the etiology of choked disc, and I, for one, believe that most of these cases are inflammatory in character though there may be, now and then, a case where it is a mechanical edema. I have been led, during the last year or two, to a consideration of the effects of toxic materials in the blood in producing inflammation. There certainly can not be an inflammation without cause, and in many of these obscure cases there is a toxin, the product, perhaps, of processes in the intestinal tract, or other parts of the body, that has a special action on the optic nerve. It is coming to be believed more and more that malignant disease—and most brain tumors are malignant—is of parasitic origin, and is it not possible that it also generates some toxic substance which produces the optic neuritis so often seen in connection with brain tumor?

DR. J. E. WEEKS, New York City—What particularly impressed me in the examination of the pathology of these cases was the fact that the ganglion cells in certain portions of the retina were destroyed, and it seems to me that the contraction of the field of vision and the concentric scotoma met with are due to destruction of the peripheral neurones rather than any direct effect on the nerve-fibers.

In regard to the diagnostic value, the study I have made has convinced me that the presence of a tumor in any particular part of the brain is not determined by the papillitis. We find papillitis most frequently accompanying tumors affecting the corpora quadrigemina, next with those of the temporo-sphenoidal lobe and next with those of the cerebellum. In 18 cases of tumor in the pituitary body there was papillitis in 50 per cent. There is, in all probability, some diagnostic value to be attached to cases where the papillitis is most marked in one eye. In 14 cases of tumor of the frontal lobe, 12 showed papillitis on the side of the tumor and 2 on the opposite side. In tumor of the cerebellum the presence of papillitis, more marked on one side than the other, is of no value whatever. I am not familiar with the study of the arterial pulse in cases of papillitis.

The question whether the condition of the optic nerve is an edema or an inflammation has engaged my attention a great deal. I am convinced that, in certain cases, it is an edema and not an inflammation. In the examination of five cases I found no evidence of inflammation.

DIAGNOSIS AND TREATMENT OF MIDDLE EAR DISEASES,

WITH A NEW DIAGNOSTIC SYMPTOM OF EUSTACHIAN STRICTURE.*

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What are we doing for middle ear troubles? Are we doing all that is possible? Are results of treatment satisfactory to ourselves and our patients? Have we made our diagnosis in a given case and decided on our line of treatment on the patient's first call, and is this satisfactory to ourselves?

If we are satisfied to be honest with ourselves and our patients, then all these questions are answered in the affirmative. There are two things necessary for our own satisfaction and consciousness that we are doing the best thing possible for our case: 1. An easy practical distinctive classification of the diseases of the middle ear. 2. The exposition of the case to the patient or his friends, where their intelligence makes it possible.

For a practical classification I have adopted the following:

OTITIS MEDIA.

Acute Catarrhal.....	
Chronic Catarrhal.....	
Acute Purulent.....	
Chronic Purulent.....	
Subacute Intumescens.....	
Subacute Cirrhotic, Hypertrophic or Atrophic.....	

With these we have only to affix the complications in any case, and a distinctive, practical diagnosis is made.

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

ACUTE CATARRHAL OTITIS MEDIA.

The acute catarrhal form is usually complicated with, or occurs in the course of, contagious fevers and acute inflammatory affections of the upper air tract, and is usually limited to the lower part of the tympanic cavity. All are familiar with its symptoms: Pain in the ears and along the jaw; tinnitus, fulness, throbbing and dulness of hearing; frequently fever. The drum shows congestion and is bulging; if perforated, there is discharge, pearly or milky white, thin, watery and abundant, the odor not offensive; if not perforated, puncture is indicated with a cautery electrode or the spear. Relief follows within a few hours. Apply dry heat to the ear, pilocarpin internally or by hypodermic syringe, and cleanse the nose and throat by wiping, not syringing, and one inflation through a catheter, when possible to use it. When the case has gone on to rupture of the drum, and you see a large hole with catarrhal discharge, cleanse the throat and nose as before. Syringe the ear daily with warm *Dobell* or weak soda bicarbonate solution, and follow with a dropper full of zinc sulphate, 7 gr. to the ounce, wipe out thoroughly dry, inflate the ear once or twice and dry again. Do not keep the ear plugged with cotton, except when the patient is out doors. Cleanse it every other day yourself, by washing—by zinc as above—and catheterization and drying, the patient using a dropper full of the zinc solution at home, three times a day, letting the water run out as well as it can after retaining it a minute. Powders are not as good as the zinc, except in cases where the hole in the drum is large enough for some of the powder to get into the middle ear, in which cases cleansing every two days, drying and using of boric acid powder will probably heal the inflammation as quickly as does the zinc. I like the latter better than the silver or permanganate solutions. It does not cause any pain, as a rule, even in solutions of 10 to 15 gr. to the ounce. I seldom use the peroxid as it does not hasten the cure, sometimes I have thought it retarded it.

CHRONIC CATARRHAL OTITIS MEDIA.

I treat the chronic catarrhal troubles, following fevers and la grippe, the same as the acute form. It is not necessary to detail symptoms for diagnosis in this class of troubles. There is a form of chronic catarrh, which I call tubercular, simply because it occurs in tubercular patients. They are very intractable, but if the constitution is good or fair these yield to treatment as well as those catarrhs occurring in the non-tubercular.

ACUTE PURULENT OTITIS MEDIA.

This is an aggravated form of catarrhal otitis, infected with pus germs. It is usually complicated, involving the attic and antrum. Pain is more intense and fever higher. The drum is ruptured sooner and the pus soon has a very bad odor, from its confinement. These patients should be treated at home, by appropriate medicines for fever and pain, and close watch on the mastoid. Puncture the attic if it is bulging. This form yields more quickly than the simple catarrhal form. The treatment is the same, except that you should treat the patients each day yourself. I have known them to get well in five or six days, while it is more common to have catarrhal forms run into the chronic or subacute, before getting well. In fact, I am always expecting the subacute ones to come back after they are apparently entirely well. This acute purulent form may run a rapid course for the patient. It is necessarily very destructive, although when healed quickly, little or no apparent injury has been done the hearing. Should you have mas-

toiditis, delay is dangerous. Wilde's incision is good practice, even on suspicion.

CHRONIC PURULENT OTITIS MEDIA.

The chronic suppurative cases are so called when there is the characteristic odor of diseased bone, even when there is scarcely any apparent discharge. These are decidedly obstinate, and yield, in the majority of cases, only on an operation, i. e., thoroughly scraping out the attic and tympanum, or even making the radical mastoid operation. No running ear is safe, although the catarrhal form is least dangerous. The purulent form is absolutely dangerous.

Chronic hypertrophic rhinitis and more or less stenosis, from spurs or deflected septum, have nothing whatever to do with causing any form of aural trouble. This may seem a remarkable statement in view of prevalent teaching. All specialists meet with stenosis, spurs, hypertrophy of turbinates extending into the posterior nares, and large soft pharyngeal tonsils in which there is absolutely no involvement of the ear, consequently, if they were causative factors, these obstructions could not exist except when complicated with aural troubles, while many cases of acute and chronic aural troubles are seen in which there are neither obstruction, hypertrophy of turbinates, nor enlarged hard or soft pharyngeal tonsils. Then, if these nasal pharyngeal troubles do exist and are corrected, the aural trouble remains the same or continues to progress, they are simply incidental and a part of the dyscrasia. Adenoids may, and usually do, cause deafness, quite as frequently, though without catarrh or discharge from the ear, as with it, and therefore their action is more mechanical, aided by the diathesis always present in these cases. While not admitting cause—except in adenoids—I always try to correct these nose and throat defects.

CHRONIC CATARRHAL INTUMESCENT OTITIS MEDIA.

I desire particularly to call attention to the distinction I shall make in describing this disease. It is this affection which, when not specifically distinguished, causes much confusion. There is such an affection and it is distinctively marked. It is always complicated by chronic soft swelling of the Eustachian tube, thick, red and soggy uvula, and pharyngeal pillars, and more or less redness and soggy or granular appearance of the upper pharynx. In this affection you will be most likely to meet with intumescent turbinates or other obstruction of the nares. The drum may be good color and but little changed in general appearance, or it may be bulging or retracted, or lax. One ear is usually much better than the other. There is another symptom which is diagnostic in this affection, although the condition outlined is sufficiently characteristic. This symptom is the subjective one of hearing. These patients hear a watch badly, but the voice is heard comparatively much more distinctly. I have had many patients who could not hear the watch except when pressed, or within an inch of the ear, yet could hear conversation distinctly. These *always* have tinnitus. The Eustachian tube is *always* obstructed. In fact, this affection is primarily a Eustachian inflammation. But changes are always found in the ear as well, and it is a true chronic intumescent catarrhal otitis media. It is most frequently found in persons who are exposed to all kinds of weather, as railroad engineers, brakemen, miners, teamsters, etc. Treatment of this affection seems of little benefit. I treat the nose, throat and Eustachian tube by astringents and dry heat, iodine, vapors of camphor, menthol and albolin, with inflation and occasionally gentle dilatation. Massage is scarcely indicated and accomplishes nothing.

Catheterization is beneficial, but should not be pursued as routine treatment. Valsalva's method should never be practiced except to coax up a spray; never by the patient at home. Pilocarpin is of service in some cases. Puncture of the drum will relieve tinnitus, until the puncture heals. Removal of the drum and malleus or incus will improve hearing. But these patients should be treated continuously for a year before operation is done, unless quite deaf, or they desire the operation. Change of climate will sometimes aid them, as low altitude, hot and dry, e. g., Old or New Mexico. Change of occupation is sometimes beneficial. These cases *sometimes* yield unexpectedly, and satisfactorily to the treatment. It is possible that they become hypertrophic, then atrophic or cirrhotic, like the following type, but if they do it is very slow indeed.

SUBACUTE, CIRRHOTIC OR ATROPHIC OTITIS MEDIA.

This affection has two stages, which can not always be demonstrated in a given case. I would liken this disease to hypertrophic rhinitis, and the preceding affection to intumescent rhinitis. It has been, I think satisfactorily proven that atrophy follows hypertrophy of the turbinates. It is also positive that in hypertrophy of the turbinates the tissues are hard and do not contract decidedly under cocaine, as does the intumescent form. Now follow this same form in the ear and Eustachian tube, and note the comparisons: The hard pharyngeal tonsil becoming finally atrophic; the hard hypertrophied turbinate becoming the atrophied turbinate; the cirrhotic hypertrophic Eustachian tube and tissues of the middle ear becoming atrophic. Do not forget the two stages of this disease and you can account for different symptoms, in cases much alike.

Symptoms.—Deafness slowly coming on, with or without tinnitus. When tinnitus is present you have the hypertrophic stage. Aerial conduction is more or less lost, hearing the watch only when very close or pressed to ear. The patient hears some better with his mouth open and still better in a noise during the first stage and up until the stapes becomes firmly locked to the oval window, when noise makes but little difference. Hearing for conversation is much worse than in the intumescent form. To appreciate this difference you must recall your cases and differentiate on these lines. The appearance of the nose and throat will likely be in line with this condition, purely incidental however. The objective symptoms are: drum retracted—may be good color but light spot displaced; Eustachian tube fairly well open or, if in the second or atrophic stage, wide open.

Treatment.—In either stage, medical treatment avails absolutely nothing. While these cases seem to remain stationary, at times the tendency is to result practically in almost total deafness. The only hope in medical treatment is to stay the progress of the disease, and this is almost a forlorn one. Operation is the best thing in either stage, and better in the earlier one. I have adopted the following course, when operation is contrary to the patient's wishes: Treat the nose, throat and tube with stimulants, and try to put some of them in the middle ear. Give iodine of potassium and strychnin internally. If massage is good in any aural affections, these are the ones, but I have little faith in it here. Inflation is of no benefit aside from its massage; indeed, it seems harmful, but it seems impossible to avoid inflation when you apply medicines in the tube and ear. You can pursue this treatment as long as the patient sticks. Always, however, bear in mind and inform the

patient that the operation is the thing which promises better hearing. This is the removal of the drum and the malleus or incus or both; the results are quite good. It is neither necessary nor good practice to remove the stapes, and besides being exceedingly difficult, it adds greatly to the danger, and its removal only temporarily increases the hearing over the other operation, because the oval window soon becomes covered with a firm cicatrix, more or less of an epidermic character, and deafness equally as great as before results.

Hearing is not as good after an operation in the atrophic stage as in the hypertrophic. In the cirrhotic the nerve also is sometimes involved. I believe all cases of middle ear trouble are due to some one or other of the dyscrasias, syphilitic, scrofulous, rheumatic, or others.

A word about tinnitus aurium: Its cause seems simple. It is due to more or less ankylosis of the joints of the ossicles with each other and of the stapes to the oval window. Sound waves striking the drum constantly are as constantly transmitted through the ossicles to the fluid of the internal ear. If you close both ears with your fingers, you are pressing the ossicles together, thus ankylosing them and the stapes firmly against the oval window. Now you note a loud rumbling noise, because of vibration through the head and from the mouth. If you talk, you get the same sound a deaf person has, as though you were talking through or into a barrel, showing conclusively that it is in the drum and ossicles that the trouble lies, and that this is due to solidification or, in other words, to more or less ankylosis. When fluid is in the ear, as in acute or chronic affections, you mechanically have the same thing, a solid body acting as a transmitter—in other words a greater or lesser loss of aerial conduction, plus pressure. I am perfectly satisfied with this explanation of tinnitus, which has been explained in so many different ways. If you take a common bass drum and glue a straight stick to the center of the head, on the inside, and strike either head, you get on the other side a muffled sound; but if you glue a chain to each head, on the inside, you get a sound free from all obstruction, in fact a clearer sound than without the chain. The explanation is that transmission of sound is conducted through the chain, instead of direct vibration, when the ear is obstructed you get direct vibration, when the ossicles are normal, you get conduction. If you puncture or remove the drum, noise stops in nearly every case, because sound waves now land against the internal ear wall and conduction is again established in the internal ear. If noise persists after puncture of the drum, it is because there is firm ankylosis or great retraction of the drum, and the hole practically does not admit the waves against the inner wall and the drum and ossicles still receive the vibrations. Conduction through the proper apparatus is absolutely essential for perfect hearing. So if the drum and ossicles are disabled or removed, you will lose some acuteness of hearing. The question arises, why, if the middle ear is stenosed by fluid, are not waves transmitted to the internal ear the same as when the ossicles are removed? The answer is that peculiarities of sound waves are lost and force is broken in transmission through fluid parts. Why do not all persons have tinnitus if ankylosis is present? This question is answered by the fact that a difference in the degree of the ankylosis is present, from that in the intumescent form and hypertrophic stage of the cirrhotic form. Where tinnitus is absent we find atrophy of the tissues covering the ossicles. Ankylosis is more *firm* and there is greater contraction of

the drum, with firm stapes—the chief cause—and the Eustachian tube is patulous and atrophic, with atrophy of the throat and nose.

Other affections of the middle ear occur, but they are usually complications of, or caused by, one or another of those treated in this paper, and are sufficiently treated in the standard text-books. My excuse for this paper is purely in its positiveness as to classification, diagnosis, and treatment. It represents condensed opinions of the best authors, with some experiences of my own. In assigning a separate class to the atrophic or cirrhotic form, it is done with a conviction that it occurs without being preceded by the intumescent or catarrhal one. In a few cases it would seem that this form is atrophic, from the beginning, but I am inclined to believe that it is a parallel condition to that found in the nose.

THE GOUTY DIATHESIS.

SOME OBSERVATIONS FROM A DIETETIC STANDPOINT.*

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In consideration of the subject of gout, of which so much is known, and withal so little, I must confess that in my hands a rigid attention to diet has accomplished better results than much medication, excluding perhaps those acute explosions in which I have found that a few good doses of piperazin or maizo-lithium for forty-eight hours have given the greatest immediate relief.

Practically, hereditary gout, as well as chronic, is a direct or indirect result of either too generous living and too much "good cheer" in the immediate ancestor, or an improper diet or indulgence in alcoholics in the subject, with also, in some cases, bad climatic influences. Arguing on this supposition, what could be a more rational procedure than to first withdraw the cause of the condition?

In the hereditary forms—and they compose about 75 per cent. of all cases—there is evidently a weakened condition of the eliminating power of the excretory organs, and a lack of physiologic resistance to the accumulation of the urates, with a consequent loss of the metabolic power. Such cases as these, "born and bred in the bone," as they are, require at our hands, a most rigid prohibition from their diets, of the nitrogeous and leguminous foods and alcoholics from infancy, and a dietetic education of the stomach and palate to a liking for green vegetables, as well as a training in the knowledge that alcoholics and nitrogenous food, and cold and dampness may eventually conclude in the gouty condition which is their inheritance. In those cases of acquired gouty diathesis we have a less persistent condition of affairs to deal with, principally a change of habits, and of these notably the diet, for I have seen as many cases of this sort in which there was no attending use of alcohol, as with it, and in most of these cases a rigid and proper attention to diet has been of more benefit than medicinal treatment where the restricted diet was persisted in.

Without discussing the various views as to the etiology of gout, we may with safety assert that there is always an excess of the urates, and whether or not it be due to a virtual excessive manufacture of them in the economy, or to a storing up of them from a deficient elimination, we know that they are stored away as deposits, and with a knowledge that either their production is

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