CONTINUING MEDICAL EDUCATION

TRANSMISSION OF LEPROSY–A REVIEW

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It is generally considered that man is the only reservoir of *Mycobacterium leprae* and that leprosy is transmitted only by leprosy patients. In recent years, there have been a number of reports suggesting the possibility of leprosy-like disease in sub-human primates. Walsh et al,[1,2] Smith et al[3] and Meyers et al[4] have reported the occurrence of lepromatoid type of leprosy in wild armadillos. Similarly, naturally acquired leprosy has been reported in chimpanzee[5] and in mangabay monkey.[6] The evidence available so far, suggests that these animals are occasionally susceptible to leprosy, but their epidemiological significance is not known.[7] Field studies carried out in India[8] have shown that atleast monkeys do not seem to play any role in transmission of the disease.

It is thus, generally accepted that leprosy is spread by leprosy patients. Among the patients, it has been considered that lepromatous cases are the main source of infection. On the basis of this, it has been difficult to explain the continued occurrence of new cases in areas where more than 90% of the cases are non-lepromatous. Careful studies conducted in Phillipines[9] have shown that when the primary i.e. the index case was lepromatous, the attack rate for contacts was equivalent to 6.2 cases/1000 persons/year. When the index case was tuberculoid, the incidence was 1.6/1000 persons/year, while the corresponding figure for non-household contact was 0.8/1000 persons/year. Thus, the estimated risk was 4 times more in close contacts of lepromatous cases than that of tuberculoid cases and risk for contacts of tuberculoid index cases was twice that of non-contacts. A 15-year follow-up of the same population group has been carried out subsequently. The observations mentioned earlier have been reconfirmed.[10]

Epidemiological studies carried out in an hyperendemic area (South India) have also shown that the attack rate for non-household contact was 1.85 as against 6.78 for non-lepromatous cases and 17.65 per 1000 per year for contacts of lepromatous patients.[11]

Workers in South Africa[12] and from Uganda[13] believe and have shown that in large parts of Africa, where leprosy is hyperendemic, lepromatous cases are too few and too scattered to account for the very high prevalence of the disease. As more than 90% of the cases in Africa are non-lepromatous, near tuberculoid, in the opinion of the above authors, tuberculoid cases must be mainly responsible for spread of the disease. A theoretical and simplified explanation may be that because of greater total exposure, the less infectious but far more numerous tuberculoid cases could be responsible for more infections than the lepromatous cases.

It is a common knowledge that smears from tuberculoid cases are generally negative. A careful search of histopathological sections may occasionally reveal a few bacilli in the nerves i.e. deep down. The bacilli are thus unlikely to escape from the surface. During reactive phases, these patients may become smear positive and the lesions may ulcerate,

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thus becoming undoubtedly infectious. Therefore, the part played by the tuberculoid cases in the spread of the disease may not depend only on their total number or the prevalence of tuberculoid leprosy but also on the frequency, duration and severity of these exacerbations that occur in tuberculoid cases.

There is almost universal agreement on the infectivity of untreated lepromatous cases. Before a person develops overt clinical manifestations, his having already spread the disease to the community is to be expected. However, there appears to be a small proportion of healthy population in the hyperendemic areas who have been shown to be smear positive without having any symptoms or signs of leprosy. In some of the studies undertaken in India,[14-16] it has been shown that 20-24% of the healthy familial contacts of lepromatous cases have AFB in the ears, in contrast to 2 to 2.5% of the healthy contacts of tuberculoid cases and none of the controls from non-leprosy area (Punjab). Thus, some information is available on the occurrence of carrier state in leprosy. The epidemiological significance of these cases is not known.

It is generally believed as shown by Rees and Valentine[17] and Shepard and McRae[18] that only the solidly stained bacteria are viable, while the non-uniformly stained ones are non-infective to mice. As a result of these, infectiousness of patients is considered not to be determined by his bacterial load or BI, but instead by MI. Even if a patient has a high BI, he is assumed to be non-infectious if his MI is zero. The experience and impression of workers in India however, is not in full agreement. Work done at Chingleput[19] and at JALMA (unpublished observations) where mouse footpad facilities are available, has shown positive takes in mice despite MI being zero, suggesting that at least some of the non-solid forms are also viable. Based on this, the Indian leprologists caution about labelling all the cases with MI zero to be non-infectious.

Discharge of bacilli

Based on the work of Muir and Chatterjee[20] and Weiner,[21] it is generally considered that the portal of exist of *M. leprae* from the human body is the skin. A confirmation to this fact is easily obtained if one takes impression smears of ulcerating lepromatous lesions. However, in recent years, it has been realized that the discharge of *M. leprae* is much more from the mucous membranes than trans-epidermal. Pedley,[22,23] using a method called 'composite skin smear technique', has shown that in reality in diffuse lepromatous patients the discharge of *M. leprae* through the skin is not significant. By taking smears of the nasal mucus, Pedley[24] has shown discharge of an enormous number of bacilli from the intact mucous membrane. In one case, 1 ml of the nasal mucus (blown out at one time) contained as high a count as 20 million bacilli. A 24-hour collection of nose-blow from another patient gave a count of 380 million bacili. The results have since been confirmed,[25] and presently, nose-blow examination is taken as a good parameter of the state of infectivity of the patients. Work done at Agra[26] has shown that even during quite respiration, a proportion of lepromatous cases throw out a significant number of *M. leprae* into the environment with the exhaled air (Table I).

We in Agra have been looking at the oral lesions and the discharge of bacilli from the mouth. In these studies,[27-29] oral mucosal affection was seen in a large proportion of the cases, and bacilli were present in the mouths of as many as 85% of lepromatous cases (Table II). The mouth-wash positivity for *M. leprae* was higher in those with oral mucosal lesions (91%) as compared to those without lesions (76.4%). The positivity appears to increase with the duration of the disease. The quantum of bacillary discharge per mouth-wash varied from 2.6×10^4 to 2.9×10^7 with a mean of

Table I. Bacilli exhaled in nasal breath and bacteriological indices of bacilli in skin smears.

	Number of patients	Skin smears	Bacilli exhaled
		Mean BI±SD	Mean number per breath
Group I (Untreated)	8	2.1±0.69	3.8×10^4 (2.25×10^3—1.1×10^5)
Group II (On treatment upto 1 month)	6	2.0±0.4	2.9×10^4 (2.25×10^3—7.1×10^4)
Group II (On treatment upto 3 months)	6	2.0±0.5	2.8×10^4 (4.5×10^3—1.3×10^5)

Table II. Discharge of *Mycobacterium leprae* from the mouth in lepromatous leprosy patients.

Groups	Number of patients	By smear		By mouth wash		Positive by either method	Negative by both methods
		Positive	Negative	Positive	Negative		
Patients with oral mucosal lesions	23(57.5%)	19 (82.6%)	4	17 (73.9%)	6	21 (91%)	2 (6.1%)
Patients with no oral lesions	17 (42.5%)	7 (41.2%)	10	10 (58.8%)	7	13 (76.4%)	4 (23.5%)
Total	40	26 (65%)	14	27 (67.5%)	13	34 (85%)	6 (15%)

Table III. Quantum of bacillary discharge per mouth wash.

Number of bacilli recovered per mouth wash	Number of patients
More than 10^6	4(10%)
10^5-10^6	15(37.5%)
Less than 10^5	8(20%)
Nil	13(32.5%)

Mean 1.6—10^6

1.6×10^6 (Table III). These findings thus show that discharge from the mouth, like the nose, contains a large number of acid fast bacilli in a significant percentage of the lepromatous cases. Further, the unhygienic habit of indiscriminate spitting, especially while chewing tobacco, betel leaves or betel nuts enhances the chances of bacillary dissemination.

From above, it is clear that enormous quantities of *M. leprae* are discharged from the nasal and the oral mucosa in lepromatous cases. As the aerosol discharge of *M. leprae* from the nose and the mouth appears to be far more as compared to that from the skin, its epidemiological significance has to be borne in mind.

Table IV. Discharge of *M. leprae* in the milk of leprosy patients.

Groups	Treatment status	Number of patients	Mean duration of disease in years	Skin smear BI (mean)	Number positive for AFB	Mean count
Tuberculoid	Untreated	10	2.0	0	1	4.3×10^4
	Treated	3	2.1	0	0	0
Borderline	Untreated	5	3.6	0.4	1	4.3×10^4
	Treated	3	6.3	0	0	0
Lepromatous	Untreated	14	4.7	2.32	9	15.3×10^4
	Treated	3	10.6	0.75	1	4.3×10^4

For breast-fed infants, in addition to their being exposed to the skin and the nose-derived bacteria, mother's milk is to be suspected as an additional source. Studies carried out by Pedley[30] and ourselves at Agra[31] have confirmed the presence of bacilli in significant numbers in the milk of untreated lepromatous women (Table IV). In 10 ml of milk from untreated lepromatous women, *M. leprae* count was found to range from 4.3×10^4 to 4.3×10^5. This indicates that in 300 to 500 ml of milk which a newborn usually takes, on an average 3.7 to 6.2×10^6 bacilli are going into the alimentary tract. The significance of such a massive daily dose of ingestion of bacteria is a matter for concern.

Lastly, though occasional *M. leprae* have been demonstrated in other excretions as well, the role played by any of these routes is definitely insignificant.

Once the bacilli are shed into the environment, these can directly infect the healthy population or can settle on fomites and subsequently get transferred to the individuals. Studies carried out in this regard in Britain[25] and Agra (India)[32] have shown that *M. leprae* can remain viable in both the temperate and the tropical conditions for as long as 7 to 9 days. This suggests that bacilli deposited on an inert surface continue to remain infectious for a week or more. However, a very low MI in untreated cases (which is usually less than 5%) indicates that as such, a very small proportion of the discharged bacilli are likely to be infective. Therefore, how significant is survival of the discharged live bacilli in the environment is to be seen.

Portals of entry

It is commonly believed that bacilli usually enter the body through wounds in the skin or following prolonged skin to skin contact. For this, it is essential to have a break in the continuity of skin secondary to microtrauma, skin infections and/or infestations. In support of this, scores of reports of solitary lesions often occurring on the exposed areas, and inoculation leprosy are available in the literature.[33-36] It appears logical to assume that these single lesions or inoculation lesprosy lesions constitute the actual sites of inoculation through the skin. Studiest done in Burma,[37] Cebu[38] and India[39] have already indicated that among those with a single lesion, exposed areas of the limbs and buttocks are involved far more frequently than the covered sites. It has been argued that if the portal of entry in leprosy was purely cutaneous, legs, feet, upper extremities and face should surely show more frequent affection than buttocks and thighs which normally are covered sites. This does not seem to be true in the developing countries.

However, despite such a strong circumstantial evidence, a case has been put forth that skin is not the usual, much less the only porta

of entry of *M. leprae*. Demonstration of *M. leprae* in sites, far distant from the solitary lesion, as has been shown by Weddell et al[40] has been taken to mean that *M. leprae* must primarily disseminate via the blood stream. It has been pointed out in the above study and by Newell[41] that the initial or the solitary skin lesions are possible even with systemic infection, the localization to a single site being precipitated by microtrauma.

The enormous discharge of *M. leprae* from the nasal and the oral mucosa suggests that the organisms may be disseminatd by aerosols. In tuberculosis and leprosy, epidemiological studies done in India,[42,43] have shown that the discharge of organisms in the sputum of open tuberculosis patients and nasal secretions in lepromatous patients is similar. The attack (disease) rates in the families and contacts are like-wise similar for both the diseases. The authors suggested by anology, that the route of infection are also likely to be the same for both the diseases. Further, Rees and McDougall[44] have shown that atleast in mice, the infection can take place by aerosols.

Coming to the clinical situation, patients with early lepromatous leprosy commonly have nasal symptoms and the commonest site of lepromatous infiltration in the nose is the anterior aspect of the inferior turbinate—a site which is first exposed to the inhaled air.[45] Does this suggest that nose is the portal of entry or does nasal (inferior turbinate) affection follows dissemination of the disease ? At the moment it is not clear. Recent studies from Karigiri, South India[46] have shown that in nferior turbinate biopsies from healthy contacts of lepromatous patients, nerve proliferation is common feature and very often one can find ranulomas in these areas. In fact in several istological sections, an indeterminate leprosy vas diagnosed. In contrast, in a study on nasal iopsies done in healthy contacts of lepromatous atients from low endemic area around Agra, one of the 57 cases showed any definite granuloma in the nose. In 16 cases however, small collections of plasma cells and lymphocytes were found in the histology specimens. No specific evidence of nerve involvement was found.[47] The work therefore needs confirmation in a larger number of carefully examined contacts from a hyperendemic situation.

In this regard epidemiological findings from Netherlands New Guinea[48,49] are important. In Netherland, where the community is not insusceptible to leprosy, even though there were 200 immigrants with lepromatous leprosy who had been moving around freely, only one indigenous case developed—an indicator against air-borne affection.

It has been pointed out earlier, that breast-fed infant of a lepromatous mother can ingest millions of bacilli in a day. The transport of *M. leprae* by flies can easily carry them through contaminated food into the intestinal tract. As in tuberculosis, intestinal mucosa, without showing any pathological lesions can serve as a point of entry for *M. leprae*. It appears unlikely, as in autopsy studies *M. leprae* were conspicuous by their absence in mesentric glands.[50]

In a recent world-wide compilation of leprosy in infants, only 11 cases of proved leprosy have been found in infants less than one year of age,[51] of these 2 were from JALMA.[52] This suggests that, the incubation period could be short, though and possibility of transplacental spread could not be ruled out. Duncan et al[53] from Ethiopia have studied serially the levels of IgA and IgM antibodies against *M. leprae* specific antigen (antigen 7). The authors have reported higher blood levels of IgA and IgM at birth, with gradual increase in the post-natal period in those who developed leprosy later, thus suggesting the possibility of intrauterine infection. Levels of IgG as expected, declined gradually after birth. Added to this, there are reports, in the non-English literature, indicating the presence of *M. leprae* in cord-blood and placenta. On

the whole, an extremely uncommon occurrence of leprosy in the infants suggests unlikelihood of transplacental transmission of *M. leprae*.

Bacteraemia in untreated lepromatous leprosy is now an accepted fact.[54-56] Likewise, blood vessel affection in leprosy is also comonly seen.[57,58] Therefore, it has been suggested that biting or blood-sucking insects like bed bugs, lice, acarinae of scabies and mosquitoes may be involved in the transmission of leprosy.[59,60] Four or more times higher attack rates in the house-hold contacts of lepromatous cases could be explained on the basis of trasmission of the disease by house-hold and house-bound or non-flying insects, all of which are extremely common in all areas where leprosy is highly endemic. Acid fast bacilli isolated from the mosquitoes have been identified as *M. leprae* using mouse foot-pad test.[61] Usually, pathogens carried by the vectors go through a cycle to become infectious as in malarial parasite or they live and multiply in vectors, like rickettsia or viruses. Such a thing does not happen for *M. leprae* as has been concluded from very extensive studies carried out in Pondicherry, South India.[62] These insects can however, serve as a vehicle as has been shown for the house fly.[63] The role played by these vectors in such a mechanical transfer of *M. leprae* does not appear to be very great, as only a small proportion of these insects inoculated in the mouse foot-pad have shown positive takes.

To conclude, in the recent years, definite evidence has been put forth to indicate that a significant discharge of *M. leprae* occurs from the nose and the mouth of lepromatous patients. Likewise, the bacillary excretion in milk of lactating lepromatous women is also significant in addition to the exit through the skin. On the other hand, some evidence has been forth-coming that extracutaneous route of infection is more important. No conclusive results are yet available to indicate that leprosy is trans-mitted by droplet infection. Therefore, a great discretion is needed in publicizing such a possi-bility. Suggestions of this nature or arthropod borne spread can have far reaching conse-quences in the community. Implicit in it is the threat that restrictive measures would have to be reintroduced for the control of leprosy. If such a thing is done, it is likely to profoundly affect the existing basis for leprosy control eradication programmes which rely on co-operation of the patients in the early stages of infection. Not only this, there will be an increased fear of leprosy in the community resulting in ostracism and hatred towards those suffering from this disease.

References

1. Walsh GP, Storrs EE, Burchfield HP et al : Leprosy-like disease occurring naturally in armadillos, J Reticuloendothel Soc, 1975; 18 : 347-351.
2. Walsh GP, Storrs EE, Meyers WM et al : Naturally acquired leprosy-like disease in the nine-banded armadillo (*Dasypus novemcinctus*) : Recent epizootio-logic findings, J Reticuloendothel Soc, 1977; 22 : 363-367.
3. Smith JH, File SK, Nagy BA et al : Leprosy-like disease of wild armadillos in French Acadiana, Louisiana, J Reticuloendothel Soc, 1978; 24 : 705-719.
4. Meyers WM,Walsh GP, Brown HL et al : Naturally-acquired leprosy-like disease in the nine-banded armadillo (*Dasypus novemcinctus*) : Reactions in leprosy patients to lepromins prepared from naturally infected armadillos, J Reticuloendothel Soc, 1977; 22 : 369-375.
5. Donham KG and Leininger JR : Spontaneous leprosy-like disease in a chimpanzee, J Inf Diseases, 1977; 136 : 132-136.
6. Meyers WM,Walsh GP, Brown HL et al : Naturally-acquired leprosy in a mangabey monkey (*Cercocebus* sp.), Intern J Leprosy, 1980; 48 : 495-496.
7. Filice G : Lack of observed association between armadillo contact and leprosy in humans, Amer J Trop Med Hyg, 1977; 26 : 137-142.
8. Hagstad HV : Leprosy in subhuman primates : Potential risk for transfer of *Mycobacterium leprae* to humans, Leprosy Rev, 1983; 54 : 353-356.

9. Doull JA, Gunito RS, Rodriguez JN et al : The incidence of leprosy in Cordova and Talisay, Cebu, Intern J Leprosy, 1942; 10 : 107-131.

10. Gunito RS, Rodriguez JN, Doull JA et al : The trend of leprosy in Cordova and Talisay, Cebu Province, Phillipines, Intern J Leprosy, 1954; 22 : 409-430.

11. Noordeen SK : Infectivity of leprosy, in : Window on Leprosy, editor Chatterjee BR : Gandhi Memorial Leprosy Foundation, Wardha, 1978; p 59-63.

12. Davison AR : The infectivity of neural leprosy, Intern J Leprosy, 1949; 17 : 247-252.

13. Kinner Brown JAK : The incidence and epidemiology of leprosy in Uganda, Trans Roy Soc Trop Med Hyg, 1955; 19 : 241-252.

14. Desai SD : Symposium : Spontaneous disappearance of skin lesions : Positive smears without lesions, Intern J Leprosy, 1955; 23 : 198-200.

15. Taylor CE, Elliston EP and Gideon H : Asymptomatic infections in leprosy, Intern J Leprosy, 1965; 33 : 716-727.

16. Chatterjee BR : Carrier state in leprosy, Leprosy India, 1976; 48 : 634-644.

17. Rees RJW and Valentine RC : The appearance of dead leprosy bacilli by light and electron microscopy, Intern J Leprosy, 1962; 30 : 1-9.

18. Shepard CC and McRae DH : *Mycobacterium leprae* in mice : minimal infectious dose, relationship between staining quality and infectivity and effect of cortisone, J Bacteriol, 1965; 89 : 365-370.

19. Desikan KV : Correlation of morphology with viability of *M. leprae*, Leprosy India, 1976; 48 : 391-397.

20. Muir E and Chatterjee SN : The infection of stratified epithelium in leprosy, Ind J Med Res, 1932; 19 : 1163-1165.

21. Weiner MA : Leprosy, report of a case with rare histopathological feature, Arch Dermatol, 1959; 79 : 709-711.

22. Pedley JC : Composite skin contact smears : a method of demonstrating the non-emergence of *M. leprae* from intact lepromatous skin, Leprosy Rev, 1970; 41 : 31-43.

23. Pedley JC : Summary of the results of a search of the skin surface for *M. leprae*, Leprosy Rev, 1970; 41 : 167-168.

24. Pedley JC : The nasal smears in leprosy, Leprosy Rev, 1973; 44 : 33-35.

25. Davey TF and Rees RJW : The nasal discharge in leprosy : Clinical and bacteriological aspects, Leprosy Rev, 1974; 45 : 121-134.

26. Green CA, Katoch VM and Desikan KV : Quantitative estimation of *Mycobacterium leprae* in exhaled nasal breath, Leprosy Rev, 1983; 54 : 337-340.

27. Girdhar BK and Desikan KV : A clinical study of mouth in untreated lepromatous patients, Leprosy Rev, 1979; 50 : 25-35.

28. Hubscher S, Girdhar BK and Desikan KV : Discharge of *Mycobacterium leprae* from the mouth in lepromatous leprosy patients, Leprosy Rev, 1979; 50 : 45-50.

29. Mukherjee A, Girdhar BK and Desikan KV : The histopathology of tongue lesions in leprosy, Leprosy Rev, 1979; 50 : 37-43.

30. Pedley JC : Presence of *M. leprae* in human milk, Leprosy Rev, 1967; 38 : 239.

31. Girdhar A, Girdhar BK, Ramu G et al : Discharge of *M. leprae* in the milk of leprosy patients, Leprosy India, 1981; 53 : 390-394.

32. Desikan KV : Viability of *M. leprae* outside the human body, Leprosy Rev, 1977; 48 : 231-235.

33. Nolasco JO and Lara CB : Histopathological study of an early case of leprosy in a young child of lepromatous patient, Intern J Leprosy, 1941; 9 : 181-192.

34. Rogers L and Muir E : Leprosy, 3rd ed, Williams and Wilkins Co, Baltimore, 1946.

35. Porritt RJ and Olsen RS : The simultaneous cases of leprosy developing in tattooes, Amer J Pathol, 1947; 23 : 805-807.

36. Lara CB and Nolasco JO : Self-healing or abortive and residual forms of childhood leprosy and their probable significance, Intern J Leprosy, 1956; 24 : 245-263.

37. Bachelli LM, Garbajosa PG, MgMg G et al : Site of early skin lesions in children with leprosy, Bull WHO, 1973; 48 : 107-111.

38. Doull JA, Rodriguez JN, Guinto RS et al : A field study of leprosy in Cebu, Intern J Leprosy, 1936; 4: 141-170.

39. Ganapati R, Naik SS and Pandya SS : Leprosy among school children in Greater Bombay : Clinical features, Leprosy Rev, 1976; 47 : 133-140.

40. Weddell AGM, Palmer E, Rees RJW et al : Experimental observations related to the histopathology of leprosy, in ; Pathogenesis of Leprosy, Ciba Foundation Study Group No 15, Little Brown and Co, Boston, 1963 ; p 3-15.

41. Newell KW : An epidemiologists view of leprosy, WHO/PA/43.64, World Health Organisation, 1964.

42. Rees RJW and Meade TW : Comparison of modes of spread and the incidence of tuberculosis and leprosy, Lancet, 1974; I : 47-49.

43. Meade TW : Growing points in leprosy research, (2) Epidemiology, Leprosy Rev, 1976; 45 : 15-21.

44. Rees RJW and McDougall AC : Airborne infection with *Mycobacterium leprae* in mice, J Med Microbiol, 1977; 10 : 63-68.

45. Barton RPE : A clinical study of nose in lepromatous leprosy, Leprosy Rev, 1974; 45 : 135-144.

46. Chacko CJG, Mathews M, Jesudasan K et al : Primary leprosy involvement of nasal mucosa in apparently healthy house-hold contacts of leprosy patients, Paper presented at XI Biennial Conference of Indian Association of Leprologists, Madras, April 5-8; 1979; Abrst. No. 63.

47. Ramu G, Malaviya GN, Bharadwaj VP et al : Studies on healthy contacts of leprosy patients, XII International Leprosy Congress, New Delhi, Feb. 20-25, 1984; Abrst. No. 369.

48. Leikar DL : Epidemiological and immunological surveys in Netherlands New Guinea, Leprosy Rev, 1960; 31 : 241.

49. Leiker DL : On the mode of transmission of *Mycobacterium leprae*, Leprosy Rev, 1977; 48 : 9-16.

50. Desikan KV and Job CK : A review of postmortem findings in 37 cases of leprosy, Intern J Leprosy, 1968; 36 : 31-34.

51. Brubaker ML and Meyers WM : Leprosy in children under one year of age, XII International Leprosy Congress, New Delhi, Feb. 20-25, 1984; Abrst. No. 1 .

52. Girdhar BK, Girdhar A, Ramu G et al : Borderline leprosy (BL) in an infant—report of a case and a brief review, Leprosy India, 1983; 55 : 333-337.

53. Duncan ME, Melsom R, Pearson JMH et al:A clinical and immunological study of four babies of mothers with lepromatous leprosy, two of whom developed leprosy in infancy, Intern J Leprosy, 1983; 51 : 7-17.

54. Drutz DN, Chen TSN and Lu WH : Continuous bacteraemia of lepromatous leprosy, New Eng J Med, 1972; 287 : 159.

55. Padma MN and Desikan KV : Bacillaemia in leprosy, Ind J Med Res, 1975; 73 : 888.

56. Sreevatsa, Sengupta U, Ramu G et al : Evaluation of bacteraemia in leprosy patients, Leprosy India, 1978; 50 : 381-387.

57. Mukherjee A, Girdhar BK and Desikan KV : Leprous phlebitis, Intern J Leprosy, 1980; 48 : 48-50.

58. Mukherjee A, Girdhar BK, Malviya GN et al : Involvement of subcutaneous veins in lepromatous leprosy, Intern J Leprosy, 1983; 51 : 1-6.

59. Dungel N : Is leprosy transmitted by insects ? Leprosy Rev, 1960; 31 : 25-34.

60. Dungel N : Is leprosy transmitted by arthropods ? Leprosy Rev, 1961; 32 : 28-35.

61. Narayanan L, Shankermanja K, Kirchheimer WF et al : Occurrence of *M. leprae* in arthropods, Leprosy Rev, 1972; 43 : 194-198.

62. Agarwal SC : Role of arthropods in transmission of leprosy, Final Report 1969-1979, Jawaharlal Institute of Post-graduate Medical Education and Research, Pondicherry, India, 1979.

63. Geater JA : The fly as a potential vector in transmission of leprosy, Leprosy Rev, 1975; 46 : 279-286.