NASO-PHARYNGEAL ADENOIDS AS A CAUSATIVE FACTOR IN EAR DISEASES.*

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Among the most interesting cases that come before the otologist are those pertaining to postnasal vegetation affecting the hearing, and there are few patients to whom more satisfaction can be rendered than to those so affected. Adenoid vegetation seems not to be restricted to countries, to climates, to sex, to color or race of man. Dr. William Meyer gives the results of his collected evidence of the existence of affections of adenoid growths in various parts of the world, and in various races of man. He says that in Greenland, in 60 Esquimaux children between 6 and 14 years of age Helms only found 16 free from adenoid vegetation. In North Dakota Quarry found adenoid vegetations frequent among the native tribes of Indians, but the growths were very little developed in adults. Cantley of Hongkong reported that native Chinese of the Mongolian race, as also those belonging to the mixed Chinese-Portuguese race, frequently suffer from them, while in Bangkok, Denietzer rarely found the disease among the native Siamese. Meyer concludes that adenoid vegetations are often to be found in varying degrees of frequency in at least three parts of the world, viz., Europe, America and Asia. The Mongolian race is almost as much prejudiced as the Aryan. A cold climate seems more favorable to their development than a warm one. Arslan of Padua, commenting on the frequency of adenoid vegetations in Italy, states that of 300 children examined in schools in Padua, he found the growths present in two-thirds of them. He attributes the preponderating rôle to heredity, a number of his patients showing that their antecedents presented exactly the same symptoms. Balme, who collected statistics on adenoid growths considered as a sign of degeneracy, in examining backward and degenerate children at the Van Cluse Colony, found that of 113 children examined 56 suffered from adenoid vegetation or enlarged tonsils or most frequently both lesions simultaneously. I agree with Meyer, Professor Stoker, Lennox Browne, and others that postnasal growths are just as common among the best classes of society as in the poorer.

Let us consider first what nasopharyngeal vegetation is. It is a hypertrophy of the lymphoid tissue situated in the vault of the pharynx bounded on either side by the orifice of the Eustachian tube, and presents on its surface several vertical furrows which partially subdivide it. The structure covers the roof of the nasopharynx back of the septum, and extends backward on the posterior wall while prolongations are often met with in the fossæ of Rossenmueller and even in the orifices of the Eustachian tubes. Many writers have impressed me by the frequency with which they found various forms of middle ear disease to depend on morbid conditions of the nasopharyngeal mucosa, and it is my opinion, based on several years' experience in the Illinois Charitable Eye and Ear Infirmary and in private practice, that the main factor in producing both suppurative and non-suppurative inflammatory conditions of the tympanic and Eustachian mucous membranes is the presence of naso-pharyngeal adenoids or the condition of the postnares consequent to their removal or absorption. The nasopharynx is an important cavity to the ear, by its relation from the nose through the Eustachian tube, and morbid conditions there readily exert an injurious influence on the ear. Adenoid vegetation may produce inflammation of the middle ear: 1, by constant irritation on account of the obstruction to the circulation of the blood by pressure; 2, by blocking the orifices of the Eustachian tube, partially or completely; 3, by their injurious effect on the general economy of the child and particularly the nerves of special sense; 4, by leaving a postnasal catarrh as a sequela, which sooner or later establishes some form of middle ear disease.

Blake advocates the theory that the first stage of inflammation, hyperemia, is produced in the middle ear by an interference with the return circulation, owing to the pressure exercised by the adenoid mass on the pharyngeal veins and those of the deep-seated tissue.

In children who suffer from adenoid vegetations the hearing is generally very sensibly impaired, and it is the common thing for a child so affected to have questions repeated often and in a louder tone of voice, say nothing of earaches and discharge from the ears. In many cases the Eustachian tube is completely blocked by dry secretions of the postnares. I have observed diminution of power of hearing on the side where the adenoid existed. On the opposite side where the postnasal space was clear the hearing was normal. I have seen cases where the hearing was seriously impaired and the drum membranes normal in appearance, yet with safety I assume the faulty hearing to be dependent on the growths in the nasopharynx. Mr. Bosworth, I believe, agrees with me in this statement.

Mouth-breathing, the most prominent objective symptom of the children affected with adenoids, has an important etiologic bearing on the subject. In this form of respiration the upper part of the lungs is never normally expanded, a fact which can be readily demonstrated by observing the child with normal respiration and one with the sucking respiration of mouth-breathers. The mouth-breather is usually found shallow through the upper part of the chest, and with very small lung capacity. The breather does not inhale sufficient air; the blood, therefore, is not sufficiently oxygenized and is surcharged with carbonic acid. This excess of carbonic acid retained in the blood causes the lassitude, headache, stupidity and sleepiness commonly met with in mouth-breathers, and gradually renders the nerves of special sense less active and responsive to stimuli. This excess of carbonic acid and lack of oxygen, I also believe to be the cause of loss of appetite, malnutrition and general cachexia so often found accompanying mouth-breathing. We frequently meet with children affected with adenoids, who are not mouth-breathers, and these children are plump, well-developed and of healthy appearance, although they usually have some ear complication.

Lennox Browne found that not only were these growths where the tonsils were not enlarged, but that in most cases of deafness formerly considered due to enlarged tonsils there was coexistence of adenoids; that failure to cure deafness after tonsillotomy would be less frequent if the vault of the pharynx were always explored and cleared of additional lymphoid overgrowth. It was also probable that adenoids do not disappear at so early an age, and several cases are quoted in which they

*Read before the Sixth International Otological Congress, London, Aug. 9, 1899.

were present and were the cause of deafness in patients over 21 years of age.

Alderton believes that disease of the nasopharynx is directly causative of middle ear disease in from 35 to 88 per cent. of all cases. White states that out of 565 cases treated for nasopharyngeal affections, he found disease of the middle ear in 197. Of the whole number, 134 had hypertrophy of the third tonsil, in which 62 of them, or 20 per cent., had impaired hearing. Bronner, before the British Medical Association, stated that on examination of 250 school children he found 20, or 8 per cent., suffering from adenoid vegetations. Eighty-five per cent. of 125 cases carefully examined showed symptoms of past and present affections of the middle ear. Laveand calls attention to cases in which adenoid vegetations produce total deafness in children. He considers that a cause of deaf-mutism, and says the removal of the vegetations may act as a cure.

A number of cases of postnasal growths in the newborn have been reported, and Sendziak believes that many children develop adenoids in the first years of their lives, and frequently become deaf from this cause, and are not able to learn to speak or forget what they do know.

According to many authors it has been shown that adenoids are much more frequent among deafmutes than among other children. In my examination of 26 children for deaf-mutism. I found only 4 free from postnasal adenoids; 16 of those examined showed marked facial deformity from mouth-breathing.

Frankenberger cites many authors who have investigated the frequency of adenoid vegetation in normal children: Meyer, 1 per cent. in 2000 cases; Schmiegenow 5 per cent. in 581 examinations; Wroblewski, 7 per cent. in 650 children; Kafemann, 9 per cent. This does not show a very large percentage, but when we arrive at the deafmutes, we find Lemcke reports 58 per cent.; Wroblewski, 57.5 per cent.; Peisson, 58 per cent.; Frankenberger, 59 per cent.; Albrich, 73 per cent.

The principal function of the Eustachian tube is to supply air to the middle ear. This equalizes the atmospheric pressure on the outer and the inner sides of the drumhead. If the postnasal cavity is blocked with the presence of adenoids, and we have a mechanical obstruction of the orifices of the Eustachian tube, either from the dried secretion or proliferation from the adenoids, the air-supply to the middle ear is partially or completely cut off. This inequality of atmospheric pressure causes a contraction of the drumhead, and such a condition will to some extent cause deafness, which will increase as the further pathologic process develops ankylosis of the ossicles and atrophy of the tympanic membrane.

Wright Wilson reports 235 cases which he had treated for postnasal vegetation. He found the proportion of sex about equal. Ten cases presented deafness as the only symptom. Wilson points out the importance of exploring the nasopharynx in every case of deafness.

Halbies found adenoids to be the cause of inflammatory process in the middle ear in 53 per cent.; Meyer, 78.8 per cent.; Hartmann, 74.2 per cent.; Ingals, 34 per cent.; Braislin, 58 per cent.; Max Schaefer, Bremen, reports a series of 1000 cases of adenoid growths of the pharynx treated by himself. Of these, 768 were treated surgically, 99 with the galvanic cautery, 81 by local applications, 52 were merely observed. In 467 cases there was deafness; in 107 otorrhea; 20 were improved and 331 cured.

I coincide with Harrison Allen and Sisson, who hold that there are many children in homes for the feeble-minded and idiots all over the world who are affected with this disease, and who by a comparatively trifling operation could possibly be restored to usefulness and their families. Interference with the condition may or may not complicate the case. Much depends on the relation of the Eustachian orifice to the vault of the pharynx. If the orifice be situated high up, a comparatively small amount of growth will block it, and cause an auditory trouble; but if it be low down, there may be extensive vegetation without the Eustachian tube being implicated. It would be obvious to mention every analogous case reported of deafmutes who, after the removal of adenoid vegetations, gave evidence of hearing and began to speak some words.

The general belief that adenoid vegetations are never present after the age of 30 is contradicted by Conetoux of Nates, who operated on a man of 65 to cure a marked unilateral deafness. Adenoids are apt to become smaller and more dense as age advances, yet I have found vegetations in ages above 60, and frequently between 30 and 40. They do not differ histologically from adenoids in children. It is not uncommon to observe these formations in the aged who are hard of hearing.

Notwithstanding all the writings of the last ten years, I can not say that the pathologic enlargement of the lymphoid tissue of the nasopharynx has received sufficient attention in the world's medical text-books.

If the symptoms of these growths were more generally recognized by the family physician, and their removal accomplished, we would not find so many chronic suppurative and non-suppurative inflammations of the middle ear with the history dating back to an attack of diphtheria, scarlet fever, measles, or other fevers. I believe that these obnoxious growths are of great importance to the health of the child, not only of the present, but of the future development of all faculties of children.

As to treatment, it is never too early nor is it too late. At the first recognition of existing growths, the operation should be performed at once, by whatever method best suits the case in hand. I do not believe in chemical cauterization or caustics, nor even a thermal cautery, especially in children, where it requires so many séances to accomplish it. Curetting is the only true basis of treatment and trustworthy of consideration. It may be done with Trautman's, Hartman's or Gottstein's curette, Meyer's ring knife, Lowenburg's forceps, and other various modifications. Such operations are recommended by Richard Hovell, Field, Lennox Browne, Holitzer, Lowenburg, Trautman, Bronner, Goldstein, Rousseaux, and many others. I am not a believer in general anesthetics in children over the age of 12 years, as local anesthesia after this age makes such an operation absolutely free from danger; but there are some cases where a general anesthetic must be administered, especially in refractory children and nervous adults. In children it is advisable to anesthetize in a sitting posture, and I prefer bromid of ethyl to any other of the numerous anesthetics. It is easily administered, anesthesia is quickly produced, and is of sufficient duration for the expert operator to remove both tonsils and adenoid vegetation, if necessary. The child recovers from the anesthetic quickly enough to clear its throat and prevent strangulation from hemorrhage. As it is common to find children suffering from adenoid vegetations to be constitutionally affected, restorative tonics, nutritious food and plenty of outdoor fresh air are indicated after the operation.

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THE PAPILLITIS ACCOMPANYING BRAIN TUMOR.*

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The papillitis accompanying brain tumor must, according to Unthoff, have reached an elevation of at least two-thirds of a millimeter, before it can be properly referred to as choked disc, and before it can be properly classified under the latter term. Although bilateral in the vast majority of cases, it is often more intense on one side than on the other, and may be unilateral. This form of papillitis, known as "choked disc," may be briefly described as follows: It is of grayish-red color, presents diffuse cloudiness, radiating stripes, knob-like elevation, borders indistinct, veins wide and tortuous, arteries small, vessels hidden in part, abruptly bent at the border of the papilla, while hemorrhages and white spots appear on the disc and in its immediate vicinity. The diameter of the papilla may be three times as great as the normal diameter. In high grades the borders of the disc may be entirely obliterated.

Macroscopic conditions.—An ampulliform swelling of the optic nerve sheath immediately back of the globe, the distension of the sheath extending for a varying distance along the nerve, is present in by far the greater number of cases. This swelling is due to the presence of fluid in the intervaginal space. Leber and Broadbent[1] have observed such ampulliform distension without papillitis. Papillitis also occurs in a few cases without marked distension of the optic nerve sheath. In some cases the optic nerve itself appears to be larger than normal.

At the inner margin of the sclera, at the foramen scleræ, the diameter of the nerve is narrowest; immediately within the opening the tissue of the disc expands, crowding the posterior layers of the retina away from the margin of the choroidal and scleral opening, and the disc forms a crater-like elevation which reaches a height of 0.5 to 2.5 mm. The depression in the center corresponds to the physiologic cup.

Microscopic appearance.—The microscopic conditions present in five cases examined by the writer are as follows, the elevations one to two millimeters:

CASE 1.—No ampulliform dilatation exists. The nerve-fibers present but little change either in the papilla—other than change of direction—or in the nerve trunk. Great distension of veins and diminution in the size of the arteries at and beyond the lamina cribrosa, is evident. The walls of the vessels are not changed, and there is no increase in the number of capillaries. There are few minute hemorrhages near the surface and in the center of the papilla; hemorrhages into the deeper layers of the retina in the immediate vicinity of the disc, evidently from capillaries. Moderate small cell infiltration about the walls of the vessels is evident, as is found in infectious inflammatory processes.

Marked serous edema of the tissues of the disc extends into the nerve-fiber layer of the retina, to a distance of 1.5 to 3 mm. from the margin of the foramen choroidæ. The connective tissue elements of the disc immediately above the choroid are greatly distended, pushing the deeper layers of the retina away from the margin of the choroid—which membrane is not disturbed—for a distance of .5 to .75 mm. The ganglion cells are intact, the pigment layer of the retina much disturbed, and there are numerous small cells in the intervaginal space.

CASE 2.—The intervaginal space is moderately distended, and there is simple extensive edema of the tissues of the disc, but no small-cell infiltration. The optic nerve trunk is edematous. The deeper layers of the retina—below the ganglion cell layer—are pushed away from the margin of the choroid at the foramen choroideæ, by the edematous tissue of the disc. There is no appreciable change in nerve-fibers, other than a moderate change in direction. The veins anterior to the lamina cribrosa are dilated, the arteries are reduced in size, and posterior to the lamina cribrosa the central artery appears to be slightly enlarged; it is certainly not smaller than normal. The ganglion cells are intact.

CASE 3.—There is marked distension of the intervaginal space, the nerve-fibers apparently unchanged, except in their course, the arteries reduced in diameter and the veins distended in the papilla and at the cribriform plate. Posterior to the cribriform plate the arteries are not reduced in size, but appear to be larger than normal. There are some small hemorrhages from capillaries in the ganglion cell layer in the vicinity of the disc. A few foci of degeneration and small-cell infiltration are found at the apex of the papilla, and some small-cell infiltration posterior to the lamina cribrosa. The ganglion cells are less numerous than normal, and some of those that remain are undergoing degenerative changes. The disturbance in the retina at the foramen choroideæ is as described in the preceding cases.

CASE 4.—This is as in Case 3, with the following exceptions: There are a number of small hemorrhages into the outer reticular and outer nuclear layers of the retina in the immediate vicinity of the disc, and the nerve trunk posterior to the lamina cribrosa is edematous.

CASE 5.—Enormous distension of the intervaginal space exists. The artery and vein posterior to the lamina cribrosa are apparently smaller than normal. At and anterior to the lamina, the arterial trunks are small and the veins engorged. This was the only case in which there appeared to be constriction of the artery posterior to the lamina cribrosa. The enlargement of the papilla is apparently due to simple edema. The edema of the nerve trunk is more pronounced between the nerve-fiber bundles, but there is evidence of limited edema of the bundles themselves. The retina is pushed away from the foramen choroideæ by the edematous tissue of the papilla. The ganglion cells are reduced in number and undergoing degenerative changes. There is no appreciable small-cell infiltration.

Summary.—Distension of the intervaginal space exists in all but one case, infiltration of the small cells in the sheath in one case and in the papilla—not very marked—in two cases. The arteries are small at and anterior to the lamina cribrosa in all cases, and small posterior to it in one case. The veins are dilated anterior to, and reduced in diameter posterior to, the lamina cribrosa, the ganglion cells undergoing degeneration in two cases. The retina is pushed away from the choroidal foramen and the retinal pigment disturbed in all cases. The choroid is intact. There are hemorrhages from the capillaries.

In three cases of papillitis which developed a few days before death, Ulrich[2] found edema of the nerve trunk and compression of the retinal vessels within the nerve.

It must be conceded that in the papillitis accompanying brain tumor there are two varieties which merge imperceptibility, the one into the other. The typical example of one form is a simple edema of the disc. The typical example of the other form is the violent inflammatory affection of the nerve and disc sometimes extending to the retina. Both may be accom-

*Presented to the Section on Ophthalmology, at the Fiftieth Annual Meeting of the American Medical Association, held at Columbus, Ohio, June 6-9, 1899.