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BONE CHANGES IN LEPROSY THEIR INCIDENCE, PROGRESS, PREVENTION AND ARREST[1, 2]

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A review of twenty-seven papers on the subject of bone changes in leprosy since the year 1889 (Paterson, 1954, thesis) shows that most of the authors of those papers considered that absorption of soft tissue and bone of the fingers and toes of leprosy patients is mainly neurotrophic in origin and progressive in character. If this is the case, then rehabilitation in leprosy is doomed to failure. The objects of this paper are as follows:

1. To describe the radiographic appearances of bone changes in leprosy, and to indicate the incidence of such changes.
2. By correlation of clinical and radiologic findings in leprosy and in other diseases, to discover which bone changes are due to the specific effects of *M. leprae* in bone, and which may be due to the effects of nonspecific influences in "neurotrophic" digits.
3. By serial radiographs and follow-up studies over a period of years to determine whether bone changes are in fact inevitable and progressive, and the extent to which these changes and the resulting deformities of hands and feet can be prevented or arrested.

METHODS AND MATERIALS

In Vellore, at the Christian Medical College in the years 1951-1954, the radiographs

[1] Presented at the Conference on Rehabilitation in Leprosy, held at Vellore, South India, November 21-29, 1960, under the auspices of the World Health Organization, the Leonard Wood Memorial, and the International Society for the Rehabilitation of the Disabled.

[2] Some of the pictures used to illustrate this article are also to appear in the second edition of the book, Leprosy in Theory and Practice, edited by R. G. Cochrane, and others in a Textbook of Practical Radiology, by J. H. Middlemiss.

of 108 leprosy patients which showed bone changes were studied. In 71 of these cases correlation of radiologic findings with Brand's clinical records were made [16]. Recently, follow-up radiographs over an eight- or nine-year period on 14 of these cases have been studied.

In Hong Kong, at the Mission to Lepers' Hay Ling Chau leprosarium, good radiographs of the hands and feet of 894 patients have been taken. Recently all admitted patients have been radiographed on admission and at yearly intervals thereafter. Follow-up radiographs over one- to three-year periods have been studied in 144 cases. A statistical analysis relating bone changes to clinical and pathologic findings in the 894 cases has been carried out.

Control radiographs of the hands of 50 apparently normal Chinese staff members were taken at Hay Ling Chau.

Radiographs of the digits of 12 nonleprosy patients suffering from osteomyelitis of fingers or toes were studied at Vellore.

The radiology of other neurologic and infective conditions that give rise to appearances similar to those seen in leprosy have been studied [16].

The arterial and venous pictures in the hands of five normal persons, 12 leprosy patients and three others has been studied by means of series of six or seven films in periods up to 120 seconds after injection of the opaque medium [15]. Post mortem studies of the arteriolar and nerve supply to phalanges have been started.

Bone biopsies of the sites of lesions that have been shown on radiographs has been possible in only three or four cases. A bone-drill biopsy apparatus has now been obtained, so that this work can be extended.

RADIOLOGIC FINDINGS

The radiologic bone changes in leprosy have been found to be of three main types. They are (*a*) specific bone destructive changes; (*b*) nonspecific bone absorptive changes; and (*c*) osteoporosis. Details of the various radiographic appearances are described. Figures in parentheses indicate the percentages of the 894 leprosarium patients in Hong Kong showing these changes (Table 1).

SPECIFIC BONE CHANGES (OSTEITIS LEPROSA)

Bone changes regarded as specific were found in 3-5 per cent of the cases studied. In 2 cases fingers were radiographed while the patients were experiencing progressive nodular lepra reactions (ENL), with painful red swellings of the fingers. In 1 case areas that previously showed just a few patches of trabecular destruction suddenly extended into larger areas of bone destruction, or "cavitation." These cavities or areas of destruction have subsequently been seen to resolve or fill up during a period of one year of DDS therapy, with remarkably little final deformity.

In 8 cases of about 1,000 studied, we have been able to observe the appearance, and the resolution under DDS therapy, of similar areas of bone destruction in fingers that were the sites of local lesions of lepra reaction. Bone biopsy in lepra reaction was not considered advisable, but biopsy after the acute stage was over was done in 2 cases (Fig. 1), and pathologic reports stated that there was evidence of lepromatous involvement of bone [9, 15].

Based on the findings in these 8 cases, and on a study of other granulomatous conditions in bone (sarcoidosis, reticuloses, tuberculosis and mycetoma), we have found that there is a radiologic picture that is almost pathognomonic of the specific action of *M. leprae* in bone. Possible specific appearances were found in 128 out of the 894 patients in

TABLE 1.—*Incidence of bone changes in 894 leprosarium patients at Hong Kong.*

Bone changes	No. of patients	Per cent
Specific (osteitis leprosa), total	*128*	*14.3*
Minimal: Honeycombing	30	3.0
Pseudocysts	46	5.0
Enlarged nutrient foramens	13	1.5
Small clear areas	25	2.8
Moderate: Central areas hazy outline	9	1.0
Subarticular hazy destruction	11	1.2
Cortical areas destroyed	6	0.7
Concentric cortical erosion	2	0.2
Healing: Sclerosis of shaft, straight	9	
Sclerosis, subarticular, straight	14	2.6
Sclerosis, shaft, twisted	10	
Sclerosis, subarticular, twisted	4	1.6
Nonspecific, total	*402*	*45.0*
Acute osteitis: Hazy area	88	1.0
Osteomyelitis: Hazy fragmentation	35	
Shaft destruction	19	5.6
Periostitis: Linear calcification	80	9.0
Irregular ossification	38	4.2
Tubular shaft	2	2.2
Neurotrophic osteitis: Concentric absorption	126	14.0
Cupping	2	0.2
Healed lesions: Terminal phalanx, tuft erosions	244	27.0
Terminal phalanx, absorbed	71	8.0
Middle phalanx, absorbed	79	8.0
Proximal phalanx, absorbed	95	10.0
Metatarsal heads, absorbed	50	5.5
Metatarsal shafts, absorbed	66	7.4
Cuneiforms, absorbed	19	2.1
Arthritis, acute: Hazy joint surfaces	42	4.7
Arthritis, chronic: Destruction of joint surfaces	82	10.0
Absorption of joint surfaces	61	6.8
Subluxation of joint surfaces	40	4.5
Arthritis, healing: Bony ankylosis	22	2.5
Other bones: Talus and navicular neuropathy	19	2.1
Osteoporosis (disuse), total	*90*	*10.0*
Minimal: Reduced subarticular trabeculation	12	1.3
Moderate: Visible cortical trabeculation	62	7.0
Advanced: Thin irregular cortex	18	2.0

Hong Kong, but of these cases only 28, or 3 per cent, showed definite active lesions; 37 cases showed evidence of healed lesions, and 114 cases had minimal or doubtful radiographic signs. It should be noted that various stages of the disease may be seen in the same hand.

Minimal signs that may be specific.—1. Honeycombing (3%): In these minimal cases a few bony trabeculae, usually at the nutrient-artery end (the distal end) of a phalanx, are destroyed. Sometimes a whole phalanx may show alteration of bone structure of a reticular character.

Clear-cut alteration of trabecular structure sometimes seen in both hands and feet may be due to other abnormalities, but hazy honeycombing has been seen in lepromatous cases with skin lesions of the fingers. Follow-up radiographs will help in such cases.

2. Pseudocysts (5%): By pseudocyst is meant a clear-cut area of loss of trabeculae or bone destruction. Again these lesions are usually seen at the distal end of a phalanx. In their healed or final state these areas develop a clear-cut sclerotic border, or they may disappear. One nonleprosy staff member showed the presence of periarticular pseudocysts following an old strain. Pseudocysts may be seen in a number of conditions other than leprosy (xanthomatosis, pneumatic-drill workers, etc.).

Pain in the patella and tibia has been frequently noted in patients in Hong Kong. One such case showed a pseudocyst in the patella that cleared up after a few months of DDS therapy.

3. Enlarged "nutrient foramen" (1.5%): In 12 out of 50 normal controls, small defects in the center of the distal end of the middle phalanges of fingers were seen. In normal people these defects were no larger than 1 mm. in diameter, usually less. It is presumed that these are holes in the cortex at the site of nutrient arteries or emissary veins. In leprosy patients, in India and in Hong Kong, large numbers of cases showed these nutrient-artery defects in the fingers and in the proximal phalanges of toes.

It has yet to be proved that nutrient vessels do enter the middle and terminal phalanges of fingers and proximal phalanges of toes, in the distal end of the bone in the midline. In proximal phalanges of the fingers, however, a nutrient canal can often be seen passing in from one side ([12]). Until further evidence is produced we regard a nutrient foramen of less than pinhead size (1 mm. diameter) as normal.

Only 13 out of 894 patients showed nutrient artery defects that were larger. Large or irregular defects at the above sites may be signs of leprous periarteritis ([5]), or periarteriolar neuritis ([11]), or of engorged emissary veins. In one patient a "nutrient foramen" defect of 2 mm. diameter was observed to be absolutely unchanged over a period of eight years (Fig. 3.).

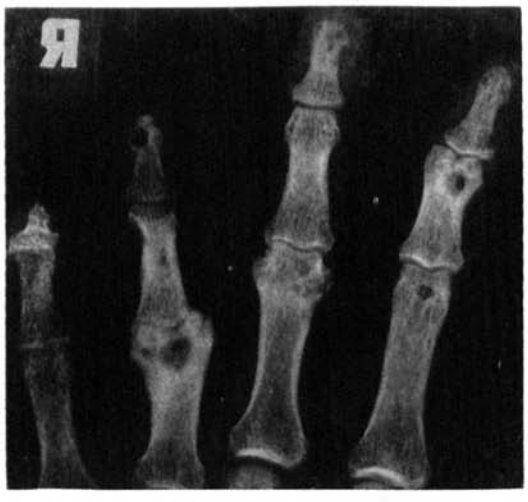

FIG. 1. Multiple dactylitis leprosa, right hand, associated with leprotic nodules. Radiograph taken 1 year after a bone biopsy (long finger, head of proximal phalanx) which revealed leprous granuloma, and after 1 year of therapy. Some sclerosis of affected areas has occurred in the interim.

Definite signs of active osteitis leprosa.—1. Multiple clear-cut small areas of bone destruction (2.8%): These may be seen in the medullary area or medial cortex of the ends of phalanges (Fig. 1). Less commonly (0.7%), clear-cut areas of bone destruction are seen in the lateral cortex of proximal phalanges at the point where we know a nutrient vessel enters the bone.

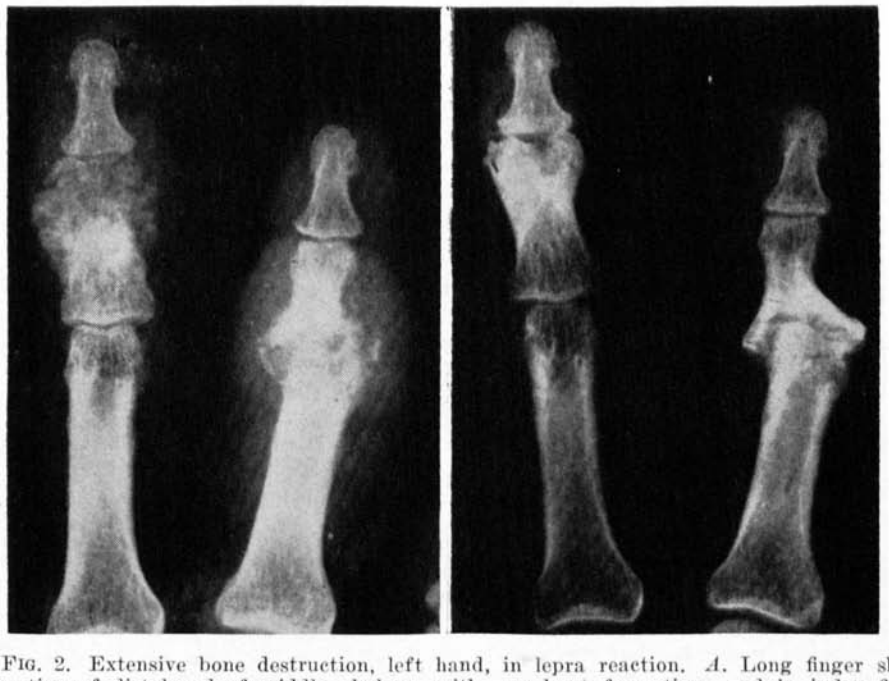

FIG. 2. Extensive bone destruction, left hand, in lepra reaction. *A*. Long finger shows destruction of distal end of middle phalanx, with new bone formation; and in index finger of the proximal end of the middle phalanx, with gross soft tissue swelling (September 1955). *B*. Healing with sclerosis and minimal deformity (January 1956). Courtesy of Dr. A. D. Thiesen, Champa, India.)

2. Ill-defined areas of bone destruction (1%): These may occur anywhere in the phalanges in lepra reaction (Figs. 1 and 2). Very rarely there may be complete destruction of the cortex, with extensive irregular subperiosteal new-bone formation, and there may be irregular destruction of one side of the subarticular bone, or destruction of periarticular cortex only.

3. Subarticular collapse (1.2%): Specific destruction of the subarticular bone does not, in our experience, involve the joint cavity or cartilage, but it does cause collapse deformity and sclerosis of subarticular bone (Figs. 1 and 2). Nonspecific subarticular osteitis may sometimes produce a similar appearance.

Healing stage; signs that may be specific.—1. Healing with sclerosis and no deformity (2.6%), with deformity (1.6%): Clear-cut round holes in bones with sclerotic borders are sometimes seen in the shafts and in the terminal phalangeal tufts. These are probably the final appearance where a larger area of bone destruction or cavity has healed, with sclerosis of its margins (Fig. 1).

Even large areas of bone destruction show a remarkable degree of healing in about a one-year period. The end result is seen to be a thickened sclerotic cortex and a diminished medullary area. If the fingers are adequately splinted, as on a round coconut-shell splint, ([2]), deformity may be prevented, but if there is no splinting the fingers may become twisted sideways at the site of the old bone destruction (Fig. 2).

2. Concentric erosion with thickened cortex (0.2%): A few cases

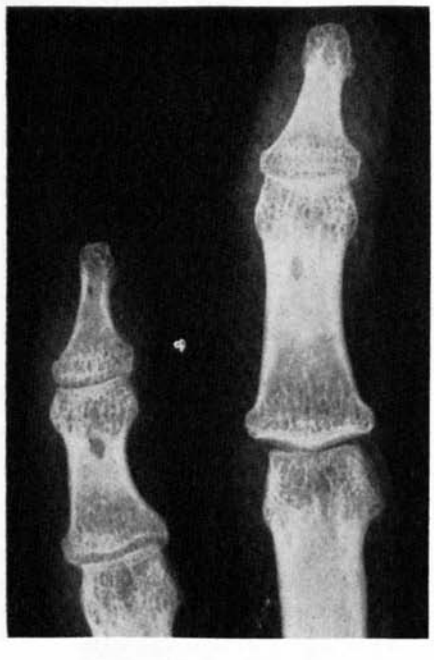

FIG. 3. Enlarged "nutrient foramens," ring and little fingers of left hand, 1.5 mm. (1952). (No changes were seen in radiograms made eight years later, in 1960.)

show the concentric type of absorption of the outer layers of the shafts of the phalanges, with no evidence of local bone destruction but with very thick sclerotic cortex and diminished medullary space. These patients have had no history of finger-tip or knuckle ulceration, but they may have had diffuse lepromatous patches or nodules in the skin of the fingers or toes. We believe that such an appearance is sometimes of specific origin.

3. Rings of cortical erosion: In 1 case, bands or rings of cortical erosion were seen in all the fingers. Perhaps these were due to old skin nodules, or reactions with resulting fibrous contracting bands around the bone (cf ainhum [18]).

4. Other bones involved in specific osteitis: In our experience it is very rare to see definite evidence of leprous osteitis in bones other than those of the hands and feet. One Vellore case showed evidence of old collapse of the subarticular tissue in the upper end of the tibia. One Hong Kong case showed local subperiosteal new-bone formation in the mid-shaft of tibia, possibly due to leprous periostitis. One case mentioned above showed a pseudocyst in the patella.

NONSPECIFIC ABSORPTIVE CHANGES

In a much larger number of cases (402, or 45%, of the Hong Kong patients) there are seen erosive or absorptive changes that usually start at the distal ends of the phalanges or metatarsals and work proximally, sometimes quickly and sometimes very slowly. Where these changes progress quickly they are exactly the same as those seen in nonleprosy patients who have whitlows or ulcers with nonspecific osteitis or osteomyelitis. Where these changes progress slowly they are exactly the same as those seen in nonleprosy patients with "neurotrophic" conditions, such as diabetic neuropathic gangrene [3] or congenital indifference to pain [17]. Similar radiographic appearances have also been noted, in the absence of clinically detectable nerve lesions, in malnutrition with chronic ulceration [8, 15], in scleroderma [10] and in frostbite [19].

These appearances have therefore been termed nonspecific bone absorptive changes. In leprosy they are almost always associated with sensory loss, trauma, and sepsis in fingers or toes. Radiographic signs of nonspecific bone absorption in leprosy are as follows:

1. *The soft tissues.*—On good radiographs the outline of the soft tissues is well shown, so that the degree of soft-tissue swelling or soft-tissue ulceration and absorption can be noted. Many cases showing reduction of the size of the pulp of the terminal phalanges or soft-tissue absorption also showed nonspecific bone absorptive changes in the terminal phalanges.

2. *Contracted fingers.*—Radiographs are routinely taken with the fingers flattened out palm down on the film. Where there is contraction

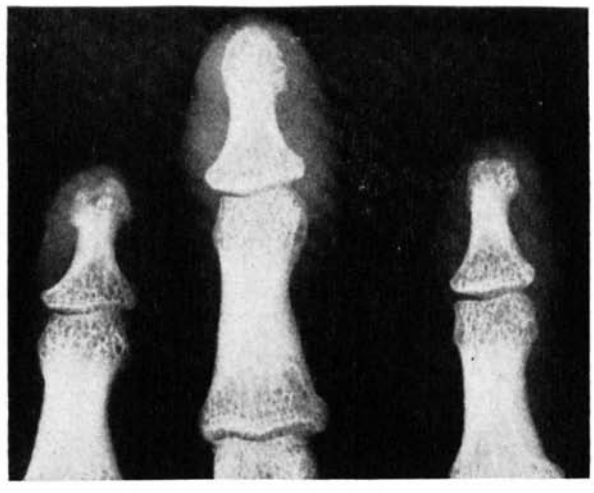

FIG. 4. Healed osteitis, right hand, with minimal erosions of the tufts of the terminal phalanges. A clear-cut nick in the tuft of the ring finger.

of soft tissues with inability even passively to extend the fingers, the fingers on the radiographs will appear curved or foreshortened. Finger-joint spaces will not be seen due to overlapping of phalangeal shadows.

3. *Nonspecific acute osteitis and osteomyelitis.*—In the presence of soft-tissue ulceration, redness and swelling, usually in the finger-tips or under the metatarsal heads, there are seen radiographically the following signs:

(*a*) Acute osteitis (1%): The appearances are exactly the same as those in nonleprosy patients: (i) A few hazy bone trabeculae. (ii) A hazy bone outline. (iii) A small area of loss of cortex and trabeculae with hazy margin (Fig. 12).

(*b*) Osteomyelitis (5.6%): Osteomyelitis in the foot is more frequent and more extensive in leprosy than in nonleprosy patients. The radiographic signs are:

(i) Fragmentation or hazy absorption of a whole bone with complete loss of structure (Fig. 7A). Occasionally fragmentation may take place in the absence of ulcer or sepsis, due to aseptic necrosis (Fig. 5A).

(ii) Extension of the area of bone destruction down the shaft (Fig. 7A).

(iii) Subperiosteal bone reaction. In severe conditions this may take the form of cloudy calcification (Fig. 7A). In some acute conditions there may just be a white line of subperiosteal calcification (Fig. 6B).

(iv) Sequestration is very rarely seen in the radiographs of leprosy patients. Clinically, sequestra are seen more often, as they are discharged through an ulcerating area. Involucrum-formation does not usually occur.

(*c*) Periostitis: Periostitis with subperiosteal new-bone formation may be secondary to osteitis as above (Fig. 6B), or primary due to spread of soft-tissue infection to the periosteal layer of bone (Fig. 6A).

Subperiosteal calcification in neurotrophic digits is usually completely reabsorbed, and no involucrum-formation takes place. Occasionally this subperiosteal calcification ossifies, with resulting irregular thickening of the cortex; later the inner layers of the cortex may be absorbed, leaving a bone with bulging or "expanded" type of cortex (Fig. 6).

These rather unusual manifestations of osteitis in neural leprosy may be due to neurotrophic hyperemia or an unusual local vascular response to infection. Local hyperemia in the presence of acute infection in one finger was shown by serial angiography in one case (Fig. 12).

(*d*) Subacute and chronic "neurotrophic" osteitis: (i) Concentric absorption (14%): When a leprosy patient with anesthetic hands or feet does not feel pain and so continues to use an inflamed finger or foot, there is apparently (in spite of hyperemia) a failure to heal the ulceration or infection completely, and there develops a slow smouldering type of subperiosteal bone erosion of the shafts of the metatarsals and phalanges. These bones are then concentrically narrowed, so that sometimes they taper off to a point. The extraordinary feature of this condition is that the thickness and density of the cortex is retained by additions to the inner aspect of the cortex at the expense of the medullary cavity. Copland ([3]) has detailed the mechanism of this type of change

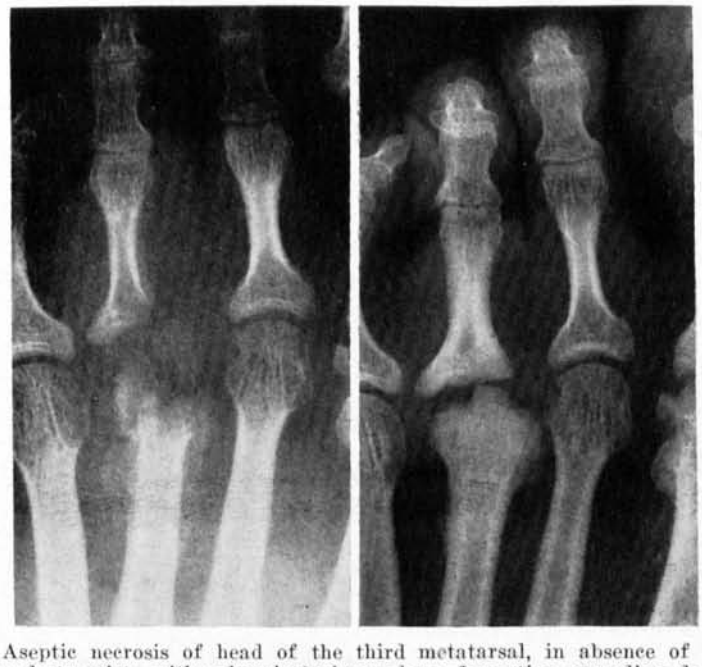

FIG. 5. Aseptic necrosis of head of the third metatarsal, in absence of plantar ulcer. *A*. Hazy bone destruction, with subperiosteal new bone formation spreading down the shaft; minimal destruction of bone of proximal phalanx, with loss of joint space (January 1959). *B*. Healing with ossification of neurotic bone; metatarsal with deformed outlines; joint space enlarged, articular surfaces irregular. (January 1960).

in diabetic neuropathy. Concentric absorption with retention of bone density is presumably a local tissue response controlled (in absence of painful sensation and higher reflexes) by a local nerve reflex only. If treatment is inadequate and plantar ulcers recur, the condition deteriorates (Fig. 7), but if rigid-sole shoes and adequate foot care is given the absorption can be arrested (Fig. 8).

(ii) Where there is neurotrophic osteitis and loss of metatarsal heads, there may also be concentric erosion of phalangeal shafts and collapse of the phalangeal subarticular bone, giving rise to a clear-cut cupping of the joint surface.

(*e*) Healing phase: In the healing phase of osteitis and osteomyelitis in "neurotrophic" digits there may be a striking reossification of aparently destroyed parts of the bone, and bone fragments may reunite with the shaft; or they may be left as completely separate, normally ossified bits of bones. These fragments are not sequestra; they apparently have a good blood supply. They need not be removed ([16]).

(*f*) Healed phase; digit absorption with clear-cut bone outline: When a normal patient's or a leprosy patient's finger-tip ulcer has healed, and when there is no longer active infection, the bone shows a clear-cut area of erosion. In conditions with sensory loss, reinfection and reulceration frequently occurs and so the bone again becomes hazy and a bit more bone is eroded. This process may go on until a whole finger is lost, or with adequate care it may be arrested at any stage.

4. *Degrees of bone absorption.*—The degree of bone-absorptive change may be recorded in three stages as follows:

(*a*) Terminal phalanges: (i) Nicks and slits in the tuft only (Fig. 11). (ii) One side or end of the tuft "sliced off" (Fig. 4). (iii) Whole tuft gone, with amputated collar-stud appearance (Fig. 11). (iv) Nothing but the base of phalanx remaining (8%) (Fig. 12).

(*b*) Middle and proximal phalanges: (i) Middle phalanx eroded or lost (8%) (Fig. 12). (ii) Proximal phalanx eroded or lost (10%).

(*c*) Metatarsals: (i) Metatarsal head eroded or lost (5.5%) (Fig. 8). (ii) Metatarsal shaft eroded or lost (7.4%) (Fig. 7). (iii) Cuneiform bones, tarsus eroded (2.1%).

The metacarpals are very rarely involved. The os calcis is often eroded secondarily to ulceration of the heel.

5. *Aseptic necrosis.*—Occasionally even in the absence of ulceration and sepsis there may be minimal swelling, blister formation, necrosis and fragmentation of a metatarsal head (Fig. 5). In the finger tips there may, as a result of trauma, develop subcutaneous hematomata and minimal bone absorption of the terminal phalangeal tufts.

In some cases of leprosy, scleroderma, frostbite, etc., there may be no evidence of sepsis but there is evidence of "devitalization" or perhaps subclinical nerve loss which leads to deep aseptic necrosis of

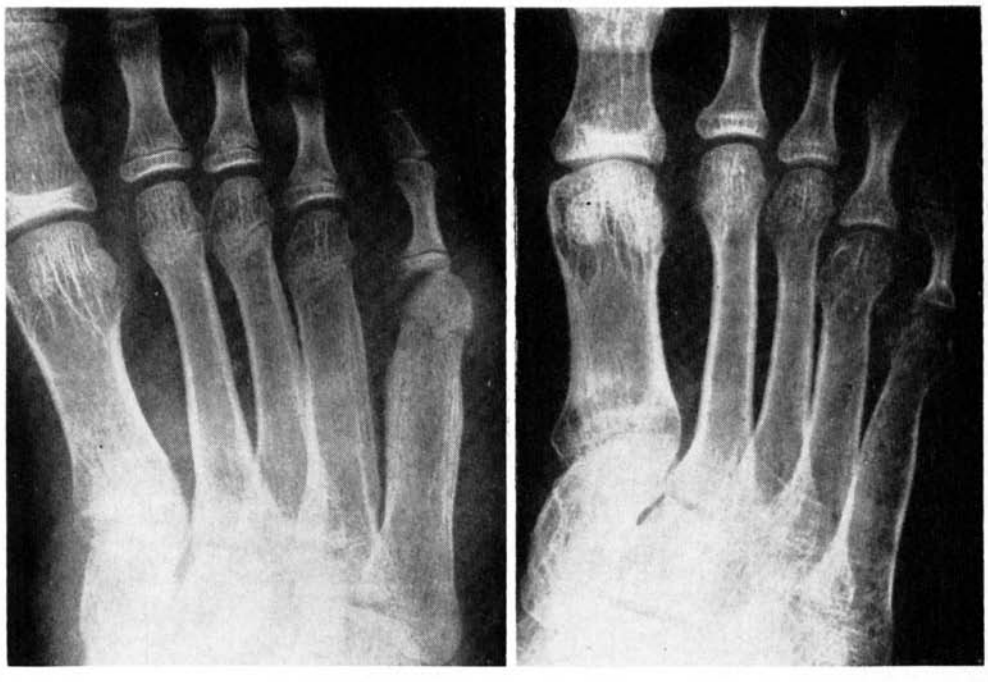

FIG. 6. Periostitis with resultant expansion of cortex. *A*. Layers of subperiosteal new bone, 5th metatarsal, with diminution of joint space, and on one side of the 4th metatarsal (October 1956). *B*. Fifth metatarsal still tubular (August 1958). (A radiograph made in 1957 showed marked haziness and an area of destruction at the base of the 1st metatarsal, which had healed a year later.)

tissues, and black discoloration of the skin. Deep tissue necrosis may lead to minimal absorption of the terminal phalanx tufts, and in some cases of scleroderma to calcification in the finger pulps.

6. *Arthritis and joint lesions in the digits.*—Where the joints are the most prominent points of the member, as in claw hand (Fig. 9), or where they are near pressure points such as the metatarsal heads (Fig. 7), they are liable to infection secondary to soft-tissue ulceration. On the feet these large "trophic" ulcers fail to heal because the patient does not feel pain and continues to walk on them. Only when there is lymphangitis and inguinal adenitis does the patient normally report sick.

In any joint the radiographic signs of arthritis are as follows:

(*a*) Acute: Haziness of articular surface with slight increase, then diminution of joint (cartilage) space (4.7%), (Fig. 6, 5th toe).

(*b*) Subacute: Destruction of subarticular bone and joint capsule (10%) with subluxation of articular surfaces (4.5%), (Fig. 8, great toe).

(*c*) Chronic: In leprosy with sensory loss and continued use infection is likely to spread, and consequently increased absorption of subarticular bone with osteitis and osteomyelitis develop (6.8%), (Fig. 7).

TABLE 2.—*Patients with bone changes in various age and sex groups. Percentages are in relation to the total number of patients examined in each age group.*

Sex and age groups	Patients with bone changes	Specific changes		Nonspecific changes		Osteoporosis		No bone changes		Total patients
(years)	No.	No.	%	No.	%	No.	%	No.	%	No.
Male patients										
0-9	—	—	—	—	—	—	—	2	—	2
10-19	40	8	10	33	39	6	7.1	44	52	84
20-29	121	34	13.8	97	38	16	6.6	126	56	247
30-39	121	41	20	103	50	15	7.3	86	41	207
40-49	59	16	18	50	56	18	20	30	34	89
50	31	7	16	24	56	14	32	12	28	43
60	13	2	12.5	10	62	6	33	3	19	16
Total	385	108	15.7	317	46	75	11	303	45	688
Female patients										
0-9	1	—		1		1		4		5
10-19	8	5		4		—		22		30
20-29	34	7		31		1		41		75
30-39	21	2		20		2		25		46
40-49	23	5		22		3		8		31
50	7			5		3		4		11
60	6	1		2		5		2		8
Total	100	20	9.7	85	41	15	7.3	106		206
Grand total	485	128	14.3	402	45	90	10	409	46	894

(*d*) Healing phase: In the healing phase the absorbed bone ends become more clear-cut. In some cases there is left a big gap between the bone ends, but in others there is bony ankylosis with complete obliteration of the joint, and bony trabeculae are seen crossing the joint space (2.5%). Fibrous ankylosis was not seen in our cases.

In severely contracted fingers a true lateral view sometimes reveals a "ditch" or anterior depression in the distal end of the proximal phalanx, which appears to be caused by pressure of the base of the middle phalanx. In such cases physiotherapy may not succeed in straightening the finger, and capsulotomy may be needed (1).

7. *Joint lesions in wrist and ankle.*—Patients with glove and stocking anesthesia (superficial nerve lesions) may also lose some of the pain sensation in the wrist and ankle joints, and so here again the course of infection or injury may be modified in a "neurotrophic" ulcer.

In the wrist, following infection and arthritis or following trauma (one case was probably tuberculous arthritis), there may develop a marked subluxation and yet very little pain. Because there is no pain

the bones may not be immobilized as they usually are in arthritis, and perhaps because of nerve lesions and "devitalized" tissue there is more bone disintegration and less new-bone formation.

In the tarsus, infection frequently spreads from ulceration of the heel to the os calcis and then to the subastragaloid joint (2.1%), the talus and the navicular bones. The talus and navicular may in this way be completely destroyed, and the ankle disorganized.

In other cases there may be no ulceration but a hot swollen ankle may appear, and a "neurotrophic" collapse of the talus and navicular bones may occur. Minor trauma to these bones at a posterior tibial transplant operation, or early weight-bearing after immobilization in plaster, may also produce talo-navicular collapse (Fig. 10). Examinations in this condition of talo-navicular collapse have not so far revealed any organism (4). It is considered that this condition may be one of aseptic necrosis or disintegration of bone following loss of sensory and other nerve fibers. An exactly similar talo-navicular collapse may be seen in diabetic neuropathy, and in congenital indifference to pain.

Neurotrophic or "Charcot" wrist and ankle joints may be the result of neglected trauma. A patient may sustain a minor fracture near a joint, but because he does not feel pain he neglects the condition and so a large hematoma forms, and the joint space is increased by this effusion and hemarthrosis. Later there is calcification in the clot, and the articular surfaces become irregular but sclerotic. The joint capsule becomes lax, and then there is subluxation or dislocation. In simple fractures there may at first be excessive callus.

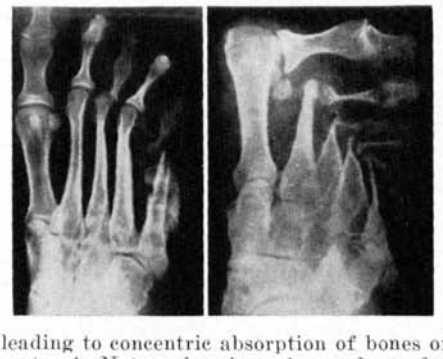

FIG. 7. Osteomyelitis leading to concentric absorption of bones of foot, due to twelve years of inadequate foot treatment. *A*. Note subperiosteal new bone formation of the 5th metatarsal, in 1948 (which had been absorbed when another radiograph was taken in 1951). *B*. Marked concentric absorption of all metatarsals except the 1st, and marked deviation of all the toes.

Charcot joint can be differentiated from neurotrophic osteitis and arthritis by the fact that, in the Charcot joint, the joint space is increased and the articular surface is disorganized and of increased density. In osteitis, the subarticular bone may collapse but the joint surfaces and space may be intact, and in arthritis the joint space is soon

diminished and the articular surfaces become hazy and osteoporotic.

Secondary to chronic ulcers of the heel the os calcis may be almost completely destroyed. With care, however, an amazing degree of soft-tissue reformation can be achieved and the condition arrested.

Sometimes plain radiographs reveal the extent of a deep soft-tissue sinus with minimal bone destruction. In other cases extensive calcification in the tendo-Achilles insertion may result from tightening and shortening of this tendon in neglected drop foot and ulceration of the heel.

Among the Chinese patients the end result of bound feet was noted in two old women. In both cases there was disuse osteoporosis, and in one there was concentric absorption of toes.

8. *Nasal bones.*—Resulting from ulceration of the nasal mucosa there may be seen absorption of the nasal spine of the maxilla, premaxilla and alveolar margin, and nasal bones. Møller-Christensen *et al.* ([14]) found such changes in ancient skeletons.

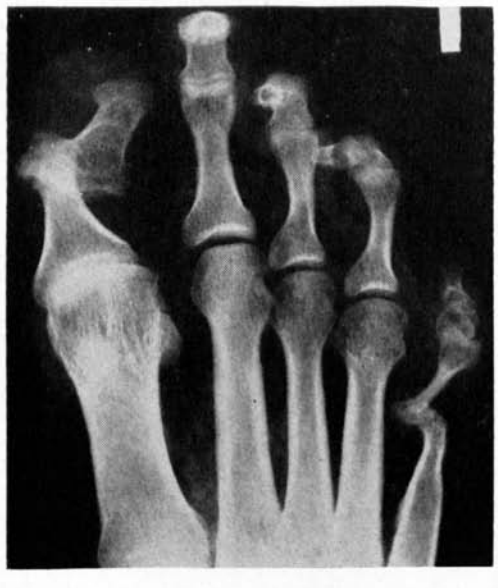

FIG. 8. Concentric absorption of bones of the foot (1953), arrested over a 7-year period by adequate foot treatment and rehabilitation (1960). Note the absorption of the distal end of the proximal phalanx, great toe, and of the 5th metatarsal and of the opposing end of the proximal phalanx.

OSTEOPOROSIS

1. *Nature and causes of osteoporosis.*—Osteoporosis is a radiologic term used to describe any condition in which there is a diminution of ossified bone substance, or a diminution in bone trabeculae. It may be general, due to malnutrition, senility or debility, or it may be local, due to disuse. Pain, tight skin lesions, edematous skin lesions, immobilization in plaster, and paralysis or contraction (Fig. 9A) all cause local disuse osteoporosis. Osteoporotic bones may easily fracture or collapse. Resp[14]- eful physiotherapy, osteoporotic bones may return).

local osteoporosis in leprosy that we have investi-

TABLE 3.—*Bone changes related to occupational trauma. Percentages are in relation to the total number of patients in each trauma group.*

Occupation	Patients with bone changes	Specific changes		Nonspecific changes		Osteoporosis		No bone changes	Total patients
	No.	No.	%	No.	%	No.	%	No.	No.
Beggars	4			4				1	5
Students	39	13	20	27	26	7	6.8	64	103
Clerical	36	6	7.7	26	34	10	13	41	77
Manual	380	103	15.7	328	49	63	9.6	277	657
Cooks	21	4	9.5	16	38	8	19	21	42
(?)	5	2		1		2		5	10
Total	485	128	14.3	402	45	90	10	409	894

gated carefully, some disuse factor has been found. In hands and feet with ulcerative lesions that would normally cause pain and osteoporosis, where there is sensory loss there is often osteosclerosis and thickening of bony cortex. In the 894 Hong Kong patients only 90 cases (10%) showed evidence of osteoporosis. About the same proportion of patients (11%) showed evidence of uniform soft-tissue swelling of the fingers. In many cases where there was uniform swelling of the fingers there was osteoporosis, probably due to restriction of movement by the swelling

2. *Stages of osteoporosis.*—The radiographic signs of osteoporosis may be divided into five stages, as done by Fontaine and Herrmann ([6]):

(*a*) Minimal subarticular: Trabeculae and cortex are reduced in thickness, with relative translucency of the subarticular bone as compared with the shaft (1.3%).

(*b*) Moderate: Reduction of density of the cortex of the shaft, with visualization of trabeculae in the cortical bone (7%). Some cortical trabeculation was seen, particularly in the fifth finger, in women and children with leprosy, and in 8 women out of 50 normal cases.

(*c*) Advanced: Uneven thickness of cortex where the inner cortical layer is eroded (2%), (Fig. 9A). This irregularity of the cortex causes a patchy appearance of the medulla (2%).

(*d*) Far advanced: Pencilled outline of cortex only remains. Very few trabeculae seen. (Only one case seen in about 1,000.)

(*e*) Very far advanced: No bone outline seen at all. (No case seen in 900).

Osteoporotic bone changes must not be confused with minimal specific changes, such as pseudocyst and honeycombing described above.

ANGIOGRAPHIC FINDINGS

Normal appearances of the digital arteries, vascular end loops, and nutrient vessels to the phalanges were noted in 5 control cases. In

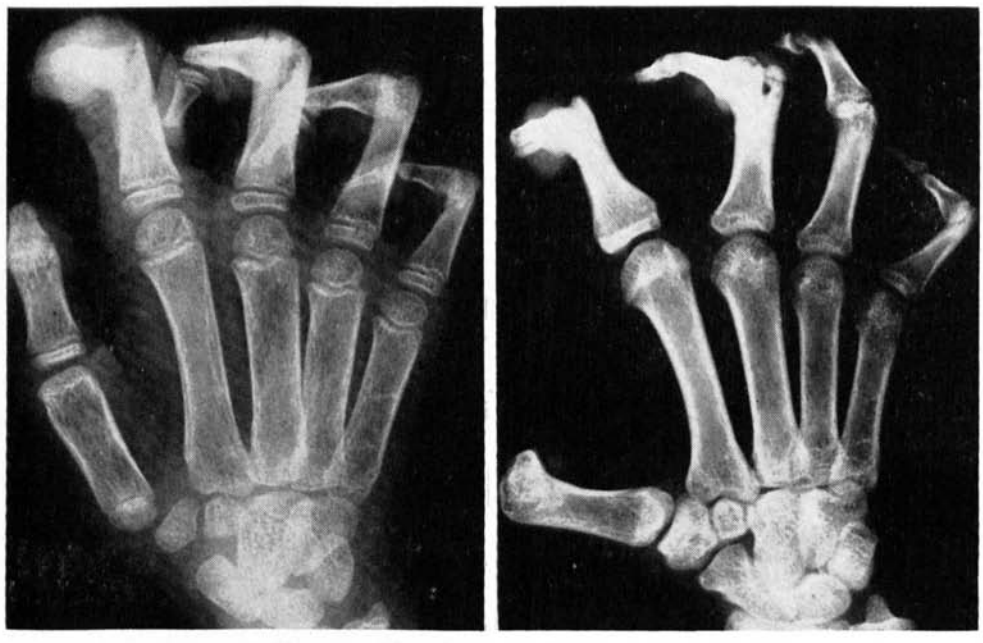

FIG. 9. Osteoporotic tubular metacarpols and subluxation in contracted finger, and marked improvement after 4 years of physiotherapy and rehabilitation. *A.* Marked thinning of cortices, with tubular shafts of metacarpals; trabeculae in cortices of phalanges (April 1954, then aged 12). *B.* Restoration of cortical bone in metacarpals and phalanges; no progression of tuft erosion (February 1958).

leprosy patients the abnormal angiographic findings were as follows:

1. In the presence of soft-tissue pulp absorption, even where there was no bone absorption, there is diminution in the vascular end loops (Fig. 11).

2. In the presence of disuse osteoporosis, with deformed absorbed fingers, etc., there is diminution in the caliber of digital arteries, but the caliber of the radial, ulnar and palmar arteries remains unchanged (Fig. 12). Similar diminution in caliber of digital arteries is described by Leb ([13]) in cases of rheumatoid arthritis.

3. In the presence of local inflammation, even where digital arteries are narrow, there is perhaps excessive hyperemia (Fig. 12).

4. In 7 out of 12 leprosy patients' angiograms there was considerable increase in the circulation time of the fingers (30 to 120 seconds). In other cases what appeared to be arteriovenous shunts were seen at the level of the proximal phalanges.

5. Evidence of endarteritis was seen in one case (Fig. 12).

CORRELATION OF CLINICAL WITH RADIOGRAPHIC FINDINGS

1. *Age and sex.*—It is shown in Table 2 that among the 894 patients admitted to the Hong Kong leprosarium over a period of about 6 years there were 688 males and 206 females, and that the majority were in the 20-40 years age groups.

TABLE 4.—*Bone changes related to type of leprosy and bacillus index. Percentages in relation to the total number of patients in each type of leprosy or each degree of bacillus index.*

Group[a]	Patients with bone changes	Specific changes		Nonspecific changes		Osteoporosis		No bone changes	Total patients
	No.	No.	%	No.	%	No.	%	No.	No.
Type of leprosy									
1. Lepromatous	328	88	14.5	265	43	66	11	279	607
2. Tuberculoid	85	17	14	80	66	10	8.3	36	121
3. Dimorphous	65	23	14.8	50	32	13	8.4	90	155
(?)	7			7		1		4	11
Total	485	128	14.5	402	45	90	10	409	894
Bacillus index									
0	194	43	13.5	175	55	18	56	126	320
1	81	25	18	67	46	9	6.2	65	146
2	67	21	14	53	35	14	9.4	85	149
3	90	22	12	71	39	30	16.5	92	182
4	48	14	16	35	40	17	19	40	88
5	2	1		—		1		2	4
(?)	3	2		1		1		2	5
Total	485	128	16	402	50	90	11	409	894

[a] No cases of the indeterminate form had been admitted at Hong Kong. Bacillus indices recorded are those found on admission.

The percentage of patients with nonspecific bone changes and with osteoporosis is seen to increase markedly with age, but the frequency of specific bone changes does not increase so much with age.

There is a higher incidence of all types of bone changes in males as compared with females.

2. *Occupational trauma.*—Patients were divided as far as possible into occupational trauma groups as follows: (*a*) dependent on society or beggar, possibly undernourished; (*b*) dependent on the family or student, usually well nourished; (*c*) clerical worker or supervisor; (*d*) manual worker or laborer; (*e*) cook, usually a housewife who may burn her hands.

In Hong Kong 657 out of the 894 patients were found to be those who worked or had worked with their hands. These ranged from factory workers, fishermen, and carpenters to waiters and coolie types. Table 3 shows that nonspecific changes occur in 49 per cent of manual workers as compared with 26 and 34 per cent, in students and clerical workers respectively.

There is seen to be a close correlation between occupational trauma and nonspecific changes, but there is no evidence of correlation between

TABLE 5.—*Bone changes related to duration of disease and duration of treatment. Percentages based on the total number of patients in each duration group.*

Duration (years)	Patients with bone changes	Specific changes		Nonspecific changes		Osteoporosis		No bone changes	Total patients
	No.	No.	%	No.	%	No.	%	No.	No.
Leprosy									
0	—	—		—		—		1	1
1	7	3		4		2		11	18
2	20	10	20	12	25	5	10	28	48
3	46	11	8.4	35	26	11	8.5	86	132
4	1	—		1		—		3	4
5	158	49	15.5	123	38	38	12	165	323
10	209	49	15.5	186	59	32	10	107	316
20	43	6	12	40	82	2	4	6	49
(?)	1	—		1		—		2	3
Total	485	128	14.3	402	45	90	10	409	894
Treatment									
0	21	4		15		6		27	48
1	39	17	23	27	36	14	18	36	75
2	51	18	15.5	34	29	16	14	64	115
3	55	13	11	43	37	15	13	62	117
4	54	15	13	45	39	11	9.6	61	115
5	62	17	15	52	46	12	10.6	51	113
6	29	6	12	27	53	3	6	22	51
7	38	12	17	32	45	3	4.2	34	72
8	135	26	14	126	68	10	5.5	50	185
(?)	1	—		1		—		2	3
Total	485	128	14.3	402	45	90	10	409	894

occupation and specific or osteoporotic changes. Even if there are extensive nerve lesions, a hand that is not used (owing to palsy or debility) will not develop absorptive bone changes.

3. *Type of leprosy and bacillus index.*—Table 4 shows that the proportion of patients showing specific bone changes remains surprisingly constant in different types of leprosy and with varying bacillus indices. However, on checking the cases that had tuberculoid and "dimorphous" (macular) leprosy it was found that only 9 out of 17 tuberculoid cases with possible specific changes had definite signs of active or healed lesions, and that the accuracy of the classification into types of leprosy was questionable. Further studies after more accurate typing are needed before any conclusions can be drawn from Table 4. All cases showing gross specific changes gave a history of recent lepromatous reaction.

Nonspecific bone changes are surprisingly common in tuberculoid

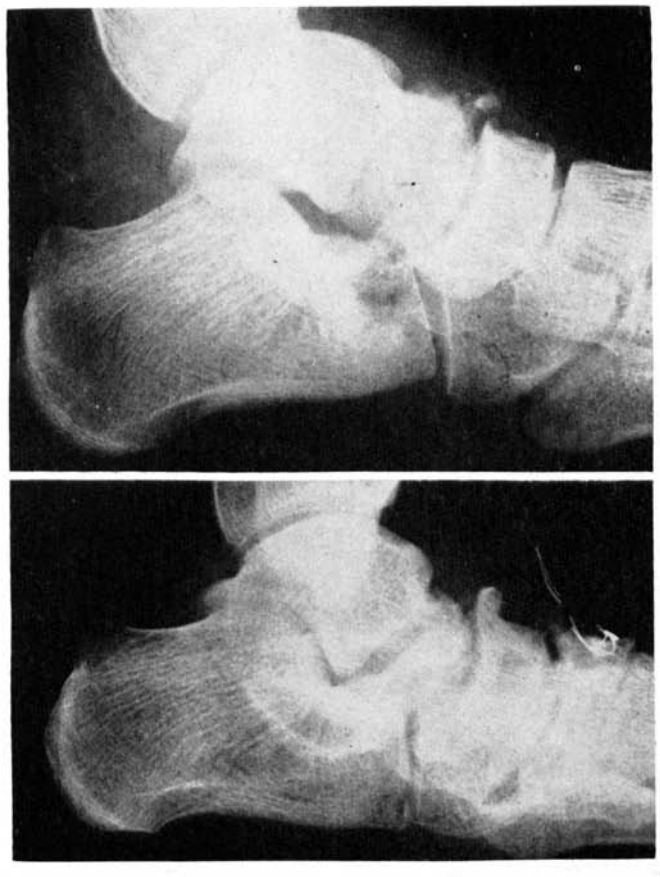

FIG. 10. Neuropathic disintegration of talus and os calcis. *A*. Areas of haziness and disintegration in upper part of talus and anterior part of os calcis (August 1959). *B*. Collapse of head of talus, and healing of cavity in the os calcis (March 1959).

cases, and in cases where there was a low bacillus index on admission. This may be due to the fact that these changes are associated primarily with nerve lesions.

Gass(7) showed the presence of *M. leprae* in the bone marrow of 17 out of 21 "mixed cutaneous and neural" cases, but that there were no *M. leprae* in the bone marrow of 48 neural cases.

4. *Duration of disease and time under treatment.*—Table 5 shows a significant increase in the proportions of cases showing nonspecific bone changes with duration of leprosy. In those cases that have been under treatment for a number of years, there is some reduction in the percentage that show specific changes, but there is an increase in the number that show specific changes. There is a reduction, with duration of treatment, in the incidence of osteoporosis.

It should be noted that a high percentage of patients showing radiologic evidence of bone change show changes (deformities or erosions) that are the end result of old disease, and that once these lesions have

TABLE 6.—*Progress of nonspecific and specific bone changes.*

Duration of X-ray survey	Patients with bone changes		Worse	Arrested or unchanged	Improved	Sclerosed or healed
	No.		No.	No.	No.	No.
Nonspecific						
6-11 months	42		3	35	4	—
1 year	102		15	69	13	5
2 years	73	244	10	42	14	7
3 years	69		18	29	14	8
4 years	3		1	1	1	—
No series taken	113					
Total	402		47	176	46	20
Specific						
6-11 months	12			11	1	
1 year	33			32		1
2 years	19	70		18		1
3 years	18		2	15		1
No series taken	46					
Total	128		2	76	1	3

occurred they will not disappear although they may be halted by treatment.

5. *Progress of bone changes under treatment.*—Table 6 was therefore drawn up in an effort to assess the extent to which bone damage had been arrested or healed during a period of leprosarium treatment and care.

In Hong Kong, patients (shown in the middle groups) had been radiographed, usually at yearly intervals, over one- two- and three-year periods. Of these, 244 patients showed bone changes of the nonspecific type, and 70 showed specific changes. Cases classified as "worse" are those in which existing lesions had increased in size or new lesions had appeared. Those classified as "improved" are those in which active hazy bone lesions had become perhaps smaller and had acquired clear-cut margins. Those classified as "healed" are those in which the margins of the lesions had become sclerotic or bone cavities had filled in.

Among the 70 cases showing specific bone changes, only 2 got worse over a three-year period, 3 showed very definite evidence of healing, and among the others in which the radiographic appearances were unchanged a fairly large number showed lesions that were already healed at the beginning of the period of x-ray survey.

Among the cases showing nonspecific bone changes, a few were seen to have got worse during the period of survey, but others were also seen to have improved or healed. Again the majority are seen to have been arrested or unchanged during the period of leprosarium treatment.

In Vellore it has been possible to follow up, clinically and radiographically, during and after treatment and rehabilitation, 14 cases over a 6-8 year period. In 10 of these cases it had been possible to arrest or heal the bone lesions. Adequate radiographic follow-up of a large series of cases has not been possible, but these 10 cases do show that nonspecific bone absorption can, with adequate rehabilitation and after-care, be healed and kept healed.

6. *Association of bone changes with nerve lesions, nonspecific ulceration, and specific lepra reaction.*—In Tables 7 and 8 we have analyzed

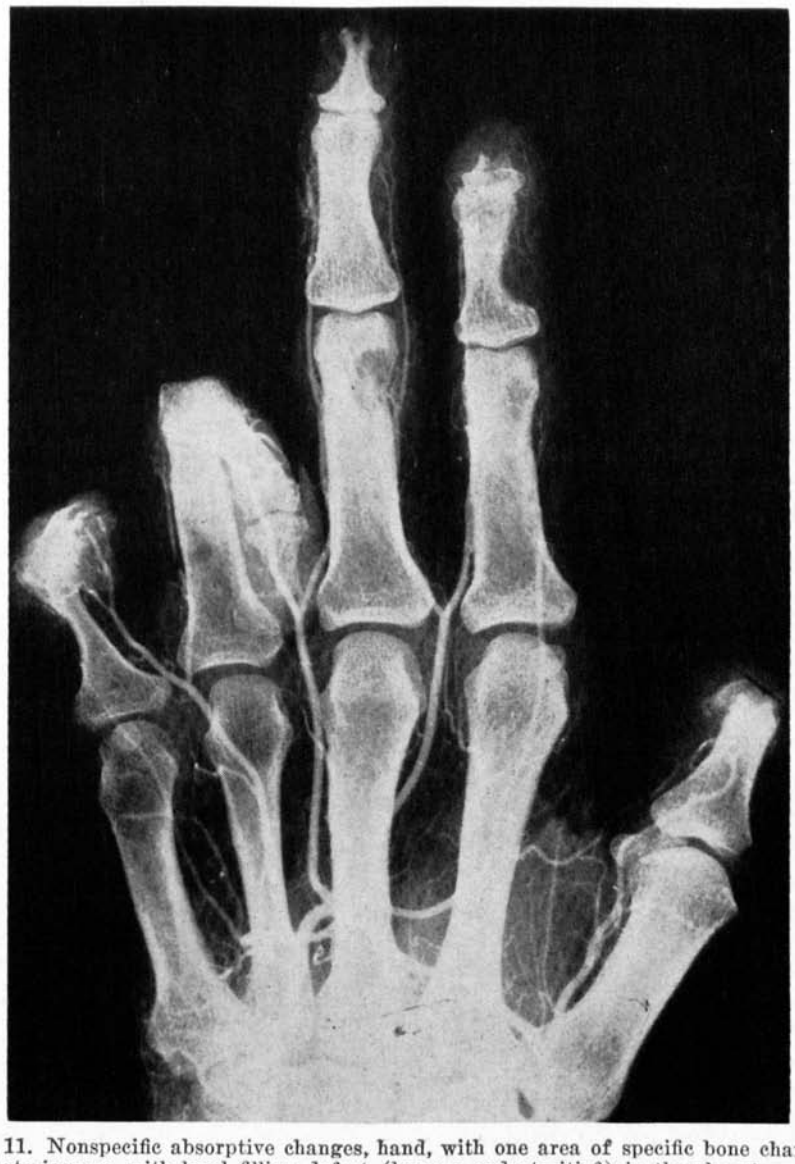

FIG. 11. Nonspecific absorptive changes, hand, with one area of specific bone change, long finger. Arteriogram, with local filling defect (leprosy endarteritis?) in thumb artery.

TABLE 7.—*Incidence of patients showing various clinical signs who have bone changes.*

Clinical signs	Patients with signs	No. of patients with signs who have:					
		Specific changes		Nonspecific changes		Osteoporosis	
	No.	No.	%	No.	%	No.	%
Nonspecific							
1. Anesthesia	626	102	16	353	56	76	12
2. Paralysis	391	69	17.5	262	67	54	21
3. Contraction	178	34	19	143	80	28	16
4. Pain	64	21	33	48	75	14	22
5. Infection and ulceration	277	47	17	242	87	28	10
6. Scarring	100	15	15	94	94	9	9
7. Trauma	31						
8. Hematoma	6						
9. Soft-tissue swelling	100						
Specific							
1. Lepra or ENL nodules	80	33	41	39	49	14	17.5
2. Tuberculoid reaction	7	2		2			
3. Dimorphous reaction	14	2		5			

the numbers of Hong Kong patients whose records show the presence of nerve lesions with anesthesia and paralysis, contractions of the soft tissues, and ulceration or scarring of the skin of the hands and feet. Records of nodules and lepra reactions affecting the hands or feet were not kept, but where general lepra reactions have been noted in the patients' records they are included in this table. Table 7 shows the numbers of patients with clinical signs who had bone lesions, and Table 8 shows the number of patients with bone lesions who had clinical records of anesthesia, ulceration, or lepra reaction.

From Table 7 we see that there were nonspecific bone changes in 56 per cent of patients with anesthesia, in 80 per cent of the patients with contractions, and in 87-94 per cent of patients with ulceration and scarring.

If we take all the 402 patients who had nonspecific bone changes, Table 8 shows that 87 per cent of them had records of anesthesia, but only 65 per cent of them had records of ulceration and scarring. Records

TABLE 8.—*Percentage of patients with bone changes who have the clinical signs indicated.*

Type of bone change	Patients	Anesthesia		Ulcer or scar		Reactions		None
	No.	No.	%	No.	%	No.	%	No.
Nonspecific	402	353	87	258	65	45	11	24
Specific	128	102	80	51	40	36	28	14
Osteoporosis	90	76	84	32	35	14	15.5	4

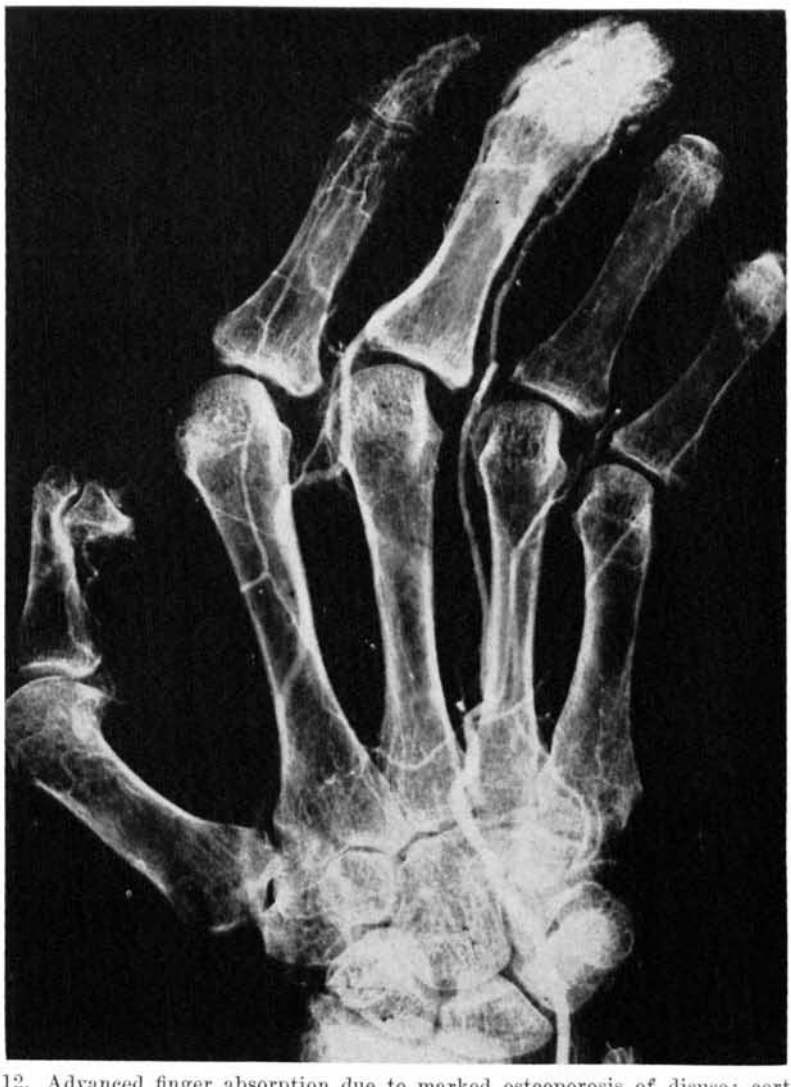

FIG. 12. Advanced finger absorption due to marked osteoporosis of disuse; cortices thin, and trabeculae absent. Arteriogram, showing hyperemia of recently reinfected long finger.

of ulceration and scarring were known to be incomplete. Records of nerve lesions were more reliable.

Table 7 shows that there were 33 per cent of patients with pain, and 41 per cent of patients with leprosy nodules or ENL nodules, who developed specific bone changes. Osteoporosis is seen to be more closely associated with paralysis, pain, and lepra reactions than with anesthesia or ulceration.

7. *Correlation of clinical signs with bone changes.*—In each patient's x-ray record the various clinical and radiographic signs of changes were recorded per digit or ray. For the purposes of statistical analysis the index and little fingers were selected as being indicative

TABLE 9.—*Correlation of clinical signs with bone changes in selected digits.*

Clinical signs and bone changes	No. of digits showing signs of lesions								No. of digits	
	Hands				Feet				Wrist	Ankle
	Right		Left		Right		Left		Carpus	Tarsus
	Index	Little	Index	Little	Hallux	Fifth	Hallux	Fifth	R & L	R & L
CLINICAL SIGNS										
Nonspecific										
Anesthesia	255	440	262	437	433	437	442	440		
Paralysis	134	256	153	256	15		13		22	149
Contraction	73	114	80	124	7		8			
Pain	19	17	19	22	23		16		2	12
Infection and ulcers	45	41	40	42	125	103	112	84		47
Trauma and hematoma	18	18	19	19	13	13	12	11		
Specific										
History of reactions	28	28	55	32	23		21		4	4
BONE CHANGES										
Nonspecific										
Soft Tissue swelling	31	27	32	26	18	11	14	7		
Osteitis (active)	3	1	3		17	13	19	9		33
Arthritis (active)	4	7	7	13	48	32	36	41	6	14
Absorption	77	71	84	65	137	97	108	85		7
Specific	11	14	19	18	36		23		3	6
Osteoporosis										
Minimal	50	60	45	65	25	25	24	22		3
Advanced	5	5	9	9	10	10	15	13		

of median and ulnar nerve lesions, and for the foot the great and little toes were selected.

On going through the radiographic records there is found a striking correlation between infected or ulcerated digits and the various nonspecific bone changes, and between those with leprosy nodules and lepra reactions and the specific bone changes.

Table 9 is an attempt to summate the findings in the selected digits, of the 894 patients. In the hands, trauma and hematoma, infection and ulceration roughly correspond to bone absorptive changes in the digits, and in the foot there is very close correlation between ulceration and absorptive bone changes. Only 50-60 per cent of digits with anesthesia developed bone absorptive changes, but almost all digits with anesthesia and ulceration develop those changes.

8. *Further research needed.*—Further research is needed on the following lines: (*a*) To determine the cause of specific bone changes, accurate clinical records of each leprosy or reaction nodule in the fingers and toes should be kept. Accurate records of any tight, whitish skin lesion or edematous skin lesion of the fingers or toes should be kept. In the presence of acute painful skin lesions, radiographs should be taken at monthly intervals, and then there should be close correlation between these skin lesions and radiographic findings. Where a bone lesion occurs, lateral radiographs must be taken of each finger separately, and bone-drill biopsies should be made.

(b) To determine the nature of the "nutrient foramens" in fingers, postmortem finger arteriograms and venegrams, using colloidal barium or colored latex injections, should be made. Microradiograms and stereoscopic microphotographs after decalcification of bone could establish the exact arterial and venous network in the phalanges. In leprosy patients with enlarged nutrient foramens, anteroposterior and oblique angiograms of the fingers should be made. Bone biopsy at the site of a nutrient foramen might be justified.

(*c*) The exact nerve supply to the bone of phalanges should be worked out by special methods of decalcifying bone and serial sections. The role of pain fibers in the mechanism of tissue repair and replacement should be further investigated.

(*d*) Nonspecific bone absorption changes should be induced in the hands and feet of experimental animals after selective nerve section or damage.

(*e*) Bone biopsies in various stages of nonspecific bone change should be carefully correlated with radiographic findings.

(*f*) The "ditching sign" in contracted fingers should be noted and correlated with the results of physiotherapy.

CONCLUSIONS

1. Three types of bone changes occur with the following incidences in leprosarium patients: (*a*) specific osteitis leprosum, 3-5 per cent;

(*b*) nonspecific osteitis, 45 per cent; and (*c*) disuse osteoporosis, 10 per cent.

2. The specific bone destructive changes are usually associated with lepra reaction. Severe destructive changes that could cause deformity are rare, only about 2-3 per cent of cases. Where a painful lepra reaction or lesion occurs in a digit, that digit should be x-rayed at about monthly intervals to see if there is bone change.

With properly-controlled chemotherapy the acute reaction stage will subside, and with adequate immobilization deformity can be avoided and healing of bone with sclerosis will take place in about one year. Joint surfaces are not usually involved.

3. The nonspecific bone absorptive changes occur in about 56 per cent of patients who have anesthesia of the hands or feet, but where trauma, ulceration and nonspecific infection occur, the incidence of bone damage increases to 80-90 per cent.

Even in the presence of anesthesia, if, by adequate care and training in occupational therapy, minor trauma and ulceration can be prevented, then infection will not spread to bone and so bone and joint lesions can be prevented or arrested. In the absence of adequate care, or if there is continued occupational trauma, nonspecific infection will be modified by neurotrophic factors and will gradually cause absorption of the digits.

4. The most permanently crippling lesions are those affecting the joints. These occur in about 5 per cent of patients' feet and in about 1 per cent of the hands. Joint lesions are usually due to a nonspecific arthritis modified and made worse by neurotrophic factors. Tuberculous arthritis may occur, and trauma may cause development of a Charcot type of neurotrophic joint.

True lateral radiographs of each affected finger are needed for the diagnosis of interphalangeal arthritis, and for assessment of the degree to which physiotherapy can help to mobilize contracted joints. True lateral radiographs of the ankle joint are needed to assess tarsal changes, and anteroposterior and oblique views of metatarsal heads are needed in cases of trophic ulcer of the foot. Check radiographs should be taken at three-month intervals.

5. The angiographic findings in a few cases suggest a possible mechanism of the pathogenesis of the nonspecific bone absorption where there is sensory loss (clinical or subclinical), trauma, and/or very chronic infection.

(*a*) Normal higher control of vasodilatation, etc., is cut off by ulnar and medial nerve palsy. Even in the absence of a clinically detectable nerve lesion, Weddell ([20]) found evidence of up to 25 per cent reduction in free nerve fibers in the skin. There may thus be a loss of the normal mechanism of tissue repair and replacement, or a devitalization of tissues.

(*b*) In the presence of infection there is excessive local vasodilata-

tion, with stimulation of osteoclasis on the outer layers of bone.

(*c*) Later, soft-tissue and periosteal vascular end-loops are obliterated by fibrosis, and both osteoblasts and osteoclasts are inhibited.

(*d*) Vascular end-loops in the bone medulla may not be so obliterated, and a local nervous mechanism (due to continued use of the anesthetic digit) causes stimulation of osteoblasts, and the laying down of new bone on the medullary side of the cortex.

(*e*) Avascularity of the skin predisposes to repeated ulceration and infection.

(*f*) Aseptic necrosis of deep tissues due to trauma of devitalized tissue may sometimes erode or spread into bone, even in the absence of sepsis. Aseptic necrotic tissue may calcify in conditions such as scleroderma.

CONCLUSIONES

1. En los enfermos de las leproserías se presentan tres clases de alteraciones óseas con la siguiente incidencia: (*a*) osteítis leprosa específica, 3-5 por ciento; (*b*) osteítis anespecífica, 45 por ciento; y (*c*) osteoporosis por desuso, 10 por ciento.

2. Las alteraciones óseas destructivas específicas suelen asociarse con la reacción leprosa. Las alteraciones destructivas graves que pueden ocasionar deformidad son raras, representando approximadamente no más de 2-3 por ciento de los casos. Cuando aparece una reacción o lesión leprosa dolorosa en un dedo, hay que radiografiar ese dedo a plazos más o menos mensuales para averiguar si existe alteración ósea.

Con la quimioterapia debidamente fiscalizada la etapa de reacción aguda cederá, y con la inmovilización adecuada cabe evitar la deformidad y aproximadamente en un año se obtendrá la cicatrización del hueso con esclerosis. Las caras articulares no suelen afectarse.

3. Las alteraciones óseas absortivas anespecíficas se presentan aproximadamente en 56 por ciento de los enfermos que tenen anestesia de las manos o los pies, pero cuando sobrevienen traumatismo, ulceración e infección anespecífica, la incidencia de las alteraciones sube a 80-90 por ciento.

Aun cuando existe anestesia, si con la asistencia adecuada y adiestramiento en la terapéutica profesional, pueden impedirse los pequeños traumatismos y la ulceración, no se propagará la infecoción al hueso y cabe impedir o estacionar asi las lesiones óseas y articulares. A falta de asistencia adecuada, o si hay continuo traumatismo profesional, la infección anespecífica se verá modificada por factores neurotróficos y ocasionará paulatinamente la absorción de los dedos.

4. Las lesiones que baldan de modo más permanente son las que afectan las articulaciones. Estas aparecen aproximadamente en 5 por ciento de los pies de los enfermos y en 1 por ciento de las cabezas. Las lesiones articulares suelen deberse a artritis anespecífica, modificada y empeorada por factorés neurotróficos. Puede presentarse artritis tuberculosa y el traumatismo puede ocasionar la formación de una articulación neurotrófica por el estilo de la de Charcot.

Se necesitan verdaderas radiografías laterales de cada dedo afectado para el diagnóstico de artritis interfalangiana y para justipreciar hasta qué punto puede ayudar la psicoterapia a movilizar las articulaciones contraídas. Son necesarias también verdaderas radiografías laterales de la articulación del tobillo para determinar las alteraciones tarsianas y vistas anteroposteriores y oblicuas de las cabezas de los metatarsianos en los casos de úlcera trófica de los pies. Deben tomarse radiografías comprobadoras a plazos de tres meses.

5. Los hallazgos angiográficos obtenidos en algunos casos sugieren un posible

mecanismo de la patogenia de la absorción ósea anespecífica cuando existen pérdida sensorial (clínica o subclínica), traumatismo e/o infección muy erónica.

(*a*) El dominio más elevado normal de la vasodilatación, etc., se ve interrumpido por la parálisis de los nervios cubitales y medianos. Aun sin haber lesión nerviosa distinguible clínicamente, Weddell [7] observó signos de una reducción de 25 por ciento en las fibras nerviosas de la piel. Puede, pues, haber pérdida del mecanismo normal de regeneración y sustitución de los tejidos o desvitalización de éstos.

(*b*) Cuando existe infección, hay excesiva vasodilatación local, con excitación de la osteoclasia en las capas externas del hueso.

(*c*) Más tarde, el tejido blando y las anas terminales vasculares periósticas se ven borrados por la fibrosis y quedan inhibidos tanto los osteoblastos como los osteoclastos.

(*d*) Quizás no queden borradas las asas terminales vasculares de la médula ósea y un mecanismo nervioso local (debido al continuo uso del dedo anestesiado) provoca excitación de los osteoblastos y depósito de nuevo hueso en la cara medular de la corteza.

(*e*) La avascularidad de la piel predispone a repetida ulceración e infección.

(*f*) La necrosis aséptica de los tejidos profundos, debido al traumatismo de los tejidos desvitalizados puede algunas veces desgastar el hueso o difundirse a éste, aun no habiendo septicemia. El tejido necrosado aséptico puede calcificarse en estados tales como esclerodermia.

CONCLUSIONS

1. Trois types de modifications osseuses surviennent chez des malades hospitalisés en léproserie. Se sont, avec leurs fréquences respectives: (a) l'ostéite lépreuse spécifique, 3-5%; (b) l'ostéite non-spécifique, 45%; (c) l'ostéoporose d'inactivité, 10%.

2. Les modifications destructives spécifiques des os sont généralement associées avec la réaction lépreuse. Les graves modifications destructives, susceptibles d'entraîner des mutilations, sont rares, seulement 2-3% environ des cas. Lorsque survient au niveau d'un doigt une réaction lépreuse, ou bien une lésion, douloureuse, ce doigt devrait être radiographié à des intervalles d'environ un mois, afin de voir s'il se produit des modifications osseuses.

La phase aigüe de la réaction regressera par un ajustement convenable de la chimiothérapie, et les mutilations pourront être évitées par une immobilisation adéquate, la guérison des os avec sclérose prenant un an environ. Les surfaces articulaires ne sont généralement pas atteintes.

3. Les modifications non-spécifiques de résorption osseuse surviennent chez 56% environ des malades qui présentent de l'anesthésie des mains ou des pieds. Cependant, là où surviennent traumatismes, ulcérations, ou infections non-spécifiques, la fréquence des délabrements osseux atteint 80-90%.

Même en présence d'anesthésie, l'infection ne se transmettra pas à l'os si les traumatismes mineurs et les ulcérations peuvent être prévenus par des soins adéquats et par la pratique de l'ergothérapie. Les lésions osseuses et articulaires peuvent ainsi être évitées ou arrêtées. En l'absence de soins adéquats, ou s'il existe un traumatisme professionnel persistant, l'infection non-spécifique sera modifiée par des facteurs neurotrophiques et entraînera progressivement la résorption des doigts.

4. Les lésions les plus irremédiablement géneratrices d'invalidité sont celles qui touchent les articulations. Elles surviennent au niveau des pieds chez environ 5% des malades, au niveau des mains chez environ 1%. Les lésions articulaires sont géneralement dues à une arthrite non-spécifique modifiée et aggravée par des facteurs neurotrophiques. Une arthrite tuberculeuse peut survenir, et le traumatisme peut entraîner le développement d'une lésion articulaire neurotrophique de type Charcot.

Des radiographies franchement de profil de chaque doigt interessé sont indispensables pour diagnostiquer l'arthrite interphalangienne et pour apprécier dans quelle mesure la physiothérapie peut être utile pour mobiliser les articulations en contracture. Des radiographies franchement de profil sont indispensables pour se rendre compte des

modifications tarsiennes, et des clichés antéro-postérieurs et obliques des têtes métatarsiennes sont nécessaires dans les cas d'ulcères trophiques du pied. Des radiographies de contrôle devraient être répétées tous les trois mois.

5. Dans quelques cas les trouvailles artériographiques suggèrent un mécanisme éventuel pour expliquer la pathogenése de la résorption non spécifique de l'os, lorsqu'il y a perte (clinique ou subclinique) de la sensibilité, traumatisme, et/ou infection chronique accentuée.

(a) Le contrôle supérieur normal de la vasodilatation, etc. . . ., est supprimé à la suite de la paralysie du nerf cubital et du nerf médian. Même en l'absence de lésion nerveuse cliniquement décelable, Weddell (7) a mis en évidence une réduction des filets nerveux libres dans la peau atteignant jusqu'à 25%. Il peut dès lors exister une perte du mécanisme normal de la réparation et du remplacement tissulaire, ou une devitalisation des tissues.

(b) En cas d'infection, il se produit une vaso-dilatation locale excessive, avec stimulation de l'ostéoclasie au niveau de la corticale externe de l'os.

(c) Plus tard, les anses vasculaires terminales dans les tissus mous et dans le périoste sont oblitérées, et les ostéoblastes et ostéoclastes sont à la fois inhibés.

(d) Dans la cavité médullaire de l'os, les anses vasculaires terminales peuvent ne pas être oblitérées de cette manière, et un mécanisme nerveux local (par suite de l'usage persistant du doigt anêsthésique) entraîne la stimulation des ostéoblastes et l'apposition d'os nouvellement formé au niveau de la corticale médullaire.

(e) L'absence de vascularisation de la peau prédispose à la répetition de l'ulceration et de l'infection.

(f) La nécrose aseptique des tissus profonds, due au traumatisme du tissu dévitalisé, peut parfois, même en l'absence d'infection, se communiquer à la surface ou à l'intérieur de l'os. Le tissu ayant subi une nécrose aseptique peut se calcifier dans certaines conditions, telle la sclérodermie.

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