THE LARYNGOSCOPE.

VOL. XVIII. ST. LOUIS, MO., JUNE, 1908.

No. 6.

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding
that they are contributed exclusively to THE LARYNGOSCOPE.)

A CONSIDERATION OF THE "HERD" THEORY AS AN ETIOLOGICAL FACTOR IN OZENA.*

BY CLEMENT F. THEISEN, M. D., ALBANY, N. Y.

In this paper the writer will limit himself to a consideration of the "Herd" theory as a cause for atrophic rhinitis. Ozena, which, according to its Greek derivation, means really a stench, should not be considered a disease by itself, but rather a symptom of some other underlying pathological process. It seems to the writer that it would be less confusing to speak of all cases in which the characteristic atrophic changes with crust formation are present, as cases of atrophic rhinitis, whatever the cause may be.

In considering the etiology of ozena or atrophic rhinitis from the viewpoint of accessory sinus disease, the "Herd" theory of Gruenwald, Hajek Loehnberg, Michel and others, I, of course, am fully aware that we can only explain a certain percentage of the cases in this way. But I am convinced that a fairly large percentage of ozena cases are associated with disease of the accessory cavities.

I would like briefly to give the results of my own observations: In 1904, in the January number of the Albany Medical Annals, a preliminary report of the results of examinations of the accessory sinuses in ozena cases was given. In this paper the twenty-one cases then reported will be briefly reviewed, and a further report of thirty-nine cases will be given, sixty in all. These were all cases in which the characteristic atrophy of the membrane with crust formation was present.

In eight of the twenty-one cases first reported, positive evidence of sinus disease was found. In four cases, chronic maxillary sinu-

*Part of a Symposium read before the Section on Laryngology and Rhinology of the New York Academy of Medicine, February 26, 1908.

itis, in two combined with chronic ethmoiditis existed. In three cases, ethmoiditis existed alone (cases of unilateral atrophic rhinitis), and in one case chronic frontal sinusitis and ethmoiditis. In the three cases in which the ethmoidal cells were found diseased, the atrophic process was practically confined to the middle turbinal body, and in these cases a direct etiological relationship certainly appeared to exist between the ethmoid disease and the localized atrophic changes. These cases were greatly benefitted by curettage of the ethmoid cells.

In the second series of thirty-nine cases, accessory sinus disease was found in six cases. In two cases ethmoid and antral disease was found, in two ethmoid disease alone, in one frontal and ethmoid, and in one ethmoid and sphenoid. In this series of cases also, in the two cases in which ethmoid disease was found, the atrophic process was practically limited to the region of the middle turbinate. The writer's cases then show fourteen cases of accessory sinus disease out of sixty ozena cases, or about twenty-five per cent.

The methods used in arriving at these results were briefly as follows: In every case as thorough an investigation as possible of the accessible sinuses was made. The nostrils of each patient examined were first freed of crusts, and thoroughly washed out, and then transillumination was employed. The maxillary antra in all cases in which the patient's consent could be obtained, were punctured in the usual way through the naso-antral wall and washed out. In the cases in which it was found that antral disease existed, decomposing and usually foul smelling secretions were washed out.

The diagnosis in the cases in which chronic frontal sinusitis was found, was made by transillumination and by washing out the sinus when possible. In one of these cases the radical operation was performed, and a greatly thickened and degenerated membrane with some pus was found. The nasal condition was greatly benefitted by the operation. In one case in which ethmoid disease had been found, a large sequestrum was removed. It was part of the left lateral ethmoid mass. The ethmoid cells were filled with a thick, foul smelling secretion. This patient was probably a syphilitic subject.

Time will not permit me to give a detailed report of every case in which sinus disease was discovered, but it may be of interest to consider one or two of the individual cases.

In the case of Miss S., aged thirty years, a foul-smelling discharge had existed since childhood. In this case ethmoid and sphenoidal sinus disease was present. The atrophic process was so extensive that the sphenoidal sinus could be readily probed.

Several operations were performed with the result that the crust formation and odor have practically disappeared. This patient I have had under observation for many years.

In another case, a boy, aged nine years, there was a history of an attack of nasal diphtheria five years before I saw him. His mother, who contracted the disease from him, stated that from the time he had diphtheria, he had suffered from occlusion of the nostrils, and an offensive yellow discharge. On examination, both nostrils were filled with badly smelling soft crusts, and the typical atrophy was present. When the crusts were removed, pus could be seen in the middle meatal region of both nostrils, and was found to come from the maxillary antra. Cultures were taken from both nostrils and examined in the Albany Bender Laboratory by Drs. Pearce and Winne, who reported that they showed the presence of typical diphtheria bacilli, in conjunction with an organism that was probably Friedländer's *Bacillus mucosus capsulatus*. The non-virulency of the organisms was proved by inoculating guinea pigs with pure cultures of the bacilli obtained from the nose. The animals all remained well.

The mother positively stated that the boy's nose had never troubled him before his attack of diphtheria, so that in this case there does not appear to be much doubt that the antral empyema caused by the diphtheria infection was responsible for the typical changes in the nose. It is also of interest that the bacilli were still in the nose after five years.

The majority of the cases of atrophic rhinitis seen by the writer occurred in children and young adults, and in many cases a history of measles, scarlet fever, or diphtheria from which the patients had suffered early in life was obtained. A common statement of the mother or father was that the child had had a yellow discharge and bad smell from the nose off and on since the attack of scarlet fever or diphtheria, and this brings us to what I believe is one of the most important etiological factors in connection with the "Herd" theory of atrophic rhinitis and a strong argument in its favor.

The discharge from the noses of these children, following their infectious disease, was caused by an infection of one or more of

the accessory cavities, and it is a reasonable deduction that in some such cases the mechanical irritation produced by the more or less constant discharge would finally lead to the atrophic changes in the membrane.

The investigations of Pearce are of interest in this connection because they show the frequency of accessory sinus disease as a complication of diphtheria and scarlet fever in early childhood. Pearce examined the accessory sinuses of fifty patients who had died of diphtheria or scarlet fever in the Boston City Hospital. Thirty-nine cases of diphtheria were examined, and in twenty-five, changes in the accessory sinuses were found; both antra in sixteen; both antra, sphenoid and ethmoid sinuses in two; one antra only in five, and the sphenoid sinus only in two. Of the eighteen double antrum cases, the exudate on both sides in three was a thick yellow pus; in three a purulent fluid with membrane, and in one, a cloudy serous fluid with membrane. Five cases of diphtheria with scarlet fever were examined, and in two changes in the antra were found; one was unilateral and contained a thick, yellow pus. Two cases of diphtheria with measles were examined, and both antra in each case contained a sero-purulent fluid. Four cases of scarlet fever were examined, one antrum was normal; in one case there was a double empyema, and in another, both antra and the sphenoid sinus contained greenish pus.

Forty-four of these children were between the ages of two and six years. The fact that cultures from the nose showed the presence of diphtheria bacilli, which in some cases on record persisted for months and years, is of great importance, because in such cases the infection undoubtedly persisted in the sinuses, and it is probable that sinuses so infected would not get well until they were opened and properly treated. Such conditions in very young children would very likely go unrecognized for a long time, and there is no good reason why the more or less constant discharge over the mucous membrane would not finally produce localized atrophic changes. There is no doubt that many of the cases of so-called purulent rhinitis of childhood, belong in this class.

Emil Mayer has also reported a case of purulent antral empyema in a child two and one-half years old, following scarlet fever and diphtheria.

These reports prove the fallacy of the theory advanced by some writers, that cases of atrophic rhinitis reported in very young chil-

dren, could not be secondary to sinus disease because the accessory sinus did not exist so early in life. It is probable that a large percentage of children who have had infectious diseases, have, as a result, a purulent sinusitis, and a certain percentage of them certainly later on have an atrophic rhinitis.

Loehnberg, who at the present time, is perhaps the most enthusiastic advocate of the "Herd" theory, states that he found the accessory cavities diseased in every one of seventy-nine ozena cases, i. e., twenty-four times an empyema of the sphenoid sinus; six times of the frontal; ten times of the ethmoidal cells and thirty-nine times of the maxillary antrum. This is certainly a strong argument in favor of this theory. Alexander quotes the investigations of other authors who reported twenty-two autopsies of atrophic rhinitis cases. The accessory sinuses were diseased in eight of these cases.

Michel believes that ozena depends mainly upon a purulent inflammation of the accessory cavities, particularly the ethmoid cells and sphenoidal sinuses, and Gruenwald also states that a considerable percentage of ozena cases are caused by purulent processes in the accessory cavities. While Hajek is of this opinion, he does not believe that this alone is sufficient to explain the pathogenesis of the whole clinical picture of ozena.

In conclusion, it appears to the writer that the "Herd" theory offers the most reasonable explanation for a fairly large percentage of atrophic rhinitis cases. None of the other theories, the rarefying osteitis theory, the theory that the atrophic process follows a previous hypertrophy of the membrane, and others too numerous to mention; give a satisfactory explanation for the large amount of secretion necessary to form the large crusts we see in some cases.

The function of the mucous glands must certainly be seriously interfered with when they become destroyed, as they do in severe cases, because microscopical examinations do not show glandular elements in tissue atrophied to the extent that it is in certain advanced cases of atrophic rhinitis. The secretions in many of these cases must come from the neighboring sinuses. If the theory that a rarefying osteitis, producing a shutting off of the blood supply to the mucous membrane, is correct, why should it in some cases produce atrophy, and in others just the opposite condition, oedema and polypoid degeneration of the membrane?

BIBLIOGRAPHY.

- MICHEL, E. Die Krankheiten der Nasenhöhle und des Nasenrachenraums, Berlin, 1876.
- GRUENWALD. Die Lehre von der Naseneiterungen. 1893.
- ABEL. Bakteriologische Studien ueber Ozaena simplex. *Centralbl. f. Bakteriologie*. Bd. XIII, s. 161.
- LOEHNBERG. Actiologie und operative Behandlung der genuinen Ozaena, etc. *Berl. Klin. Wochenschr.*, No. 11 to 13, 1900.
- HAJEK, M. Pathologie und Therapie die entzündlichen Krankheiten der Nase, s. 267, 1899.
- ALEXANDER, A. *Arch. f. Laryngol. u. Rhinol.*, Heft 1, Bd. 14, 1903.
- PEARCE, RICHARD M. The Bacteriology of the Accessory Sinuses of the Nose in Diphtheria and Scarlet Fever. *Jour. of the Boston Soc. of Med. Sci.*, March, 1899.
- MAYER, EMIL. Empyema of the Antrum of Highmore in Young Infants. *Trans. Amer. Laryngol. Ass.*, 1901.
- 172 Washington Avenue.

A Case of Vincent's Angina in which the Larynx and Trachea were Involved. H. W. BRUCE. *Lancet*, Oct. 12, 1907.

Describes a case in a man, aged 47, in which Vincent's angina not only involved the fauces, but attacked the mucous membrane of the larynx and trachea. The characteristic bacilli were present in large numbers in smears taken from the slough, but no spirilla were discovered. Laryngeal obstruction necessitated laryngotomy. At the end of five days, although respiration was relieved, the tissues of the neck around the wound were attacked by a sloughing process. The skin and subcutaneous tissues were converted into a gray foetid material; the gangrenous process spread outwards and downwards as far as the clavicle, the lungs became involved; and the patient died eleven days after admission. Post-mortem examination showed sloughing of the uvula. A thin green slough covered the ary-epiglottic folds, the mucous membrane below the false cords, and the trachea almost down to the bifurcation. The author remarks on the unusual features of the case, and compares it with the mild form of phagedaena or hospital gangrene.

THOMSON.