CORRESPONDENCE

This department is provided for the publication of informal communications which are of interest because they are informative or stimulating, and for the discussion of controversial matters.

SPONTANEOUS DISAPPEARANCE OF SKIN LESIONS; POSITIVE SMEARS WITHOUT LESIONS

In this department of the last issue there appeared a letter from Dr. Felix Sagher, of Jerusalem, posing two questions: (1) What is the importance of impermanent hypopigmented patches in bacteriologically negative contact children, and if they are leprous does their disappearance indicate permanent cure? (2) Should contacts without visible lesions but with positive smears be regarded as patients and be given treatment, or may it be assumed that the condition will clear up spontaneously? In an editorial note readers were invited to contribute—as they still are. In the meantime, Dr. Sagher's inquiry was sent to several persons who it was thought might contribute, and the following represents the replies received.

In a second letter on the subject Dr. Sagher wrote:

I am surprised that cases with bacteriologically positive findings but without clinical lesions have not been studied more extensively in endemic countries than they seem to have been. So far, I have had five certain cases which I have been following carefully for three years. There was quite a discussion in our group as to whether they should be treated or not. There was one patient with 3-plus findings in his skin and nasal mucous membrane, and I felt that this was an "open" case; so I decided to treat them all and follow them up bacteriologically. The observations are not yet ready for publication, but I hope that can be done in the not too distant future.

From Drs. Lauro de Souza Lima and Nelson de Souza Campos, São Paulo, Brazil.—Replying to the questions of Dr. Felix Sagher, of Jerusalem:

1. It is very common, in children from 6 to 12 years of age, with or without contact with leprosy patients, to find hypochromic spots, usually situated on the face, without disturbances of sensation and with the complete (i. e., negative) histamine reaction, which are not of leprous origin and which sooner or later disappear spontaneously. In our country these lesions, known as *dartro volante* or "pitiriasis alba," are of streptococcic origin and heal quickly with local treatment with a solution of iodide base. A positive lepromin reaction in such a case is due either to contact with leprosy or to tuberculin sensitivity.

The hypochromic lesions of leprous origin, of the indeterminate group, besides presenting disturbances of sensation and the incomplete (i. e., positive) reaction to histamine, have specific clinical manifestations and usually do not heal spontaneously.

2. We have never had the opportunity of observing a clinically negative contact with positive smears from the nose or skin, although such cases have been mentioned by some authors. Certain cases clinically without individualized lesions but presenting very inconspicuous diffuse infiltration may give positive bacteriological findings, but

their clinically lepromatous type must always be confirmed by biopsy. This done, they should be considered as patients and treated as such.

From Dr. S. D. Desai, Acworth Leprosy Home, Bombay, India.—About the questions asked by Dr. Sagher, I will confine myself to presenting observations bearing on the second one.

During the last few years we at this institution have made a careful study of the spread of leprosy in families of patients, and have examined a total of 1,852 contacts clinically, bacteriologically and immunologically. Our primary concern has been the detection and investigation of persons without skin lesions but positive for acid-fast bacilli. We have found many of them, as will be seen, and have actually observed lesion-free contacts who were bacteriologically negative to start with but who later showed repeatedly a few bacilli in the skin. The bacilli in these lesion-free persons were obtained from the earlobe, or from the skin of the back, the arm, the forearm or the thigh. These findings have been reported in two articles [*Indian J. Med. Sci.* **3** (1949) 253-265, reprinted in THE JOURNAL **18** (1950) 59-66; THE JOURNAL **19** (1951) 165-172]. In searching for bacilli, since 1952 we have used instead of the deep biopsy method the chloroform extraction method of Figueredo and Desai [*Indian J. Med. Sci.* **6** (1952) 296-301].

Regarding our terminology, we call "primary lesions" those of a kind that we have seen to develop in some of the positive contacts. These occur as one or more small, circular, hypopigmented areas 1/4"-1/2" in diameter, flat or slightly raised, without sensory changes, but positive for a few acid-fast bacilli by the chloroform extraction method; the lepromin reaction is positive at this stage. Some of these primary lesions later develop sensory impairmant, and then they are called "persistent lesions-early neural" [here designated PL-EN]. In some patients these lesions have become tuberculoid, or the "simple macule" of the Cairo classification. A few lesions have been seen which we call "persistent lesions-early lepromatous" [PL-EL]; these are the "prelepromatous" macules of Cochrane. A description of these lesions for medical practitioners has been published [*Indian J. Child Hlth.* **1** (1952) 285-295].

The distribution of findings in the 1,852 contacts examined was:

(1) 385 Established leprosy (325 N, 60 L);
(2) 4 "Persistent-neural" lesions;
(3) 3 "Persistent-lepromatous" lesions;
(4) 194 Primary lesions, bacteriologically positive;
(5) 610 No lesions, bacteriologically positive;
(6) 656 No lesions, bacteriologically negative.

The lesions in Group 4 and the cases in Group 5 were repeatedly positive for bacilli, 5-10 in number, and all of the patients were lepromin positive except 14 children in Group 5; 12 of them, when retested, also reacted positively. The cases of Group 6 were repeatedly negative for bacilli and nonreactive to lepromin except for a few that had nodules less than 3 mm.[1]

Continued observations: The later findings in those cases that we could keep under surveillance were as follows:

Of 67 negative contacts (Group 6), 34 later on gave positive smears repeatedly, and the lepromin reaction turned positive; 18 of them developed primary lesions. Of the 67 there were 18 others who developed primary lesions without our having observed

1 The lepromin used has been of the Dharmendra type. Uninfected contacts give no early reactions, at most slight erythema less than 5 mm. in diameter and without edema; they have shown no nodule up to four weeks, although in a few there were small lumps less than 3 mm. in diameter and palpated with difficulty. In infected contacts the early reaction, with edema, is always more than 5 mm., and the late reaction nodule is 3 mm. or more.

the bacillus-positive stage; 3 of them progressed into the PL-EN stage, 1 becoming tuberculoid.

Of 71 positive contacts (Group 5), 27 developed primary lesions. In 2 of the 27, those lesions were found to have vanished after a few months. On the other hand, 5 of the 27 progressed to the PL-EN stage and 4 of those later became tuberculoid. Two of the 71 developed the PL-EN condition without observation of the primary lesions stage, and 1 progressed to simple macular. In one case the first lesions seen were tuberculoid.

Of 48 contacts originally with primary lesions (Group 4), those lesions vanished in 4 cases; 7 progressed to the PL-EN stage, 1 later becoming tuberculoid macular; and 1 was seen with tuberculoid lesions without observations of the PL-EN stage.

Of the 4 cases of Group 2 (PL-EN when first seen), one progressed to the tuberculoid stage, the others remaining unchanged.

Supposed noncontacts: During the past two years we have also examined 756 persons not suffering from leprosy but sent to us as suspects. Only 31 of them (4.1%) showed a few acid-fast bacilli, 2 to 9 in number, on the first examination (chloroform method). Whether or not they were contacts of leprosy patients could not be determined. Of 20 who could be reexamined, 4 showed a few acid-fast bacilli on two further examinations and 16 were negative on the second examination. There is a significant difference between 4.1% among these cases (only 0.53% on repeated examination) and the 33% among contacts, and this provides an objective basis for the opinion that the bacilli found in the contacts are *M. leprae.*

General conclusions: 1. That contacts without lesions but harboring acid-fast bacilli in the skin are infected persons. Most of them are lepromin positive.[2]

2. That contacts without lesions and not harboring acid-fast bacilli in the skin are uninfected persons. They are lepromin negative.

3. That when an uninfected person gets infected, the lepromin reaction changes from negative to positive.

4. That because the lepromin reaction is positive in the majority of the infected contacts and negative in the uninfected contacts, the positive response does not signify resistance to attack but only resistance to the progress of the disease when it is acquired.

5. That the majority of infected contacts develop "primary lesions," which usually progress toward the well-established neural-type macules, the lepromin reaction being positive throughout.

6. That lepromatous leprosy does not arise as such from the beginning, but is a development after the primary-lesion stage.

With reference to the last statement, we have not encountered a single lepromin-negative contact that has shown signs of infection, developing lesions, and has remained negative to lepromin at the early-lesion stage; they have all been lepromin positive. We have seen only two children and one adult with very early lepromatous lesions (PL-EL), similar to the prelepromatous macules of Cochrane, positive for a few bacilli by the section method and negative to lepromin; but we have not been able to trace the origin of these lesions.

Treatment: Originally, our uninfected and infected contacts without lesions were kept under observation without treatment. Recently we have been giving DDS twice a week, in doses of 10-25 mgm. for children and 25-50 mgm. for adults. The period of treatment has been 4-14 months.

Of 11 uninfected contacts (6 adults and 5 children) receiving treatment regularly, none has yet become infected. In the untreated control group (7 adults and 5 children), one child now shows signs of infection, and 2 children have developed primary lesions.

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[2] Khanolkar has called this stage the "silent phase of the disease." (Studies in the Histology of Early Lesions of Leprosy, Indian Council of Medical Research Special Report Series, No. 19, 1951.)

With regard to infected contacts, 7 adults and 1 child out of 35 adults and 19 children were rendered bacteriologically negative by the treatment. In the untreated control group, only 4 adults and 3 children were examined regularly; 2 adults and 1 child developed primary lesions.

Additional staff is to be engaged to extend this experiment in order that we may come to definite conclusions regarding the efficacy of DDS in preventing infection to uninfected persons, and in removing infection from the infected contacts without isolation of the infector.

From Dr. Dharmendra, Calcutta, India.—1. The first question of Dr. Sagher really comprises two, first about the nature of the impermanent hypopigmented patches in contact children, and then the significance of their disappearance. Such lesions are well known to many workers as suspicious of being leprotic but without any definite signs of that infection, although by some they are described as "primary" or "basic" lesions of leprosy. The question is the grounds on which they can definitely be considered as leprotic, in the absence of all known signs of the disease (sensory disturbances, acid-fast bacilli, and histological changes), especially when similar-looking lesions can be seen in unexposed persons and can be produced by nonleprotic affections of the skin. The fact that some of the cases react strongly to lepromin is of no significance, for the test is not diagnostic and positive reactions are nonspecific.

It is not denied that some of these lesions may be due to leprosy, but it is impossible to distinguish them with certainty from others that are not. It is also true that such suspicious patches in children in close contact with infectious cases of leprosy are of graver import than similar patches in children without such contact, since the likelihood of their being due to leprosy is greater, but not all can correctly be ascribed to leprosy. Use of a concentration method for finding bacilli may sometimes be helpful in determining lesions that are of that nature.

It can therefore be said, in reply to the first part of the first question, that not all "impermanent hypopigmented patches in bacteriologically negative contacts (children) of leprosy patients" necessarily "represent an early clinical manifestation of the disease."

The rest of the question, about the disappearance of the lesions, obviously refers to those that are leprotic. On that basis it may be said that in general their disappearance usually means spontaneous cure. Occasionally, however, one does see a case in which there is a reappearance of the disease later on. In this connection I am reminded of a patient first seen in 1939 at the age of 6 with slight hypopigmentation on thighs and legs. No definite signs of leprosy were found, and the lesions disappeared after a short local treatment. (They might have disappeared as well without any treatment.) He was kept under observation, however, and in 1946 ill-defined and irregularly thickened smooth patches were seen on thighs, knees, and legs, which showed loss of sensation and positive bacteriological and histological findings.

2. The second question of Sagher necessarily leads to consideration of the significance of the finding of acid-fast bacilli in the skin of healthy contacts. Search for bacilli in the nasal mucosa, the skin, and even the superficial lymph nodes of such contacts has been made to detect latent infection, and acid-fasts encountered have often been regarded as leprosy bacilli. It is necessary to examine this interpretation critically.

As for positive findings in the nasal mucosa, some early workers reported the finding of acid-fast bacilli in this site in varying proportions of healthy contacts, but that has not been confirmed by extensive work of some later investigators. Regarding the superficial lymph nodes, positive findings have been reported but with significantly less frequency in recent reports than in the earlier ones. Moreover, one cannot be very sure of the significance of positive findings. If acid-fast bacilli

are found in either location, the greatest caution should be exercised in deciding to call them leprosy bacilli.

The matter of positive findings in the skin needs more attention. It is generally accepted that the usual portal of entry in leprosy is the skin, and it is to be expected that during the long latent period the bacilli may lie dormant there—somewhere. Because this structure is so extensive, and because of the difficulty of identifying any acid-fasts encountered in the healthy skin, there was little search of this site until the recent reports of certain workers in Bombay. They have reported positive findings in as high as 80% of healthy contact groups! Certain other workers, including myself, have confirmed the finding of acid-fast bacilli in the skin of healthy contacts, although less frequently. The significance of these findings, however, is not clear.

The bacilli so found by the Bombay workers have been described as "acid-fast micro-organisms possessing the morphological character of leprosy bacilli," and have been presumed to be of that nature. These "positive contacts" have been considered as infected persons with the disease still latent, and the tissue changes have been described as "histological findings during the silent phase of the disease." My own view, however, is that available evidence does not justify the conclusion that the bacilli so encountered are really leprosy bacilli, and that because acid-fasts other than leprosy bacilli may sometimes be present in the skin of healthy persons, these findings have to be interpreted with great caution.

According to the Bombay workers, their diagnosis is supported by: (1) correlation between lepromin positivity and the presence of these bacilli; (2) phagocytosis of these bacilli by macrophages in the skin; and (3) subsequent development of leprosy lesions in a number of the positive contacts. These observations need to be examined critically.

(1) The reported correlation between positive bacillus findings and positive lepromin reactions would, if correct, no doubt provide strong support for the presumption that the acid-fasts found are really leprosy bacilli, and are responsible for the positive lepromin reactions in these individuals. However, in view of the absolutely nonspecific nature of the lepromin reaction (except for the specific negativity of lepromatous cases), and the fact that large proportions of people in nonendemic regions, never exposed to leprosy, give positive reactions, it is difficult to understand this reported correlation. In a similar study reported from Calcutta there was no such complete correlation.

(2) The finding that the acid-fast bacilli seen in the skin of normal contacts had been engulfed by the macrophages does not unequivocally signify that they were leprosy bacilli, or that they were not mere saprophytes. It is at least quite possible that other acid-fasts of variable degrees of virulence (e. g., paratubercle bacilli) might get into the skin and be disposed of in this manner.

(3) The subsequent development of leprous lesions in bacillus-positive contacts is no doubt a strong point in favor of the presumption, but it is far from conclusive. The fact that when the lesions develop bacilli are often absent from them and from other parts of the body weakens this evidence. Their disappearance when the lesions appear would be difficult to explain. It is not possible that the bacilli seen earlier had no causative relationship to the leprosy lesions which appeared later? It has not been shown that persons in whom these acid-fasts cannot be found in the skin may not also develop leprosy.

From the foregoing it follows that the evidence on which the Bombay workers have based their presumption regarding the nature of the acid-fast bacilli found in the skin of healthy contacts is not definite and clear-cut, especially in view of the ubiquitous nature of saprophytic acid-fasts. The absence of control observations of persons living in nonendemic areas contributes further to this uncertainty.

With this background the obvious reply to Sagher's second question would be that "contacts without any clinical signs of the disease but with bacteriologically positive smears" should not be regarded as cases of leprosy. The question of their treatment as patients should therefore not arise.

There is another and related question which may be mentioned, one about which some leprosy workers have shown concern, namely, the prophylactic use of sulfone drugs in all contacts as a means of controlling the spread of the disease. This matter doubtless should be investigated, with respect to method and results, but until that is done it would not be correct or desirable to advocate the large-scale treatment of all contacts. Sulfones are toxic drugs. There is justification for their prolonged use in persons suffering from a serious disease in which the beneficial effects outweigh the harmful ones, but that cannot be said regarding persons who are not suffering from the disease and many of whom will never suffer from it.

From Dr. Casimiro B. Lara, Culion, Philippines.—The question of contact children with hypopigmented patches is of interest to me and I wish to offer some remarks. The other question, that of contacts without clinical signs of leprosy but with positive smears from the skin and/or the nasal mucous membrane, I prefer not to discuss.

Hypopigmented patches are of course well-known as among the early manifestations of leprosy. This type of lesion and other initial kinds are discussed in some detail in an article on leprosy in infancy and childhood I published in 1948 [*Mo. Bull. Bur. Hlth.* (Manila) **24** (1948) 61-89; also, Mem. V. Congr. Internac. Lep., Havana, 1948; Havana, 1949, pp. 414-431].

Our experience with contact children in Culion indicates that the genesis of the hypopigmented patches is variable. A few of them, in very young children regularly observed since birth, have been found to arise as such. After the last war, a much larger proportion of the early lesions found among the pre-school children who had been observed only infrequently during the war years consisted of similar hypopigmented patches. On the other hand, quite a few of the children who have been regularly and frequently observed up to the time they reach the school age, both before the last war and since then, have hypopigmented patches that had their origin from slightly raised, pinkish or reddish hypopigmented macules, or from wheal-like papules, and, more rarely, even from papulonodules.

Hypopigmented areas, flat or very slightly raised, with clear borders and negative for *M. leprae* in ordinary smears, if due to leprosy, usually show more or less definite tuberculoid histology, and the Mitsuda reaction is positive. A certain proportion of such lesions show no specific type of reaction or pattern histologically, only round-cell collections with or without a few scattered epithelioid or small tuberculoid foci, which may be found from the basal epithelium down to the deeper structures of the dermis. Some proportion of these cases give negative or weak Mitsuda reactions.

Hypopigmented macules of clinically recognizable tuberculoid leprosy in children usually persist for one or even a number of years before showing a tendency to fade. Other types of (flat) hypopigmented macules, although equally persistent, are liable to show alternating periods of increased and decreased activity, usually with pinkish color and perceptible slight elevation of part or the whole of the macule during the active periods. Eventually, however, these kinds of hypopigmented macules will also fade, irrespective of the subsequent arrest or general progress of the case. Those cases with a tendency to progress give positive smears with increasing frequency preceding the onset of more definite manifestations of lepromatous leprosy. They are not, however, to be confused with the faint, well-delineated, flat hypopigmented lepromatous macule, which usually shows a burnished or coppery sheen and many acid-fast bacilli on smears, and usually occurs in large numbers over extensive areas of the surface.

It should further be pointed out that, even among contact children, faint hypopigmented macules without other definite manifestations of leprosy may not necessarily indicate a lepromatous nature if their negative histopathology and the absence of acid-fasts (as observed by our Dr. J. O. Nolasco, of Culion) can be taken as reliable criteria.

From Dr. José N. Rodriguez, Manila, Philippines.—The impermanent hypopigmented patches found among children who are family contacts of open cases of leprosy, described by Dr. Sagher, are quite familiar to leprosy workers in the Philippines, where such patches stand out distinctly on the light brown skins of our children. The neurological examination is seldom satisfactory, due to their young age. The bacteriological findings are almost always negative, as said by Dr. Sagher, and histologically the pathologists report only perivascular round-cell infiltration.

In one special group of 80 children ranging in age from 1 to 8 years which I examined in 1924 (not reported) about 18% showed this type of lesion, known to us as the "simple macule." I was able to reexamine some of these same children at various intervals ranging from 10 to 16 years later at Welfareville in Manila, where they had been transferred from Culion. These included 9 of those who had shown only the "simple" hypopigmented macules in 1924. None of the children previously with simple macules had become lepromatous, judging from the available records.

To answer specifically Sagher's first question, the importance of such simple hypopigmented patches in contact children is that they are an indication that those children should be followed up. Some of these lesions later become tuberculoid; very few become lepromatous; in most instances they disappear completely. This is my experience up to the age of about 20 years, at least; I do not know what happens after that.

Do they represent an early clinical manifestation of leprosy? Yes, I believe so; I have not seen typical lesions of this kind among children in the normal population.

Does their disappearance mean a spontaneous cure, or may the disease reappear after a latent period? I do not know, but, as already said, in our experience with cases now up to around 20 years of age the disease has not reappeared.

With reference to the second question, about family contacts without clinical signs of the disease but with positive smears, I have no personal experience. The Leonard Wood Memorial is planning to do some work along this line at Cebu. On general grounds, however, I am inclined to believe they should be given active treatment.

From Drs. Felix Contreras and Javier Guillen, Fontilles, Spain.—The questions of Dr. Sagher are interesting, arising from observation of cases such as are seen by all who are concerned with the early diagnosis of leprosy and especially in the study of leprosy in childhood.

It is classical in dermatology to consider all macules as stages of evolution of very different lesions or syndromes which may also assume this form during the evolution to recovery. Hypochromic macules are very frequent in childhood, especially in the lower social classes, and it is there where we should be especially careful about the diagnosis of the initial manifestations of leprosy.

Dermatologically these macules are given little importance because in most cases they are only indications of streptococcic or other banal dermatoses. However, because they can be the initial manifestations of such a major infection as leprosy they should be dealt with as of major importance.

The proportions that can be attributed to leprosy or to more banal causes vary greatly, depending on the endemicity of a given area and the nature of the clinic or dispensary in which the study is made. In general dermatological work, not con-

nected with leprosy, the great majority of the hypochromic macules seen have no relation to that disease, whereas leprosy investigators in endemic foci more often find a relationship of these macules with leprosy infection.

In the meticulous study of such lesions consideration must be given the history and origin of the patients, and the investigation must include tests of sensation, the histamine test, and the bacteriological examinaion. In cases in which these tests are negative they are, as usual, of no value; but the investigation should be continued without worrying the patient about the disease we have in mind. Those coming from endemic regions should be given the lepromin test, which enables us to ascertain the degree of specific resistance of the organism against the probable causative agent, and also the Fernandez test, by subcutaneous or intramuscular injection of 1 cc. of lepromin, which is of most value in precisely these cases.

In some cases, in spite of all methods of examination, we cannot determine the etiology of the macules. When they disappear without our first being able to diagnose them, without knowing whether they were banal or specific, the uncertainty persists with respect to the question of whether or not they may reappear. We have cases exemplifying the various possibilities, which imposes the necessity of submitting all these children to careful and long-term observation.

With respect to Sagher's second question, in leprosy as in all infections there can be carriers of the germs without clinical manifestations, which cases can be explained as *latent* infections or *abortive* clinical infections, according to the eventualities described by Wade and admitted by all. We believe that these cases are infrequent, possibly exceptional. They may perhaps explain some cases of leprosy the infection of which is extremely difficult to understand.

What should be done with these cases depends not only upon the clinical and bacteriological findings; but the immunological condition, the response to lepromin, is also very important. We agree with the norms indicated by Fernandez in his book "La Infección Leprosa en el Niño."

From Dr. Jacinto Convit, Caracas, Venezuela.—With reference to the significance of the hypopigmented patches about which Dr. Sagher inquired, in our experience the primary, hypopigmented lesions are among the less frequent indications of early clinical leprosy. They have been the initial manifestations in only 12% of the cases in our records. More often these spots or patches have been observed here as a secondary development of papulate or nodular plaques.

On the other hand, in dealing with the problem of a bacteriologically negative hypopigmented patch, histologically a chronic, perivascular round-cell infiltration, where impaired sensation may be difficult if not impossible to determine, as in the case of children, we would consider the following possibilities: (1) To diagnose the patch as an incipient leprosy lesion, (*a*) when it presents clinical or localizational characteristics, and/or (*b*) when it gives an abnormal histamine reaction. (2) Not to diagnose it as an early lesion, if the clinical characteristics and the histamine reaction are at variance. (3) To regard the case as still doubtful when it appears clinically to be incipient leprosy but gives a normal histamine reaction. In considering these three possibilities I have not mentioned the investigation of sensation, because of the difficulty that it involves in children.

With reference to the first alternative indicated above, we would have to consider whether or not there is a definitely positive Mitsuda reaction. If so, we would give adequate sulfone treatment, and "consolidation treatment" after the lesion has disappeared. If not, on the other hand, we would begin sulfone treatment and also give BCG vaccination to induce lepromin positivity. The treatment will be prolonged, and: (*a*) If the Mitsuda reaction becomes persistently positive we proceed as in the former case, but (*b*) if the Mitsuda reaction continues negative we continue the treatment

until at some time the reaction does become positive, without attributing special importance to the disappearance or the persistence of the hypopigmented patches.

In the event of the third alternative above, the contact should continue under observation and control as advised by the Madrid congress.

As for the question of whether or not the spontaneous disappearance of hypopigmented patches should be interpreted as a spontaneous cure of the disease, and of the possibility of the disease reappearing after a period of latency, my reply is fundamentally contingent upon the nature of the lepromin reaction in such contacts. I have observed contacts with hypopigmented patches and a decidedly positive lepromin reaction, in whom the patches recovered their natural color after treatment with chaulmoogra oil—which is not a very active medicament—and who have not subsequently shown any new signs of the disease. These cases did not receive sulfone treatment, which has been used in Venezuela only since 1945. On the other hand, I have observed contacts with hypopigmented patches and no other apparent manifestation of the disease and with negative lepromin reactions, who in spite of chaulmoogra treatment subsequently developed lepromatous leprosy. Also, I have had Mitsuda-positive contacts whose hypopigmented patches have persisted and increased in area. There is one point which I believe is important, and that is whether or not the contact continues to live with the source of infection. It may be assumed that those who do so are in greater danger of developing the disease than those who do not.

As for Sagher's second question, I have had almost no experience in this matter, as the cases we have had under observation are few. I think that the bacilli should be found in a fairly numerous group of contacts to permit the establishment of criteria, but we have seen very few such individuals. Until these findings have been properly appraised, the control and protection of such contacts should follow the standards accepted by the Madrid congress.

[Note: One well-known leprologist to whom the questions were sent preferred not to contribute a communication or to be quoted, because he has not especially studied the matter. However, he said that he has read some of the publications on this subject "with misgivings and some astonishment." He has been surprised sometimes to see how vague lesions of the skin are definitely classed as leprous, with no clinical or bacteriological support. He feels that to make a definite diagnosis of leprosy "with neither clinical nor bacteriological evidence of a convincing nature is practically never justified."]