaction, and erythema nodosum leprosum to occur in papular than in nodular lepromatous leprosy.

The last grouping, Nodular Lepromatous Leprosy is manifested by larger infiltrations than those seen in the papular leproma. Here the majority of the lesions are larger than 5 mm. diameter, and there may be large nodules and plaques of infiltration. In this variety there is less likelihood of early complications, but slow unremittant neural involvement is usual.

To the far left of the spectrum is the Established Leproma, already discussed. The only means of differentiating this from the Nodular Leproma is in the clinical course of the disease. (It may be that with time and experience a histological differentiation will be possible.) In the fixed leproma there is a slowly progressive disease with no period of exacerbation or remission. There is a steady increase of areas of peripheral anaesthesia which goes unnoticed by the average patient. Without therapy there is a steady down-hill course and increasing evidence of serious complications. If treatment is given there is a slow and gradual improvement which is not marked by episodic complications. However, treatment will undoubtedly be required for life as there appears to be no attempt to develop bodily defences in this variety of leprosy.

As yet we have not mentioned Neuritic (or polyneuritic) Leprosy. Cochrane (1964) states that potentially there are neuritic patients associated with all divisions of the spectrum, which patients do not show obvious cutaneous changes. However, I am inclined to agree with Leiker (1966) that probably there are no neuritic patients which do not at some stage show cutaneous lesions. If sought for assiduously such lesions or their residua can invariably be found. I believe that patients most commonly called neuritic actually arose initially as dimorphous macular lesions in which the evidence of cutaneous disease has disappeared but there is still activity or the results of activity such as scarring and fibrosis in the large superficial nerves. Thus in the categorization of neuritic disease one should attempt to determine the original type of skin involvement, and place the patient in the pattern of disease most appropriate.

In order to demonstrate how extremely important is a correct classification of leprosy in order to draw any significant conclusions about this multi-faceted disease, let us again refer to Dr. Mallac's 'Onset and Pattern of Deformity in Leprosy' (1966). According to Dr. Mallac, indeterminate leprosy has the least potentiality for deformity, yet in actuality, indeterminate, if correctly defined, is a group of patients in whom deformity *never* occurs unless it is transformed into one of the definitive

types of leprosy. Dr. Mallac uses the conventional definition for indeterminate leprosy which is little more than a 'waste basket' for macular lesions.

He also records that deformity on the average occurs in $2\frac{3}{4}$ years in tuberculoid leprosy. It would be preferable to divide the tuberculoid into its varieties and note specifically the ones where deformity is a common occurrence. It never occurs in maculo-anaesthetic leprosy, properly defined, and it never occurs in Minor Tuberculoid patients. In major tuberculoid it may occur as related to an extremity on which lesions are found. Thus it may occur on one limb alone, and seldom involves all 4. Dr. Mallac's usage of Borderline is like that of the South Americans. Thus it coincides with only a very limited part of the Dimorphous spectrum. This definition includes only relatively few patients, whereas dimorphous properly conceived includes a large grouping, and also one in which there is a high incidence of deformity. This should include the dimorphous macular lesions as well as those which are infiltrated.

After reviewing the literature Dr. Mallac concludes that 'no single test emerges as a reliable means of predicting the potential for deformity', and on this we fully concur. However, he does not carefully relate incidence of deformity to the spectrum of disease accurately conceived, which process aids immeasurably in determining a prognosis concerning disability. This work is a very real contribution to the literature on rehabilitation in leprosy, and the stress he lays on early diagnosis and treatment is quite correct. Just one thing more should be added to his work that is of extreme significance, and that is that with correct classification of a patient, leading then to therapy related to the type of disease, *at an early stage*, deformity is preventable in all except a small minority of dimorphous patients.

It is of utmost importance to remember that there are no absolute boundaries dividing the various types and varieties of leprosy, there is simply a gradual progression from one category into the next.

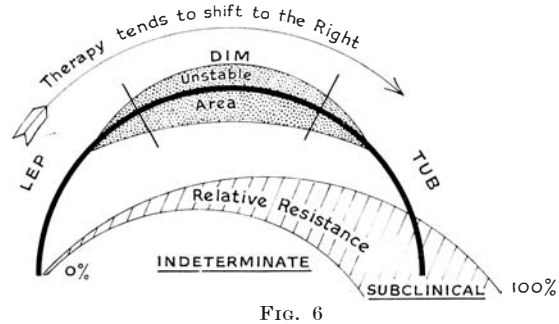

FIG. 6

There are three factors related to this composite diagram of Classification that can be readily superimposed on to the picture to add to its significance. These all relate to matters already referred to, and need little comment. The factors are: 1. host resistance, 2. disease type stability, and 3. response to therapy, and these may be added to the diagram as in Fig. 6.

The resistance of the individual to disease is high on the right side of the spectrum, so high in fact that in the great majority of exposed individuals no obvious disease becomes manifest. Thus we have the 'Sub-clinical' category. Host resistance gradually lowers as we proceed toward the left, and thus disease becomes more and more severe.

In consideration of type stability we note that the central area of the spectrum is the least stable, and a patient can shift in his manifestations quite readily. On the other hand the

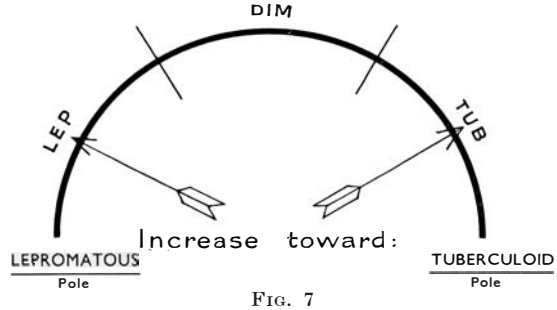

FIG. 7

1. Number of lesions.
2. Symmetry.
3. Bacilli.
4. Smoothness and lustre.

1. Definition of edge of lesion.
2. Hypopigmentation.
3. Size of individual lesion.
4. Anaesthesia.
5. Lepromin positivity.
6. Response to therapy.

disease at the very poles of the spectrum is absolutely stable. The closer to the centre of the spectrum, the more likely a loss of 'balance of power' between disease and host. The disease may shift to the left with any stress such as intercurrent illness, undue physical activity or even psychic trauma. Conversely, an increase of resistance in an individual tends more to the type of disease to the right.

Finally, concerning therapy we can state that adequate and effective treatment tends to shift the type of disease to the right hand side of the spectrum.

A further diagram which may aid in determining the site of a patient in the spectrum of disease is noted in Fig. 7. Note that the characteristics of leprosy lesions which increase as disease moves towards the left or becomes more nearly lepromatous, are the number of lesions, the symmetry of lesions, the number of bacilli present, and the smoothness and shininess of lesions. The farther to the right we go in the classification the more marked are the following characteristics: definition of the edge of the lesion, hypo-pigmentation, size of individual lesions, anaesthesia, lepromin positivity, and the response to therapy. Alterations which appear in the histology as a patient appears to the right or left in the diagram could also be discussed here, but we have not included it in our present considerations.

SUMMARY

The similarity of opinion held by a majority of present-day workers on the matter of classification of leprosy is noted, and an attempt is made to bring together various divergent viewpoints into a united whole by a co-ordination of the thinking of several authorities.

Emphasis is laid upon the logical basis for a spectral concept of leprosy related directly to the establishment of a balance between host resistance and bacillary multiplication.

The logical classification is diagrammatically presented in order that workers may develop a uniform concept of the spectrum of leprosy on which further information can be developed as it becomes clear.

The types and varieties of leprosy making up the total spectrum of disease are briefly described, and their relationship to one and another are established in the spectrum.

It is pointed out that without an adequate understanding of a proper classification of leprosy, many aspects of this disease, most importantly as related to management and therapy, remain unclear. Thus a plea is made for still further development and elucidation of the subject of classification.

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