THE VASCULAR LESIONS OF LEPROSY

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INTRODUCTION

It has been recognized for many years, at least since the work of Joelsohn in 1893 (6), that the various blood vessels related to leprous nodules and infiltrations of the skin may become involved in the pathological process, with bacilli appearing in their lining endothelial cells. There are a number of isolated accounts of such lesions in the literature, by Phillippson, in 1899 (9); Uhlenhuth, in 1900 (13); Sakurane, in 1902 (11); Herxheimer, in 1923 (5); Riecke, in 1925 (10); and Klingmüller, in 1930 (7). Many other writers have made statements like the recent one of Cowdry (1), that "bacilli commonly occur in the endothelial cells of blood vessels." The present paper presents the results of the examination of lesions from 77 cases of leprosy, including 10 autopsies, to determine the nature, frequency and significance of vascular involvement in this disease.

MATERIAL

A rough cross-section of leprosy as it occurs in Hawaii is presented, cases of all types being included. Most of the lesions were biopsy specimens of skin. Amputated fingers and toes, which frequently show no intrinsic leprous changes, have been omitted. Several cutaneous nodules obtained from Dr. I. D. Hirschy at the settlement at Kalaupapa, Molokai, are included without clinical data. The data on the cases and material involved in this study are shown in Table 1.

Most of the material was fixed in equal parts of 95 percent alcohol and 10 percent formaldehyde. A few blocks were fixed in 10 percent formaldehyde, Zenker's fluid with formaldehyde, or 5 percent mercuric chloride in 80 percent alcohol. Sections of all specimens were stained for acid-fast bacilli (2).

FREQUENCY OF INVOLVEMENT OF BLOOD VESSELS

Blood vessels were seen to be affected in lesions from 32 of the 77 cases included in this study. If the cases are roughly

TABLE 1.—*Cases and material examined.*

Case				Form of [b] disease	Duration yrs.	Lesions examined	Bacilli present	Vessels involved
No.	Age	Sex	Race[a]					
1	14	M	J-H	Infiltr.	2	Nodule	4+	Terminal
2	13	F	H	Nodular	6	Nodules (2)	4+	Terminal
3	50	M	P	Nodular	15?	Nodule	4+	Terminal
4	18	F	H	Nodular	1	Infiltrations (3)	4+	Terminal
5	11	M	H	Mixed	3	Nodule	3+	Terminal
6	11	M	F-H	Mixed	1	Nodule	4+	Terminal
7	12	M	H	Macular	5	Macule	4+	Terminal
8	19	F	J-H	Mixed	12	Nodule	4+	Terminal
9	8	F	H	Macular	1	Macules (2)	3+	Terminal
10	13	M	J-H	Nodular	3	Nodules (2)	4+	Terminal
11	9	F	H	Macular	3	Macules (2)	4+	Terminal
12	23	M	P	Mixed	(?)	Infiltration	3+	Terminal
13	10	M	J	Nodular	2	Infiltrations (2)	4+	Terminal
14	10	M	H	Mixed	1	Nodule	4+	Terminal
15	—	—	—	—	—	Nodule	4+	Terminal
16	—	—	—	—	—	Nodule	4+	Terminal
17	45	M	F	Mixed	5	Nodule	4+	Terminal
18	59	M	H	Mixed	12	Nodule	4+	Subcut. veins
19	24	F	H	Macular	2	Infiltration	4+	Small artery
20	62	M	C-F	Nodular	15?	Nodule	4+	Subcutaneous
21	15	M	H	Infiltr.	4	Autopsy	4+	Subcutaneous
22	19	M	F	Nodular	8	Autopsy	2+	Superficial
23	12	F	H	Nodular	3	Nodules (2) Nasal mucosa	4+	All kinds
24	32	M	H	Nodular	5?	Nodule	4+	Subcut. veins
25	56	M	P	Infiltr.	15?	Autopsy	4+	Subcutaneous
26	60	M	K	Nodular	10?	Nodules (5)	4+	Subcut. veins
27	33	F	H	Mixed	20	Nodule	4+	Subcutaneous
28	36	M	P	Mixed	20	Autopsy	1+	Small
29	18	M	H	Mixed	8	Nodule Lymph node	4+	All kinds
30	25	M	P	Neural	(?)	Subcutaneous vein	0	Tuberculoid leprous thrombophlebitis
31	19	M	P	Mixed	6	Tuberc'd nodule	1+	Terminal
32	—	—	—	—	—	Nodule	3+	Artery
33	10	M	H	Macular	3	Macule	0	None
34	10	M	H	Macular	5	Macule	0	None
35	7	M	J	Macular	1	Macule	0	None
36	17	M	H	Mixed	3	Nasal mucosa Macule	2+ 0	None
37	29	F	H	Tubere'd.	7	Macule	0	None
38	60	M	Ca-H	Neural	7	Macule	0	None
39	33	F	H	Macular	2	Macule	0	None
40	27	M	H	Neural	2	Macule	0	None
41	41	M	F	Macular	?	Macule	0	None
42	47	M	H	Neural	17	Autopsy	0	None
43	55	F	H	Neural	30	Autopsy	0	None
44	56	F	H	Neural	18	Autopsy	0	None
45	40	M	J	Neural	30	Autopsy	0	None
46	69	M	K	Neural	15?	Autopsy	Nerves only	None
47	55	M	H	Macular	(?)	Macule	Rare	None
48	22	M	H	Mixed	3	Macule	3+	None
49	34	F	J	Neural	9	Macule	2+	None
50	42	M	H	Macular	2	Macule	1+	None
51	14	M	H	Macular	3	Macule	2+	None
52	28	F	H	Macular	15	Macule	1+	None

TABLE 1.—*Cases and material examined (cont.).*

Case				Form of [b] disease	Duration yrs.	Lesions examined	Bacilli present	Vessels involved
No.	Age	Sex	Race[a]					
53	9	F	H	Macular	3	Macule	Rare	None
54	58	M	H	Neural	15?	Tuberculoid mac.	Rare	None
55	38	F	K	Macular	5?	Tuberculoid mac.	1+	None
56	31	F	H	Mixed	3	Tuberculoid mac.	1+	None
57	23	F	J	Tubercd.	1	Tuberculoid mac.	1+	None
58	8	M	Ch-H	Tubercd.	½	Tuberculoid mac.	Rare	None
59	25	M	F	Tubercd.	(?)	Tuberculoid mac.	Rare	None
60	37	M	H	Neural	19	Tuberculoid mac.	0	None
61	18	M	Ch-H	Macular	6	Tuberculoid mac.	0	None
62	23	F	H	Macular	1	Tuberculoid mac.	0	None
63	39	M	F	Macular	(?)	Tuberculoid mac.	0	None
64	8	F	P-F	Tubercd.	1	Tuberculoid mac.	0	None
65	35	M	P	Tubercd.	1	Tuberculoid mac.	0	None
66	25	M	Ch	Macular	(?)	Tuberculoid mac.	0	None
67	28	M	H	Mac. Inf.	2	Autopsy	4+	None
68	9	M	Ca-H	Tubercd. Nodular	3	Tuberculoid mac.	Rare 4+	None
69	12	M	H	Mixed	4	Ulcerated macule	3+	None
70	10	M	H	Macular	3	Macule	4+	None
71	76	M	Ch	Mixed	½?	Infiltration	3+	None
72	56	M	P	Nodular	36	Nodule	4+	None
73	33	M	Ch	Nodular	12	Testis, scrotum	4+	None
74	25	M	P	Mixed	11	Nodule Nasal mucosa	4+	None
75	35	M	P	Nodular	(?)	Nodule	4+	None
76	15	M	H	Nodular	6	Granulation tiss.	4+	None
77	—	—	—	—	—	Granulation tiss.	4+	None

[a] H = Hawaiian; P = Portuguese; K = Korean; Ch = Chinese; F = Filipino; J = Japanese; Ca = Caucasian.

[b] This classification indicates the predominant type of lesion. "Infiltr." means diffuse infiltrating nodules. "Mixed" signifies the presence of both macules and nodules in considerable numbers.

divided according to severity of the disease, vascular lesions were seen in 30 of 41 severe ones, and in 2 of 36 light ones. This is a simple statement of their preponderant occurrence in the presence of abundant bacilli.

In view of the usual multiplicity of cutaneous lesions in leprosy, and of the demonstration in the autopsied cases that not more than two-thirds of the specimens taken show vascular involvement, actual affection of the blood vessels in leprosy is undoubtedly greater than the figures indicate, and the conclusion appears justified that there is vascular involvement in nearly every severe case of the disease.

HISTOLOGICAL AGE AND ITS RELATION TO THE FREQUENCY OF VASCULAR LESIONS

It has been recognized from the study of these lesions that there is a clear relation between their age and the occurrence

of changes in the vessels. In one sense, the older the lesion the more likely it is to show vascular involvement.

There is a distinction to be drawn between clinical and histological age of a lesion, with respect to both the nodules and the borders of macules. In the latter case it is obvious that the active border of a spreading macule of ten years' duration is not actually ten years old; the central part regresses spontaneously while the border advances. In stationary nodules or infiltrations, there are several changes which are associated with different states of activity. During the acute or highly active stage the bacilli occur abundantly in small masses and packets, and tend to infiltrate in the neighboring tissues; during the chronic stage of activity the lesions tend to become organized, and large numbers of bacilli develop in globi; added to this is the state of regression of the lesion, with disappearance of bacilli and sharp demarcation of the foci.

It is during the state of chronic activity that the blood vessels appear to become infected. This is illustrated in Case 2, in which two biopsy specimens were obtained from the same lesion, the first one during the subsidence of an acute reaction and the second one four months later. No vascular involvement was seen in the first specimen, while infected vessels occurred in the other. Bacilli had not decreased in number, and, although clinically the lesion had flattened, histologically it appeared not inactive but older.

Many nodules of advanced clinical age present quite a variable appearance. Some parts of a lesion may be active, while other, not distant, areas may be obsolete, and all stages of activity may be said to be present in a single nodule. In general it is usual to find the active areas close to the surface and the deeper foci undergoing regression. In the lesions of this type, vascular involvement appears in great variety. In several cases the vascular lesions have been the main features of the picture in areas from which bacilli had largely disappeared from the surrounding lepromatous tissue.

The significance of this feature of histological age and the frequency of vascular lesions lies in the fact that they do not appear at the start of the lesion, and Herxheimer's designation of some such lesions as early foci conflicts with this observation. No correlation is seen with the age of the patient or the duration of the disease as a whole.

CAPILLARY INVOLVEMENT

The most difficult lesions of blood vessels to identify are those of capillaries, owing chiefly to the difficulty of recognizing these vessels in uninjected tissues.. Large, solid lepromas are actually rich in capillaries. In two cases they were so filled with red blood corpuscles that they were clearly delineated, and they were seen to be so plentiful that scarcely a bacillus-containing cell in the lesion did not have some point bordering on such a vessel. This abundant capillary bed goes unrecognized in most sections, and the observer looking for evidence of capillary involvement may see none or much according to his fancy.

In this study nothing has been called a capillary lesion unless the vessel itself was plainly recognizable. From this viewpoint, therefore, lesions of capillaries may be more common than described, in view of the impossibility of identifying more than a small fraction of these vessels that are actually present in the lesions.

The statement has been made that there is usually a widespread capillary involvement in leprous nodules, and that this is a common or intrinsic feature of those lesions. That this is not the case, and that capillary involvement is not much more extensive than described, would appear from the following observations. (*a*) The most common involvement of blood vessels in leprosy is that of the terminal vascular loop, including the arteriole, capillary and efferent venule. When bacilli were found in capillaries, they were also found in the terminal arterioles and venules in the neighborhood. In the latter situations their existence in the endothelial cells is readily determined. (*b*) Bacilli in lining endothelial cells are often found in more or less discrete areas; large parts of a nodule may show none, yet in one small area they may be plentiful. This suggests that when one part of a terminal loop becomes infected, the infection spreads readily to other parts and to branching vessels of relatively the same size. (*c*) In the most exuberant types of leprous tissue, such as young granulation tissue which appears just below the epidermis or in ulcerating lesions, in which both bacilli and cells appear to be flourishing and reproducing readily, there are abundant newly formed vessels and capillaries, with large endothelial cells which make them easy to identify. Bacilli do not commonly occur in these endothelial cells.

The so-called capillary embolus, in which the lumen of the

capillary is occupied by a chain of intermeshed extracellular organisms, is much less frequent in occurrence than the simple presence of bacilli in capillary endothelial cells, and, unlike the diffusion through the endothelium to larger vessels, it appears to end abruptly with the capillary. It is curious that the endothelial cells of the vessels containing these emboli may contain no bacilli.

In contrast to the relative infrequence of bacilli in capillary endothelium is their uniform occurrence just outside the capillaries, usually within cells but not always so. These pericapillary infiltrations are conspicuous about the margins of active foci, in such numbers that they appear to constitute one of the important paths by which infiltrations and nodules enlarge in size. They are abundant during the more acute stages and absent around old regressive lesions; and the bacilli or bacillus-containing cells are so closely applied to the capillary as to leave little doubt as to the importance of the relationship. The same thing can be seen in the experimental cutaneous lesions of rat leprosy (3).

The endothelial cells containing single bacilli usually present no other abnormalities. When they are more heavily laden the organisms may be grouped about the nuclei. Still larger numbers occur in loosely arranged masses in hollow vacuoles, less compact and not matted together as is commonly the case in the ordinary globi.

LESIONS OF LARGER BLOOD VESSELS

The various changes seen in larger vessels may be grouped in several types, of which more than one may appear in a single vessel.

1. Diffuse infection of the endothelium, with every cell containing bacilli, was seen in both arteries and veins; it seemed to result from spreading of the bacilli in the endothelium. Scattered bacilli in endothelial cells were usually found in connection with other lesions and sometimes constituted the only change.

2. The occupation of smooth muscle cells by bacilli in large groups occurred only in small arteries. Like other cells harboring the microorganisms, the cytoplasm was largely replaced by masses of loosely arranged bacilli, all of the muscle cells being affected. Such lesions were seen in six cases.

3. In larger vessels it appeared that the infection often spread by way of the vasa vasorum in their walls, and that the

infection of the vessel was dependent upon this. This accounts, perhaps, for the involvement of groups of large vessels in the subcutaneous plexus, both arteries and veins, when the smaller vessels are unaffected. Bacilli were not identified in the endothelium of the vasa vasorum, and the spread appeared to be along and not within those vessels.

4. Subendothelial or intimal accumulations of bacilli occurred in one case, apparently in connection with a general peripheral arteriosclerosis, but in two other cases they appeared in connection with the infiltrations along the vasa vasorum, and in another one they appeared to follow heavy infection of the endothelium.

5. In one case a lepromatous mass was found projecting into the lumen of an artery.

6. In one case there was a lepromatous thickening of the valve of a large vein.

7. In one instance tuberculoid changes were found in a large thrombosed vein, as the only residual condition in a mild case of leprosy of limited extent. Several of the cases of leprous involvement of large veins reported in the literature have consisted of massive lepromatous organization of thrombosed vessels, but no such lesions were found in the present group. With this single exception no thrombi have been seen in any of the infected vessels, a fact which seems remarkable.

LESIONS OF BLOOD VESSELS IN OTHER TISSUES

Vascular involvement was seen in the testis once, and in the nasal mucosa twice. The peripheral nerves and lymph nodes in the autopsied cases have not shown such lesions.

LESIONS OF LYMPHATIC VESSELS

Although many of the foci of leprosy apparently arise in the perivascular lymph spaces around arteries, veins and nerves, ordinary histological sections do not demonstrate the lymphatic vessels themselves. In only two instances, Cases 10 and 28, were bacilli seen in lymphatic channels, in the lining endothelium. Many of the lymphatics in those lesions were widely distended, in contrast to the usual state of collapse.

Lesions similar to the vascular capillary emboli occur occasionally in terminal lymphatic spaces (see photographs). These spaces do not have a demonstrable endothelial lining, and the appearance within them is of extracellular ramifying masses of

bacilli. These were seen in four cases, all in active infiltrations of subcutaneous fat globules, between individual fat cells. They were traceable to neighboring intracellular masses, from which they appeared to arise. Their absence in connection with large masses of bacilli in globi lends further support to the opinion that globi are not derived from terminal lymphatics.

In heavily infected lymph nodes the lymphatic channels may be obliterated by the mass of leprous granuloma, but bacilli have not been observed in the lining of the peripheral sinuses or in blood vessels within the nodes.

EFFECTS OF THE VASCULAR LESIONS

It is probable that veins of which the endothelial cells are laden with bacilli discharge organisms into the circulating blood. In view of the frequency of such lesions it would appear that there is a continual discharge, of variable degree, in most instances of nodular leprosy. That this is the case is well supported by the evidence of the heavy seeding of the liver and spleen with bacilli. In thirteen autopsied cases[1] in which bacilli were demonstrable in the liver and spleen, the seeding of the Kupffer cells and the splenic pulp cells with bacilli constituted a much more extensive part of the picture than the miliary lepromata. In three of the cases the lepromata in the liver were rare and inconspicuous in size, while every oil-immersion field showed four or five bacilli in Kupffer cells, frequently as single organisms, occasionally as two to five bacilli in a single cell, or rarely more. Grossly calculated, this would be about one billion bacilli for the entire liver. Apparently the life of the bacilli in the Kupffer cells is limited; certainly few proliferate to form miliary lepromata, although these may be abundant.

TABLE 2.—*Occurrence of visceral lesions in leprosy.*

Source	Nodular leprosy		Neural leprosy	
	No. of cases	Visceral lesions	No. of cases	Visceral lesions
Hansen & Looft (1894).....	89	67	36	0
Mitsuda (1937).....	131	127	19	0
Present cases.....	13	12	7	0

Though Table 2 is too simplified, and there are recorded exceptions to its conclusions, the figures shown there further in-

[1] Six of these cases were from the U. S. National Leprosarium at Carville, La., obtained through the courtesy of Dr. R. D. Lillie.

dicate that visceral foci are common in that type of leprosy in which bacilli and vascular foci are abundant.

CONCLUSIONS

In cutaneous lesions from 32 out of a total of 77 cases of leprosy of all types, some involvement of blood vessels was present, usually with bacilli in the lining endothelial cells. Infection of the terminal vascular loop was the most common occurrence, while larger vessels were involved in a variety of ways. Infection of the vessels occurs with prolonged activity of the lesion, and is seen especially when bacilli are abundant. The vascular infection effects a continual intravenous autoinoculation of lepra bacilli.

The vascular involvement is always focal in distribution, except that, when larger vessels are involved, the infection may spread through many of the smaller branches of those vessels. Arteries and veins appear to be involved with equal frequency, and it is probable that the infection of large vessels is by way of the vasa vasorum in many instances.

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DESCRIPTION OF PLATES

(NOTE: With one exception, the photographs are from sections stained for acid-fast bacilli and counterstained with hematoxylin and van Gieson's stain. The counterstain in many instances is faint, in order to accentuate the bacilli. Dark masses appearing in the photographs are masses of lepra bacilli.)

PLATE 13

FIG. 1. An apparently early leprous focus. The bacilli lie outside of the capillary, and not in endothelial cells. Case 9, ×900.

FIG. 2. A late leprous focus in a terminal capillary loop. The endothelial cells are all infected and dark masses of bacilli project into the lumen. The vacuolated cells embedding the vessel contain only a few granular bacilli. Case 22, ×900.

FIG. 3. A diffusely infected venule lying at the margin of a nodule. Case 21, ×900.

FIG. 4. A small vein forming the center of a small nodule. Masses of bacilli occur both in the endothelium and beneath it. The nodule itself, composed of vacuolated cells, contains comparatively few bacilli and is apparently a lesion of long standing. Case 22, ×200.

FIG. 5. Leprous lymph node. The peripheral sinuses are relatively unaffected and stand open. Case 21, ×120.

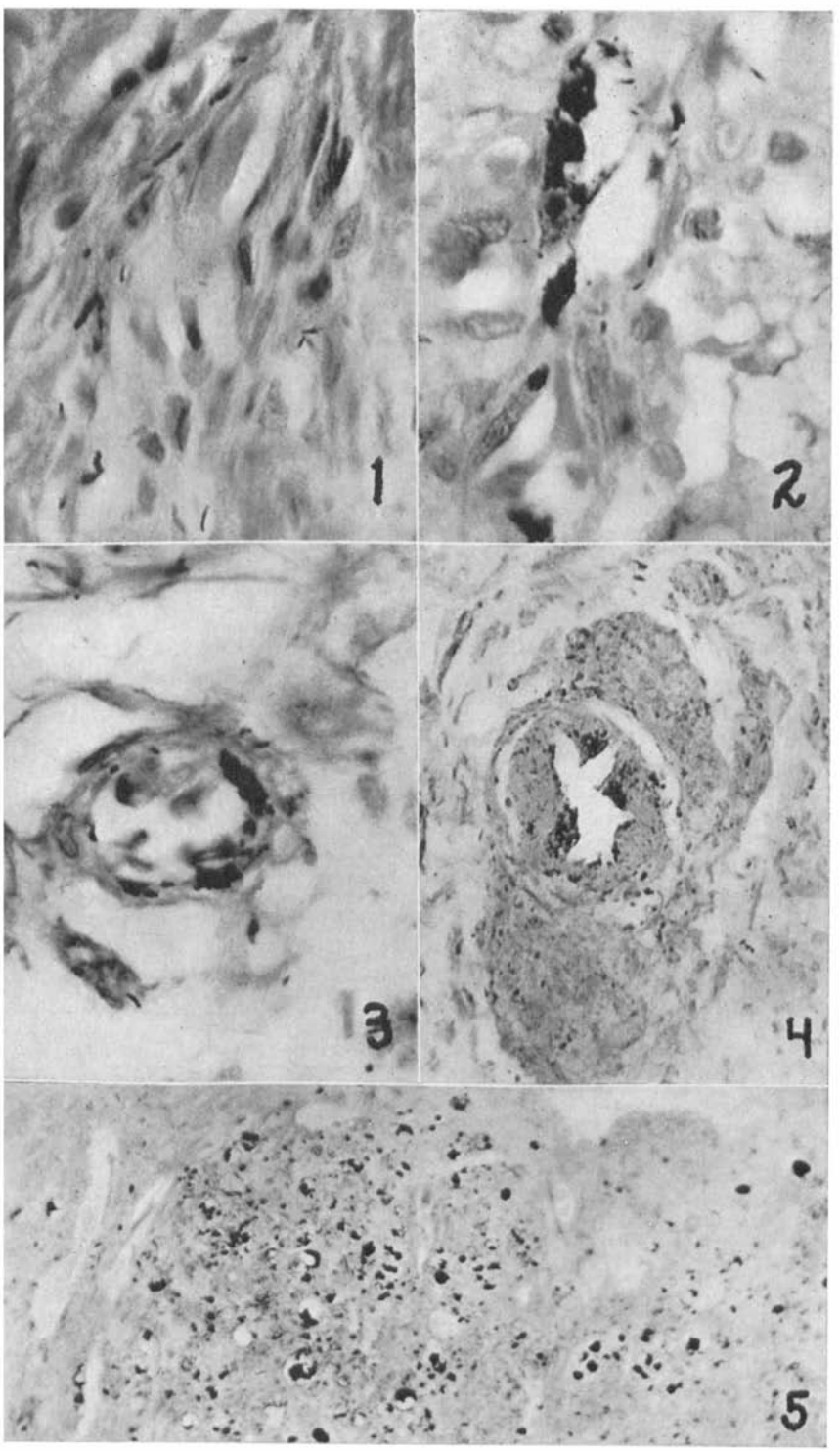

PLATE 13

PLATE 14

FIG. 6. A small leprous vein in the rete cutaneum. The large numbers of lepra bacilli in the endothelium clearly outline the inner wall of the vessel. Case 20, ×120.

FIG. 7. Another example of an affected vein, with heavy leprous infiltrations surrounding it. Case 20, ×200.

FIG. 8. The clear zone about the infected endothelium represents the wall of the vessel, which is relatively uninvolved. Case 18, ×200.

FIG. 9. A small leprous vein at higher magnification. Case 27, ×900.

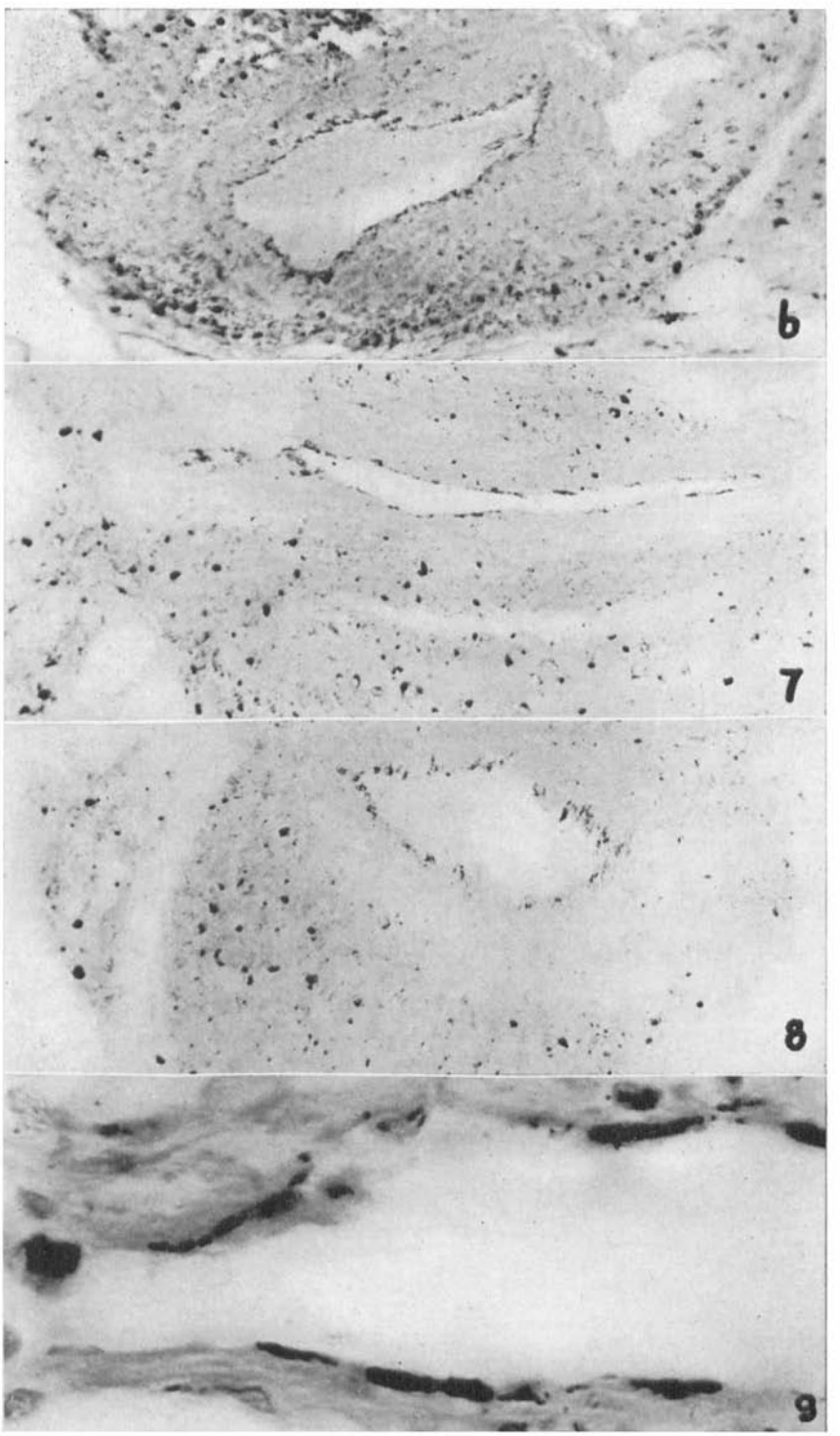

PLATE 14

PLATE 15

FIG. 10. A leprous vein from the nasal mucosa. Case 23, ×900.

FIG. 11. Lepromatous mass projecting into the lumen of an artery. The vessel was embedded in a large nodule, and there is an interruption in its wall at the point of attachment of the subendothelial mass. Hematoxylin and eosin. Case 32, ×200.

FIG. 12. Masses of bacilli within globi. The rings of bacilli about the margins are not uncommon. Case 21, ×900.

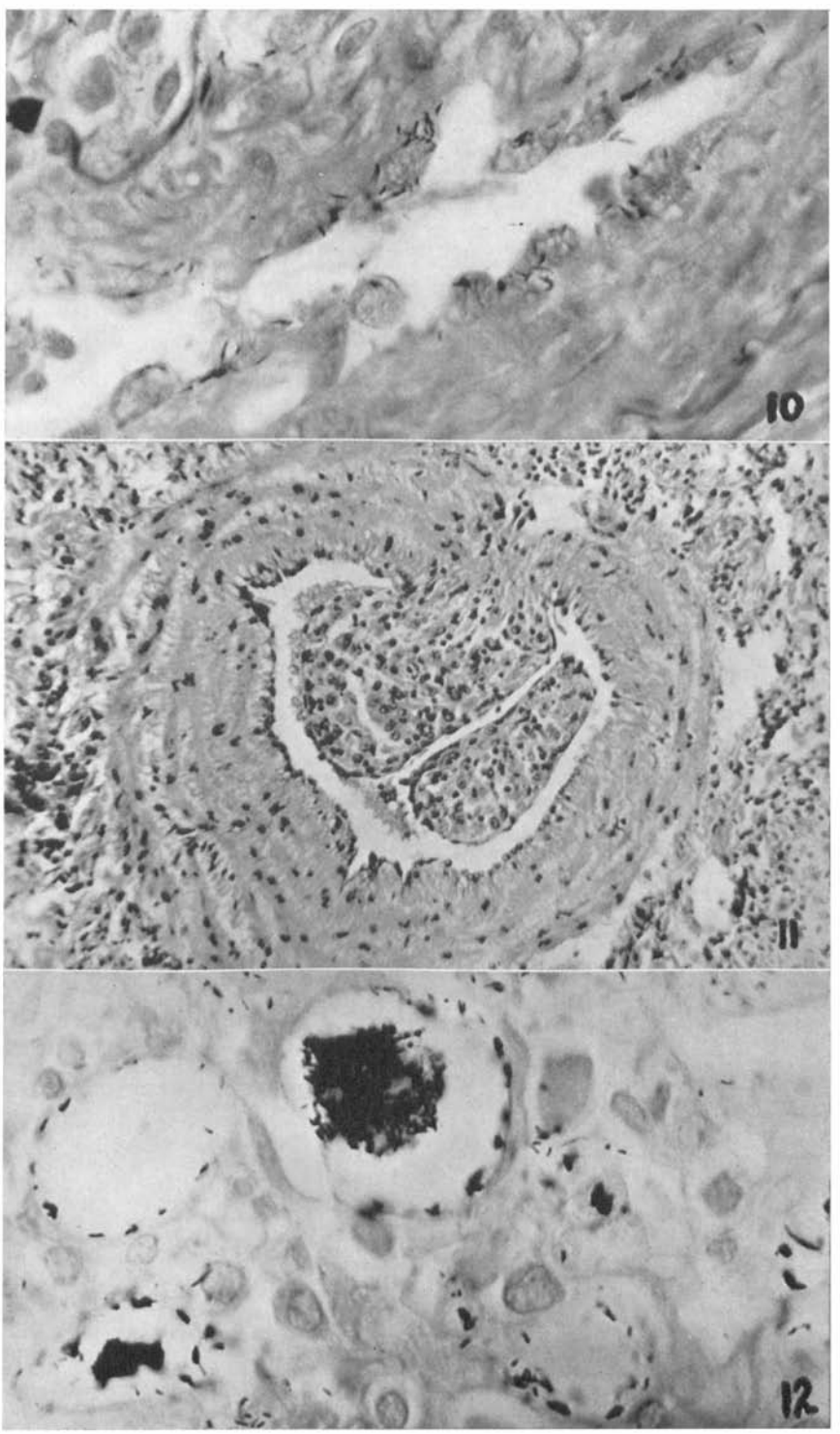

PLATE 15

Plate 16

Figs. 13 and 14. "Lymphatic capillary emboli." No cells are demonstrable in connection with these masses of bacilli, which ramify between fat cells. Case 26, ×900.

Fig. 15. A small lepromatous nodule in the spleen. Case 21, ×900.

Fig. 16. A leprous arteriole with bacilli in the muscular coat. Another section close to this one showed a single infected endothelial cell, from which the infiltration apparently arose. Case 31, ×900.

Fig. 17. Periarterial deposits of lepra bacilli in the spleen. Case 22, ×900.

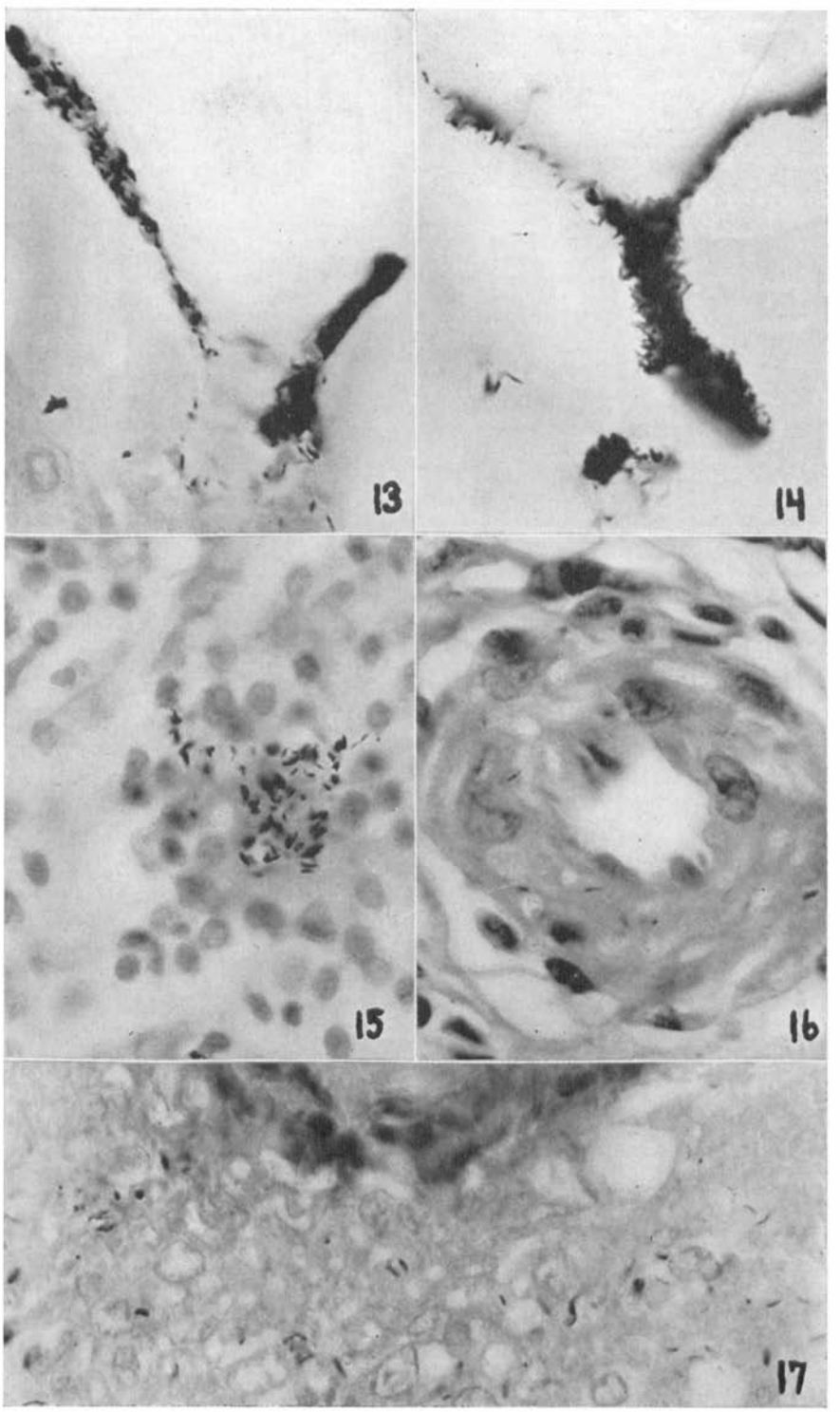

PLATE 16

PLATE 17

FIG. 18. Diffuse infection of the endothelium of a fibrous-walled blood vessel, embedded in a large chronic nodule. Case 23, ×650.

FIG. 19. Heavy infection of the endothelium of a capillary. Case 24, ×650.

FIG. 20. Chain of bacilli within the central capillary of an arrector pili muscle. The endothelial cells, not shown in the photograph, were flat and apparently not infected. The entire embolus was about three times as long as the part illustrated. Case 26, ×900.

FIG. 21. Exuberant leprous granulation tissue, with small, newly formed vessels uninvolved in the heavily infected tissue. Case 77, ×200.

FIG. 22. An infected venule. Case 29, ×650.

FIG. 23. Subendothelial accumulations of bacilli in the thickened intima of a subcutaneous artery. Case 21, ×200.

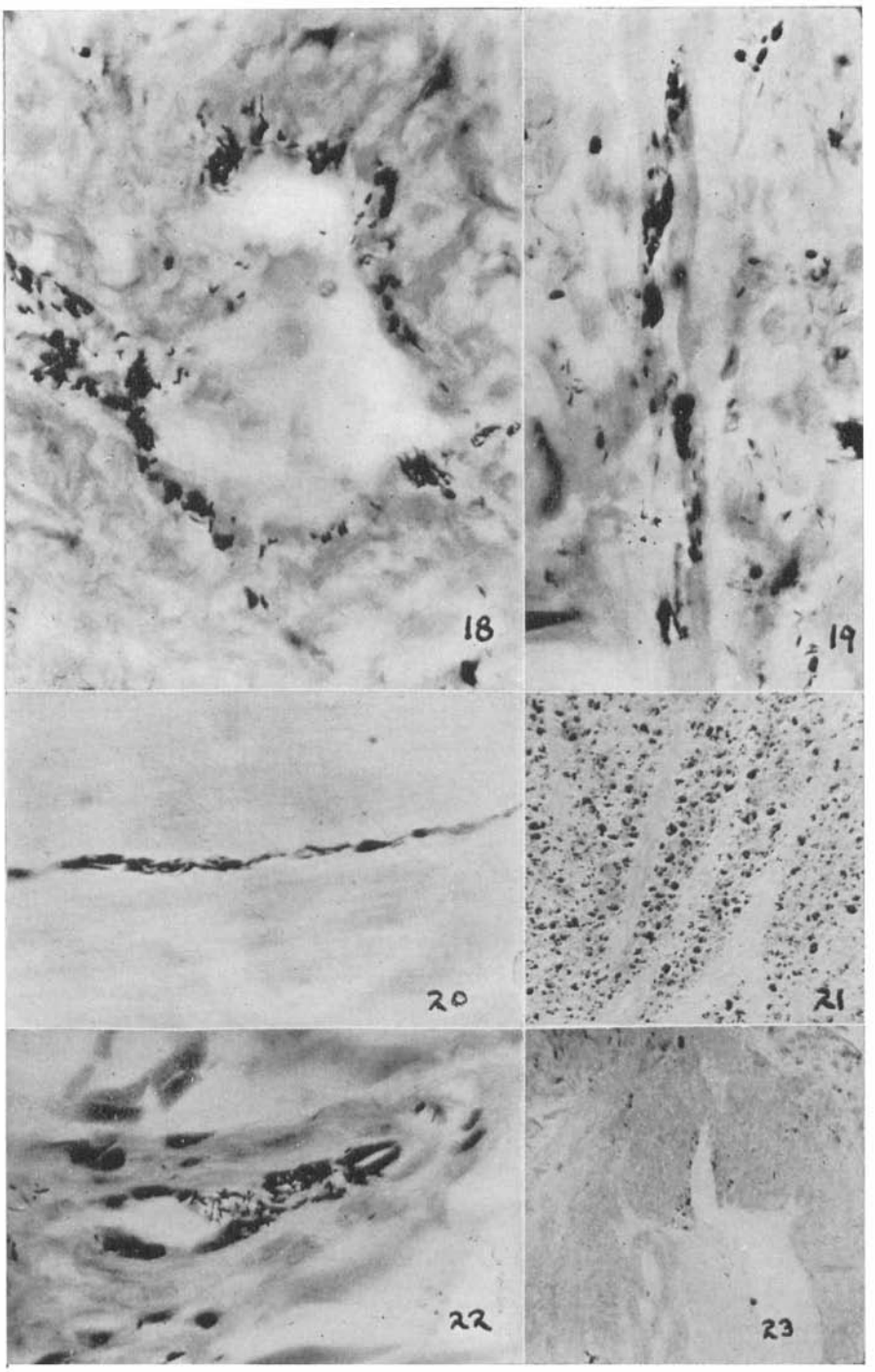

PLATE 17

Plate 18

Figs. 24 and 25. Heavily infected terminal vessels with swollen, vacuolated, endothelial cells. In sections stained with hematoxylin and eosin, these have patent lumens. In Fig. 25 the area above the vessels shows the old lepromatous tissue which constituted the bulk of the lesion, with its weakly-staining, beaded and fragmented bacilli. Case 29, ×650.

Fig. 26. A leprous vein. The thickening of the wall appears to be due chiefly to the bacillary infiltrations. Case 25, ×650.

Fig. 27. An infected venule. Case 28, ×900.

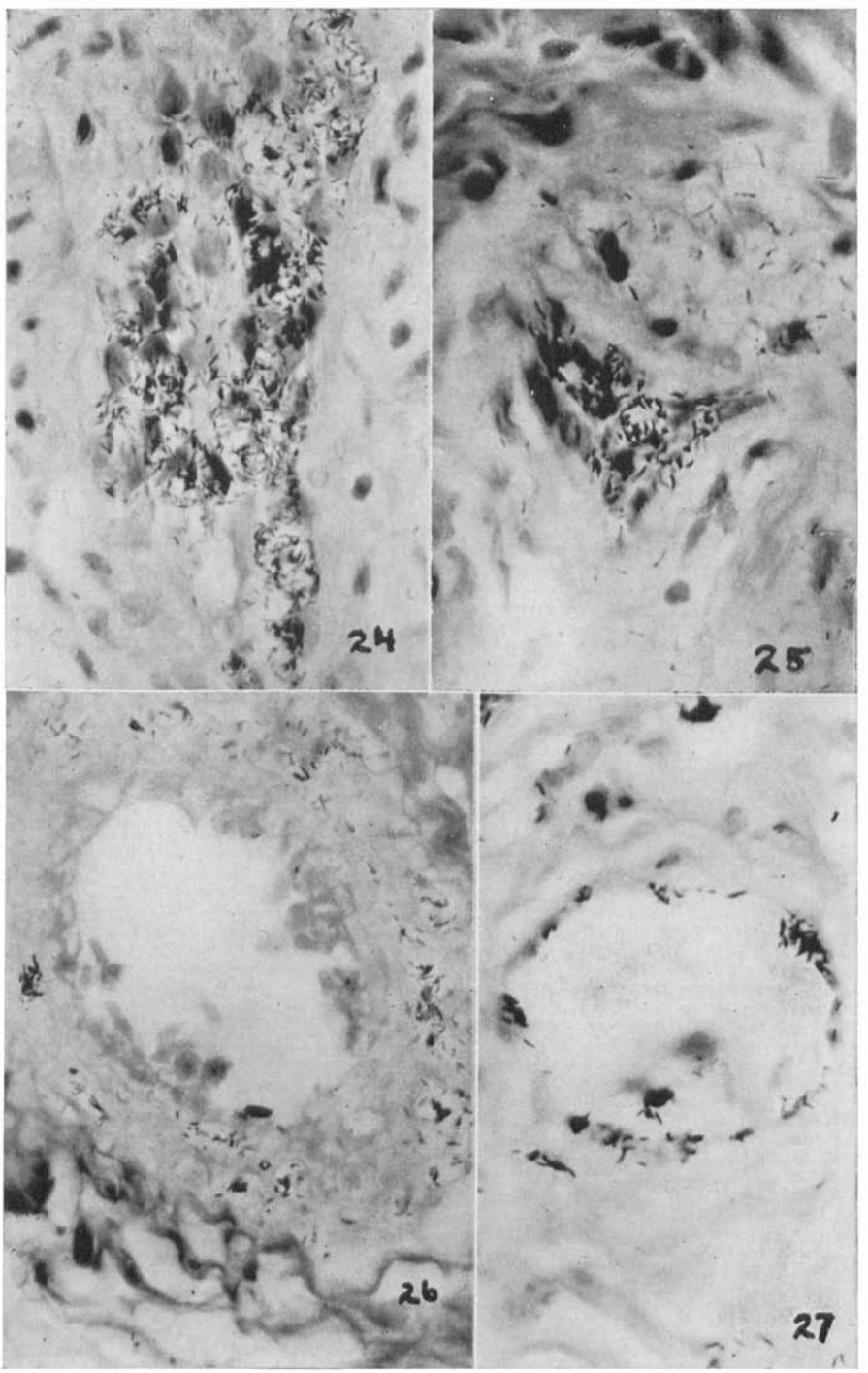

PLATE 18