MODERN CONCEPTS OF LEPROSY

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TO JEANNE

PREFACE

It is hoped that dermatologists and others may find this monograph a happy medium between the brief chapters on leprosy to be found in most general textbooks, and (for them) the unnecessarily detailed accounts found in books devoted exclusively to leprosy. It is written for them, and for students in their field, and not for leprologists. I am not a leprologist myself, but a student and practitioner of dermatology who has had the opportunity to see a good many cases of leprosy and to follow some of them over a period of years. I hope that what my background lacks in experience with leprosy will be balanced to some extent by what it gains—for the reader—from the dermatologic point of view. Where this work is too brief or too superficial, it should be supplemented by reference to the practical literature or the texts of Cochrane, Chaussinand, Rogers and Muir, and others. It is not intended to supplant any of these.

I wish to acknowledge my indebtedness to the students of leprosy enumerated in the bibliography, from whose writings I have freely borrowed, and in particular to those who have stimulated my interest and added to my knowledge by personal contact: H. W. Wade, Fernando Latapí, José M. M. Fernandez, Vicente Pardo-Castelló, Robert G. Cochrane, F. E. Rabello, Jr., Norman R. Sloan, W. Lloyd Aycock, Edwin K. Chung-Hoon, N. E. Wayson, and my associates, E. A. Fennel and Irvin L. Tilden, of the Pathology Department of the Straub Clinic. It is to Dr. Tilden that I am indebted, too, for the photomicrographs and some of the other photographs, as well as for specific suggestions in the preparation of the manuscript, in which Drs. Fennel, Sloan, Aycock and Wade were also most helpful.

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MODERN CONCEPTS OF LEPROSY

Chapter One

INTRODUCTION

LEPROSY is at once one of the most ancient, one of the most chronic and intractable, one of the most feared, and one of the most puzzling, of all the chronic infectious diseases.

For 15 centuries or more, leprosy was generally regarded as a highly contagious disease; it was one of the first diseases to be so regarded. Yet students of the disease have known for a century past that it actually behaves as if it were only very slightly contagious, in most circumstances; indeed, vigorous arguments were being waged half a century ago as to whether it was contagious at all, or merely hereditary. And the late great Sir Jonathan Hutchinson wrote a book to prove that it was caused solely by eating imperfectly cured fish, and died under the shadow of this thoroughly discredited conviction. Now, in 1952, the degree of its contagiousness is still a moot question.

Leprosy is beclouded with other misapprehensions. Most textbooks describe it as manifesting itself in two forms, leprosy of skin and leprosy of nerves. Yet ever since Armauer Hansen, leprosy workers have been aware that each of the two forms of the disease almost always attacks both skin and nerves.

There are few bacterial diseases in which the presumably causative organism is as easy to find as it is in the lepromatous form of leprosy; yet there are few in which it is as difficult to find as it is in the tuberculoid form (except during reactions).

Many physicians are under the impression—and many textbooks state—that loss of pinprick sensitivity is an early sign of leprosy; actually it is in many cases a relatively late sign, appearing only after other manifestations of the disease have been present for several weeks or months.

It is often stated that the finding of acid-fast bacilli in scrapings or smears from the nasal septum is diagnostic of leprosy—which it is not—and one often encounters the implication that their absence from such preparations rules against a diagnosis of leprosy, which is equally untrue.

It is not generally known that treatment with chaulmoogra oil and its esters was abandoned in some leprosaria two decades or more ago, as being ineffective. Armauer Hansen himself so regarded it, as long ago as 1902, though its efficacy in the hands of some leprosy workers cannot be denied. Neither is it generally known that for the past decade, relatively effective treatment has been available in the form of the sulfone group of drugs (see Chapter 8).

Finally, there is a widespread impression that leprosy is an extremely rare disease, so rare that it is of no great importance. It is not rare; it is uncommon, in many areas, but not so rare that it can be ignored. There are perhaps five million persons in the world who are afflicted with it; and though it is true that the great majority of these live outside the U.S.A., our country still contains a good many unrecognized cases.

The availability of effective treatment imposes a new and grave responsibility upon physicians in general and skin specialists in particular to be sufficiently alert and well informed to make the diagnosis of leprosy, and make it early, before the disease is so far advanced that it will take years of treatment to eradicate it. It is primarily to this end that this monograph is dedicated.

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Chapter Two

ETIOLOGY

THE CAUSATIVE organism of leprosy is Mycobacterium leprae, an acid-fast bacillus which was described by Dr. Gerhard Armauer Hansen in 1874. It has never been possible to prove its relationship to the disease by fulfilling all of Koch's postulates, because the organism has never been successfully grown on artificial media, or successfully inoculated into animals or human volunteers. The several reports of successful cultivation have all failed of confirmation. Still, it is found invariably and in enormous numbers in every lepromatous case of leprosy, and in small numbers in a great many cases of the tuberculoid variety of the disease; and no serious doubt is entertained of its being the actual cause of leprosy.

The organism is usually seen in smears or sections as a straight or slightly curved acid-fast rod about 3 to 5 micra long and 0.2 to 0.4 micra thick. When many bacilli occur together they tend to be aggregated into packets (traditionally likened to "bundles of cigars"), and in much larger aggregates, with spheroid contours, known as globi (Fig. 1). It is probable that the latter represent masses of bacilli which have been ingested by (and have multiplied within?) phagocytic reticuloendothelial cells, in the course of the generalized foreign-body-reaction-like process which characterizes lepromatous leprosy and accounts for a great many of its curious and characteristic clinical features.

Scores of attempts to transmit leprosy to human volunteers, by inoculating them with fresh, presumably infective material, have almost uniformly failed. The only noteworthy exception, an inoculation of lepromatous tissue

made by Edward Arning into the skin of a Hawaiian named Keanu, seems to have been successful. Its importance has been questioned because Keanu is known to have had leprous relatives, and therefore might have been infected by the usual routes; nevertheless, the fact that he developed a leproma at the site of inoculation suggests that the experiment was successful.

The simultaneous occurrence of tuberculoid leprosy in two tattoos in 1943 received by two young American Marines from Michigan (a wholly nonendemic area) on the same day and in the same shop, in Australia, during World War II, seems to constitute two more examples of successful infection of humans by inoculation.

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Chapter Three

EPIDEMIOLOGY

THE EPIDEMIOLOGY of leprosy is a challenging puzzle, full of curious contradictions and paradoxes. Historically, as we have said, leprosy has been regarded on the one hand as highly contagious, and on the other hand as purely hereditary. Perhaps the truth lies, as it so often does, somewhere between the two extremes. The uncertain and probably highly variable incubation period makes epidemiologic studies particularly hard to interpret. It probably varies from as little as a year or two (in infants chiefly) to five or 10 years.

AGE

Children have long been regarded as much more susceptible to leprosy than adults. Cochrane's figures for 2,000 cases seen in a seven year period at Chingleput, in Madras, indicate that about a third of new cases occur before the age of 15, and that the attack rate drops sharply above the age of 24. Lara at Culion in the Philippines found 200 cases of leprosy among 770 children between the ages of one and five, living with their leprous parents. Chung-Hoon's figures for 316 new cases in Hawaii indicate that about a third have their first reported symptoms before the age of 20.

But as Lloyd Aycock of Harvard points out, many infectious diseases are comparatively prevalent among children, and the high incidence of leprosy among children may be in considerable part a result of their having been exposed within their families, or in scuffling or wrestling with schoolmates (Wayson), before they became adults. Lara

notes that no case has yet been seen at Culion among some 600 children of non-leprous employees living in close proximity to the leprosarium. And Chung-Hoon found about a third of new cases beginning after the age of 40. Certainly children are susceptible—but adults are susceptible too. There are many instances of leprosy in individuals clearly exposed for the first time in adult life, though such an occurrence is rare, for some reason, among attendants in leprosaria. It is not certain that age is a major factor in the epidemiology of leprosy.

SEX

Leprosy workers the world over have observed that in general, leprosy is twice as common among males as among females. There are certain curious and perhaps significant exceptions to this remarkably uniform preponderance, however. Hopkins and Faget found no such disparity in Negro admissions to the United States Marine Hospital at Carville, of whom, in a studied period, there were 27 women and 25 men. Rodriguez and Lara, in the Philippines, observed that among children the sexes are attacked equally. Cochrane found that among 5,000 persons examined in Madras, the ratio of males to females was 51:32 (per 1,000) among the adults, and only 77:68 (per 1,000) among the children. Chung-Hoon's figures on 145 newly diagnosed cases of leprosy seen in Hawaii during the period 1945-1949 show a male-female ratio of approximately 2:1 among the lepromatous cases, but only 1.1:1 among those of tuberculoid type.

RACE

It is virtually impossible to evaluate racial susceptibility to leprosy because it can hardly be separated from such other factors as diet, opportunity for contagion, individual inheritance of susceptibility, and so forth. Still, racial varia-

tions in incidence are so striking that they deserve mention, whatever the basis for them may be. They are even more remarkable when one considers race in relation to type of leprosy; for example, Cochrane has reported that lepromatous eye lesions were three times as common among Anglo-Indians, in Madras, as among Indians, and laryngeal lesions occurred 30 times as often in the former group as in the latter. Muir has suggested that this sort of disparity may result from the circumstances under which infection occurred, that is, the amount and site of entry of the inoculum, and other factors.

Chung-Hoon has summarized the average morbidity rate in Hawaii by races for a 10-year period, in 316 cases. It ranged from 70.2 (per 100,000 per year) for the Hawaiian group, to 1.1 for the Caucasian (a single case). Part-Hawaiians showed a rate of 13.9 (27 cases), Filipinos 13.6 (20 cases), Chinese 4.3 (eight cases) and Japanese 2.6 (six cases).

OPPORTUNITY FOR CONTAGION

This too is a difficult factor to evaluate in such a disease as leprosy. For example, leprosy workers are not unanimously agreed on the question of whether a bacteriologically negative case—that is, one in which no acid-fast bacilli are found in one or more skin lesions or in nasal scrapings—can transmit the disease to others. In general, it is believed that the danger of transmission of infection by such a case is extremely slight, and in most countries and states, such patients are not isolated.*

The exact risk of contact with a bacteriologically positive case—an "open" case—is not known, though we know from Doull's survey in Cebu, and other evidence, that it is many times greater than that of contact with a negative one. Conjugal infection, of one spouse by the other, is notoriously infrequent. Leprosarium workers are infected only

* See Chapter Nine.

very rarely. And yet the relatively high incidence of leprosy in exposed children, described by Cochrane, Lara, and many others, contrasts sharply with the low incidence of leprosy in children removed at birth from leprous parents, as observed in India, Hawaii, and elsewhere.

Cochrane states that in children with multiple lesions, household contact has occurred in nearly 40 per cent of cases; whereas in children with only one or two lesions, it has occurred in less than 20 per cent. He further shows, in an analysis of over 300 cases in which known contact had occurred, that the ratio of room contact to mere house contact was 3:1 for "neural" (chiefly tuberculoid?) cases and over 20:1 for lepromatous cases, suggesting that intimate contact with the latter is relatively dangerous. Lampe and Boenjamin in Indonesia came to exactly the same conclusion.

It is odd, at least on the face of it, that of Chung-Hoon's series of cases already referred to, approximately 60 per cent denied any knowledge of contact with the disease. Moiser, in Africa, reports exactly the same observation. Doull and his associates, in Cebu, obtained no history of contact in 62 per cent of cases, and Lampe and Boenjamin, in Indonesia, in 31.4 per cent. Some of these denials must be discounted; still, fear of leprosy and of the consequences of being found to have it have long been at a relatively low level in Hawaii, especially during the past five years or so, since the availability of effective treatment has been publicized. Most patients, if not all, give their correct names, and there is excellent cooperation with the investigating authorities, who go about their work in an atmosphere of friendliness and helpfulness. Presumably, in some cases, contact with the disease is denied merely because the patient was unaware of the existence of infection in the individual who infected him.

Still, in summary, we must admit that this figure of 60

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