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In infections of the ear, it is well to bear in mind the mechanical features of the eustachian tube, tympanum and mastoid. The system consists of connecting air chambers of varying sizes and shapes with a blind end in the mastoid and an open end in the nasopharynx. The normal physiology of the ear depends upon this mechanism remaining intact. The eustachian tube and adjacent portions of the tympanum are lined with ciliated epithelium which sweeps secretions down into the nasopharynx. The tube is constructed to open upon yawning and swallowing, and, in this manner, maintains the air pressure at the proper level in the middle ear.

Once infection invades this mechanism its normal function becomes impaired. At the orifice in the nasopharynx, at the junction of the cartilagenous and bony portions of the eustachian tube, and at the additus ad antrum which connects the middle ear and mastoid antrum are strategic points where barricades may be erected by nature to prevent the further invasion of the infection. We must determine in each case, (1) how far the invasion has progressed, and (2) how to prevent further spread of the infection. When the infection passes a barricade, that barricade becomes an obstacle to recovery because it blocks the drainage of pus back into the nasopharynx.

The first line of defense is at the opening of the eustachian tube into the nasopharynx. Here the normal secretions of the nose are swept above and below the opening by the ciliated epithelium of the nose and nasopharynx. If infection gains access to the nasopharynx and pus accumulates around the tubal opening, the tissues in the region become edematous and tend to close off this opening. The symptoms vary from referred pain and momentary stuffiness in the ears to a definite mild impairment of hearing, depending upon the severity of the infection and the amount of edema.

Treatment at this stage should be directed to the nose and nasopharynx. The nasal passages should be opened and kept open by the use of cocaine solutions or ephedrine. In the early serous stage the oily solutions are preferred because of the protection the oil gives the raw mucous membranes. The nasopharynx should be painted with 10 to 20 per cent argyrol or some other mild antiseptic solution. The argyrol should not be more than a week old, because after a week it undergoes disintegration and may be extremely irritating. If argyrol is prescribed for the patient to use himself, he should be warned against its continued use over a long period of time. Too many cases of argyrosis from continued self-medication with argyrol have been reported recently.

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The continued use of ephedrine solutions also should be discouraged. Many patients develop a sensitization to ephedrine after long or very frequent use. The turbinates and septal mucosa become very swollen and pale, and there is marked nasal obstruction which is no longer relieved by ephedrine.

If the infection gains access to the lower end of the tube, a barricade is erected at the junction of the cartilagenous and bony portions. Because the tube is narrower here and the mucosa is in folds, very little edema of the tissues is required to close off this portion completely. At first the pain in the ear is somewhat more marked and the stuffiness more pronounced. In the early stages the drum still may have a normal color and lustre, and may show only some retraction. If the obstruction becomes complete and lasts long enough, some injection may appear around the rim of the drum; the air in the middle ear cavity may become absorbed and be replaced by a serous transudate. At this stage, an acute serous otitis media develops. The drum still is quite transparent and one may see a fluid level with air above.

As long as there is no bulging of the drum to indicate increased pressure within the middle ear, treatment should be directed toward the nose and nasopharynx. The application of 20 per cent argyrol directly to the eustachian tube openings in the nasopharynx often is very helpful and will tend to open up the lower end of the tube and to draw the secretions down out of it. The application of argyrol can best be accomplished by passing a curved probe, tipped with cotton, through the nose in the same way that one uses the eustachian catheter. This should be left in place for about five minutes.

Under no circumstances should the tubes be inflated in the presence of an acute infection. Such a procedure would only result in blowing infected material through nature's barricade into the middle ear. After all acute inflammation has subsided, inflations should be used to promote drainage and to restore normal ventilation. When fluid appears in the middle ear and is not under pressure, a 5 per cent solution of phenol in glycerine or auralgan will be very helpful if dropped into the external canal and allowed to remain in contact with the drum for five to ten minutes. This tends to ease the pain and to promote osmosis of the serum through the drum. If there is a definite bulging of the drum, a myringotomy should be done immediately. Increased pressure within the tympanum should be avoided if possible because if it is not relieved, it will cause some devitalization of the tissues. It also may spill over into the mastoid cells and produce a further extension of the infection.

When the actual infection breaks through the barrier in the eustachian tube, it progresses rapidly into the middle ear. There usually is a rise in temperature and in the leukocyte count. The pain may be quite severe

and persistent. The deafness is more marked and of the conductive type. The drum is edematous, thickened, and injected. The landmarks become obliterated and as the pressure within the middle ear increases, the drum bulges. At this stage a myringotomy should be done. Care should be taken to incise the drum down to the floor to allow an adequate opening for the escape of pus. A culture of the fluid should be made immediately so that the type of organism may be determined. The most treacherous of all the infecting organisms is the pneumococcus, type III. With this type of organism, the middle ear and drum may gradually clear up while the infection actually is progressing and burrowing deeper into the mastoid. Such a case should be watched very carefully. The different strains of streptococci will vary in their virulence from year to year and in different localities.

Chemotherapy has done a great deal to help control ear infections and their complications. By taking a culture early, the physician will know the type of infecting organism, and therefore will be able to select the proper chemotherapeutic agent to be used. If large doses of the drug are administered, the patient should be hospitalized, and the blood level of the drug and the blood count should be watched carefully. These drugs often mask symptoms which otherwise would serve to indicate the progress of the infection. Therefore, such a patient should be watched more carefully than one who is not receiving any of these drugs.

After the myringotomy the ear should be cleaned and inspected every day, especially if there is any fever. The dry method of treating acute ears, which is merely gently drying out the canal by sopping up the pus with cotton on the end of a probe, is to be preferred. Gentle irrigations of the canal may be given if the secretions are too thick to drain out properly, or if it is impossible for the physician to see the patient often enough to keep the ear clean. The nose should be treated until the ear is dry. The use of Haskin's suction is of great help in treating children who are too young to keep the nasal passages clean by blowing. Suction is delivered through a small soft rubber catheter, the end of which has been cut off and the sharp edges of the rubber softened and rounded by being passed through a flame. The catheter then is oiled and gently passed into each side of the nose back to the nasopharynx. One is often amazed at the amount of pus which can be aspirated.

The temperature and leukocyte count should be watched. If the perforation tends to become closed by the swelling of the surrounding tissues, 5 per cent phenol in glycerine may aid in keeping it open. It may be necessary to repeat the myringotomy. The presence of a profuse discharge may be very misleading. With increased pressure in the middle ear it is possible for a large amount of pus to escape through a very small opening. In such a case the ear may dry up rather rapidly after an ade-

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quate opening is made. For this same reason a spontaneous rupture of the drum may give inadequate drainage. It is advisable to do a myringotomy on all cases which show a drum that is still bulging.

In most of the severe infections of the middle ear there is some inflammatory reaction in the mastoid, which may occur without any actual invasion of the infecting organism. There may be some mastoid tenderness in the first few days and a roentgenogram of the mastoid may show some clouding of the cells. If these symptoms do not persist after adequate drainage is established through the drum, and if the temperature does not remain unduly high, conservative treatment should be continued.

Once the infection breaks through the barriers at the additus, between the antrum and mastoid cells, and actually invades the mastoid, surgical intervention may be required. An increasing number of cases, however, are responding to the conservative treatments already outlined when they are combined with some form of chemotherapy.

The invasion of the mastoid usually is proclaimed by a further elevation of the temperature and leukocyte counts or a re-elevation, if they had returned to normal after the initial infection of the middle ear. There is pain in the ear and mastoid, and edema and tenderness over the mastoid. There is often a sagging of the posterior superior canal wall, which is manifested by a narrowing of the canal lumen from above and behind. A general toxic reaction occurs which in infants may take the form of severe prostration, diarrhea, dehydration, rapid loss of weight, and a high fever.

Where the infection becomes serious and the patient obviously is losing ground rapidly, surgical intervention may be necessary without delay; however, in most cases, it is best to delay operating upon the mastoid for a few days to allow some local resistance to the infection to develop. If operated upon too soon, new avenues for the absorption of toxins are opened and a severe septicemia and stormy convalescence may ensue. The advent of sulfanilamide and its allied compounds has greatly influenced this phase of the problem. It will frequently convert a surgical mastoid into a non-surgical one. Those that have to be operated upon do better if the patient is given chemotherapy for two or three days before the operation.

In cases in which delay is possible, the temperature usually subsides to a certain extent in a few days. If the indications for interference still are present, the operation should be performed. The roentgenogram may not always show breaking down of the intracellular septa, but when it does, operation should be performed immediately.

When the patient is recovering from his mastoid infection, usually on the tenth day of convalescence, the tonsils and adenoids should be removed, if present. Any sinus infection also should be corrected in order

to hasten convalescence and to prevent a recurrence of ear infection. If these procedures are delayed until the mastoid wound is practically healed, it is best to wait two or three months, because at this stage, the removal of the tonsils and adenoids may stir up the ear infection at a time when there will be no adequate drainage, and may necessitate reoperation.

The management of a mastoid infection is very difficult. One very dangerous situation is found in the cellular mastoid with a very thick cortex, which imprisons the infection in the deep cells of the mastoid. It may prevent tenderness or edema over the mastoid even when a very severe infection is progressing beneath it. A feeling of false security may be attained by the absence of tenderness over the mastoid and dire consequences may result. A brief outline of one case will serve to illustrate this situation.

A young physician developed an otitis media about five weeks before Christmas. He immediately consulted an otologist who performed a myringotomy. The following day the other ear was involved and also was opened. The ears continued to run for the ensuing five weeks and there was some elevation of temperature, but no tenderness over the mastoid. Because of this lack of tenderness, roentgenograms of the mastoid were not taken. He then drove two hundred miles to his home. Several days later there was a sudden elevation of temperature, headache, nausea, and vomiting. Still there was no tenderness over the mastoid. At first, intestinal influenza was suspected but when he failed to improve after a few days he was seen by a neurologist who immediately sent him to the hospital. Upon arrival he was found to have a well-developed meningitis with streptococci in the spinal fluid. Roentgen examination showed badly infected mastoids, with some breaking down of the cells, and an extremely thick cortex. This was verified at operation when both mastoids were completely cleaned out. However, it was too late to save his life and he subsequently died. This occurred before the development of sulfanilamide. His physicians were all led astray by the absence of tenderness over the mastoid.

If the acute condition subsides in any of the stages already outlined, the mechanism of the ear may return to a normal or nearly normal condition. There may be complete healing even after a mastoid infection. If there has not been great destruction of tissue, the function may return to normal. The perforation of the drum may heal and the scar may be pushed off to the periphery of the drum so that later inspection may show a perfectly normal appearing drum. This is especially true if a myringotomy has been done.

If the healing is not complete, a chronic condition ensues, such as tubal catarrh, in which deafness of the middle ear persists. Contrary to the textbook teachings, some of these cases may show a greater loss for the

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higher tones than for the lower tones, giving the picture usually attributed to a nerve deafness. The drum is retracted and may show no other evidence of pathology. When the acute infection has completely subsided, the eustachian tube should be inflated. These cases also should have nasal treatments with tampons soaked in ephedrine solutions and the application of a fresh solution of 20 per cent argyrol to the eustachian openings in the nasopharynx. This usually will give relief.

If the hyperplasia of the mucous membrane extends into the middle ear cavity, a hyperplastic otitis media results. This usually is due to repeated mild and acute attacks. The drum is thickened, retracted, and shows a loss of lustre. Here again the treatment is directed toward the eustachian tube in an attempt to restore its patency by inflations and nasal treatments. Foci, such as tonsils, should be removed and nasal obstruction from a badly deviated septum should be corrected to prevent a recurrence of further acute episodes which will lead to further loss of hearing. If there are bands of adhesions within the middle ear, some deafness will remain and no treatment has been found yet which will combat it successfully.

An acute suppurative otitis media and mastoiditis may progress to a chronic otorrhea. This should not be allowed to continue for more than three or four months because the loss of hearing will be too great. In all chronic otorrheas there is some involvement of the mastoid cells, if any are present.

The patient with a chronic otorrhea should be examined carefully. First the adequacy of the perforation should be determined. It often is too small, or is located too high off the floor to give sufficient drainage. In such a case a myringotomy should be done. Sometimes a polyp will be found which protrudes through the perforation and fills the canal. This blocks the drainage and should be removed. These simple procedures may change the entire course of the infection.

The nose and throat should be examined for any possible foci which may contribute to the infection. Very often the removal of the tonsils and adenoids will result in healing of the ear condition. Any infection present in the sinuses should be adequately treated, and surgery performed if necessary. If the otorrhea still persists, further treatment depends upon the type of mastoid which is present, whether there is a cellular infected mastoid or a sclerotic noncellular one. The roentgenogram alone will differentiate the two types. If the mastoid contains infected cells, a mastoidectomy should be done. This type of chronic otorrhea, which is one of the most dangerous of all, and those with cholesteatoma are the ones which most often develop intracranial complications with acute exacerbations. As long as such a patient has otorrhea, the chances of serious complications are very great.

If the mastoid is sclerotic and noncellular the ear can be treated conservatively. One per cent thymol in seventy per cent ethyl alcohol is perhaps the most effective drug. This is instilled into the ear after it has been dried out carefully. Care should be taken to get the solution through the perforation and into the tympanum. A gentle negative pressure in the external canal will usually draw the air out of the tympanum and allow the fluid to enter. This can be done daily. Thymol iodide powder also is very useful and can be used later when the drainage decreases.

If the otorrhea persists and there are granulations or cholesteatoma in the middle ear cavity, and if the hearing is gone, a radical mastoid operation should be done. The radical operation does not always produce a dry ear but, by converting the mastoid and middle ear into one large cavity connected with the external canal, the danger of subsequent serious complications is removed.