DEVELOPMENT OF MAJOR TUBERCULOID LEPROSY

A REPORT OF CASES[1]

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In the great majority of leprosy cases, whether resident in an institution or attending a clinic, the affection progresses in uneventful and monotonously familiar ways. Occasionally, however, one is encountered that presents features which merit special notice, especially with reference to the phenomena of change of variety or of type of the disease. That we believe to be true of a few of the group of neural-type cases which we have observed jointly since 1933 and 1934 (6, 3). Since a fuller understanding of such phenomena must necessarily depend upon detailed study of the cases concerned over long periods of time, and since the modern literature of leprosy is decidedly poor, to say the least, in observations of the kind, these cases will be reported in some detail.

In the two cases presented here the major tuberculoid form of the disease—the form ordinarily called "tuberculoid leprosy"—developed from lesser forms or stages. In both of them it ultimately receded, as it usually does. One is now apparently recovered and has been paroled. The other, after a rather long period of apparently complete recovery, suddenly developed extensive lesions of extraordinary appearance and nature, the case becoming apparently borderline between tuberculoid and lepromatous. In both instances the major tuberculoid lesions, bacteriologically positive and so necessitating hospitalization of the patients, developed in the course of lepra reaction of the sort to which Wade first called attention (5) and which Schujman (4) has called "tuberculoid reaction" but which, rather oddly, Muir (1) has recently chosen to rename "recovery (or

[1] This article is the second of a series that deal with follow-up observations of neural-type cases of leprosy at Cebu, in the Philippines.

convalescent) reaction." The fact that a reaction-like onset is very common in the evolution of at least the more severe cases of this variety of the disease is well known, but there is little definite information concerning the conditions that existed in cases before that development took place.

Both of these patients were first seen more than five years ago. Since that time actual recorded observations have been made at rather infrequent intervals, but they suffice to define the main events. Regarding the underlying processes that were responsible for the changes that occurred we can contribute no information; no special tests or examinations of any kind, immunological or biochemical, were made. When it is considered how small is the chance that any particular case will undergo unusual developments it is apparent that, in order to have such information if one of them should become interesting, provision would have to be made for such examinations of large numbers of routine patients over long periods of time. The conditions prevailing at Cebu have never permitted doing work on such a basis. Our purpose in presenting this report is partly to record observations of the sort that, we believe, must be accumulated if we are to advance beyond broad generalizations in this matter, and to indicate problems relative to the cause and nature of the changes that occurred.

CASE I. CONVERSION OF A SIMPLE MACULAR CASE TO ACUTE MAJOR TUBERCULOID, WITH APPARENT RECOVERY

The history of this case—thus far—has three phases: (*a*) one in which there were present only ordinary simple, indolent macules; (*b*) one of reaction, in which the macules were converted into conspicuous, bacteriologically positive, major tuberculoid plaques, with later a generalized eruption of coarse papular lesions; and (*c*) one of recession and recovery with only residual effects remaining, in which the patient was paroled.[2]

FIRST PHASE.—The patient, P. E., male Filipino, 33 years of age, first appeared at the Cebu Skin Dispensary in March, 1933 (record by Dr. F. C. Plantilla), with ordinary flat, simple macules. Two young sons had similar leprids. On the right upper arm were two lesions, one 6.5×7.5 cm. and the other about 1.0 cm. in diameter; they were somewhat hypopigmented centrally with irregular, slightly pinkish borders, in which were fine papulations, and, outside, several larger, palish, slightly pinkish papules.

[2] The earlier developments in this case were reported by one of us (J.N.R.) in a note on the occurrence of open lesions in previously negative cases (2).

On the left knee and extending downward for about 15 cm. was a large, faint macule, the border ill-defined and patchy except on the medial surface, where it was pinkish. Sensory disturbances present. Bacteriological smears (three) negative.

The disease, the patient stated, had commenced about four years before (1929, age 29) with a small (2 cm.) pale macule below the left knee. Some time previously numbness of both legs had been noticed. The lesion enlarged, the center becoming more or less repigmented. Some two years later the macule just above the right elbow developed, the smaller one above it afterward.

Examination: When seen jointly, in August, 1934, the lesions seemed inactive but they had enlarged, those on the arm having fused to cover the lower half of the posterior surface (Plate 12, fig. 1). The one on the leg covered the upper third anteriorly, extending well onto the sides. These two areas were much alike, with very irregular ("streaming") edges in advance of which there were separate small areas or spots ("colonial development") without papulation. They were classified as "simple, quiescent."[3] Smears (2) negative. Regarding treatment, the patient had taken only 16 injections in the 17 months since the first examination.

Biopsy: Anterior edge of the arm lesion. Histology: moderate round-cell infiltration present. In the first sections examined a single tuberculoid focus was found in a subcutaneous nerve, but in serial sections more such foci, all small, were seen at various levels. No bacilli found.

To this point the skin lesions were of the nonelevated, clinically "simple" variety, except for the papulation recorded in 1933. They were of very mild grade, both clinically and histologically, but they were active and enlarged slowly. The treatment taken could not have affected their course materially.

SECOND PHASE.—The patient did not appear again at the clinic until sent for 19 months later, in March, 1936, when it was found that the lesions had undergone a remarkable change. He stated that they had improved for a time and became less evident but reappeared about a year before as red, infiltrated patches which enlarged. The lesion on the arm was considerably larger than before, and the one on the leg now involved the lower part of the thigh. Both were thick, reddish plaques, for the most part sharply delimited, irregular-surfaced and anesthetic. There were also deep-seated firm nodulations about the elbow and knee, and nodular swellings of both earlobes and helices, most marked on the right side. The earlobes were turgid and the face flushed. Ulnar nerves palpable, great auriculars slightly so, peroneals not; apparently no actual thickening. About five weeks later the nodulations of the ears had become smaller, the left ear being practically normal; otherwise the condition was unchanged. Smears repeatedly positive, usually 4+, with numerous small globi; one from the right nasal septum 2+. Patient admitted to the Eversley Childs Treatment Station as an "open" case, recorded as C1-N1 on the assumption that the lesions were now lepromatous.

[3] Lesions with the characteristics noted we would not now class as entirely inactive.

Reexamination, September, 1936: At this time, during a follow-up examination of the entire study group, it was recognized that the change that had occurred was really conversion from "simple macular" to the major tuberculoid condition, obviously by a kind of reaction phenomenon.

The plaques had changed since March from red to dark brown, with a violaceous tinge, and had increased in thickness, though parts of the leg lesion were thin in comparison with that on the arm (Plate 12, figs. 2 to 4). The surfaces of the plaques were still irregular, but on palpation the tissue felt velvety and even. In the lower central portion of the arm lesion were coarse folds of peculiar appearance, probably due to the effect of flexation on the thickened, inelastic tissue but suggestive of the possibility of lepromatous change. Complete anesthesia to superficial pain, light touch and temperature, though deep pain felt in some parts and sense of deep touch present everywhere.

An interesting new feature consisted of several hundreds of discrete, superficially located papules, ranging in size from a pin point to a split pea. They were very numerous on the trunk and the extremities, particularly the upper ones (Plate 13, figs. 5 and 6), and a few were on the face. In general their appearance was as if they had been produced by sharply localized foci of proliferation in the superficial portion of the dermis. Some of them, especially the largest, were rounded, pinkish and succulent in appearance, but others were strikingly pale and flat or nearly so, evidently in retrogression. According to the patient these lesions first began to appear about two months previously and came out in successive crops. At the outset he felt "heavy and hot" for a few days, but he believed that there had been no fever.

Nerve trunks negative. No enlargement of superficial cutaneous nerves except that the left infrapatellar branch of the saphenous, under the original lesion, was barely perceptibly thickened.

Of six smears from various places, one from the suspicious-looking area above the right elbow was 2+, with some globi; the others were negative. This was in striking contrast with the previous findings, but some six weeks later all of four smears from different sites were positive (2+).

Five *biopsies:* edge of arm lesion and suspicious-looking central area, and papular lesions in different stages from both forearms and the left scapular region. Smears from both biopsy sites in the old arm lesion were positive (1+ only), the other three negative. Histopathology: All specimens are tuberculoid, though in the massive arm lesion there are pseudo-lepromatous effects produced by local injection of chaulmoogra ethyl esters. The tuberculoid condition is of marked major degree, of the atypical form that is commonly seen in such lesions of acute reaction origin and which may perhaps be called "intermediate" with respect to its approach in certain morphological features to the nonfoamy type of leproma, without implication of actual conversion to that condition. On the whole they differ rather widely from the ordinary chronic tuberculoid leprids, in which the granulomatous foci are more or less distinct if not actually separated from each other and almost universally contain characteristic focal masses of epithelioid cells. In the present material such foci are found only occasionally. For the most part the granuloma is more diffuse and less differ-

entiated, the component cells not distinctly of the epithelioid form or if so not aggregated in characteristic foci; giant cells are not numerous or conspicuous or typical. Bacilli in small numbers were found in four of the specimens; in the one that had most, five or six isolated intracellular ones were found in one section and one group of three or four in the wall of a capillary. In both specimens from the large arm lesion acid-fast drug particles were present, varying from large globules to fine granules.

In this stage the old, indolent simple macules were converted by a reactional process to conspicuous raised plaque-like lesions which enlarged with relative rapidity and were found to be bacteriologically positive. Between the time of admission to the leprosarium and the last examination there occurred a secondary eruption of papules of the same nature histologically as the main leprids, obviously arising through metastatic distribution of the causative agent by the blood stream.

THIRD PHASE.—Improvement began and progressed rapidly. Only six weeks later (October, 1936) both of the plaques had subsided greatly, the edges being at most only slightly raised. However, smears were still positive (2+). Many of the papular lesions had become only ill-defined small pale areas; some had pin-point micropapulations in their peripheries and the largest of them, it was noted, looking like "typical lichenoid patches."

Reexamination, June, 1937: The principal lesions now seemed almost residual. That of the leg was hyperpigmented, flat, not infiltrated. That of the arm was in parts elevated (Plate 13, fig. 7), but on palpation the tissue was soft, with no suggestion of actual infiltration. On the body and extremities the larger of the previously existing papules were now represented by flat pale spots; no trace remained of the smallest ones, or of those that had been on the face (Plate 13, fig. 8). Multiple smears were all negative, as were those made a month previously.

Two *biopsies:* edge of arm lesion and its lax central part. Histology: Nothing is to be found in either specimen except pseudolepromatous and other effects of local injection, and rather marked loss of elastic fibers in the second specimen. The previous tuberculoid condition, marked as it was, seems to have disappeared completely.

Last examination, September, 1938: The patient was virtually negative. The lesion-area on the arm still bulged and had the same soft, lax feeling as before, obviously due to destruction of the elastica of the reticular layer. The area on the leg, to the contrary, was rather scarred and not elevated. The arm area was anesthetic to pain and light touch, the leg one not definitely so. Numerous palish spots were the only evidence of the previous papular lesions. Ears quite normal. Ulnar, peroneal and great auricular nerves slightly thickened.

In this 15-month interval the patient had continued to take treatment quite regularly, though in September, 1936, he was excused for a while because of a "reaction," of which no clinical note was made. Smears had been made 14 times and on 6 occasions had been found positive, usually slightly so and never in more than one or two of the several smears that

were made each time. The last four examinations were all negative. No further biopsy seemed indicated.

The major tuberculoid condition as a whole was evidently at its height when the patient was examined in September, 1936, for retrogression occurred rapidly thereafter, and in June, 1937, the lesions showed no evidence of activity and biopsy specimens showed little more than the effect of local drug injections. The appearance was much the same at the last examination (1938), but in the 15-month interim small numbers of bacilli had been found several times. Some thickening of nerve trunks apparently occurred during this period. Since our last examination the patient has been paroled (May, 1939).

CASE II. EVOLUTION OF A TUBERCULOID CASE WITH APPARENT RECOVERY BUT SUBSEQUENT RECRUDESCENCE

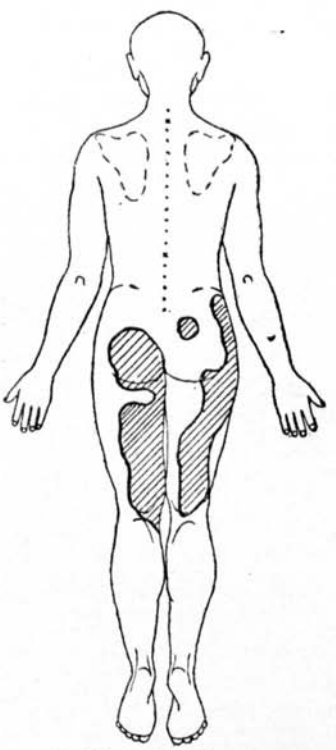
The history of this patient, to date, is divisible into four phases: (*a*) that of onset to the time he was first seen, when there were extensive marginate minor tuberculoid macules, found bacteriologically positive; (*b*) that of exacerbation during a febrile reaction period, with a flare-up in the old lesions and appearance of new ones of major tuberculoid grade, after which the patient was hospitalized; (*c*) that of apparent recovery, in which the patient was paroled; and (*d*) that in which there suddenly developed, also as a reaction phenomenon, widespread lesions of atypical appearance and apparently lepromatous structure. This last phase will be dealt with only briefly here.

FIRST PHASE.—This patient, T. C., a male Filipino, then 20 years old, was encountered in September, 1933, during a survey of the population of Cordova, Cebu. About 18 months previously a small (1 cm.) raised reddish area had appeared on the left buttock. It enlarged rapidly, and when it was about 3 cm. in diameter the center became pale and receded, leaving a typical annular minor tuberculoid lesion. A similar one soon appeared on the right buttock.

These lesions spread so rapidly that at the time of examination they covered extensive areas of both thighs to the popliteal spaces, the right one extending upward onto the hip (Text-fig. 1). The borders of these areas were in places infiltrated and pinkish, and centrally they were entirely anesthetic to touch, pain and temperature. Smears from the elevated margins in two places were positive, bacilli numerous (3+); the nasal septum was negative. The case was classified at the time as moderately advanced cutaneous type leprosy.

Biopsy: margin of the buttock lesion. Histopathology: tuberculoid, active and rather marked. Numerous tuberculoid foci, more or less conglomerate, mostly in the papillary layer. Smaller foci in the reticular layer, but none in its lower half. Bacilli found, in places numerous.

This case was undoubtedly of the minor tuberculoid variety from the outset, and the condition progressed with extraordinary rapidity. In connection with that fact, the positive bacteriological findings at the time of examination may be significant. No history has been obtained suggestive of a reaction condition to which the findings could be attributed.

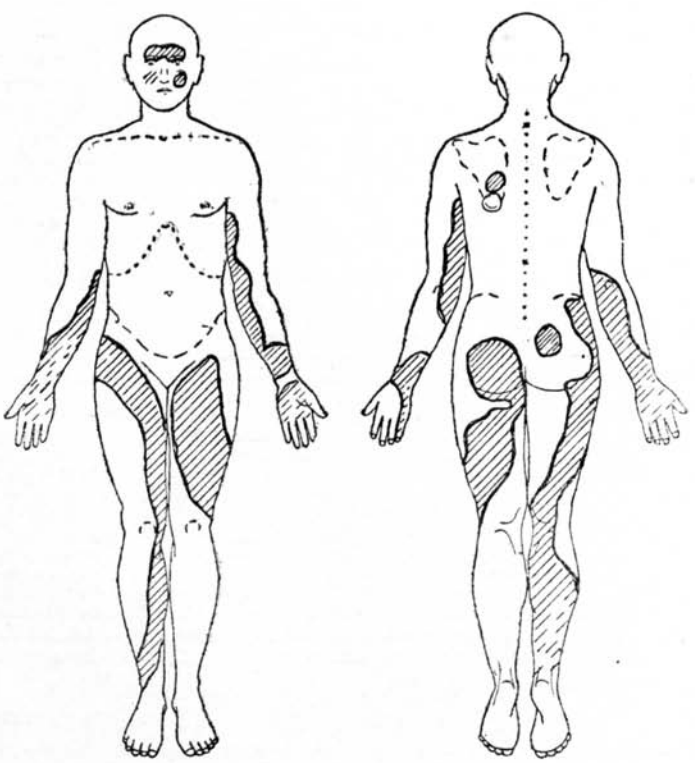


TEXT-FIG. 1. Diagram of Case 2, showing roughly the location and extent of the marginate minor tuberculoid lesions—including the recovered areas—at the time of first examination in September, 1933, supposedly about 18 months after onset. (As drawn by Dr. R. S. Guinto at Cordova; but see Text-fig. 2 with reference to distribution on left thigh.)

SECOND PHASE.—Six months later (March, 1934) the patient presented himself at the dispensary in Cebu in bad condition. In December, shortly after attending a town "fiesta," he had had an acute reaction with fever and was bedridden for about two months, losing 16 lbs. in weight. The attack started with fever for ten days, accompanied and followed by the appearance of new erythematous and elevated lesions on the face, earlobe, arms and the thighs anteriorly. Contracture of the fingers began during this period.

When examined the greater part of the macules seen previously—then largely inactive to residual—were involved in the flare-up; and they, to-

gether with the many new ones, constituted extensive infiltrated patches and gyrate areas and bands on the forearms and thighs, the last forming irregular bands extending down on the right leg (Text-fig. 2). The areas on the arms were especially marked, raised and red. Penis affected. A patch on the forehead extended to both upper lids. Left earlobe contained a nodule, left cheek red. Nasal septum ulcerations, both sides. Bilateral orbicularis oculi paralysis. Ulnar nerves much thickened, especially the right; peroneals slightly enlarged. Atrophy of both hands, especially right, with quite marked contracture of fingers and some absorption of right index and middle fingers; anesthesia of right hand to wrist, right foot to ankle, left foot and leg to knee; perforating plantar ulcer, left. Smear from left earlobe positive (2+). It being assumed that the lesions were now lepromatous, the patient was admitted to the leprosarium classified as C2-N2, later to be transferred to the Culion Leper Colony.



TEXT-FIG. 2. Diagram showing roughly the location and extent of the lesions, now of major tuberculoid grade, when Case 2 was hospitalized in March, 1934. (As sketched by Dr. J. G. Tolentino, at the Eversley Childs Treatment Station.)

Though no histological examination was made at the time it is beyond doubt that the development that occurred during the febrile, reaction period was a conversion of the previously

minor tuberculoid condition to the major form, with marked exacerbation of the disease as a whole. The rapidity of development of polyneuritic manifestations is decidedly unusual.

THIRD PHASE.—Retrogression of the reaction condition was rapid and at Culion four months later (July, 1934) there were, besides the trophic changes, only pale thickened macules on the arms and forearms, similar but uninfiltrated areas on the right scapular region, buttocks and lower extremities, and slight infiltrations of earlobes and cheeks. After another three months (ten months after the reaction occurred), with the clinical condition still more retrogressive, the patient was once found bacteriologically negative. In a reexamination, however, positive smears (1+ and 2+) were obtained from two of the numerous sites examined, and sixteen months later the same places were again positive (1+) though ten others were negative. In the meantime the macules had in general continued to clear up, so that when examined in connection with this study in September, 1936, little was to be seen but residual changes aside from the sequelae of nerve involvement, and some of the areas shown in the 1934 diagram were quite indistinguishable. The patient was paroled in March, 1937, having repeatedly been found negative over a period of about nine months.

Clinical subsidence of the lesions was rapid, as could only happen with tuberculoid leprids, and ultimately many of them quite disappeared. Again as in such leprids, bacilli quickly became difficult to find, though a few were encountered from time to time long after all clinical evidence of activity had disappeared, and parole was delayed thereby.

Eighteen months after parole the patient was seen again in our follow-up examination of the study group. In the interim, he stated, there had been no manifestation of the disease until three weeks previously. At that time a large boil had developed over the left knee, and very promptly extensive new lesions had developed. They were most extraordinary, with some features of the lepromatous condition but others that were highly atypical of it—apparently borderline. It seems best to postpone discussion of it until further observations can be made.

SUMMARY AND DISCUSSION

In the first of the two cases here presented there were, as late as the fifth year of the disease, only two simple flat macules which at most had been very indolently progressive and which had shown only a very slight tuberculoid condition histologically. Suddenly they flared up in an obvious "reaction" process to become active major tuberculoid plaques, smears showing numerous bacilli, often in small globi. A few months after the patient was hospitalized an eruption of multitudinous small,

papular lesions began to appear in crops, evidently as a further reaction condition, the causative agent obviously distributed metastatically by the blood stream; they never attempted to expand to produce macules, but subsided. Histologically both the plaques and the papules consisted of a massive tuberculoid granuloma of "intermediate" or reaction type and the papules were of the same nature. From that point, in 1936, retrogression of the lesions was rapid, so that in 1937 they all seemed entirely quiescent if not residual, but until 1938 an occasional smear contained a few bacilli. The patient has now been paroled.

Except that it was studied, both clinically and histologically, before as well as after the tuberculoid development there is probably nothing unique about this case. Of interest, however, is the striking papular eruption, which is not a recognized feature of tuberculoid leprosy though as will be shown later (7) it is not uncommon in the Philippines. There is also something of interest in the way in which the major tuberculoid condition developed, and in the contrast between the persistent indolence of the original macules, which had not been overcome in five years, and the rapid recession of the tuberculoid lesions once that active condition had reached its maximum.

The second case is more complicated. It differed from the first one at the outset in that there seems to have been no simple macular phase, the disease having evidently been of the minor tuberculoid variety from the outset—unusual in the extraordinary rapidity of its progression, the positive bacteriological findings at the time of the first examination, and the early appearance of polyneuritic changes. The subsequent course (the flare-up of the lesions to the major tuberculoid state following a reaction, the recession of that condition, and in time the parole of the patient) essentially parallels that of the first case. The last phase to date, the sudden development during another severe reaction, after many months of apparent recovery, of an extensive eruption that was clinically atypical and histologically not distinguishable from the atypical lepromatous granuloma—apparently a borderline condition—is unusual.

The events in connection with the original major tuberculoid development in these cases are of interest not only with regard to the question of the relationships between the different varieties of leprids but also that of causation of change from one to another. The circumstances were not alike in the two.

The first patient reported no coincident illness. The second was ill for weeks with a reaction condition, but just what the relations are as regards causation and effect between it and the previous and subsequent states of the disease cannot be said. In the first case the new development was confined to the regions of the old macules, except for the papular eruption that occurred some months later; in the other case the old lesions were involved but new ones also appeared. There being apparent differences in the processes by which the maximum stage was attained, there were presumably differences in the underlying conditions, immunological or other, that were responsible for those processes; to suggest that the differences are only quantitative and not essentially qualitative is pure speculation. At any rate the similarities as regards the tuberculoid condition at its height, the rapid clinical and bacteriological improvement of the lesions after hospitalization, and also the persistence of a few demonstrable bacilli long after the lesions had subsided to apparent quiescence, all bespeak a similarity if not identity of the processes in those stages.

Another feature that the cases had in common is noteworthy, namely, the manner in which the areas that had been involved by the less marked lesions long before, were affected by the reaction process by which they became major tuberculoid. One of the outstanding characteristics of the ordinary (i.e., nonreactional) progression of the leprids is that where the active advancing margins of two neighboring ones meet, the activity dies out. There is no more attempt for the margin of one lesion to continue on into the area of another than for a patch of fire in grass to continue into another patch already burned over. The same thing is seen in the area inside of the active margin of a leprid; normally there is no tendency to centripetal reinvasion of that region once it is healed. Apparently the affection as it heals leaves the tissue with a local immunity of some sort —which condition is presumably responsible for the healing. Certainly it is difficult to hypothesize an exhaustion of any element of the tissue required to maintain the infection.

Yet when the major tuberculoid reaction occurred in these cases there was no such respecting of tissue previously involved by the lesser-degree processes, either simple macular or minor tuberculoid. On the contrary, in the first case the entire areas (and only those areas) of both old lesions were affected, though they were several years old and most of their central portions had

presumably been entirely residual for a long time; and in the other case extensive parts, though not all, of the older lesions were similarly affected. It seems almost as if, with respect to the major tuberculoid condition, the old areas had been sensitized rather than immunized. Probably related to this phenomenon is the rare occurrence of reinvasion of a macule to produce a peculiar, double-margined lesion, as is shown in the illustrations of Case 3 of the report of one of us on cases studied in South Africa (5). That case, too, was major tuberculoid in a state of "severe," but very protracted, reaction.

If there is evidence that ascending grades of the leprids possess ascending degrees of pathogenicity, of ability to reinvade tissue once affected and recovered, there is correlative evidence that in the more marked varieties of this form of the disease a particularly intense and effective antagonism to the infection usually develops in due course, resulting in the rapid recession of the lesions that is so frequently seen. Treatment cannot be held solely responsible for the recession in our patients, for it is well known that in many such cases similar improvement takes place without treatment. At most we can only believe that medication may hasten improvement, and perhaps help to make it permanent. It would seem as if the flare-up resulted ultimately in the activation of the defense factors in a way or to a degree that the original condition could not do.

Yet the effectiveness of this antagonism is only relative, and it exhibits certain peculiarities. In the most conspicuous granulomatous masses that the immediate reactability of the tissue gives rise to, the forces of resistance frequently fail to prevent the development of bacilli in sufficient numbers to be easily demonstrable in smears; and they may actually become as abundant as in lepromata and may even form small globi. But even in that condition, with perhaps large numbers of metastatic lesions appearing, the anergy of the lepromatous form of the disease does not follow and the lesions do not become lepromatous in behavior, however much they may resemble that condition histologically. Sooner or later they subside, and they may do so rapidly; but even when they may seem all but residual, small numbers of bacilli may persist for long periods. In that stage an exaggerated tissue response no longer occurs.

Even after apparent complete recovery, clinical and bacteriological, has taken place the ultimate outcome may be uncertain.

In the general run of tuberculoid cases the prognosis is apparently good, but that is not necessarily true of the more severe forms of the condition. We refer here to the matter of relapse within the range of the neural type, not to the question of transformation to the lepromatous form of the disease which comes up in connection with the most recent changes in the second case. The relapse that occurred in that case after a total of more than two years of clinical and bacteriological negativity—a phenomenon that also occurred in certain of the cases to be reported later—emphasizes the fact that we have as yet no means of knowing whether or not a case has been actually cleared of the infection. It gives emphasis to the absolute necessity of following individual cases for many years if we are to appreciate the vicissitudes of this form of the disease.

Acknowledgment is made of indebtedness to Dr. José G. Tolentino resident physician of the Eversley Childs Treatment Station, Cebu, to Dr. Fidel C. Plantilla, in charge of the Cebu Skin Dispensary, to Dr. Ricardo S. Guinto, junior epidemiologist of the Leonard Wood Memorial at Cebu, and to members of the staff of the Culion Leper Colony, for the use of records made by them and for valuable cooperation in other respects.

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DESCRIPTION OF PLATES

PLATE 12

FIG. 1. Macule on arm, Case 1, as seen at time of biopsy in 1934. Composed of two separate ones that had enlarged and fused since the patient had first appeared at the clinic 17 months before. The irregular "streaming" outline and the colonial development outside are incompatible with complete quiescence. Histology: very slight tuberculoid—slight perivascular round-cell infiltration, with scattered small epithelioid foci.

FIG. 2. The same lesion when seen in September, 1936, after it had undergone conversion to the major tuberculoid condition, then at its height. The portion suspected, erroneously, of having undergone leprqmatous change is central toward the lower end. Some of the innumerable papular lesions that had appeared recently are seen, especially on the back; most of them are rounded, not retrogressed (compare with Figs. 5 and 6).

FIG. 3. Showing especially the abrupt edge of the plaque on the arm, typical of the condition.

FIG. 4. The inner aspect of the less exuberant lesion of the left knee and leg, which by this time had extended well up onto the thigh.

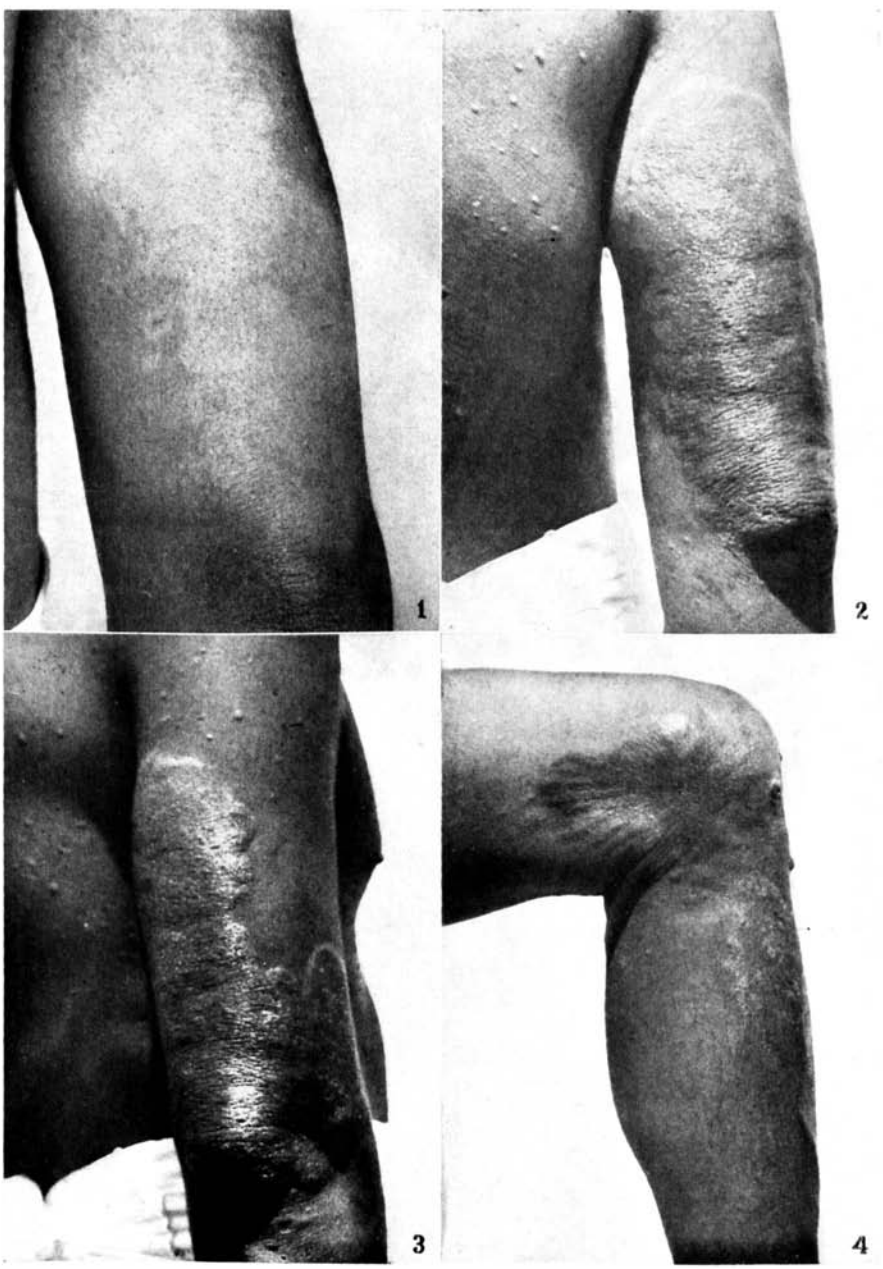

PLATE 12

PLATE 13

FIG. 5. Anterior aspect of the body and the left arm, showing the papular eruption. Papules in various stages, many much retrogressed and flattened, especially on the upper chest.

FIG. 6. The papular eruption on the back. The several lesions of this kind that were examined were histologically of the same "reaction tuberculoid" nature as the large plaques, though less marked, but they were bacteriologically negative.

FIG. 7. The plaque on the arm as seen 9 months later (June, 1937). The continued elevation of this area was not due to persistence of the tuberculoid condition, for sections showed that that had cleared up. It is a soft flaccid bulging due to permanent damage, chiefly wholesale destruction of the elastic fibers throughout the dermis.

FIG. 8. The anterior surface of the body and left arm in 1937, showing (as also in Fig. 7) the complete recession and, usually, disappearance of the papules, with only occasional palish spots resulting from the largest of them.

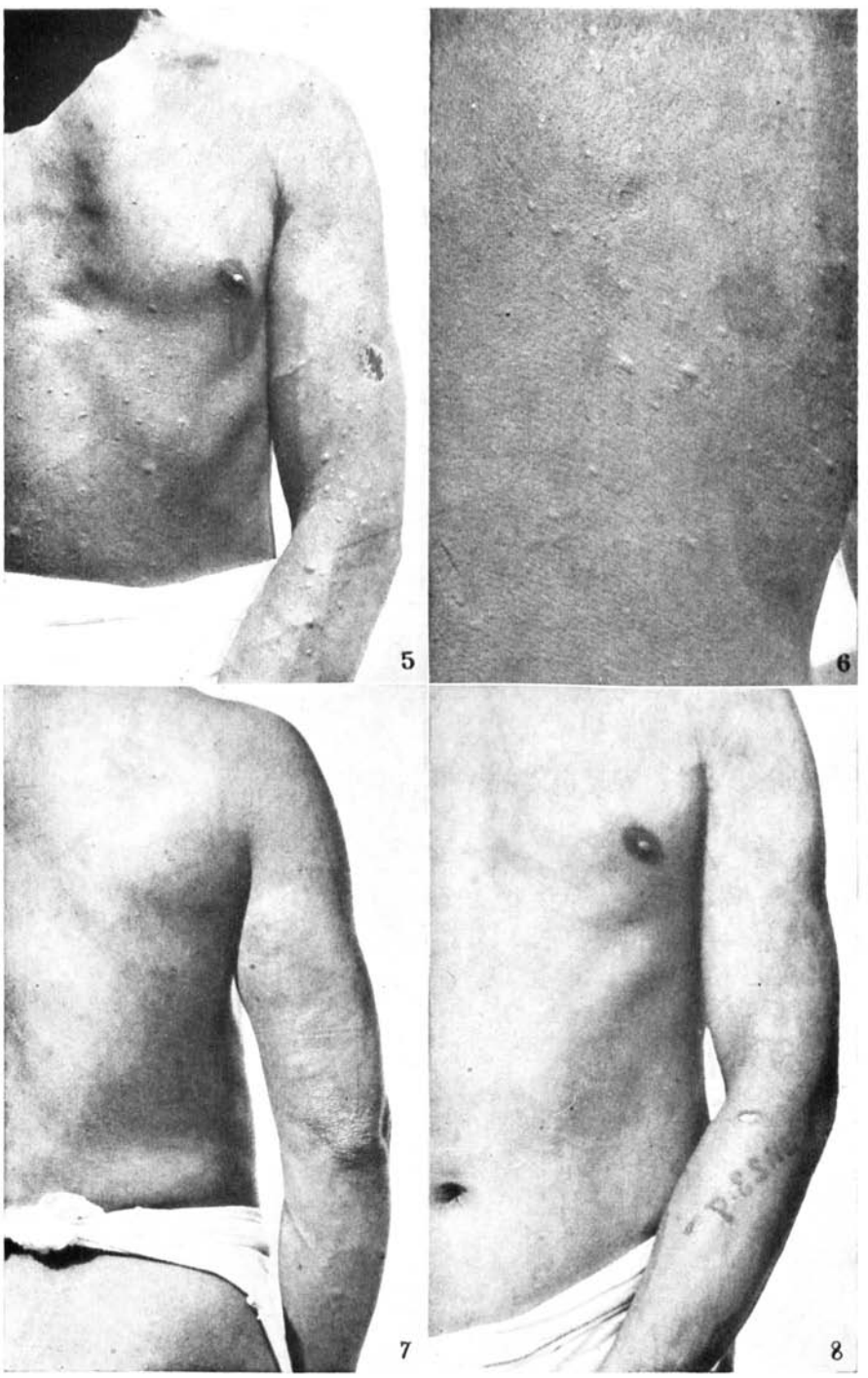

PLATE 13