CAUSES OF DEATH OF LEPROSY PATIENTS INFLUENCE OF LEPRA REACTIONS AND RENAL DISEASE

CARLOS M. BRUSCO, M.D.
AND JORGE G. MASANTI, M.D.
Sanatorio Baldomero Sommer
Buenos Aires, Argentina

The study of leprosy must cover all the facets of the disease, and therefore must deal with conditions related to it.

The general health aspects of the disease, and its epidemiologic, immunologic, and diagnostic features have been and are being intensively studied, with various results, by specialists. These are mostly dermatologist-leprologists who, however, have tended to neglect the clinicopathologic aspects of the disease related to internal medicine. In the treatises on leprosy, little importance is placed on the clinical and pathologic study of the viscera, and little space is devoted to those subjects. This fact is also noticeable on reading the titles of papers presented before specialized medical societies and at national and international congresses.

In some diseases, less frequent and of minor social significance, such as disseminated lupus erythematosus, nodular periarteritis, and dermatomyositis, the visceral lesions are much better known than are those of leprosy. Nevertheless, the visceral lesions of leprosy patients are of great importance *quoad vitam,* and the prognosis of the disease depends greatly on them.

In the present study of the causes of death in leprosy patients, the influence of the type of leprosy and of lepra reactions are considered especially, and also the influence of these factors on the development of kidney diseases and the incidence of kidney complications.

MATERIAL AND METHODS

The data of this report have been taken from the clinical histories of 630 patients who died from 1941 to June 1960 in the Baldomero Sommer Sanatorium. Of that number, 132 histories were discarded for lack of essential data, leaving 498 for analysis. The data obtained from these histories are the following: clinical form, age of the patients at the time of death, sex, length of time from the onset of the disease to death (calculated according to the answers to questions asked the patients on admission plus the duration of segregation), presence or absence of lepromatous reactions in the course of the disease, presence or absence of kidney complications as indicated by the occurrence of persistent albuminuria, and the clinical diagnosis of the cause of death. We were not able, unfortunately, to include postmortem findings since autopsies were not done except in exceptional cases.

Regarding the 132 discarded histories, it will be seen that special importance is given in this report to the occurrence of lepromatous reactions in the course of the disease, and all of the histories from which we were unable to ascertain whether or not the lepromatous patients had had reactions were discarded. In general, the discarded histories pertained to patients who had been interned in the sanatorium for only short

periods. The 498 cases whose histories were used in this study were divided into 3 groups:

1. *Nonlepromatous leprosy* (T.L.I.).—In this group are included all the patients with the tuberculoid, *limitrofe* (borderline), and indeterminate forms of leprosy; i.e., those conditions which did not transform to lepromatous. There are 35 cases in this group.

2. *Quiescent lepromatous leprosy* (without reactions).—In this group are included all of the patients with the varied manifestations of the lepromatous form of leprosy who, according to their histories, did not have reactional outbreaks. This group comprised 255 cases.

3. *Lepromatous leprosy with reactions.*—In this group are included all the patients whose histories contain evidence of the occurrence of reactional manifestations at some time during their evolution. Only patients who had isolated ocular reactions were not included. This group comprised 208 cases.

On the basis of the available data the following problems are discussed:

(1) Immediate causes of death, according to the clinical diagnoses.

(2) Age at the time of death. The average age of the total group was determined, and the cases were divided according to sex and clinical form with respect to the factor of reactional manifestations. The average ages of the total group and of the different groups considered were calculated by the grouped series method ([10]), the results of which did not differ significantly from the arithmetic averages calculated by the usual procedure. (A comparison of the results of the two methods is shown in Table 4.)

(3) The incidence of kidney complications in the different groups, and their influence on the age at the time of death.

In some instances cases had to be excluded from one or another of the groups because of lack of certain data. This explains why the figures in some tables and the totals are not in agreement.

To ascertain the statistical significance of the differences found in these studies, the Pearson Chi-square test was applied ([10]).

CAUSES OF DEATH

The tables of the posthumous diagnoses are not included in this report, for they are very complicated. However, the following data are drawn from them. Before proceeding, it is necessary to make clear the fact that the diagnoses were clinical, and therefore subject to usual errors of this type of diagnosis.

Noteworthy is the frequency of the diagnosis of uremia as the cause of death, which was made in 21.6 per cent of the cases with reactional leprosy, and 11 per cent of those without reactions. In the former group, especially the patients below 50 years of age, uremia was the most important cause of death. Heart disease was the most important cause of death among patients older than 50 years.

With the data available it was often difficult, not to say impossible, to correlate the clinical diagnosis with the immediate cause of death. However, according to data discussed later, it was evident that some of the causes of death are related more or less directly to leprosy, without implying that the leprosy bacillus was directly responsible for the lesions which led to death.

Some of the clinical diagnoses deserve comment. The so-called leprous cachexia is, very probably, a consequence of a complication of leprosy which, in turn, is indirectly the cause of death. The diagnosis of acute leprosy as the direct cause of death also seems defensible. The

diagnosis of intestinal amyloidosis was made in 8 cases, but in none of them did it have a firm clinical basis. It would not be surprising if what was diagnosed as acute leprosy, intestinal amyloidosis, and leprous cachexia are related to a common cause.

PROBABLE LENGTH OF LIFE

From Tables 1 and 2 it is seen that the length of life seems to be shorter among the lepromatous patients than among those with the relatively benign clinical forms (lepromatous: 51 years 9 months; nonlepromatous, 66 years 5 months). In Table 3(a) there are data which confirm this indication. In the more detailed study there is evidence that, among both the men and the women, lepra reactions had shortened the life of the patients (patients with reactions: men, 49 years 6 months,

TABLE 1.—*Average age at death of the groups of patients studied.*

Patient group	No. of cases	Average age	
		Arithmetic mean	Grouped series method
Nonlepromatous, total	35	67y.6m.	66y.5m.±11y.
Males	18	—	67y.2m.±11y.
Females	17	—	65y.7m.±10y.6m.
Lepromatous, total	463	54y.	51y.9m.±15y.6m.
With reactions	208	49y.3m.	47y.7m.±15y.1m.
Without reactions	255	57y.8m.	55y.3m.±13y.1m.
Sex in lepromatous cases			
Males			
With reactions	124	—	49.6m.±14.6m.
Without reactions	187	—	55y.9m.±14y.8m.
Females			
With reactions	81	—	44y.10m.±14y.
Without reactions	68	—	54y.3m.±13y.7m.

TABLE 2.—*Average age at onset of the disease, and duration at the time of death.*

Patient group	Age at onset	Age at death	Duration
Nonlepromatous, total	55y.11m.	66y.5m.	10y.6m.
Males	56y.4m.	67y.2m.	10y.10m.
Females	55y.3m.	65y.7m.	10y.4m.
Lepromatous total	40y.9m.	51y.9m.	11y.
With reactions	37y.7m.	47y.7m.	11y.
Without reactions	44y.11m.	55y.3m.	10y.11m.
Males, lepromatous			
With reactions	37y.11m.	49y.6m.	11y.7m.
Without reactions	44y.5m.	55y.9m.	11y.4m.
Females, lepromatous			
With reactions	34y.9m.	44y.10m.	10y.1m.
Without reactions	44y.2m.	54y.3m.	10y.1m.

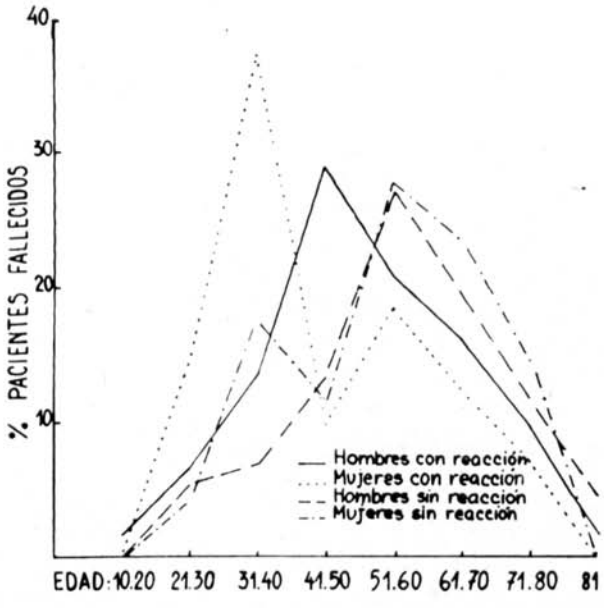

FIG. 1. Age distribution of deceased lepromatous patients, by grouped series.

women, 44 years 10 months; patients without reactions: men, 55 years 9 months, women, 54 years 3 months). These findings are confirmed on comparing the mortality earlier than 50 years (Table 3(a), (b), (c) and (d)). These differences are statistically significant. From Table 3(e) and 3(g) it is seen that women with reactions die earlier than the men, a fact which seemingly does not occur when the sexes of the patients with lepromatous leprosy without reactions are compared (Table 3(h); Figs. 1 and 2). In summary, it can be stated that lepromatous leprosy shortens the life of the patients; this is more evident in the patients who experienced reactions (Fig. 3), and especially among the women with this phase of leprosy.

DURATION OF THE DISEASE

A noteworthy fact shown in Table 2 is that the duration of the disease was practically the same in all of of the groups of patients studied.

AGE AT ONSET

Taking into account the age at the time of death and the duration of the disease, the age of the patients when the disease appeared has been calculated for the different clinical groups (Table 2).

The data indicate that the disease may appear at an early age in those patients with lepromatous leprosy who present, in the course of the disease, reactional manifestations, especially among the women.

If this observation is correct, and can be applied generally without committing error, we should be able to accept one or the other of the following hypotheses: (1) The earlier the onset of lepromatous leprosy

TABLE 3.—*Mortality before* (a) *50 years and* (b) *40 years of age, and statistical significance of the differences.*[a]

	Patient group[b]	Patients total	Patients dead	Value of P.[c]
Death before 50 years				
(a)	Nonlepromatous	350	3	<0.001
	Lepromatous	460	184	
(b)	Lepromatous with reactions	205	113	<0.001
	Lepromatous without reactions	255	71	
(c)	Males with reactions	124	63	<0.001
	Males without reactions	187	48	
(d)	Females with reactions	81	50	<0.001
	Females without reactions	68	23	
(e)	Males with reactions	124	63	<0.001
	Females with reactions	81	50	
(f)	Males without reactions	187	48	Not significant
	Females without reactions	68	23	
Death before 40 years				
(g)	Males with reactions	124	27	<0.001
	Females with reactions	81	42	
(h)	Males without reactions	187	23	Not significant
	Females without reactions	68	15	

[a]Chi-square method of Pearson.
[b]All lepromatous except for the first item.
[c]The difference significant except where stated otherwise.

occurs, at least in the sexual age, the greater the possibility that reactional forms will develop, or (2) the patients who are potentially reactional are less resistant to the disease and therefore the onset is earlier in them.

KIDNEY COMPLICATIONS

Among the lepromatous patients with reactions, it is seen that 59 per cent had, at the time of death, kidney disease as evidenced by persistent albuminuria; among the nonreactional ones the proportion was 26 per cent. The difference between the two groups is statistically significant, as can be seen in Table 4(b). The difference between the nonreactional lepromatous patients (26%) and those of the nonlepromatous group (8%) in Table 4(a) is also significant.

In Figure 4, we see that there are two decades of life in which the greatest incidence of kidney disease occurs (31-40 years, and 51-60

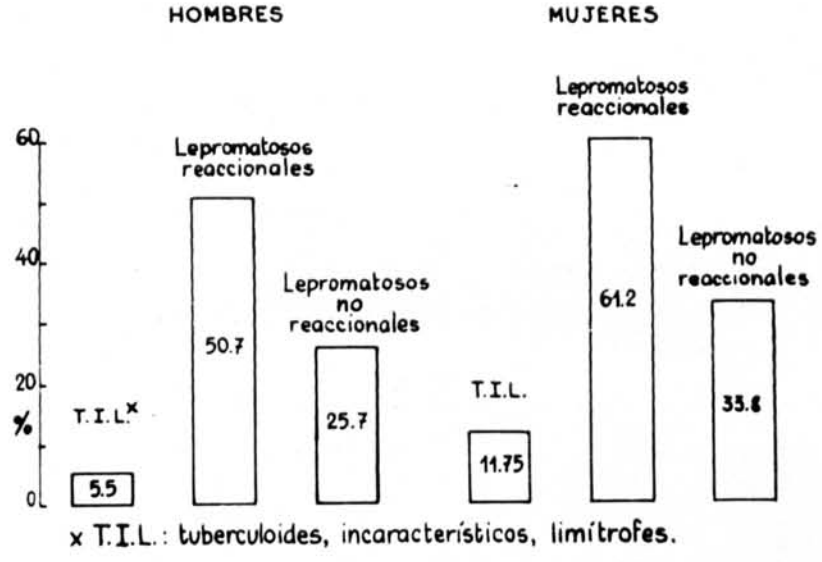

FIG. 2. Patients dead before 50 years of age, men and women grouped as nonlepromatous (T.L.I.) and as lepromatous with and without reactions.

TABLE 4.—*Incidence of renal disease and statistical significance of the differences.*

	Patient group	No. of patients	No. with renal disease	Value of of P.[a]
(a)	Nonlepromatous	35	2	<0.001
	Lepromatous	384	148	
(b)	Lepromatous with reactions	144	85	<0.001
	Lepromatous without reactions	240	63	

[a]Significant in both instances.

TABLE 5.—*Distribution of the ages of the dead patients with renal disease.*

Age group (years)	Lepromatous cases		Nonlepromatous cases
	With reactions	Without reactions	
10-20	2	0	0
21-30	7	6	0
31-40	24	10	0
41-50	16	8	1
51-60	19	18	0
61-70	13	13	0
71-80	4	6	0
81	0	2	1
Totals	85	63	2

years). This suggests that kidney disease before 40 years of age may, in general, be different from that observed after 50 years of age. It is therefore possible that, in the fourth decade of life, one type of kidney disease (amyloidosis?) may predominate, and another type after 50 years of age (pyelonephritis?). If this is true, it must be accepted that reactional conditions (which produce the greatest number of kidney diseases before 40 years of age) favor one type of kidney disease and that lepromatous leprosy itself favors another. In turn, lepromatous leprosy without reactions, by means of aggregate infections (e.g., chronic osteomyelitis), undoubtedly may also produce renal amyloidosis.

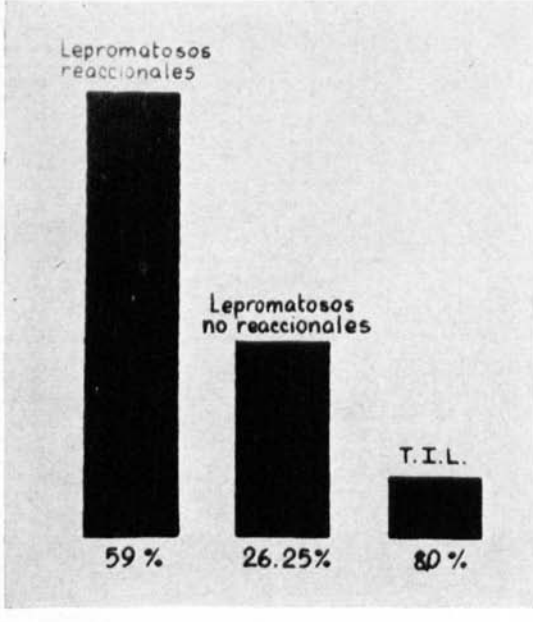

FIG. 3. Incidence of renal disease in the total groups of deceased patients.

INFLUENCE OF KIDNEY DISEASE ON THE AGE AT DEATH

From Table 6(a) it is seen that kidney diseases shorten the life of lepromatous leprosy patients, since within this group those with kidney diseases die before 50 years of age in a larger proportion than those without kidney lesions. The difference is statistically significant.

Also among the lepromatous patients without reactions (Table 6(b)), a greater proportion of those with renal lesions die before 50 years of age, although the difference with respect to those without kidney lesions does not have statistical significance.

DISCUSSION

Lepromatous leprosy, whether quiescent or with reactions, shortens the life of the patients. The unsettled problem is to what lesions the actual cause of death is to be attributed.

Certainly, it is not the dermatologic lesions directly that cause the

TABLE 6.—*Influence of renal disease on the death of lepromatous patients before 50 years of age.*

	Patient group	Number of patients	Number of deaths	Value of of P.
(a)	With reactions, with renal disease	85	49	<0.001
	With reactions, without renal disease	120	43	
(b)	Without reactions, with renal disease	63	24	Not significant
	Without reactions or renal disease	192	47	
(c)	With reactions, with renal disease	85	49	Not significant
	Without reactions, with renal disease	63	24	

death of the patients. From biopsy and autopsy studies the existence of specific granulomas in the different visceral organs (spleen, liver and kidneys) has been described ([1-4, 8, 11, 15, 20]). Furthermore, it has been found that, at least in the liver, there occur acute reactions similar to those observed in the skin of lepromatous patients with reactions. The importance *quoad vitam* which these specific visceral changes may have has not been established ([1, 20]).

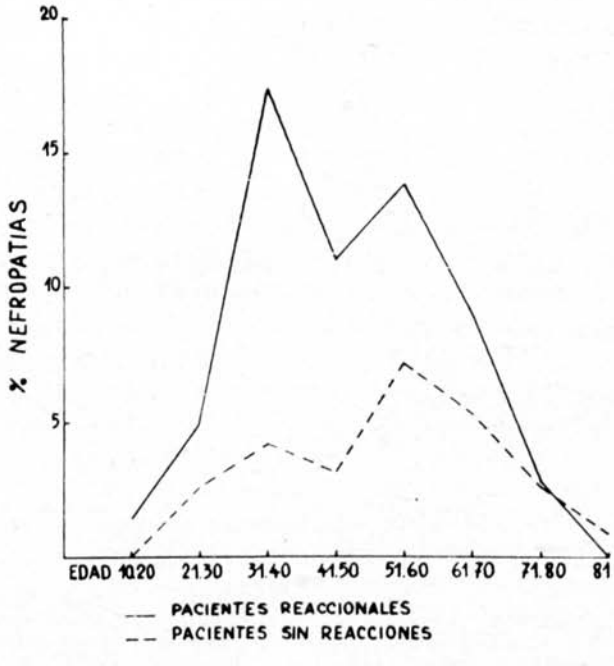

FIG. 4. Distribution by age of deceased patients with renal disease, with and without episodes of lepra reaction.

The patients with repeated reactional episodes die in a few months or years. This is seen with relative frequency in young individuals in whom the fatal outcome cannot be attributed to causes completely independent of leprosy. In some of these cases we have found that, in the course of the reactional phase, amyloidosis develops. In some cases it was noted that, once albuminuria had appeared, an indicator of renal localization of the amyloidosis, pathologic changes in the kidneys developed in a relatively short time (1½, 3 or 5 years), and the patients died with uremia ([16]).

It is accepted in general pathology that renal amyloidosis is of particularly bad prognosis, whereas amyloidosis of the liver, spleen and suprarenal gland may have no important functional significance. Intestinal amyloidosis generally is present in particularly severe cases with that complication, and it may precipitate cachexia and death.

Although leprosy may shorten the average life of the patients, in general it does not kill in an acute manner (average evolution 11 years), permitting the amyloidosis to make a complete cycle, a fact which is not common in other amyloid-producing diseases. Some patients who, due to repeated reactional outbreaks, developed disseminated amyloidosis became cured of that condition after the disappearance of the reactions. In cases which had no renal amyloidosis, there remains a residual kidney lesion with negative Bennhold test ([18]).

Amyloid disease of the kidneys may cause death due to uremia; records of infectious complications in lipoidic nephrosis are not common. In a previous report ([17]) it was pointed out that amyloidosis was seen frequently in leprosy patients with reactions or with chronic infections. Shuttleworth and Ross ([24]) found, among 20 successive autopsies, 12 cases with amyloidosis, of which 10 had reactions and 2 had none; of the other 8 cases without amyloidosis, only 2 had reactions. One of us (J.G.M.) has demonstrated that renal amyloidosis causes hypertension when renal insufficiency sets in ([16]). It is relatively common to find patients with kidney disease and hypertension, with or without uremia, with negative tests for amyloidosis and generally with histories of repeated reactions. Some, at least, of these patients might have had postamyloid disease of the kidneys ([21]), and others might have had some other type of kidney disorder as yet not well differentiated (perhaps a pyelonephritis). It is possible that diseases of the kidneys which lead to hypertension may culminate in cardiac insufficiency in ways independent of renal insufficiency.

The importance of kidney diseases as a cause of death has been well demonstrated by the high rate of incidence of those conditions at the time of death of the patients, an incidence which is much higher than among the general run of patients in the sanatorium (nonlepromatous group: deaths 8%, total population 3%; lepromatous without reactions: deaths 59%, total population 19%) ([19]).

Besides the kidney affections, there are other visceral changes of vital significance. The following perhaps played some role in the causes of death. Aguirre and others ([1]) reported leprotic cirrhosis of the liver demonstrated anatomically (hypertrophy without ascites, histopathologically of the Marchand-Mallory type), which was given a poor prognosis on long duration. Harada and Takashima ([9]) described a leprous meningoencephalitis. Kean and Childress ([12]) found cholelithiasis in 11 per cent of autopsies in leprosy patients, against 3 per cent among the general population.

Before we finish with the problem of the causes of death we wish to suggest a study possibility. Patalano, Marenzi and Masanti ([22]) observed by a chemical technique an increase of the different lipidic fractions of the blood serum. Although this is in contradiction to the observations of other authors ([13, 26]), it seems to us that it might be worthwhile to ascertain whether or not leprosy is capable of producing atherosclerosis. Biochemical, clinical and pathologic investigations may, by confirming the assumption, explain and relate to leprosy certain causes of death apparently independent of the disease.

Schujman ([23]) and other leprologists believe that lepromatous reactions are beneficial to the patients. In this study as in others, for example those of Contreras *et al.* ([5]), Lewis and Edwards ([14]), and Davison and Kooij ([6]), the evidence indicates that lepromatous reaction, far from improving the prognosis, worsens it. With regard to our series, it may be objected that it only includes the cases with unfavorable evolution, whereas those which were benefited, perhaps the majority, had been discharged and therefore are not included in the statistics, for which reason our findings would be nullified. Against this objection are the statistical data of Contreras *et al.* and Lewis and Edwards, according to which patients with reactions take longer to cure, have higher bacteriologic indices, die in greater proportions, are less benefited by paroles, and present higher rates of kidney complications. Davison ([7]) agrees with this view of the matter.

It must be accepted that in some who at certain times present reactional episodes, the subsequent evolution is notably benign, and that in other cases after severe reactional outbreaks with necrosis of the acute elements, rapid cure or evident improvement of leprosy was achieved. These cures, however, are sometimes comparable to Pyrrhic victories, since most of these patients still have visceral lesions which in the long run may be fatal, or have multiple cutaneous scars which considerably affect the cosmetic aspect of the patients.

SUMMARY

The causes of death, the average duration of life in the different groups of patients, the incidence of kidney disorders, and the importance of these conditions and of lepra reactions with respect to the

evolution of leprosy, have been studied by reviewing the clinical histories of leprosy patients who died in the Baldomero Sommer Sanatorium.

1. The lepromatous patients, both men and women, whose histories show that they have at some time experienced reactional conditions, die earlier than those without reactions, while the latter die earlier than the patients with nonlepromatous forms of the disease.

2. Women who experience reactions die earlier than men with the same experience.

3. Kidney complications shorten the life of lepromatous patients, especially those with reactions, among whom these complications seem to be the important cause of death.

4. The evolutive course of leprosy seems to have connections with the age of onset of the disease.

5. Lepromatous reaction, far from benefiting the patients, rather makes them worse.

RESUMEN

Repasado las historias clínicas de los leprosos fallecidos en el Sanatorio Baldomer, se han estudiado las causas de muerte, la duración media de la vida en los diversos grupos de enfermos, la incidencia de las nefropatías y la importancia de estas dolencias y de las reacciones leprosas con respecto a la evolución de la lepra.

1. Los enfermos lepromatosos, tanto hombres como mujeres, cuyas historias muestran que han experimentado en alguna ocasión estados reaccionales, méren más tempranamente que los que no tienen dichas reacciones, en tanto que los últimos fallecen antes que los los que padecen de las formas lepromatosas de la enfermedad.

2. Las mujeres que experimentan reacciones fallecen antes que los hombres afectados en formaigual.

3. Las complicaciones renales acortan la vida de los lepromatosos, sobre todo si tienen reacciones, pareciendo que en ellos esas complicaciones constituyen la causa importante de muerte.

4. El curso evolutivo de la lepra parece guardar relaciones con la edad al iniciarse la dolencia.

5. La reacción lepromatosa, lejos de beneficiar a los enfermos, má bien los empeora.

RESUMÉ

Une revue des histoires cliniques des malades de la lèpre décédés au Sanitorium Baldomero Sommer a permis d'étudier les causes de mort, la durée moyenne de la vie dans les différents groupes de malades, l'incidence des troubles rénaux, et l'importance de ces conditions ainsi que des réactions lépreuses par rapport à l'évolution de la lèpre.

1. Les malades lépromateux, hommes ou femmes, avec antécédents réactionnels relatés à un quelconque moment de leur histoire, meurent plus tôt que ceux sans réaction. Quant à ceux-ci, ils meurent plus tôt que les malades atteints de formes non lépromateuses de la maladie.

2. Les femmes avec antécédents réactionnels meurent plus tôt que les hommes qui se trouvent dans la même condition.

3. Les complications rénales abrègent la vie des malades lépromateux, et particulièrement de ceux avec réactions, parmi lesquels ces complications paraissent être la cause majeure de mort.

4. Le cours évolutif de la lèpre semble en rapport avec l'âge auquel la maladie est apparue.

5. La réaction lépromateuse, bien loin d'être d'aucun bénéfice au malade, semble plutôt rendre son état pire.

REFERENCES

1. Aguirre, M., Gomez Orbaneja, J., Garcia Perez, A. and Lopez Garcia, E. Lesiones hepáticas en lepra. Mem. VI Congr. Internac. Leprol., Madrid, 1953; Madrid, 1954, pp. 773-776.
2. Arning, E. Tuberculoid changes in the viscera. Internat. J. Leprosy **4** (1936) 102-103 (correspondence).
3. Bechelli, L. M. and Rotberg, A. Compendio de Leprología. Rio de Janeiro, Serviço Nacional de Lepra, 1951.
4. Bru, P. and Rollier, R. La ponction-biopsie du foie dans la lèpre. Étude de 38 cas traités ou non traités. Maroc Méd. **36** (1957) 1194-1205.
5. Contreras, F., Guillen, J., Terencio, J. and Tarabini, C. G. Pronóstico de las leprorreacciones. Rev. Leprol. Fontilles **4** (1958) 317-323.
6. Davison, A. R. and Kooij, R. Is erythema nodosum a favorable occurrence? Internat. J. Leprosy **25** (1957) 91-98.
7. Davison, A. R. Erythema nodosum leprosum. Leprosy Rev. **30** (1959) 112-113.
8. Gomez Orbaneja, J. and Garcia Perez, A. Lepra. Madrid, Paz Montalvo, 1953.
9. Harada, Y. and Takashima, S. Histopathology of lepromatous leprosy, especially, studies on leprous vasculitis and leprous meningo-encephalitis. La Lepro **25** (1956) 1-20.
10. Hill, A. B. Principios de Estadística Médica. Segunda Edición. Buenos Aires, El Ateneo, 1958.
11. Hueck, W. and Büngeler, W. Patología Morfológica. Buenos Aires, Labor, 1944.
12. Kean, B. H. and Childress, M. E. A summary of 103 autopsies on leprosy patients on the Isthmus of Panama. Internat. J. Leprosy **10** (1942) 51-59.
13. Kusaka, T. Alterations in the lipid content of the blood and tissues in leprosy patients. La Lepro **27** (1958) 228-232.
14. Lewis, R. A. and Edwards, R. A statistical study of 1,000 cases of leprosy observed in Rangoon for approximately two years. Internat. J. Leprosy **26** (1958) 236-251.
15. Llombart, A. and Alcacer, F. La siderosis hepática del enfermo de lepra. Mem. VI Congr. Internac. Leprol., Madrid, 1953; Madrid, 1954, p. 730-742.
16. Masanti, J. G. Nefropatía amiloidea: insuficiencia renal e hipertensión asociadas con amiloidosis renal secundaria. Medicina (Buenos Aires) **18** (1958) 61-70.
17. Masanti, J. G. Amiloidosis secundaria en la lepra; reacción leprosa y amiloidosis. Medicina (Buenos Aires) **19** (1959) 1-10.
18. Masanti, J. G. Curación de la amiloidosis secundaria de los enfermos de lepra. (In preparation.)
19. Masanti, J. G. and Melamed, M. (Unpublished data.)
20. Okada, S. Pathological studies on the changes in the livers of leprosy patients and murine leprosy rats by means of biopsy. La Lepro **24** (1955) 9-17 (supplement).
21. Patalano, A. and Masanti, J. G. La nefropatía postamiloidea. (In preparation.)
22. Patalano, A., Marenzi, A. and Masanti, J. G. (Unpublished data.)
23. Schujman, S. Influencia benéfica de la reacción leprosa en la evolución de los casos lepromatosos. Rev. argentina Dermatosif. **31** (1947) 506-512.
24. Shuttleworth, J. S. and Ross, Sr. H. Secondary amyloidosis in leprosy. Ann. Int. Med. **45** (1956) 23-38.
25. Souza Campos, N. and de Souza, P. R. Rectional states in leprosy. Lepra reaction, tuberculoid reactivation (tuberculoid lepra reaction), reactional tuberculoid leprosy, borderline (limitantes) lesions. Internat. J. Leprosy **22** (1954) 259-272.
26. Tarabini C, G. Breves consideraciones sobre la patogenia de los transtornos lipoideos en la enfermedad de Hansen. Rev. Leprol. Fontilles **4** (1958) 363-364.