

external jugular. The second sound was loudest midway between the nipple and sternum, over the fourth interspace. The edge of the liver could not be plainly felt, but there was more resistance on the right side than on the left. The upper line of the liver dulness corresponded to the fifth rib in the axilla. There was no œdema of the legs. The child grew gradually worse and died a few months later with symptoms of cardiac failure. No autopsy was permitted; so that the probable diagnosis of mitral regurgitation, with, perhaps, some communication between the two sides of the heart, could not be ratified.

Owing to the fact that the diagnosis of the cardiac condition in the 18 cases which have been just cited was confirmed by autopsy in but four instances, and that the descriptions of the changes in the eye-grounds in most cases are too meagre from which to draw inferences regarding the precise lesion in the fundus which the various cardiac lesions may occasion, it has not seemed worth while to attempt to summarize the facts which have been given, or to seek to prove the truth of Nagel's assertion from them. As indicating the value of ophthalmoscopic examination in cardiac cases of this type, it is of interest, however, in addition to the case of Babinski, which has already been cited, to refer to Carpenter's second case in which there was no cardiac bruit and but slight cyanosis, and the true nature of the complaint was determined only after the discovery of the typical changes of cyanosis retinæ by the ophthalmoscope.

## SCARLATINAL OTITIS.

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STRANGE as it may seem in these days of advanced thought in medical science, there are yet a few physicians who are firmly adherent to the old notions of tentative medicine.

After all that experience has taught as to the dangers to the functions of hearing and of life itself by ear inflammation complicating scarlet fever, yet there are many of our profession who assume an attitude of expectancy and contented helplessness, and a peculiar sense of satisfaction when an ear aches, breaks, and runs during the course of this infectious disease.

This is unfortunate for the science of medicine and for the human race, and inexcusable in these days of enlightenment.

We might excuse the doctor of past generations, who felt that scarlet fever must leave some "corruption" in the system and felt

that the organ of hearing could be spared better than the kidneys or other organs, so he welcomed with delight the appearance of pus in the auditory canal, feeling that now the system was safe from harm.

Otitis media is the most frequent complication of scarlet fever, and, everything considered, the most serious. Like the general infection, its severity varies with the epidemic, the climate, and the seasons of the year. It is more common in the winter and spring months and in the colder climates. Statistics show that from 3 to 9 per cent. of the cases of scarlet fever have ear complications, and in about 50 per cent. of these cases both ears are affected. Bezold, of Munich, collected 640 cases of aural disease secondary to scarlet fever covering a period of eleven years in which 984 organs of hearing were affected, about one-half of them being bilateral. During the same time he estimated, from other statistical sources showing 17,087 cases, that 3.75 per cent. of all cases had ear complications. In 530 of Bezold's cases 263 had a continued discharge from the ear lasting over eight years; and a total destruction of the drumhead in 25 per cent. of these cases. In another report of 185 cases he shows that in 30 there was an entire destruction of the drumhead with the loss of one or more of the ossicles; in 59 the perforation in the drumhead comprised two-thirds or more of the membrane; in 13 there were small perforations; in 44 granulations and polypi; in 15 total loss of hearing on one side, and in 6 cases total loss on both sides; in 77 cases the hearing distance for whispered voice was less than half a metre.

Of 4397 cases reported by Finlayson, otitis occurred in 10 per cent., and of 1008 by Caiger, 13 per cent.; in Burkhardt-Merian, 33 per cent. The greatest importance is attached to the inflammation of the middle ear, although the affection of the labyrinth or otitis interna are of no little moment, when one considers the large number of cases of total deafness, and the large percentage of inmates of deaf-mute institutions where otitis is due to scarlet fever.

Dr. Blau found from statistics of eight authors 14,045 cases of otitis interna with 55 cases, or 3.8 per cent., due to scarlet fever. Bruckner gives 7 per cent.

In a survey of the deaf-mute asylums of Europe, in 1887, the smallest percentage was found in Italy, 1.5 per cent., and the largest in Norway, 27 per cent., representing the ratio of the cases due to scarlet fever and showing the relative frequency of the disease in the warm and cold climates.

May has collected statistics in New York of 5613 deaf-mutes, 572 of whom, or nearly 10 per cent., owed their condition to otitis of scarlet fever. Holmes, of Cincinnati, reports that in the deaf-mute institute in Jacksonville, Ill., of 500 cases 7.2 per cent. were due to this disease. In the Rhode Island School for the Deaf out of 201 cases admitted 44, or 21.3 per cent., were due to scarlet fever.

In the Clarke School of Northampton, Mass., 105 out of 640, or 16.4 per cent., were due to scarlet fever.

On the other hand, statistics at large show that of all cases of suppurative otitis media about 12 per cent. are due to scarlet fever, thereby showing that a disease which impairs and often destroys the function of one of the most important organs of special sense, which is the focal point of infection in many cases of septicæmia, pyæmia, meningitis, brain abscess, and is a constant menace to the patient's life, is a complication of scarlet fever which is often ignored or even welcomed by the physician with content.

For illustration in the study of this disease I wish to use a season's experience in the scarlet-fever wards of the Rhode Island Hospital. Beginning about the middle of January, 1904, and continuing till the middle of June, I had the privilege of watching some 60 cases. The ears and throats of each case were systematically examined at regular intervals. During these five months 7 cases of suppurative otitis media developed, and 3 cases were in active progress when the patients were admitted, making 10 cases in all, or about 17 per cent. Seven cases were bilateral and 3 unilateral. Of the 7 which developed after admission, 4 were characterized by pain in the ear and rise of temperature and increased rapidity of the pulse. Three of the 7 had no pain whatever, the only condition which attracted attention being a sudden rise in temperature which could not be otherwise accounted for. These cases were discovered by examination of the ear, in accordance with a standing order that in the event of a sudden rise of temperature or increase of fever symptoms in any of the cases, I was to be notified immediately, it being my purpose to forestall the ulcerative spontaneous perforation of the drumhead by a good, free incision, assuring immediate depletion of the parts and adequate drainage. Of the 17 suppurating ears, 2 went on to suppuration and necrosis of the mastoid. These were operated and made an uneventful recovery. One of the cases, a girl of ten years, who had a very severe general infection, streptococcic pharyngitis and nephritis, the ear is still discharging a little at intervals.

In 4 other cases, in which paracentesis was done, the drumheads healed perfectly and hearing returned to normal condition in a very few weeks. In one of the severe cases, which had nephritis but no severe pharyngitis, the drumheads of both ears and two of the ossicles (malleus and incus) of the left ear were swept out as clean as if eaten out by acid, in spite of the most careful and energetic treatment. In this case the ears were still discharging when seen ten months later. The other 3 cases were lost track of after discharge from the hospital.

The bacteriological study of these cases was intensely interesting. The cases were very carefully followed in the laboratory by Drs. Fulton, McElroy, and Barrows.

Four cases on which paracentesis was performed showed streptococcus in pure culture in the discharge which was taken from the canal immediately after the incision was made—these were the cases which healed the most rapidly—the ear returning to its normal condition in a few weeks. All of these cases, however, showed a mixed infection within a week after the first culture. An examination showed staphylococci and other organisms with the streptococci in each case. One of these also had a diphtheria-like bacillus in combination with the others. It is interesting to note that this case healed the quickest of any case in the series, and it is also interesting to note that the case which had so extensive destruction of tissue and ossicles had exactly the same combination of organisms.

The diphtheria-like bacillus which was found in 3 cases differs from the Klébs-Loeffler bacillus, so this was not considered a diphtheria infection. I might add that while we had a few cases of the double infection, diphtheria with the Klebs-Loeffler bacillus and scarlet fever, yet in these cases under consideration the Klebs-Loeffler bacillus was not found. As yet no specific organism has been isolated as being peculiar to the otitis of scarlet fever; and bacteriology has not yet given us any specific organism for this very common infectious disease. Leutert,<sup>1</sup> a former assistant at Schwartz's clinic, who has done considerable research in this line, is of the opinion that the malignant forms of scarlatinal otitis characterized by rapid destruction of tissue are due to streptococcus, and he likewise thinks that streptococcus is the germ that gives to scarlet fever the picture of a severe disease, while the so-called specific germ of scarlet fever seems to have no relation to the general infection and suppuration. Kurth frequently found a kind of streptococcus laid together in chains forming a lump-like colony which he called streptococcus conglomeratus.

Before bacteriology revealed to us the possibilities of the infection in scarlatinal otitis it was always considered as diphtheritic, and nearly all authors up to the present time classify this form of otitis as diphtheritic or scarlatinal-diphtheritic; and the natural inference is that it is necessary to have a diphtheritic infection of the throat in order to have the otitis. In our cases there was no diphtheria infection in either throat or ear.

Clinically there are three forms of scarlatinal otitis which I prefer to classify as follows: 1. Acute serous. 2. Acute suppurative. 3. Acute necrotic. The severity of the otitis seems to be dependent on the severity of the general infection on the one hand, and the constitution of the patient on the other. Anatomical conditions and the structure and condition of the vasomotor system are important factors. In children previously healthy and with

<sup>1</sup> Archiv. f. Ohrenheilkunde, Bd. xlv. and xlvii.

good firm skin, normal lymphatic system, and good resistance generally, the disease assumes the milder form if the general infection is not too severe. In children of the lymphatic diathesis, so-called scrofulous tendencies, subject to swelling of the glands of the neck, enlarged tonsils, and hyperplasia of the adenoid tissue of the pharynx, we see the severe forms of the disease.

**ACUTE SEROUS INFLAMMATION.** The mild forms of otitis occur mostly about the time of the eruption or within the first ten days, or while the prodromal pharyngitis is active. The symptoms are similar to those of ordinary acute catarrhal otitis media. If the patient is old enough he complains of a feeling of fulness in the ear, pulsating noises, slight deafness, and varying degrees of pain, some slight, some severe, and in some there is no complaint of pain at all. In the young children we are only led to think of the ear by an increase of fever symptoms and evidence of discomfort by restlessness and crying; and perhaps by the discovery of a discharge from the ear.

Examination of the ear with the head mirror and speculum under a strong light reveals, in some cases, a slight redness and swelling of the fleshy portion of the canal, according to the period of the scarlet fever and the condition of the skin of the body. If desquamation has begun, the canal is usually coated with scales of epidermis, and the wax glands seem to have an increased activity at this time while the opening of the canal is usually well coated with wax. The drumhead varies from a dull, flat appearance to that of intense congestion and bulging. After three or four days of the subjective symptoms, a discharge of serum is noticed escaping from the canal, the pain subsides, the fever symptoms diminish, and if the ear receives no secondary infection it heals after a few days. Unless these cases are under close observation, as in a hospital or by a trained nurse, they are rarely seen until the drumhead has ruptured. Probably most of these simple cases become secondarily infected and continue on into the purulent form. When this is the case the discharge of serum takes on a purulent character and becomes more profuse, the patient becomes restless and more feverish, with a general increase of toxic symptoms, and suffers more or less pain deep in the ear and mastoid bone.

**ACUTE SUPPURATIVE INFLAMMATION.** This form of otitis usually occurs somewhat later in the course of the scarlet fever than the previous form, about the second week, although it is probably dependent upon the severity of the general infection. The subjective symptoms are much more severe; the sudden increase of feverish symptoms is very pronounced, the temperature often rising to 103° or more. The pain in the ear is lancinating and throbbing in character, radiating from the depths of the ear to different parts of the head and neck. Swelling of the glands of the neck with stiffness of the muscles and torticollis are fre-

quently present in these cases. In some there is evidence of cerebral irritation shown by convulsions and vomiting, so that meningitis may be feared. We must bear in mind, however, that these cerebral symptoms are often among those which usher in the general infection, but when they occur after eruption is complete, or later, they may accompany kidney or ear complication.

*Objective Symptoms.* In cases where the drumhead has not ruptured, the canal is scaly and at its entrance abnormally coated with wax, and is often swollen. The drumhead is oedematous, swollen, varying in color from a bright to a dusky red, and bulging in parts or as a whole, to a greater or less degree, sometimes protruding a fourth of an inch or more into the canal. Even after spontaneous perforation has taken place this enormous protrusion of the membranes of the drum cavity is sometimes seen.

ACUTE NECROTIC INFLAMMATION. This malignant form of otitis is commonly called the diphtheritic form. This type of the disease is analogous to the throat condition known as scarlatinal diphtheria, although Klebs-Loeffler bacillus are not found in the discharges, nor are there any of the parietic sequelæ so common to diphtheria.

The term diphtheritic which infests the traditional teaching of disease of the throat and ear makes it very difficult to estimate the true conditions which have existed in the history of these membranous and exudative forms of disease of these organs; but bacteriology has done much, and we hope soon will do more, toward clarifying this befogged condition. Many cases of the so-called scarlatinal diphtheria, characterized by severe infiltration of the lymphoid tissue of the throat and membranous deposits on the tonsils and posterior and lateral walls of the pharynx and of the mucous membrane surface of the nose, show pure cultures of streptococci or staphylococci, or the mixture of the two, Klebs-Loeffler bacilli being absent.

This malignant or membranous form of otitis is to be considered the most severe form of ear disease, the infection being intensely virulent, and the destruction of tissue very rapid, often sweeping out the entire drumhead and the ossicles in a few days. If it is uninterrupted by treatment, the necrotic process destroys the inner membranes of the tympanum, those of the round and oval windows, invades the labyrinth, destroying the membranous labyrinth and organ of Corti, thus destroying the organ of hearing and disturbing the equilibration of the body causing a rolling, staggering, or tottering gait for a long time after recovery. Cases have been reported, and I have seen two, where parts of the labyrinth and cochlea were exfoliated in the necrotic process and removed as a sequestrum at operation.

In other cases the infection travels on through the internal auditory canal, damaging both the auditory and facial nerve, the latter

causing facial paralysis, and on into the meninges of the brain causing meningitis and death. Post-mortem examinations in these cases have revealed necrosis of the cochlea and deposits of membranous and necrotic tissue in the vestibule and other parts of the labyrinth.

The appearance of this type of otitis, as seen through the speculum at the early stage of the inflammation, is similar to the violent suppurative form, but the drum membrane soon becomes livid, or has a yellowish-white pulpy look having the appearance of a false membrane which doubtless is present, similar to the false membrane of the pharynx, and contains quantities of streptococci. The secretion at first is not abundant, in fact very little, but in a few hours a brownish, purulent fluid with a very fetid odor of tissue necrosis is present. The hearing soon lessens, the drum membrane dissolves away, and the drum cavity is filled with this membranous exudate with a foul odor. When the labyrinth is affected its onset is signalized by sounds like the ringing of bells or the clanging of steel, and by dizziness. In some cases the labyrinth becomes involved primarily.

**CAUSE.** The cause of scarlatinal otitis is evidently a streptococcus infection, the method of invasion being still a matter of discussion; formerly it was thought that the avenue of invasion was through the Eustachian tube, and some hold this view to-day. More recent investigations suggest that it is hæmatogenous, the infection carriers finding their way to the tympanum through the blood current.

It seems to me that in some of the milder forms infection is carried through the Eustachian tube, and in the severer forms through the blood current or through the lymphatic system of the pharynx and tympanum, or, perhaps, like other cavities with small outlets, the Eustachian tube becoming closed by the pharyngeal inflammation, a vacuum is produced in the tympanum, causing it to fill with serum, and in the presence of infection a suppurating process is established.

**PROGNOSIS.** Prognosis as regards both the continuity of the organ of hearing and of life is largely dependent on prompt treatment and proper care. The simple forms are favorable, the severe ones not so favorable, especially as regards hearing, for in some a large part of the hearing is lost, and the case goes on to necrosis of tympanum, ossicles, and mastoid, in spite of the most careful treatment. Cases where adenoids in the nasopharynx and enlarged tonsils are present, also children of scrofulous diathesis, are apt to have a severe time of it. And while the outlook, as far as immediate danger to life is concerned, may be good, there is always the loss of the hearing function or its impairment to be feared, and the establishment of a chronic otorrhœa which is a nuisance to the patient and his family and friends, and with all its attending dangers it is a constant menace to life itself.

**TREATMENT.** Treatment is naturally surgical and antiseptic. We are dealing with an infectious process caused by some one or a number of varieties of micro-organisms, and this simple or mixed infection, as the case may be, must be removed as soon as possible, and the part kept as free as possible from them until healing is complete.

Some recommend preventive treatment by irrigating the nose and throat daily to keep the amount of infection reduced to a minimum. This procedure I think is one attended by great risk, as there is danger of washing infection through the Eustachian tube into the tympanum, and thereby setting up the inflammation one is trying to avoid. The patient should be kept in a warm room night and day, avoiding rapid changes in the temperature, draughts, and chilling of the body after bathing; this will do much toward preventing congestion of the ear and mucous membranes of the body, for the less the circulation in these parts of the body is disturbed the less will be the danger of inflammation. When the lymphatics of the neck show signs of swelling, ice should be applied in a throat or ear ice-bag and kept on constantly. Iodide of lead ointment is also useful in reducing the inflamed glands.

If spontaneous rupture of the drumhead takes place and the ear begins to discharge serum, this should be removed by pledgets of sterilized cotton and *not* by syringing, as any disturbance of this condition as by an unsterile syringe or other instrument might result in a secondary infection. When the discharge has become purulent then it should be syringed every two or three hours, in order to keep the canal as free from pus as possible, and avoid an infection of the deeper parts and the external glands of the ear and lymphatics surrounding it.

When a case of scarlet fever is first seen by the physician, instructions should be given to the parents or nurse regarding the ear complications, and if the patient complains of pain in the ear, or manifests any discomfort in the ear by putting the hand to it or rubbing it, moving the auricle, or refusing to lie on the affected side, or if a sudden rise of temperature occurs, strict orders should be given that the attending physician be notified at once; and he should immediately examine the ears, and if any of the appearances of the drumhead above described should be present, he should at once incise it freely. If the drumhead is to be incised, the canal must first be sterilized by syringing with 1:2000 bichloride and carefully dried with sterile cotton, and a sterile instrument used. The canal should then be closed by sterilized cotton, and changed as often as it becomes saturated, great care being taken to see that the hands and instruments are clean. If the discharge becomes purulent, the canal should be syringed and any one of the following solutions may be used: Sodium bicarbonate solution 1 drachm to the pint, lime-water, normal salt solution, carbolic acid,



1: 40; saturated solution of boric acid, or 1: 500 permanganate of potassium solution. After cleansing, the canal should be dried with sterile cotton and a solution of boric acid in 60 per cent. alcohol instilled into the canal. In the severe cases a 1 per cent. solution of bichloride of mercury in 60 per cent. of alcohol, or 2 per cent. nitrate of silver solution may be dropped into the ear, these solutions having been previously warmed.

If swelling of the mastoid lymphatics or tenderness over the mastoid region occur, the aural ice-bag should be applied, and if relief is not had within a few hours, leeches should be applied over the tip and upper part of the mastoid bone. These measures will usually give prompt relief and often stay the progress of the inflammation. If the disease goes on to suppuration of the mastoid, operation will be necessary, and should be performed without delay; yet if the indications for operation are not especially urgent, I prefer to wait until after desquamation is complete, as in my experience if the operation is performed during desquamation, or if within six or seven weeks from the onset of the fever, the repair process is extremely slow, even after a week has elapsed following operation, there will be little or no formation of new tissue in the bone cavity. The discharge from the wound is very profuse and intensely acid, excoriating the parts wherever it touches, ulcerating the fresh cut surfaces and sloughing out the stitches and destroying the new healthy granulation tissue that may have formed. The large open wound resulting from this destructive process affords an unprotected area for septic infection, and with the virulent nature of the discharge, which contains quantities of streptococci and staphylococci, the system is exposed to great danger. One of our cases developed a pronounced septicæmia, unquestionably from this cause.

A word regarding the contagious nature of the discharge would not be amiss. A recent experience at the hospital will furnish a good illustration of the care needed in handling a discharging ear immediately after the scarlet fever has had its run. There were three children representing three families who had fulfilled the demand of the Board of Health regarding quarantine and returned to their respective homes, each child with suppurating ears. Within ten days after their discharge from the hospital one other child from each family was admitted with scarlet fever, and it is believed, after careful investigation, that these new cases were started by coming in contact with the discharge from the ears of the children who had just returned home. I believe that the discharge from a scarlatinal otitis serves as a good infection carrier, and cases should be detained in an intermediate station, and from other children at least two weeks after desquamation is complete; during this period the ears should have thorough antiseptic cleansing. As a safeguard to the community I think it is essential to consider the

discharging ears in the determination of raising quarantine in scarlet fever cases.

In conclusion, let me emphasize the importance of early recognition of the ear complications of scarlet fever; we should not expect and wait for the ear to "break and run," as is so often done, for this is but little short of criminal negligence, but, through prompt treatment by paracentesis of the tympanum as soon as there is the slightest indication of inflammation in the tympanic membrane or exudate in the tympanic cavity, relieve the distressing symptoms and place the patient in the safest possible condition as regards systemic infection and intracranial complications, and do all in our power to preserve the function of one of the most important organs of special sense.

## CONCERNING THE CAUSES OF GALLSTONES.<sup>1</sup>

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My purpose in presenting this short paper to this Society is two-fold. First, I wish to indicate a new method of investigating a very difficult problem, the causation of gallstones, and to show what results this new method of investigation has borne in my hands. Secondly, I want to interest the members of this Society, who are in a position to examine organs derived from a large number of autopsies, in this method of investigating, so that more cases may be brought together and studied, and the correctness of my data be either verified or denied.

Without going into the earlier history of the theories of gallstone production, which would lead us far astray and tend to obscure the points that I wish to emphasize, I would like to unfold to you the development of the present theory of gallstone production. This development is closely bound to the Strassburg school of medicine, to Naunyn and his pupils. The work of this school gave us our present conception of the etiology of gallstones.

Before the publication of these modern views the whole question of gallstone production was unsettled—just as unsettled as the questions of the etiology of pulmonary consumption prior to Koch's discovery. Diathesis, heredity, age, sex, were regularly named as the causes of cholelithiasis, while the actual processes at work were scarcely considered. In the early nineties Naunyn attacked these

<sup>1</sup> Read before the New York Pathological Society, March, 1905. Since reading this paper I have been able to add a new case to my series.