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**THE HISTOLOGY OF NASAL ACCESSORY SINUS
SUPPURATION.**

(A Comparison of the Simple Inflammatory Processes in the Upper Respiratory Tract, including the Middle-Ear Cleft.)

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THE object of this paper is to show that the changes which occur in the mucous membrane of the accessory sinuses in simple acute and chronic inflammatory conditions are analogous to those which take place in the nose in catarrhal and suppurative rhinitis, and in the middle-ear cleft in catarrhal and suppurative otitis media; secondly to bring forward evidence in favour of the view that these processes occur first of all in the nose and affect the pharynx, accessory sinuses, and middle ear by direct extension, and further that there is no essential difference between what is called "catarrh" and what is called "suppuration," as one condition passes insensibly into the other.

Under normal conditions the accessory sinuses contain air, while the lining membrane is coated with a delicate layer of mucous secretion; it is important to remember that the cavities are lined by mucous membrane continuous through the ostia with that of the nasal cavities. We are all aware of the fact that an attack of coryza is frequently followed by more or less catarrhal pharyngitis, laryngitis, and even tracheitis, and it seems probable that the

mucosa of the sinuses takes part in any changes which the mucous membrane of the nose may undergo. Harke, E. Fränkel, and Oppikofer on the one hand think that sinus suppuration is the direct consequence of the causative disease, whereas Zuckerkandl, Hajek, Lack, and Goetjes are of opinion that most cases are due to the spread of a catarrhal process from the nose. It is hardly necessary to say that the accessory sinuses are not always affected by an attack of acute rhinitis, just as the middle-ear cleft is not involved in every case of measles or scarlet fever. On the other hand it frequently happens that although the catarrhal process in the nose may have subsided, the inflammatory condition has continued in the maxillary antrum or in the tympanic cavity. It has been frequently stated that cases of maxillary antrum suppuration, which have been treated and apparently cured by alveolar puncture, often relapse during an attack of coryza, and this fact supports the view that the mucous membrane of the sinuses takes part in the inflammatory processes which have invaded the nasal cavities. In regard to the physical conditions which obtain in the accessory sinuses, it is necessary to call attention to the fact that the ostia are very small in proportion to the size of the cavities they drain, and further that these ostia are, in the case of all except the frontal sinus, very badly situated for drainage by gravitation. Under normal conditions drainage is probably carried on by ciliary action alone, but in diseased conditions the epithelium is not infrequently damaged, and under such circumstances gravitation syphonage and suction may come into play (Yankauer).

HISTOLOGY OF THE NORMAL MUCOSA OF THE ACCESSORY SINUSES.

In dealing with specimens obtained from the *post-mortem* room it is an exceedingly easy matter after formol fixation to strip the mucous membrane from the underlying bone with a small blunt dissector such as that used in the submucous resection of the septum, and even during operation on a diseased sinus it is often possible to detach large areas of thickened mucosa in the same way. Oppikofer lays stress on the examination of large pieces, as varying pathological conditions may be found at different parts of the mucous membrane obtained from the same sinus; this statement is confirmed by Goetjes. The normal mucous membrane consists of two layers—superficial epithelium and submucous connective tissue, separated by a delicate basement membrane (Figs. 1 and 2). The surface layer is composed of ciliated cylindrical cells with a deeply

FIG. 1.—Normal mucous membrane from maxillary antrum. $\times$ about 50 diam. 1. Ciliated mucous membrane. 2. Submucous connective tissue.

FIG. 2.—Normal mucous membrane from maxillary antrum. $\times$ about 400 diam. 1. Normal mucous secretion. 2. Superficial layer of columnar ciliated cells. 3. Intermediate layer of spindle-formed cells. 4. Deep layer of cubical cells. 5. Basement membrane. 6. Connective tissue.

FIG. 3.—Normal ostium of sphenoidal sinus. $\times$ about 17 diam. 1. Epithelial lining of sinus. 2. Mucous glands in connective tissue.

FIG. 4.—Normal middle turbinal, showing air-cell lined by healthy mucous membrane. $\times$ 6 diam. 1. Lining membrane of cell in middle turbinal. 2. Bony core of turbinal. 3. Glands in submucous tissue on nasal aspect. 4. Mucous membrane of nose.

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placed nucleus; a varying amount of granules is also present in these cells. In some the amount is so large that the nucleus is hidden, in others the granules are scanty, and in others again they are absent and the cells appear as "ghosts." Hajek has shown that in the nasal mucous membrane this latter condition is associated with a discharge of mucus, and it is almost certain that the same things holds good in the accessory sinuses. Between these superficial ciliated cells and the basement membrane there are one or two rows of cubical or spindle-shaped cells, the nuclei of which are large in proportion to the size of the cell. In normal cases the basement membrane is a very delicate structure and is represented by a very fine homogeneous line just beneath the deepest layer of the epithelium. Goetjes states that under normal conditions a basement membrane is not present. The submucous tissue consists of several layers of elongated connective-tissue cells with their long axes parallel to the surface of the mucous membrane. Eschweiler has divided this layer into two—a looser superficial and a denser deep layer, the latter forming the periosteum of the subjacent bony wall. It is in this latter that the blood-vessels run, while in the superficial stratum are occasionally found the coiled mucous glands which form such a prominent feature of the submucous tissue of the inferior and middle turbinal bodies. It is usual to find an aggregation of these glands in the neighbourhood of the ostium (Fig. 3). It should perhaps be pointed out that there is not the same necessity for a large number of mucous glands in the accessory sinuses as there is in the nose itself; the interchange is very slight between the air contained in the sinuses and that passing to and fro in the nasal cavities; the drainage of the sinuses would also be extremely difficult if a large quantity of mucous secretion were poured out in these cavities. Goetjes states that a certain amount of round-cell infiltration is to be found in the normal submucous tissue of the sphenoidal sinus just as in the nasal mucous membrane. I have not been able to confirm this observation.

CHANGES IN THE MUCOUS MEMBRANE IN ACUTE AND CHRONIC INFLAMMATION.

Nineteen cases have been examined, distributed as follows: antral mucosa 9, ethmoidal 8, frontal 1, and sphenoidal 1; for two of the antral cases I am indebted to Dr. Logan Turner.

ACUTE INFLAMMATION.—Only one case of acute inflammation in the mucous membrane of a sinus was met with (Fig. 7), this was

in the sphenoidal sinus mentioned above, which was obtained at the autopsy on a case of acute pneumonia. The mucous membrane was considerably thickened and deep red in colour; the surface was velvety, and the sinus contained some thick muco-pus, but no blood. On microscopical section the prominent feature was the extensive hæmorrhage in the submucous tissue and the engorgement of the vessels; small-cell infiltration was not so marked as in chronic cases, and the œdema was slight in amount; the superficial epithelium was in places markedly disintegrated. Goetjes states that in acute inflammatory conditions the number of beaker cells, and consequently the production of mucus, is greatly increased; finally, the superficial layer of cells desquamates or the whole of the epithelial covering may be lost. In four cases of acute inflammation of the mucous membrane of the sphenoidal sinus Goetjes found marked round-cell infiltration, most intense just under the epithelium, but also present in the deeper layers around the glands and vessels. Submucous hæmorrhages were present in three out of four cases. The round cells consisted mainly of lymphocytes, but eosinophiles, polynuclear and plasma cells and isolated phagocytes were also present.

CHRONIC INFLAMMATION.—In the first place a more or less thick layer of catarrhal or purulent secretion is present on the surface (Figs. 14 and 15); it is important not to remove this layer during the process of preparing the specimen for examination, because, if it is present, we can exclude with certainty the fallacy of an artefact when talking about the condition of the superficial layer of cells. The secretion or exudation consists of mucus and leucocytes in various proportions, with occasional epithelial cells.

Changes in the Superficial Layer.—It is remarkable how often this layer appears to be normal, although there are marked changes in the submucous tissue. Not infrequently, however, the number of ghost-cells in the most superficial layer is very large, while in other cases this ciliated layer is absent in parts, and only the spindle-shaped or cubical cells beneath are left. Occasionally the basement membrane is entirely denuded of epithelial covering (Fig. 10). In most of the specimens leucocytes may be seen making their way between the epithelial cells to the surface, there to mix with the mucous secretion and form the *muco-purulent* or purulent exudation characteristic of sinus catarrh or suppuration (Fig. 9). In one and the same specimen mononuclear cells may be seen at one point between the epithelial cells, and at another only polymorphonuclear cells; eosinophiles may also occasionally

FIG. 5.—Early inflammatory edema of middle turbinal (nasal polypus). $\times$ about 6 diam. 1. Edema of submucous tissue of air-cell in middle turbinal. 2. Bone. 3. Pendulous masses of edematous connective tissue (polypi). 4. Dilated gland ducts. 5. Mucous glands.

FIG. 6.—Edema of middle turbinal in case of ethmoidal suppuration. The normal tissues are crowded towards the bone; the thickened lining membrane of the cell in the middle turbinal is well seen, and the small-cell infiltration of its mucous membrane. $\times$ 6 diam.

FIG. 7.—Mucous membrane from sphenoidal sinus showing acute inflammatory changes. $\times$ 50 diam. 1. Engorged blood-vessels. 2. Hemorrhages. Notice disintegrated condition of surface epithelium.

FIG. 8.—Polypoid mucous membrane from floor of maxillary antrum. $\times$ 9 diam. 1. Apparently normal epithelium on surface. 2. Stalk of the polypus. 3. Large connective tissue spaces filled with serum.

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be found in the same situation. To a smaller extent this process may be observed in the nasal mucosa in cases of chronic catarrh, but in this condition the leucocytes which have come through the epithelium on to the surface are diluted by the large quantity of mucous secretion poured out by the vast number of mucous glands in the nose, and consequently the discharge is only *mucopurulent*, whereas in the accessory sinuses the mucous secretion is scanty and the leucocytes at least equally numerous, so that the discharge is more definitely *purulent*. With regard to metaplasia of the epithelium, after going very carefully over all my specimens I found small areas in three of the nine antral cases (33 per cent.) in which the superficial cells approximated more closely to the squamous than to the cylindrical type (Fig. 9). Oppikofer found this condition present in 41 per cent. of antral cases. The question of the relationship of cholesteatoma of the accessory sinuses and middle ear to this metaplasia of the epithelium is not yet settled; so far a case of cholesteatoma has not been recorded in which there was no possibility of the epithelium coming in from a surface covered by squamous epithelium (mouth, skin, external auditory meatus), but cases have been described in which this metaplasia of the epithelium was regarded as the cause of the cholesteatoma formation. In none of my specimens did the loss of superficial tissue extend deeper than the basement membrane, so that nothing in the nature of ulceration was met with in the accessory sinuses. Goetjes states that it is not possible to distinguish acute from chronic inflammatory processes by the epithelial changes.

The *basement membrane*, which is extremely delicate and may apparently be absent in the normal accessory sinuses, is, as a rule, markedly thickened in cases of chronic suppuration.

Changes in the Submucous Tissue.—The thickness of the mucous membrane varies considerably in different parts, being greatest in the hollows and least over the convex surfaces; this variation depends on the amount of submucous tissue. In chronic inflammatory conditions this tissue is usually much increased in depth, the increase being mainly due to œdematous infiltration, though fibrous changes, vascular engorgement and small-cell infiltration no doubt play a part. Goetjes looks on sclerosis of the submucous tissue as an indication that cure had resulted in a case of chronic inflammation (Fig. 16). I agree with Oppikofer in thinking that the degree of thickening of the submucous tissue is not a measure of the severity of the suppurative process, and that a slightly thickened mucous membrane may suppurate freely and cause the

same symptoms as a sinus with enormously thickened polypoid mucosa. In fact I think that many cases of this latter type are not clinically or pathologically severe, and are more allied to conditions of simple nasal catarrh and polypus formation (compare Fig. 8 with Fig. 11).

The œdematous infiltration results in a widening of the delicate connective-tissue network, so that large spaces are formed which are filled with serum; the process is in fact exactly similar to that found in the middle turbinal in cases of nasal polypus formation and in the inferior turbinal in so-called "chronic hypertrophic rhinitis." This polypoid character shows itself especially in the dependent parts of the sinuses, *e. g.* along the floor of the maxillary antrum; the condition is not difficult to explain, for we have only to remember that the mucous membrane accurately fitted the antrum when the submucous tissue was of normal thickness; if, now, this tissue increases tenfold in depth it follows that if the epithelium on the surface is to remain of the same superficial area it must become folded on itself, or, in other words, polypoid. As seen in section this tendency is naturally most marked in the dependent parts of the sinus (Fig. 8).

Small-Cell Infiltration.—This varies markedly in situation, in degree, and in the character of the cells; in some cases there is marked œdema and marked small-cell infiltration. Again, the infiltration may be confined to that part of the submucous layer which lies just under the basement membrane (Fig. 6), or it may be more or less evenly distributed over the whole thickness of this tissue, or it may be in places aggregated into lymph-nodules. I have not observed it especially marked in the deepest layer of the submucosa, but such appearances have been reported by Oppikofer. In some cases the submucosa resembles embryonic connective tissue (Fig. 14). The great majority of the cells are lymphocytes; this is interesting, because cytological examination of the fluid washed out of the maxillary antrum and nose by puncture through the outer wall of the inferior meatus frequently shows the cellular elements to consist almost entirely of polymorphs. I do not pretend to be able to explain this discrepancy, but I think that it is important in these cases to use a stout hollow needle which can be fitted on to the barrel of an evacuating syringe, so that any discharge present in the antrum may be withdrawn into the syringe and examined without the risk of contamination by the muco-pus and micro-organisms present in the nose.

Glands.—The gland-ducts in the submucous tissue may be

FIG. 9.—Superficial epithelium from case of chronic maxillary antral suppuration. $\times 1000$ diam. 1. Leucocytes making their way to the surface through the epithelial layer. Notice the catarrhal exudation on surface.

FIG. 10.—Mucous membrane from maxillary antrum from a case of chronic suppurative catarrh. $\times 300$ diam. 1. Metaplasia of superficial epithelium—approach to squamous type. 2. Complete loss of epithelium—the basement membrane, however, remains.

FIG. 11.—On the right is seen the infiltrated lining membrane of the air-cell in the middle turbinal. From a case of chronic ethmoidal suppuration. $\times 50$ diam. 1. Small-cell infiltration is very dense, but oedema is slight. 2. Columnar epithelium. 3. Small vein showing fibrous thickening of wall.

FIG. 12.—Chronic suppurative catarrh of the maxillary antrum. $\times 300$ diam. 1. Small-cell infiltration of connective tissue. 2. Catarrhal secretion in gland duct. 3. Condensation of fibrous tissue around duct.

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dilated and filled with mucus, degenerated cells, and leucocytes; in one case I observed marked fibrous thickening round the ducts (Fig. 12). Cysts are occasionally found in the submucous tissue (Fig. 13); these are filled with homogeneous "colloid" material and lined by cubical cells; they are of course due to blockage of the gland-ducts, and are said to be more frequent in the antrum than in the frontal sinus; they are not diagnostic of chronic suppuration, according to Oppikofer. Goetjes frequently observed cysts in the sphenoidal sinus mucosa.

Changes in the Vessels.—The great engorgement characteristic of acute inflammation is not present in chronic cases to the same extent, but still a certain amount of vascular dilatation is to be observed. Thickening of the vessel walls was observed in one of my cases, and Oppikofer has described hyaline changes in the walls in otherwise healthy (presumably non-syphilitic) people. I have observed one such case. The small-cell infiltration is usually very marked round the vessels. Old hæmorrhages are not infrequently seen in the submucous tissue, and deposits of pigment are also found (Fig. 15).

Changes in the Bone.—In the cases of antral, sphenoidal, and frontal suppuration the bony wall of the sinus was not included in the tissue examined, but out of the eight ethmoidal cases osteoclasts were found in only one instance. Bone disease may be present in accessory sinus suppuration, but it is not an essential change, and is due to the more or less gradual spread of the inflammatory process from the surface through the mucous membrane. Grünwald found bone disease in 18 per cent. of antral cases and in 60 per cent. of ethmoidal cases, and Oppikofer states that the bone often appears to be eroded with numerous lacunæ-like depressions, that lamellar formation is irregular, and bone-cells are enlarged. He states that osteoclasts are very rare; I have observed them in one case of ethmoidal suppuration.

Micro-organisms.—I have not been able to find organisms in the tissues in a single case, although numerous sections of all the cases were examined after staining by Gram's method in addition to the ordinary stains. In the discharge lying on the surface organisms could occasionally be seen. Dr. Turner reports that Mr. Wade found organisms present in each of four antral cases, and in three of these the bacteria were present in the cells of the mucous membrane, while in one case there were small abscess cavities crowded with cocci in the submucous tissue. Lack expresses the view that there is no definite evidence at present to

show that a particular organism or mixture of organisms is associated with a definite sort or degree of inflammation.

What is wanted, if we are ever to obtain useful knowledge as to the prognosis and method of treatment in a given case of sinus suppuration, is an extensive investigation into the subject. If we could, from a large series of cases, obtain a table giving the cause, duration, symptoms, rhinoscopic appearances, cytological and bacteriological examination of the discharge, along with an account of the histology of the mucous membrane if the case came to radical operation, we might be able to make a considerable advance.

Comparison of the Simple Inflammatory Processes in the Upper Respiratory Tract, including the Middle-Ear Cleft.—In a paper published in the JOURN. OF LARYNGOL., RHINOL., AND OTOL. (August, 1908) I showed that in nasal polypus formation and in inferior turbinal hypertrophy the condition was essentially one of chronic inflammatory œdema or serous exudation into the connective-tissue spaces under the basement membrane; the amount of leucocytic infiltration is slight, while the superficial epithelium is, as a rule, practically normal. On the other hand, from the microscopical examination of the nasal mucosa in cases of ozaena it is apparent that the change in the superficial epithelium is very marked—large tracts are converted into squamous epithelium and keratinisation of the superficial cells is sometimes present; the glands are atrophied and there is intense leucocytic infiltration of the submucous tissue; œdema, on the other hand, is absent. We find, however, not infrequently that the inferior turbinal shows typical atrophic changes, while the middle turbinal shows a polypoid condition.

In the accessory sinuses similar changes may be met with; thus two of my cases show œdema of the submucous tissue with intact epithelium and little or no leucocytic infiltration (Figs. 5 and 8). Kahn also remarks on the similarity of one of his cases to nasal polypus formation. On the other hand many sinus cases that come to the radical operation show changes which closely correspond to such conditions as suppurative rhinitis or ozaena. In these cases we have dense leucocytic infiltration with marked changes in the superficial epithelium (loss of ciliated layer, etc.). It is of course obvious that the accessory cavities are not, like the nose, subject to the drying influence of the air-currents and are not so liable to mixed infection; consequently we do not meet with the foul-smelling dry crusts in the accessory sinuses.

FIG. 13.—Mucosa of maxillary antrum from case of chronic suppurative catarrh. $\times$ about 20 diam. 1. Superficial epithelium. 2. Dense small-cell infiltration of submucosa. 3. Cystic space lined by cuboidal cells. 4. Colloid secretion in the cyst.

FIG. 14.—Mucous membrane of antrum from case of chronic suppuration. $\times$ about 300 diam. 1. Leucocytes passing through epithelial layer to the surface. 2. Purulent secretion. 3. Epithelium showing loss of ciliated layer. 4. Engorged blood-vessels.

FIG. 15.—Mucous membrane of maxillary antrum from case of chronic suppurative catarrh. $\times$ 60 diam. 1. Catarrhal secretion on surface. 2. Superficial epithelium. 3. Haemorrhages in submucous tissue.

FIG. 16.—Mucous membrane of frontal sinus from case of rhinitis caseosa. $\times$ 50 diam. Shows fibrous change in submucous tissue. 1. Loss of superficial layers of epithelium. 2. Almost normal epithelium.

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Catarrh of the mucous membrane of the accessory sinuses is much more common than is usually supposed. Oppikofer found that about half of his ninety-four *post-mortem* cases of sinus inflammation might be classified as *catarrh* and half as *suppuration*. All specialists are well acquainted with the type of patient who comes complaining of nasal discharge and obstruction, with perhaps a heavy feeling over the bridge of the nose or glabellar region. On examination the turbinals are found to be enlarged—the middle turbinal polypoid—and there is excess of muco-pus in the nose. The antra fail to illuminate, though the frontal sinuses as a rule light up well. On proof puncture of the antra only a little mucus or flaky muco-pus is washed out. The number of cases which fail to illuminate is too large to be accounted for by anatomical causes, such as thick bony wall. I do not, however, mean that accessory sinus catarrh is present in all cases of nasal catarrh, or that nasal suppuration is to be found in all cases of sinus suppuration. The position of the ostia of the accessory sinuses, while it renders infection of the mucosa of the sinus difficult, also tends to prevent the spontaneous cure of a severe sinus infection. The nasal mucosa on the other hand is frequently affected by catarrhal processes, but the drainage conditions in the nose are much more efficient than those in the accessory sinuses. In short, the nasal mucosa, though frequently infected, has a great chance of recovery, while the accessory sinuses, though not so exposed to simple or suppurative catarrhal processes, are less able to throw off these conditions once they have occurred.

Finally, although the number of my specimens of acute and chronic middle-ear inflammation is small—four acute and nine chronic cases—I think that it is possible to show that the changes here are similar to those in the nose and in the accessory sinuses; in other words, we have acute catarrh, chronic catarrh with oedematous swelling, acute and chronic suppurative processes. The difference between catarrhal and suppurative processes is only one of degree, and there are borderland cases. Otologists are all familiar with the type of acute otitis media which occurs in children with adenoids, where the tympanic membrane is slightly congested, cloudy, and bulging; such cases may go on to suppuration or they may not, and, even if perforation does occur, there is only for a day or two the escape of a thick *muco-purulent* discharge in which the mucous element predominates. Such cases are on the borderland of what is called “catarrh” and what is called “suppuration,” and from this mild type we have all grades of

severity up to those malignant cases in which within a few days the tympanic membrane is almost entirely lost and bone disease occurs in the ossicles and in the walls of the middle-ear cleft, more especially in the mastoid cells, where the drainage is least efficient. Nager has confirmed the observations of Burckhardt-Merian and others that there is a parallel—if not indeed an ætiological connection—between the severe nasal and pharyngeal affection of scarlet fever and the different forms of middle-ear suppuration. Nager has very seldom observed a severe scarlatinal diphtheria of the nose and pharynx without participation of the middle ear. Again, we know the type of case in which after signs of acute inflammation have been observed in the tympanic cavity the membrana tympani and hearing power return to normal, but the mastoid inflammation continues and an abscess forms. Neumann and Rutin have shown that the *Diplococcus mucosus capsulatus* is the organism present in many of these cases. Such cases appear to correspond to those of maxillary antral suppuration in which the nasal cavities present normal appearances. Finally, I venture to suggest that in the classification of acute and chronic simple inflammatory processes in the upper respiratory tract (including the middle-ear cleft) it would be well to consider the changes not only in the submucous tissue, as Eschweiler has proposed, but also in the epithelial covering. Under the heading of *Simple Catarrh* we might include cases with œdema of the submucosa, with slight alteration of the superficial epithelium, and only moderate leucocytic infiltration of the submucous tissue. Under the heading of *Suppurative Catarrh* we might include four more or less distinct conditions: (1) Cases with little or no œdema and only slight epithelial changes, but with marked leucocytic infiltration (Fig. 11). (2) Cases similar to the above, except that the epithelium has undergone metamorphosis, or is almost entirely absent over large areas. (3) Cases with marked œdema and leucocytic infiltration, but with slight epithelial changes. (4) Cases with marked œdema and leucocytic infiltration, but with extensive changes in the epithelial layer.

In conclusion, I should like to express my thanks to the Royal College of Physicians (Edinburgh) for kindly allowing me to work in the Laboratory, to Dr. James Ritchie for his advice and help in the preparation of this paper, and to the Carnegie Trust for enabling me to reproduce photomicrographs of my preparations.

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STATISTICAL TABLES.

Being Part III of the Report for the year 1908 from the Ear and Throat Department of the Royal Infirmary, Edinburgh, under the charge of Dr. Logan Turner.

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