to the tympanum, so that colder air coming up the Eustachian tube would be immediately mixed with warmer air from the antrum? The tympanic orifice of the tube points toward the aditus, and, although the ossicles come between, the incoming air will go toward the wall on which opens the aditus. Every time we perform Valsalva's experiment the tympanic membrane goes outward and warm air must necessarily be sucked out of the antrum.

REFERENCES.

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OTOSCLEROSIS.[1]

BY DR. GUSTAVE BRÜHL (Berlin).

(Abridged translation by DAN MCKENZIE.)

OF recent years it has been generally agreed that the deafness characteristic of otosclerosis is the result of an osseous ankylosis of the stapes in the oval window, the middle ear in other respects being free from abnormality. With regard to the ætiology of the disease, however, there is still much difference of opinion. Questions have been asked as to the original seat in the bone of the lesion which induces the ankylosis. Is it to be found in the periosteum or in the capsule of the labyrinth? And if in the latter, in which layer of the bone, the internal or the external, does it occur?

All the agencies with which we are acquainted as causing bone disease in general have, at one time or another, been looked upon as being of importance in the production of otosclerosis. But none of these assumptions have satisfied the requirements.

Quite recently, indeed, doubts have actually been thrown upon the significance of the ankylosis of the stapes itself, and the classical view which looks upon the fixation of that ossicle as the *causa causans* of the symptoms of otosclerosis has been submitted to searching criticism. The grounds for this criticism lie in the fact

[1] From *Les Archives Internationales de Laryngologie, etc.*, January–February, 1911, p. 1. We desire to express our indebtedness to the editor of that journal for kindly permitting us to use the blocks of the diagrams illustrating the article.

that osseous changes similar to those which exist in and around the ankylosed stapes have been found elsewhere in the petrous bone. And, indeed, such widespread lesions may occur without the stapes being itself affected. The disease in which, up to the present, these changes have been found to be most prevalent, is congenital or hereditary atrophy of the nerve elements in the labyrinth. Moreover, in cases where the stapes has undergone ankylosis without any sign of bone disease elsewhere, atrophic changes in the labyrinth are by no means uncommon. The old idea was that these atrophic changes were secondary to the ankylosis, and that they were of the same nature as the labyrinth degenerations which so frequently follow other diseases of the middle ear. But it has lately been suggested [1] that the atrophy of the labyrinth is the prime factor in the situation, and that the ankylosis of the stapes is only a secondary or accidental phenomenon. That is to say, that otosclerosis is nothing but one of the varieties of a disease of the internal ear—of degenerative hereditary deafness, to wit. Thus, in spite of all the clinical and microscopic research of the last twenty years, the picture of otosclerosis is still blurred and indistinct.

My own personal views on the essential nature of the disease are the outcome of a series of anatomical investigations upon the auditory organs of eight individuals, whose hearing had been carefully examined during their life-time. The specimens include examples of simple ankylosis of the stapes as well as examples of atrophy of the labyrinth without ankylosis.

Before going on to describe what I found in these specimens I shall give a brief sketch of the normal condition of the region of the stapes.

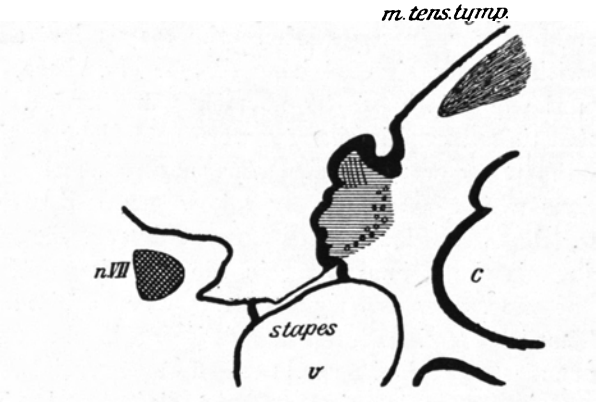
The osseous capsule of the labyrinth is composed of two layers of bone; first, an external layer, covered with connective tissue, and contiguous to the mucous membrane of the middle ear; and secondly, an internal layer derived originally from cartilage, and extending into the periosteum lining the interior of the labyrinth. In this internal layer there are numerous islets of unossified cartilage even in the adult bone. The foot-plate of the stapes in the oval window is invested with a delicate covering of mucous membrane, the deeper layers of which merge into and become identified with the periosteum of the ossicle.

In otosclerosis certain areas of the bone undergo a process of porosis or spongification, the tissue of which is characterised, *inter*

[1] Manasse, *Verhandlung. der Deutsch. Otolog. Gesellsch.*, 1909.

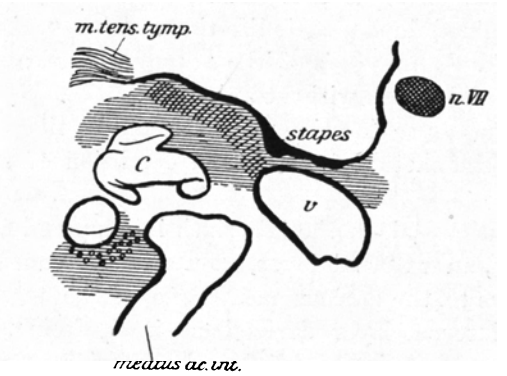
alia, by a strong affinity for microscopic stains. These disease-areas or foci are separated by a clear line of demarcation from the hard, eburnated, and compact bone around them. The new bone is very vascular. It is rich in cell-constituents, and the cells are

FIG. 1.



large and irregularly distributed. In addition to those features, the bone of the disease-areas presents a considerable difference in the size and contents of its medullary spaces, and there is also an obvious difference in its age. The clinical effect of the lesions,

FIG. 2.



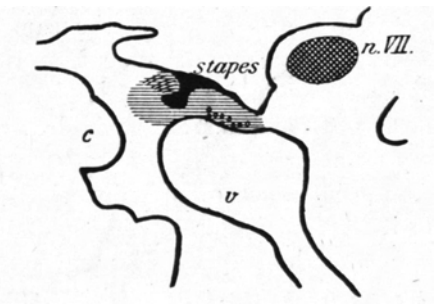
like those of certain pathological changes in the central nervous system, depends upon their situation and upon their extent.

My preparations fall into four groups, according to the situation of the hyperostoses: (1) Those in which the changes in the bone lie in the vicinity of the stapes, *without* ankylosis of the foot-plate (Fig. 1); (2) those in which the focus is limited to the region of the oval window and stapes, *with* ankylosis of the foot-plate

(Fig. 2); (3) those in which multiple and extensive hyperostoses are present *with* ankylosis (Fig. 3); and (4) those in which the hyperostoses are situated some distance from the stapes and in which ankylosis has *not* occurred (Fig. 4).

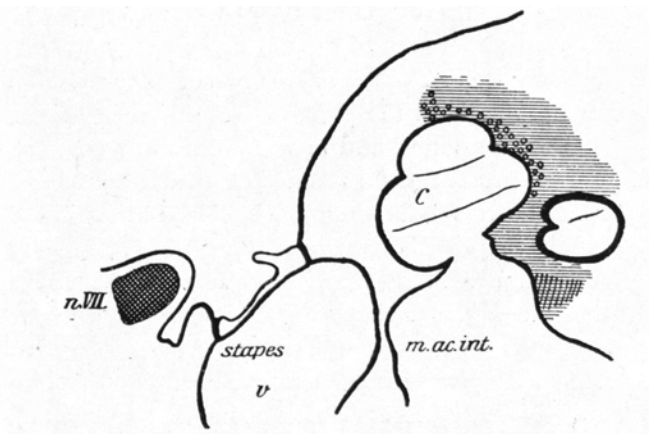
The first point that calls for remark in examining my specimens

Fig. 3.



is the observation, not, of course, a new discovery, that the bone in front of the fenestra ovalis is always more seriously affected than that lying posterior to it. The second is that the site of origin of the change in the bone appears to have been at the spot where the bone is oldest—that is to say, superficially, just under the peri-

Fig. 4.



osteum. Further, in almost all the specimens, in addition to those foci of proliferation which are situated at the region of the older bone, we find others extending far beyond the region of the stapes and promontory. In all the specimens, also, obvious new formation of bone by osteoblasts and resorption by osteoclasts is visible. This observation applies particularly to the younger areas of

diseased bone. The disease-areas present, as it were, the appearance of an enucleable foreign body.[1] In only two of the specimens (Fig. 4) are the changes confined to that part of the bony capsule which is developed from endochondrium.

We find, then, first, deafness affecting patients in whose petrous bones osseous new formations, occurring in close proximity to the oval window, have led to ankylosis of the stapes; and secondly, deafness occurring in patients in whom the same kind of bony transformation is situated at some distance from the oval window, without having induced any ankylosis.

Do these facts disturb the conventional ideas of otosclerosis? In my opinion they do not.

With regard to the influence of atrophy of the labyrinth, we cannot ascribe to it any particular ætiological importance in the production of ankylosis of the stapes. To begin with, such atrophic or degenerative processes in the labyrinth are constantly present in suppuration and other diseases of the middle ear. But no one would dream of attributing to these nerve-lesions any *rôle* in the causation of the suppuration. Moreover, in young patients the stapedial lesion is so definite and striking that the nerve degeneration can have but a secondary and subsidiary place in the evolution of the malady. It is, indeed, impossible to imagine by what means atrophic changes in the labyrinth could provoke such a definite and unmistakable lesion as that of ankylosis of the stapes.

The cases, on the other hand, in which foci of bony transformation are distributed throughout the petrous bone without affecting the neighbourhood of the stapes, and in which atrophic changes in the labyrinth are also present, seem to me to be different, ætiologically speaking, from those in which ankylosis of the stapes exists. In the former we have probably to do with trophic disturbances, for such disturbances are more likely to affect bone formed from endochondrium, seeing that that kind of bone is poorly nourished, and exhibits a marked proclivity to undergo resorption. Still, even when we grant this possibility, it is difficult to establish any link between the disseminated lesions in the bone and an atrophy of the labyrinth. We may, to be sure, refer both these conditions to one and the same disturbance of nutrition. But it must be remembered that the bone disease may exist without any sign of disease in the labyrinth. At all events, if deafness is present it is due to the atrophy of the nerve elements in the labyrinth, and so does not, of course, present the same characters as the typical middle-ear deaf-

[1] Alexander, *Arch. f. Ohrenheilk.*, lxxviii.

ness of otosclerosis. If the atrophy of the labyrinth were the initial lesion in otosclerosis, then the sufferers from that disease would come to us with the signs, not of middle-ear deafness, but of nerve deafness.

In the pathological picture of otosclerosis there is only one constant concomitant to the stapedial ankylosis, and that is a fibrous thickening of the mucous membrane of the middle ear close to the disease focus in the bone, and without doubt secondary to it.

The porotic change in the osseous tissue cannot be regarded as inflammatory. Dr. Orth, after an examination of my specimens, came to the same conclusions with regard to the osseous lesions as Starem did after examining Siebenmann's preparations,[1] namely, that the change in the bone consists in a spongy hyperostosis produced probably as follows: The vessels in the periosteum enlarge, and at the same time the bone, which is developed from periosteum, undergoes resorption, its place being taken by new-formed porous bone.

To call a process such as this "periostitis" or "osteitis" would not bring us any nearer to a solution of the problem. Besides, periostitis and osteitis in the bone of the ear are conditions so well defined that it would be better to avoid using these terms in discussing a lesion so different from them as that which induces ankylosis of the stapes.

Bony ankylosis of the stapes as a result of inflammations or adhesions in the middle ear may doubtless occur, but it must be very rare. And when it does occur it can only be looked upon as a purely accidental consequence of the inflammatory disease.

For these reasons, the best name for the lesion present in otosclerosis is *spongy hyperostosis in the oval window*. Siebenmann's term, *spongy transformation of the petrous bone*, may be used to designate that group in which foci of new-formed bone develop some distance from the oval window, and do not necessarily lead to ankylosis.

We now come to consider why it is that the bone in the neighbourhood of the stapes is so liable to this transformation. Are there any conditions in this region which favour such a change?

In order to elucidate these problems I submitted my preparations to Prof. Gebhardt, of Halle, the well-known authority upon transformations of bone. His opinion coincided with mine. And my opinion is, that the changes in the bone which lead to the typical

[1] *Zeitschr. f. Ohrenheilk*, xxxiii.

ankylosis of the stapes are the result of the action of forces of traction and compression. The neighbourhood of the articulation of the stapes is a likely spot for the development of proliferation in the bone for the following reasons: Immediately in front of the oval window the tendon of the tensor tympani passes over the "arciform fasciculus" on its way to the malleus; secondly, the movement of the stapes is more extensive in the anterior than in the posterior pole of the oval window. The continual pulling of the tendon together with the friction of the foot-plate of the stapes on the anterior border of the foramen ovale produces an incessant movement of the periosteum and an irritation of the bone lying between the tendon and the annular ligament of the stapes. The cartilaginous covering of, and the cartilaginous residua in, the bone at this place render it very liable to transformation. To sum up, the traction and friction induce hyperæmia and a tendency to proliferative processes in the periosteum and in the bone.

As a rule, the formation of hyperostosis remains limited to the oval window, but in some varieties (Fig. 3) isolated areas of transformed bone may also be found elsewhere in the capsule of the labyrinth. In such cases the focus in the vicinity of the stapes is the oldest. We may therefore assume that the production of the diffused foci is set agoing by the initial deposit at the oval window.

The fact that in typical cases the ankylosis of the stapes is, however, limited to the neighbourhood of the oval window favours my theory of the ætiology. For as soon as the articulation becomes ankylosed the irritating movements come to an end, with the result that there is no further proliferation. Another point in its support is that otosclerosis is rarely found in diseases of the tympanic cavity, in which the stapes or the tensor tympani have been immobilised by adhesions, etc.

That only comparatively few individuals suffer from otosclerosis may be accounted for by supposing a peculiar predisposition like that which leads to the formation of multiple osteomata elsewhere in the body—a predisposition which seems to be hereditary.[1] To this we may add the activity of growth about puberty, and the influence of syphilis, trauma, etc.

This theory of mine has some bearing upon treatment. When an

[1] Recent surgical opinion as to the causation of multiple osteomata refers these outgrowths to the influence of rickets, which, by disturbing and distorting the regular lines of ossification, leads to the displacement and isolation of islets of ossifying cartilage. From these displaced islets the osteomata arise.—*Trans.*

articulation is threatened with ankylosis as a result of constant irritation the obvious remedy is rest to the irritated parts. To this point Panse has already drawn attention. If, then, we were able to diagnose the presence of a hyperostosis at the oval window before the onset of deafness—say, by observing obvious discoloration of the membrane or hyperostoses elsewhere—we might arrest the disease by dividing the tendon of the tensor tympani, or by removing that part of the malleus to which it is attached.

In cases of ankylosis with an intact labyrinth, it might be advantageous to perform a mastoid operation, and to open a new fenestra at the tuberosity of the ampulla (of the external semicircular canal?), as hyperostosis rarely occurs at this place.

But before these suggestions can be confidently adopted, it will be necessary to carry out many more *post-mortem* investigations upon people whose hearing has been carefully tested during life.

ENDOTHELIOMA OF THE FRONTAL BONE.

By Neil Maclay, M.B., C.M.,
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W. F——, aged sixty-seven, the subject of illustration, consulted a doctor on February 15, 1911, on account of a painful swelling over his right orbit. The practitioner describes the condition as a red, fluctuating, circumscribed lump, the size of a walnut, situated over the inner limit of the right eyebrow. The presence of pus was diagnosed and an incision made; only blood escaped, and a friable new growth projected from the wound.

Four days afterwards I saw the patient and found a fungating mass, in situation already named, which entirely obscured any evidence of incision and extended fully $\frac{1}{4}$ in. from skin surface; the centre of growth corresponded with position of anterior wall of right frontal sinus.

A probe could be passed easily into sinus and down through fronto-nasal duct. Great destruction of bone had taken place; not only two thirds of anterior sinus wall but a good part of posterior wall had disappeared, and the growth extended $1\frac{1}{2}$ in. towards frontal lobe.

The pathological report submitted by Prof. Stuart McDonald described the structure as an endothelioma with much evidence of necrosis.

Surgical interference was not advised, and in spite of rapid growth little or no pain existed till a fortnight before death, which