

# New Knowledge of Leprosy through Paleopathology

Vilm. Møller-Christensen, M.D.<sup>1</sup>

I shall now give a short survey of some new knowledge of leprosy, obtained by use of a special research method that I call osteoarcheology, which is a further development of paleopathology.

In November 1964 I opened a new department of the Copenhagen University Medical Historical Museum, which is now named The Museum of the History of Leprosy, Tuberculosis and Syphilis. It is illustrated in Figures 1 and 2.

The exhibit is informative especially on two main problems, viz., (a) the *facies leprosa*, and (b) the *usura orbitae*.

<sup>1</sup>Prof. of Medical History, Medical Historical Museum, Copenhagen University, Copenhagen K, Denmark.

## *FACIES LEPROSA*

A skull with *facies leprosa* shows atrophy of the anterior nasal spine in 83 per cent of the cases, combined with central atrophy of the maxillary alveolar process. These changes are always accompanied by inflammatory changes of the superior surface of the hard palate. Figures 3 and 4 illustrate cases of *facies leprosa*.

The nasal spine atrophy occurs in three degrees: (a) a well-defined reduction of the anterior nasal spine, (b) an advanced nasal spine atrophy, although a distinct but very small nasal spine remains, and (c) complete obliteration of the nasal spine.

Atrophy of the maxillary alveolar process also has been classified in three groups ac-



FIG.1. The Museum for the History of Leprosy, Tuberculosis, and Syphilis, erected in a building from the year 1792.

cording to degree: (a) the first is characterized by unquestionable recession upward of the prosthion and a slight exposure of the roots of the central incisors; (b) the second is marked by extension, in both depth and breadth, of the atrophic bone area, whereby the prosthion recedes still further upward, thus exposing the roots of the central and lateral incisors to such a degree that looseness of one or more of these teeth must be expected; (c) in this group the bone atrophy is severe, and at least one tooth, generally one of the central incisors, has presumably been lost *in vivo*, or the alveoli have been obliterated.

The inflammatory changes of the nasal surface of the hard palate are also classified in three groups according to degree: (a) the first represents the earliest recognizable pathologic changes, viz., small, closely spaced pits, usually localized near the transverse palatal suture or at the lateral surface of the nasal crest; (b) the second shows more advanced stages of these changes, which may be interpreted as reactions ascribable to the local periosteal inflamma-

tion extending into the bone itself; these changes may be of a destructive or reactive nature, or a combination of the two; (c) the third shows destructive changes so advanced that irregular perforations of the bone occur.

In cases of leprosy, pathologic changes in other parts of the skull, except in the orbit, the *usura orbitae*, have never been observed.

In syphilis we find the skull cap to be the part of the skull predominantly showing characteristic changes, i.e., *calvaria syphilitica* (Fig. 7). Such changes must be distinguished from destructive changes occurring after burial (Fig. 8).

In 1953 I published a description of the changes in *facies leprosa* (<sup>1,2</sup>). This syndrome seems to represent specific changes in advanced cases of lepromatous leprosy. In the meantime several leprologists have tried to explain the pathogenesis of *facies leprosa*. In 1953 Professor Erik Waaler of Bergen, Norway, published the results of an examination of four ancient specimens with leprosy from the Armed Forces Institute of



FIG. 2. The interior of the leprosy section with a horseshoe-shaped exhibition case for demonstration.

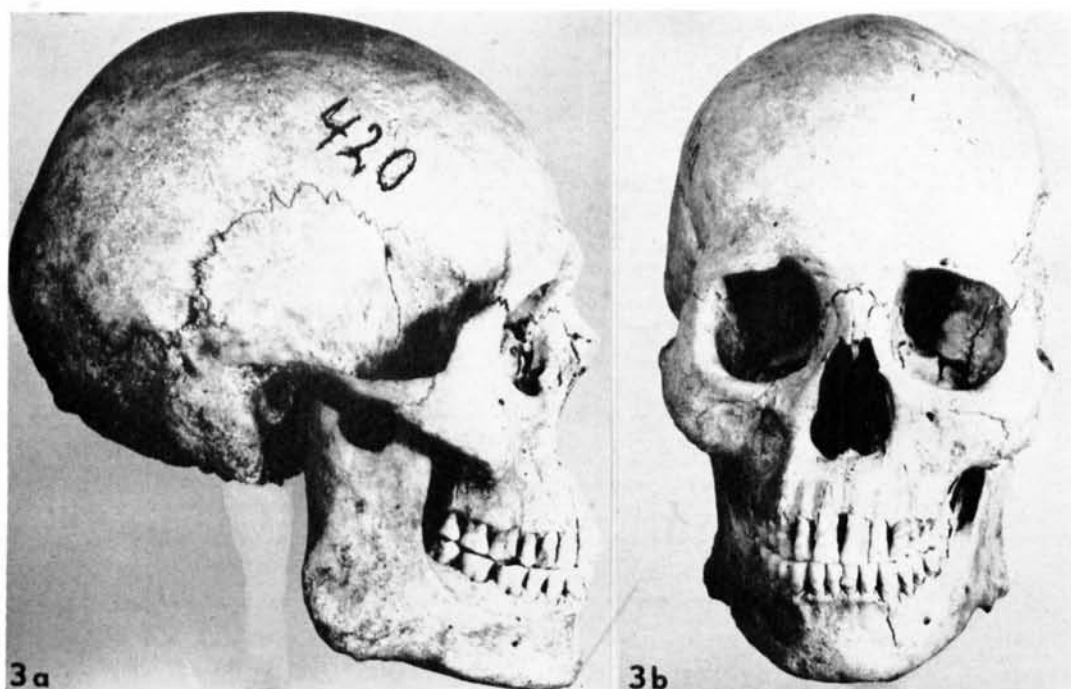


FIG. 3 a & b. Case with *facies leprosa*. (Ref. 1—Plate II, Nos. 7, 8).<sup>1</sup>

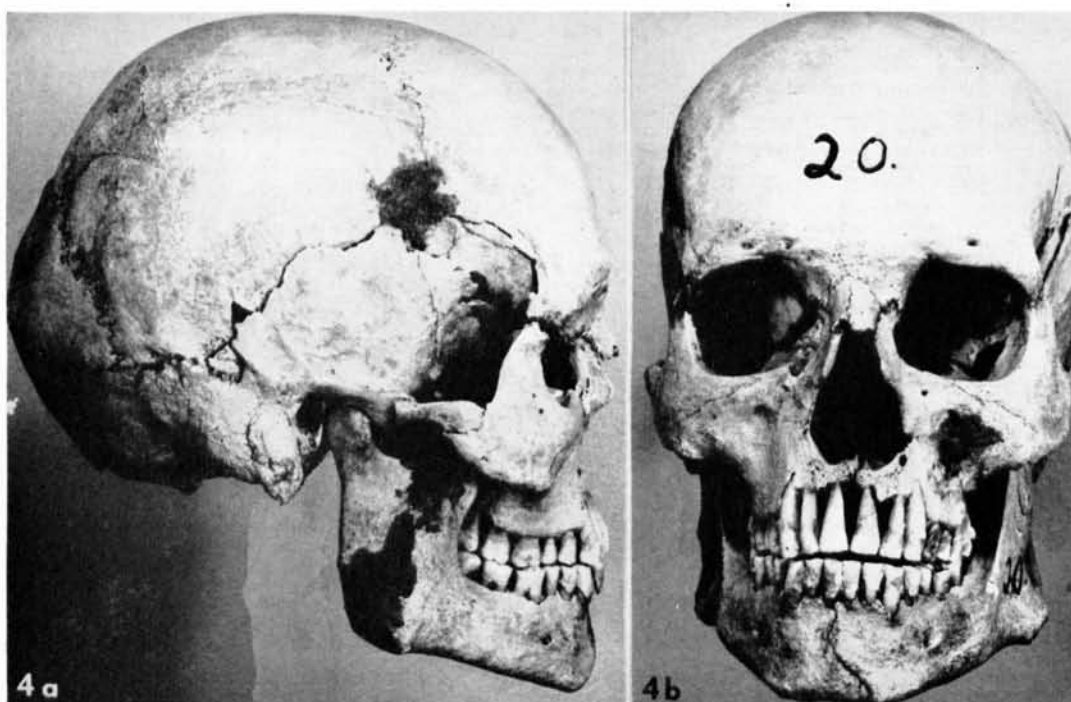
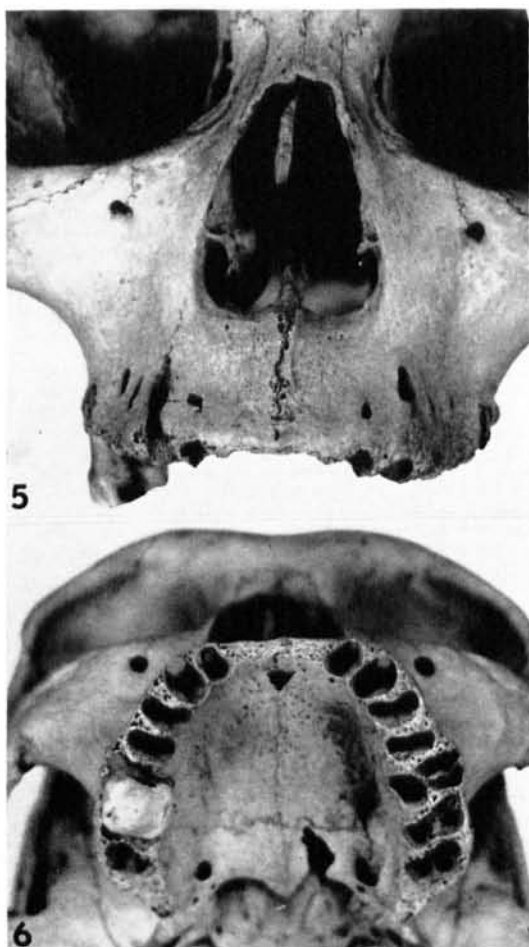


FIG. 4 a & b. Case with *facies leprosa*. (Ref. 1—Plate II, Nos. 17, 18).<sup>1</sup>

<sup>1</sup>Ref. and plate numbers in parentheses in these and subsequent figures refer to Møller-Christensen (1).



FIGS. 5 & 6. These illustrate a normal skull with antemortem traumatic loss of the upper incisors. The anterior and the posterior walls of the alveoli are joined to form a compact bridge between the lateral incisors without loss of bone. (Ref. 2, 12).

Pathology in Washington, D.C., U.S.A. In one of these cases he noted that the atrophy of the maxillary alveolar process was identical with that found in one of the medieval skeletons from Naestved. As the teeth here had been preserved *in situ*, the bone atrophy could not be considered due to any inactivity of the jaw. Waaler also demonstrated that the atrophy of the anterior nasal spine had in no case resulted from an inflammation of the bone.

Findings from the specimens he examined showed exclusively atrophy and bone resorption, combined with chronic in-

flammation of the underlying connective tissue. A specific nerve affection of a leprous character was also observed in the small nerves in the mucous membranes.

Waaler has suggested two explanations of these lesions, viz., (1) They may be a consequence of the nerve affection and thus pathogenetically of the same type as the characteristic bone atrophy seen in fingers and toes. (2) They may represent traumatic atrophy caused by persistent chronic inflammation of the adjacent soft tissue.

A fellow-member of Waaler's medical team, R. Melsom, examined 5 out of 7 patients in Bergen with *facies leprosa*. It was not possible in any of the cases to demonstrate an anesthesia indicating that nerves in this region were affected.

Waaler's conclusion was that it is hardly justifiable on the basis of Melsom's clinical studies, mentioned above, to assume that the disturbance is of a neurotrophic nature; however, the nerve affections demonstrated here indicate a possibility that this is the case. Whether one or the other of the theories of pathogenesis will come to the fore cannot be decided on the basis of the present material. The affection, however, is hardly a destructive leprous one, and it is certainly not a specific leprous inflammation.

According to Waaler this lesion cannot be considered a specific finding of leprosy, but rather a characteristic symptom of the disease. He enumerates the following diseases in which bone changes in the facial aspect are encountered: harelip with cleft palate, lupus vulgaris, and syphilis. In their further manifestations, however, these show highly individual characteristics not to be confused with the changes seen in *facies leprosa*.

Later on, Professor J. Michman from Jerusalem, who visited Copenhagen a few years ago and had occasion to study some of the skulls, told me that in his opinion the atrophy of the maxillary bone was due to the innumerable microtraumata caused by mastication; he noted that when the incisors bite, the pressure they exert can be as much as 60 pounds by actual measurement. In an area already infected by leprosy, the



FIG. 7 a & b. A skull with *calvaria syphilitica* (Ref. 2, 12).

persistent trauma of the act of biting may well bring on central resorption that results eventually first in loosening and then in loss of the incisors.

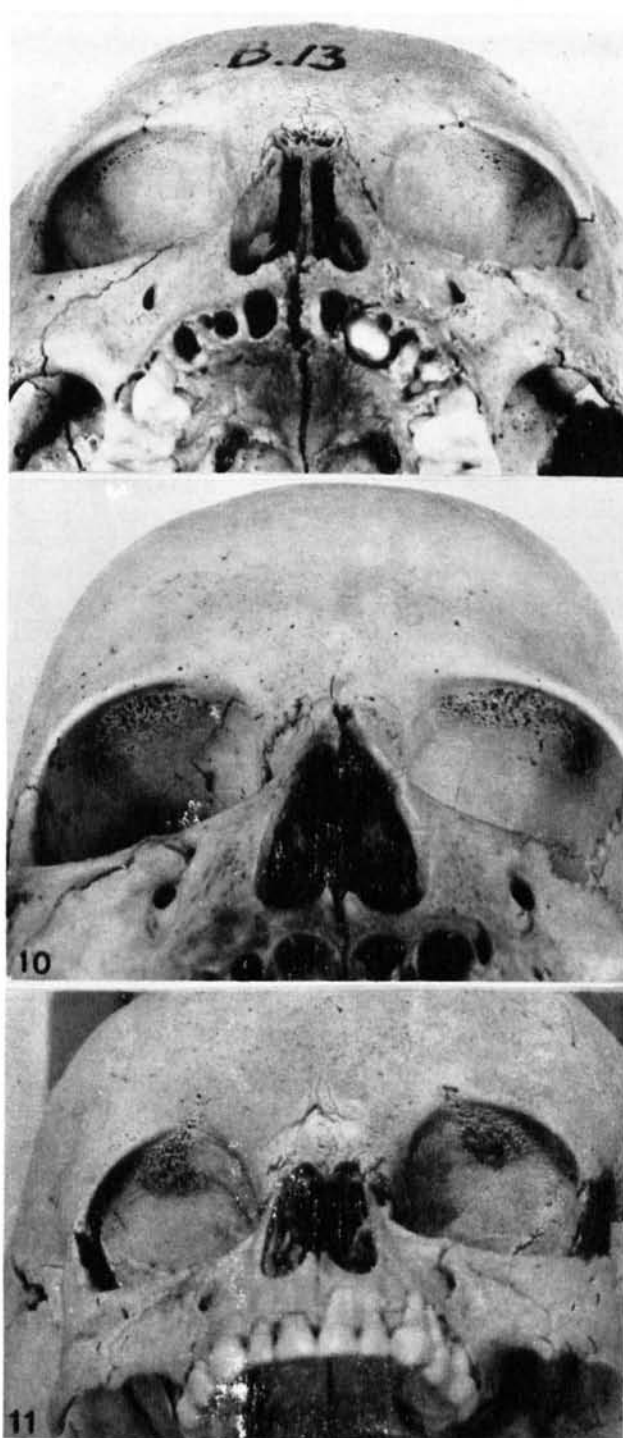
Three years ago, while visiting Copenhagen, Professor Paul Brand gave me the following explanation: When leprosy patients experience the first inflammation in the nose, which blocks it up, they begin to breathe through the mouth. This respiration through the mouth must result in cooling of the central part of the alveolar maxillary process. Leprosy bacilli grow best in cool areas. Breathing through the mouth gives these bacilli an ideal temperature in which to flourish in the bone tissue. So the act of biting, with its innumerable microtraumata may set off the typical bone resorption of *facies leprosa*.

To prove this hypothesis scientifically it will be necessary to make microscopic examinations, either postmortem or by biopsies.



FIG. 8. A skull with postmortem changes probably due to beetles, crabs, ants, etc. (Ref. 2, 17).





FIGS. 9, 10 & 11. Skulls to show increasing severity of *usura orbitae* (Ref. 3, 127; (1) V, 9).

### USURA ORBITAE

In the leprosy material *usura orbitae* or *cribra orbitalia*, i.e., the presence of bilateral cribriform changes in the orbital roof of symmetric appearance and occurrence mainly in the region of the lacrimal fossae, was noted in 67.9 per cent of skulls from 100 skeletons with leprotic changes in hands and feet.

In the Danish material there is no difference in sex incidence. In groups belonging to the same historic period and area, *usura orbitae* is found 134 per cent more frequently in leprotic than in nonleprotic skulls. Obviously leprosy or one of its complications is responsible for this difference, and this indicates that *usura orbitae* has some pathologic significance.

If *usura orbitae* were found only in leprosy skulls, the position would be clear; it is, however, in no way so restricted. If it is the result of pathologic changes, then other diseases than leprosy must have an etiologic significance and it may be that the lacrimal gland is affected in some way. Leprosy patients sometimes show marked protrusion of the upper eyelids, and it is known that leprosy may affect primarily or secondarily the eyelids, cornea, uveal tract, and the lacrimal sac.

I am not aware of any published accounts of the appearance of the lacrimal gland in leprosy, but it is known that tuberculosis, sarcoidosis, and syphilis rarely affect the orbital contents. The lacrimal gland is sometimes involved in disease processes that affect the salivary glands primarily; most of these conditions are rare. If *usura orbitae* is the result of a disease process, that disease must be a common one, affecting wide areas of the globe and known to be of great antiquity. The higher incidence of *usura orbitae*, which we find in primitive people, might reflect only poor nutrition, indifferent hygiene, and a higher morbidity rate. The only diseases that fulfill these criteria are mumps and, possibly, smallpox. These are common diseases, widely spread among all races and known from the time of ancient Greece. All age groups may be infected, but the diseases are commoner in childhood (<sup>3</sup>).

These remarks remain entirely speculative. In my opinion, one way to study the problems of *usura orbitae*, in a case of advanced lepromatous leprosy, is to conduct a postmortem examination, open the *cavum cranii*, remove the orbits with contents, and then make a proper histopathologic examination. Probably it would be possible to find the same bone changes that we have found in about 70 per cent of the skulls with *facies leprosa*.

### REFERENCES

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3. MØLLER-CHRISTENSEN, V. and SANDISON, A. T. *Usura orbitae* (cribra orbitalia) in the collection of crania in the Department of Anatomy, University of Glasgow. Pathol. & Microbiol. (Basel) **26** (1963) 175-183.

**Dr. Binford.** Thank you very much, Dr. Møller-Christensen. The next time any of you are in Denmark, you must visit Professor Møller-Christensen and see his interesting display. I meant to say at the beginning that this work of Professor Møller-Christensen probably establishes for the first time anatomic evidence that leprosy truly existed in Europe in medieval times. Some medical historians have minimized the importance of leprosy in the so-called lazarettos or leprosy hospitals in Europe during this period. They have said that these hospitals housed patients with all kinds of disease, and that the leprosy prevalence among the inmates has been greatly exaggerated. Professor Møller-Christensen's work in Denmark shows not only that persons with leprosy were interred in these burying grounds, but that as many as 70 per cent of the skeletons in the grounds showed evidence of the disease. We know today that many patients with leprosy do not show x-ray evidence of the disease; therefore one can assume that many of the inmates whose skeletons make up the remaining 30 per cent also had the disease.