Patients who have been operated on for empyema of antrum or other accessory sinus and in whom discharge persists or recurs I have ionised, some with pot. iod., some with soda salicylate with kathode in the tube, some with zinc sulphate with anode in the tube, but the results on the whole have been very poor. In only one case was the suppuration definitely stopped.

From my slight experience of the method I am led to believe that ionic medication will prove useful in some nasal conditions, and my purpose in describing my tubes is to bring before my *confrères* an inexpensive and simple means for applying the treatment.

## THE ETIOLOGY OF OTOSCLEROSIS.[1]

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### Part I.—Ætiology of Otosclerosis.

*Internal Secretions.*—Manasse has found in cases of goitre bony changes similar to those in otosclerosis. Improvement in otosclerosis has been noted on the administration of thyroid extract. Alexander, Siebenmann, and Mayer have observed deafness and bony changes in cretins exactly resembling those present in otosclerosis.

Sohier Bryant thinks that the cerebral pituitary, the suprarenals, the parathyroids, and the ovaries have an intimate relation to otosclerosis.

Axhausen says that changes in the internal glandular secretions —especially the thyroid—determine the dystrophic processes of bone. Epidemic osteomalacia occurs in goitre districts.

Ovarian extract may have an effect similar to thyroid extract, and it is noteworthy that some observers hold that the ovaries are the cause of osteomalacia. Hyperactivity of the ovary is believed

[1] For the making of this abstract Dr. Bryant has kindly supplied an English translation of his article on otosclerosis ("Die Ätiologie der Otosklerose," "Osteodystrophia petrosa Bryant") which appeared in the *Monatschrift für Ohrenheilkunde*, 1913, pp. 436 and 584. For the references the reader is referred to the original article. Dr. Bryant's article is interesting because it attempts to bring otosclerosis into line with other non-suppurative lesions of bone.—J. S. F.

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to cause reflex dilatation of the vessels. Cases of osteomalacia may be cured by ovariotomy.

It is supposed that the trophoneurosis is transmitted to the bone by way of the sympathetic fibres.

Bossi maintains that the suprarenal gland is the cause of osteomalacia and has reported cases of cure of osteomalacia in pregnant women by the use of suprarenalin.

Axhausen holds that the parathyroids influence osteodystrophic process and that the cerebral hypophysis may act in the same way.

*Female Sex.*—It is generally admitted that in otosclerosis the female sex is more affected than the male. This is probably due to parturition, disorders of menstruation, or prolonged lactation. Further, women suffer more often from anæmia.

Bezold's statistics show that from 57 to 66 per cent. of cases of otosclerosis occur in women, while only 42 per cent. of all ear diseases are found in the female sex. Seven per cent. of all ear cases suffer from otosclerosis.

*Intoxication due to Pregnancy or the Puerperium.*—The temporal bone may be affected by the bony changes which occur in the puerperium, and in this way otosclerosis may be produced. Politzer first suggested the relation of otosclerosis to the puerperium. He thought it was a true infective puerperal osteitis.

Veterinary surgeons note that milch cows alone are affected by osteomalacia.

Osteophytes on the skull occur in pregnancy.

Osteomalacia is rare in males, and after puberty osteomalacia occurs only among women.

*Infection from the Nasopharynx by way of the Eustachian Tube.*—Sohier Bryant holds that the commonest source of infection is the nasopharynx. He believes that toxins from this region affect the thyroid and also the hypophysis through reflex paths. In the same way puerperal pelvic infections cause dystrophic bone changes in the pelvis. Bryant believes that treatment of the nasopharynx may improve otosclerosis. He says that chronic infection of Luschka's tonsil is the most important factor, as this structure supplies the toxic material which affects the sympathetic nerve-supply of the pituitary bodies and thyroid gland. The results are tropho-toxic changes affecting the vessels of the petrous pyramid and resulting in the bone lesion of the labyrinthine capsule.

Mueller and Heimann also hold that abnormal conditions in the nose and pharynx are ætiological factors in otosclerosis. Other writers hold that repeated attacks of cold in the head and naso-

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pharyngeal catarrh may lead to otosclerosis, while others believe that exposure to severe cold may bring on the disease.

*Otitis Media.*—Lucae held that otosclerosis cannot be distinguished from chronic middle-ear catarrh. Sohier Bryant says that otosclerosis may follow suppurative otitis media and calls this the *secondary form of otosclerosis.* According to Bryant the majority of cases of otosclerosis, even of those with a history of hereditary deafness, belong to this secondary type. Habermann and Katz believe that middle-ear inflammations predispose to otosclerosis. Siebenmann has shown that the bony outer wall of the labyrinth is partly supplied by vessels from the middle ear. The redness of the promontory in otosclerosis suggests that certain cases of the affection arise from the tympanic mucosa. Habermann, Katz, and Manasse hold that otosclerosis may be induced as a secondary change after a simple inflammation or catarrhal condition of the middle ear. According to Katz the primary pathological process in otosclerosis is invariably inflammatory, and is situated in the muco-periosteal lining of the tympanum. It is transmitted to the bone along the vascular walls in the same way as the chronic vascular osteitis of Volkmann.

Moeller states that otosclerosis occurs first at the point of entry from the tympanic periosteum. Ferreri also says that otosclerosis may arise in the periosteum of the middle ear.

On the other hand, Bruehl believes that the thickened periosteum is probably secondary to the bone change.

*Auto-intoxication.*—Gastro-intestinal auto-intoxication is a frequent cause of otosclerosis, and the subjective symptoms of this condition may be relieved by treatment of the digestive affection. Ferreri and Yearsley both lay stress on the importance of auto-intoxication.

*Circulatory Disturbances.*—O. Mayer holds that functional disorders of the vasomotor system are of importance in the production of otosclerosis. The otosclerotic foci are symmetrical and correspond in location to the terminations of the nutrient arteries of the pyramidal bone. The internal auditory artery supplies not only the membranous labyrinth and the endosteum, but also the cartilage bone which lies next to the endosteum and surrounds the hollow spaces of the labyrinth. Mayer holds that this bone is not the seat of otosclerosis. According to Mayer, the process is as follows: There is an insufficient blood supply in the terminal vessels owing to constriction of the vessels. This leads to a state of malnutrition of this part of the bone, with the resulting forma-

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tion of fibrous marrow. If the circulatory disturbance continues, there is resorption of the fibrous marrow resulting in the production of cysts in the interior of the bone. If, on the other hand, the vasoconstriction is followed by an increased blood supply, there is a new formation of bone in the marrow spaces together with hyperostosis and exostosis.

*Arteriosclerosis.*—Cornet reports that 50 per cent. of his otosclerotic cases had arteriosclerosis. In many cases of otosclerosis we find on microscopic examination that there is atrophy of the stria vascularis and of Corti's organ, just as in senile deafness due to arteriosclerosis. O. Mayer has found atheroma in the internal carotid and auditory arteries in otosclerosis.

[It is curious that otosclerosis is common in young and otherwise healthy girls. Surely these cases cannot be due to arteriosclerosis. —J. S. F.]

*Anæmia.*—Many authors lay stress on the importance of anæmia in the causation of otosclerosis.

*Nervous Affections.*—Severe psychic traumatism, *e.g.* shock, fright, grief, anxiety, mental exhaustion, and neurasthenia favour the development of otosclerosis.

Some authors are of opinion that a nerve lesion is the primary factor (Baginsky). In almost all cases nerve changes have been found in the inner ear on microscopic examination, even in cases where the foci of diseased bone were very small (Manasse). Others hold that the nerve changes in the labyrinth are due to the bone lesion (Siebenmann).

Osteomalacia is also said to be a trophoneurosis dependent on disturbances in the female sexual organs, especially in the ovary. These disturbances are transmitted to the bone by the sympathetic nerves.

Lumbar puncture may ameliorate the symptoms of otosclerosis.

The diminution or absence of the sensibility to tickling in the external auditory meatus indicates the presence of a nerve lesion (Froeschels).

According to Panse four cases diagnosed as otosclerosis were found on examination to have degeneration of Corti's organ and rigidity of the stapes, but no bone lesion.

*Infections.*—Infectious diseases are causative of secondary periostitis and osteitis of the labyrinth capsule (Hammerschlag).

The scleroses produced by certain infections are limited to particular vascular areas, *e.g.* sclerosis due to variola affects the

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pulmonary arteries. Hence the possibility exists that certain infections may influence the pyramidal bone in a similar way.

*Rheumatism, Gout, and Arthritis.*—Many authors have noted a connection between these diseases and otosclerosis. Sohier Bryant holds that though these diseases are often found in cases of otosclerosis, it is more probable that all the conditions are due to a common cause than that rheumatism, gout, etc., give rise to otosclerosis.

*Syphilis.*—Gruenert, Manasse, Heymann, Heine, Katz, Shambaugh, Heimann, Gradenigo, and Paludetti all regard syphilis as a predisposing cause. Habermann, who reports a case with marked luetic changes in the vessels, holds that the syphilitic cause of otosclerosis is shown chiefly by the histological findings of chronic inflammation extending to the bone from the periosteum by way of the vessels, without suppuration or necrosis. The presence of hyperostoses and also the porous composition of the bone, favour a syphilitic origin.

Bezold, from an experience of over a thousand cases, does not believe in the causal relationship of syphilis. Beck and others have tested the Wassermann reaction in many cases of otosclerosis and found it positive in a very small percentage (five times out of forty-four cases). Gray notes that syphilis is more common in men, while otosclerosis is more common in women.

*Osteitis.*—Scheibe, Heimann, Katz, and Habermann consider that the bony lesion in otosclerosis is an inflammatory osteitis. Mayer holds that otosclerosis is closely allied to the osteitis fibrosa of von Recklinghausen. Manasse compares otosclerosis to the vascular ostitis of Volkmann. Sohier Bryant says that both in otosclerosis and in osteitis we find increased vascularisation, displacement of the normal bone by osteoid tissue, exostosis and hyperostosis, the line of demarcation or Pommer's cementing line, the origin in the endosteum or from the periosteum, and finally resolution by new atypical dense bone.

*Osteomalacia and Rickets.*—Ferreri says that in many cases of congenital deaf-mutism the loss of hearing is due to an early attack of capsulitis labyrinthi *in fœtal life*. This attack is coincident with maternal intoxication or other affections such as rickets or osteomalacia which are due to diplococci. All the osteomalacic patients seen in Arcangeli's clinic had evident otosclerosis. The administration of a vaccine improved the osteomalacia and also the otosclerosis. Ferreri claims that rachitic lesions of the labyrinth capsule can be recognised in the new bone. Rickets and osteo-

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malacia are ætiologically the same. The differences are merely quantitative and depend on the predominance of halisteresis and the proliferation of osteoid tissue.

*Old Age.*—Bryant has observed that otosclerosis has a tendency to appear in old people—probably as a result of tropho-toxic changes.

*Tuberculosis.*—According to Katz the scrofulous constitution bears a causative relation to otosclerosis.

*Traumatism.*—J. Moeller states that constant exposure to noise or traumatism may be the predisposing or exciting causes of otosclerosis. Mayer points out that vasomotor neuroses may follow trauma. According to Bruehl the anterior margin of the oval window is specially vulnerable on account of the greater movement of the anterior part of the foot-plate of the stapes, and also because of the presence in this region of the pulley of the tensor tympani muscle.

*Thrombosis.*—Gray holds that otosclerosis is due to thrombosis of the small blood-vessels and that the areas of otosclerosis are really areas of aseptic necrosis. The bone dies and, as it is not infected, becomes absorbed. Bruehl states that the diseased areas present the appearance of an enucleable foreign body. It is possible that syphilitic changes in the vessel wall may lead to thrombosis, and Yearsley points out that the puerperium favours stagnation in the capillaries.

*Otosclerosis an Idiopathic Primary Bone Disease.*—This view is held by many great otologists, *e. g.* Politzer, Bezold, Siebenmann, Möller, Bruehl, and Schwartze. These writers deny that otosclerosis is an inflammatory condition. Gray also states that in cases of otosclerosis there are no signs of past or present inflammation in the tympanic cavity. Politzer and Bruehl point out that the otosclerotic foci are separated from the periosteum by layers of normal bone, and that they may even occur in the region of the internal meatus where there is no mucosa. Ankylosis of the stapes is by no means always present.

Under normal conditions growth should cease in the labyrinth capsule at or soon after birth, though it continues in other bones. Siebenmann views otosclerosis as the continuance of a process of growth in the labyrinth capsule, in the regions where remnants of embryonic cartilage most abound.

Taniyama has found marked remains of the primitive cartilaginous capsule of the otic vesicle in the bone in the anterior margin of the oval window in the fœtus at various ages, and also in children and even in adults.

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*Heredity.*—Before discussing the question of heredity it is necessary to again remind the reader that there are probably two forms of otosclerosis: (1) primary otosclerosis, which is markedly hereditary, and (2) secondary otosclerosis, which follows inflammatory conditions in the middle ear.

Statistics with regard to the frequency of a family history of otosclerosis in patients suffering from this disease vary considerably, *e. g.* Siebenmann, 35 per cent ; Denker, 40 per cent.; Bezold, 82 per cent. Mayer holds that, as in arteriosclerosis and the vasomotor neuroses, the causative factor in otosclerosis is transmitted. Woakes held that this causative factor was a defect in the sympathetic ganglia. Hammerschlag says that otosclerosis is congenital and is due to abnormally differentiated germinal cells. Hammerschlag classes hereditary congenital deaf-mutism, hereditary nerve deafness, and primary otosclerosis as different forms of the same disease. Yearsley states that otosclerosis is transmitted in the female line. Alexander believes that the congenital predisposition to otosclerosis only begins to develop at puberty.

## Part II.—Pathology of Otosclerosis.

*Blood-vessels.*—In otosclerosis the first change is increased vascularity of the affected bone and of the thickened muco-periosteum overlying it. The normal bone is replaced by very vascular osteoid tissue. The vessels are merely dilated capillaries. In the later stages the number of the vessels is diminished. In osteomyelitis, rickets and osteitis fibrosa, the initial change is an intense vascularisation of the marrow.

*Endosteum.*—The dissolution of the compact bone by the proliferation of the marrow is the essential feature of otosclerosis. In the affected parts the marrow is lymphoid and only later becomes fibrous. In osteomalacia and rickets the disease process begins with endosteal proliferation and vascularisation of the marrow, while in osteitis fibrosa the process consists in a disintegration of the bone starting in the marrow.

*Haversian Canals.*—Otosclerosis begins in these preformed vascular spaces which become larger and are filled with new connective tissue and new blood-vessels, *i. e.* granulation tissue. In some cases buds grow from the periosteum into the Haversian canals and absorb the bone. Katz says that in otosclerosis new medullary spaces are formed. In the older parts of the lesion the spaces are smaller, the cells fewer, and the fibrous tissue more

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marked. In osteomalacia and rickets one of the initial changes is the dilatation of the Haversian canals, while in osteitis fibrosa there is a marked new formation of these canals.

*Absorption of Bone.*—In otosclerosis the dense petrous bone is traversed by newly developed vascular channels, and the absorption of bone proceeds from these marrow spaces, through the proliferation of connective tissue cells and the formation of granulation tissue. The cartilaginous bone (interglobular space bone) is absorbed without the presence of osteoclasts and is replaced by new bone. The absorption is due to atrophy caused by the pressure of the new tissue. In its turn the new osteoid tissue is absorbed by osteoclasts. In osteomalacia and rickets, the softening of the bone starts from the medullary canals.

*New Bone Formation.*—In otosclerosis there is a true formation of new bone or osteoid tissue and not merely a transformation of the old bone. Manasse has observed a bunch-like mass of new bone projecting into the old compact bone from the wall of a vascular space in the normal labyrinth capsule. The new formed bone has a strong affinity for basic stains such as hæmatoxylin and therefore appears dark blue. [New formed bone laid down in the deep layer of the periosteum of the mastoid air cells in cases of subacute mastoiditis does *not* take on the basic stain, but stains pink with eosin.—J. S. F.] In the later stages of otosclerosis osteoblasts form osteoid tissue in the walls of the enlarged lymph spaces in the new bone. Thus in time the new bone becomes sclerosed and consists of a network of irregular lamellæ which stain strongly pink with eosin. The cavities in the older parts of the new bone contain fewer cells and larger vessels, as well as a large amount of connective tissue; in other words, the cavities contain fibrous marrow which may become fatty marrow or may calcify. The new bone has no cartilaginous interglobular spaces, and finally becomes sclerotic and entirely free from marrow spaces. In osteomalacia and rickets the newly formed bony tissue is absorbed (osteoporosis). In osteitis fibrosa the disintegration starts in the marrow and is followed by reformation.

*Osteoporosis.*—Rarifying osteitis is the essential characteristic of otosclerosis. Katz found spongy bone in the squamous portion of the temporal bone of a cat, whose labyrinth capsule showed otosclerosis. In this case also, the malleus, incus, and stapes were all affected by osteoporosis. Cysts may occur in the otosclerotic lesions. In Paget's osteitis deformans, in osteomalacia and in rickets we find osteoporosis, as also in the early stages of syphilitic

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affections of bone, osteo-arthritis, and those changes in bone associated with pernicious anæmia.

*Hyperostosis and Exostosis.*—In otosclerosis the periosteum forms new bone in the shape of irregular hyperostoses. In the region of the oval window this in time leads to ankylosis of the stapes. In syphilis, in osteitis deformans, and in rickets hyperostoses occur.

*Character of the Cells.*—In the new osteoid tissue of otosclerosis the connective tissue cells are very numerous; round, spindle, and cubical cells are present as in ordinary granulation tissue. Myelocytes and nucleated red blood-corpuscles are always absent, and so the tissue is not true marrow. Later on the cells become fewer, and occasionally true fat cells are found.

Osteoclasts are rare at the margin of the otosclerotic tissue, but occur in the central part of the new bone. In the later stages osteoclasts disappear, thus showing that the process can terminate.

*Connective or Fibrous Tissue.*—Mayer maintains that the proliferation of the connective tissue cells of the marrow causes the bone absorption. In the older portions of the new bone there is a great increase in connective tissue due to the multiplication of the cells of the enlarged perivascular lymph space. These cells fill the space between the central vessel and the bony wall. This fibrous marrow is transformed into coarse connective tissue and may give rise to cysts. Mayer states that the periosteum is very much thickened in the niche of the oval window.

*Formation of Lamellæ.*—The osteoid tissue has at first no lamellated structure; this only appears later on, and then the lamellæ are not arranged in a regular manner. The new osteoid tissue is, as a rule, sharply marked off from the normal bone by Pommer's cement line.

*Is the Condition Inflammatory?*—Opinions are divided on this point, but the onset of the disease with the formation of granulation tissue, the alternation between apposition, absorption, and renewed apposition, the formation of giant cells, all point to a chronic inflammatory process similar to osteitis fibrosa and Volkmann's vascular ostitis. The transformation of the cellular marrow into fibrous marrow, the ultimate production of solid compact bone, and the formation of exostosis are conditions which are found in chronic inflammations of other organs.

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