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LABORATORY AIDS IN OTOLOGY*

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Otology, during the past twenty years, has been greatly enriched by the products of research. From a narrowly restricted field it has so broadened that it now encroaches upon several of the specialties to which it formerly bore only a remote relationship. In its growth, it was only natural that the confusion and awkwardness of adolescence should accompany the new lore. Now that it has passed that stage, it is imperative that it classify its knowledge, cross-index the new with the old and discard the worthless. We will then be ready for further additions, which, like the ordered parts of a mosaic, can be fitted into their proper places.

While otological surgery has progressed remarkably during these years, it is in the utilization of the numerous advances in laboratory medicine that an even more remarkable progress has been made, particularly in the field of diagnosis. But here the cross-indexing is not yet complete; and I propose, by correlating the laboratory aids at our disposal with our already well indexed clinical data to clarify some of the confusion which is manifest.

The diagnosis of the surgical diseases in otology has far surpassed our ability to recognize the non-surgical conditions. It is therefore evident that the laboratory has aided us more accurately in the recognition of the former types of lesions. By study of the blood, the cerebrospinal fluid, the bacteriological flora and the roentgenographic pictures of the mastoid process, clinical diagnosis has been rendered a great deal more precise in surgical otology. In the non-surgical diseases of the ear, while laboratory studies have as yet yielded scant information, here too great promise is held forth. I shall speak first of the laboratory aids in the diagnosis of the surgical diseases of otology.

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Blood Studies:

The routine estimation of the *red cell count* has not been accorded the position its importance warrants. I have found it of great diagnostic value. Examination of the red cells, however, is never complete without an estimation of the hemoglobin percentage. The two go hand in hand as a diagnostic aid, both before and after an operative procedure. I have repeatedly emphasized the distinction between the two types of acute mastoiditis, the acute coalescent mastoiditis and the acute hemorrhagic mastoiditis. The former is the one most generally encountered. There is a breaking down of the intercellular walls, a coalescence of the cells and the formation of pus and granulations. The hemorrhagic type is more rare. It is characterized by a multiplicity of thrombi in the minute venules of the lining of the mastoid cells. In this type we never see a coalescence of the cell walls with a consequent formation of pus. The lesion, no matter what its stage at operation, always exhibits intact cell walls. Clinically, the marked tenderness over the mastoid process, the severe prostration and the septic temperature presented at the onset of the ear lesion are never in accord with the otoscopic picture where no sagging of the canal wall and no profuse discharge are evident. Those of you who have seen such cases during the late influenza epidemic will recall what were considered to be unusual findings at operation. But the sharp recovery after operation on the mastoid process warranted the continuance of surgical intervention in these cases, although they presented none of the classical signs of acute mastoiditis.

Bacteriological investigations showed this type of mastoiditis to be due to the streptococcus hemolyticus. It is in the hemorrhagic mastoiditis, particularly, that repeated examinations of the red cells and the hemoglobin percentage will result in early recognition of the lesion. In a patient who presents an acute purulent otitis media, accompanied by septic temperature and severe prostration from the outset of the disease, a daily decrease in the number of

red cells and in the percentage of hemoglobin speaks for a diagnosis of acute hemorrhagic mastoiditis, and indicates an immediate simple mastoidectomy where all other conditions have been eliminated. Since the lesion in this type of mastoiditis is present in the small vessels within the mastoid process, the hemolytic streptococci present within the infected thrombi destroy the hemoglobin and the red cells of the blood stream. This is evidenced by the gradual fall in the number of red cells and in the percentage of hemoglobin.

After a simple mastoidectomy in this type of mastoiditis, thorough exenteration of the mass of infected thrombi is followed by a subsidence of the sepsis and a gradual return of the red cells and hemoglobin to normal. Should the reduction in the red cells and hemoglobin continue after the simple mastoid operation, a further extension of the disease into the lateral sinus should be suspected, and a transfusion of whole blood is in order. If the reduction still continues, the lateral sinus should be obliterated and the internal jugular vein ligated regardless of whether or not the outer wall of the sinus is diseased. Since the sinus thrombosis in this type of mastoiditis is produced by the intravenous extension of the minute thrombi directly into the lateral sinus, there will rarely be found any erosion of the sinus plate or any evidence of a periphlebitis. In this type of mastoiditis, continued reduction of the red cells and the hemoglobin after operation furnishes a better diagnostic aid than the appearance of the sinus wall.

In the acute coalescent mastoiditis, a daily study of the red cells and hemoglobin throughout the period of hospitalization subsequent to operation will always lead to an earlier detection of a sinus thrombosis where a hemolytic organism is the offending factor. In these cases, the earliest appearance of a thrombosis will be manifest in a gradual daily decline in the number of red cells and the percentage of hemoglobin. Where the hemoglobin falls steadily, and where the clinical signs of sepsis are absent, a transfusion will replace the lost hemoglobin and so add to the patient's

resistance. On the other hand, a persistent unilateral headache after operation, accompanied by an increased leucocytosis and a gradual fall in the number of red cells and the percentage of hemoglobin, should lead one to inspect the interior of the sinus for the presence of a thrombus. The utilization of the red cell and hemoglobin estimation as a routine in every case of mastoiditis will afford an early guide in the detection of otitic sepsis, whether it be due to an acute hemorrhagic mastoiditis or to a complication of an acute coalescent mastoiditis or to a chronic mastoiditis.

The White Cell Count:

The study of the *white cells* furnishes us with a diagnostic as well as a prognostic aid. In the ordinary white cell count, the otologist is interested in the total number of white cells and the percentage of polynuclear cells. The former is the index of the resistance of the individual while the latter indicates the severity of the infection. Utilizing this as a foundation, McKernon drew up a chart whereon the total number of leucocytes could be compared with the percentage of polynuclear cells. He determined that with a given number of leucocytes a patient could present a certain maximum percentage of polynuclear cells before his resistance was overcome by the virulence of the infection. Once this was determined, we had at our disposal an excellent prognostic guide. Knowing the maximum number of polynuclears that is compatible with any given leucocytosis, we can determine at a glance whether or not the patient has sufficient resistance to combat the infection. Where the percentage of polynuclears exceeds the maximum allowed for any given number of leucocytes, the patient's resistance is succumbing to the infection. On the other hand, where the polynuclear percentage is less than, or equal to, the maximum allowed for any given total leucocytosis, the resistance of the patient is good. The use of these resistance charts will be found of inestimable value in prognosticating the outcome of any otitic infection.

The white cell count is also of value as an indication of an impending complication. After a simple mastoidectomy, the white cell count should show a rapid decline toward normal. A persistent leucocytosis with an increase in the percentage of polynuclear cells is indicative of a continued infection. A careful observation of the patient is necessary in such cases to determine the source of the infection that is causing the leucocytosis to persist. Most frequently it is due to the continuation of purulency in the middle ear; and an increased white cell count may be noted until the middle ear infection has subsided. If, however, the increased leucocytosis is accompanied by a unilateral headache, an extension of the disease beyond the mastoid process should be suspected. Where, following operation, the leucocytosis subsides only to recur after an interval of time, accompanied by unilateral headache, a sinus thrombosis should be strongly suspected. I have had several cases of sinus thrombosis subsequent to the acute coalescent mastoiditis wherein the only signs that led to a diagnosis of sinus thrombosis were the persistent unilateral headache and the increased leucocytosis. In these cases I had no other signs or symptoms of sepsis on which to base a diagnosis.

Recently I have had experience with the method of counting the white blood cells devised by Schilling and introduced at Beth Israel Hospital by Dr. Arthur Weiss. In brief, the count is made as follows: The total number of cells is tabulated as heretofore. In the differential count, however, particular attention is paid to the various morphological forms of the polynuclear cells. One notes the percentage of mature polynuclear, that is, those in which the nucleus has undergone complete segmentation, and the percentage of young polynuclears, wherein the nucleus is still unsegmented and appears in the form of a rod. The latter type are called the "staff cells." In a normal count, the young forms, or staff cells, number less than 5 per cent. At the onset of an acute infection which produces a polymorphonuclear leucocytosis, there occurs a marked increase in the number of these immature staff cells. This

is due to the fact that the bone marrow is stimulated to produce an excess amount of polynuclears to combat the infection and these cells are thrown into the general circulation before their nuclei have had time to undergo complete segmentation. In addition to this, the percentage of lymphocytes diminishes. This picture has been termed a "shifting to the left." As the infection subsides, the number of staff cells also becomes less until "normal" is finally reached. The blood picture does not have any relationship to the temperature curve but is in direct relationship to the virulence of the infection and the resistance of the patient. Where, following a simple mastoidectomy, the number of staff cells continues high, that is, above 15 per cent., it is indicative of a continuation of the infection. It is not diagnostic of any complication but it should put the surgeon on guard to watch for the clinical signs of a complication. The continuance of a high staff count, or the reappearance of one after it has returned to normal subsequent to aural surgery, should be viewed with the utmost suspicion.

I have found this method of counting the white cells of inestimable value in determining the procedure to be followed in the so-called borderline cases. Where a mastoiditis is presented and one is in doubt as to whether or not to operate, the staff cells will furnish an excellent guide in helping one to reach a decision. Where the staff cells increase in number on several daily examinations and reach 12 per cent. or more, operation is indicated. On the other hand, where the percentage of staff cells remains stationary below 12 per cent., or shows a gradual reduction, operation can be postponed with safety and the patient kept under further palliative treatment.

Blood Culture:

Before discussing the results of blood culture and the importance to be attached thereto, I should like to discuss as briefly as possible the pathogenesis of a sinus thrombosis. In the acute coalescent and the chronic mastoiditis, there occurs a gradual destruction of the mastoidal

structure until the inner table is reached. The inner table overlying the sinus is then subjected to pressure from the pent-up pus and undergoes necrosis. The outer wall of the sinus is thus brought into direct contact with the purulency within the mastoid process. The pressure against the wall of the sinus causes a narrowing of the blood channel with a consequent slowing of the blood stream. In addition, the outer wall of the sinus becomes infected by the pus in the mastoid process and we then have a sinus periphlebitis. If the mastoid is operated upon at this point, there is found a protective layer of granulations over the sinus wall at the exposed area. On the other hand, should this periphlebitis be permitted to progress so as to involve the endothelial lining of the sinus, the injury presented to this lining membrane, plus the narrowing of the channel and the slowing of the flow of blood, offer all the elements necessary for the production of a thrombus formation. This thrombus is protective in nature and is at first sterile. If the round cell infiltration and granulations on the outer coat of the sinus are not sufficient to keep out the organisms from within the sinus, the thrombus will become secondarily infected and we then have an infected thrombus within the sinus. This thrombus grows with and against the blood stream and only its central portion is infected; its extremities are sterile. Therefore, if a blood culture is taken at this stage of the thrombosis, no growth of organisms will be noted. If nature is successful in localizing the infection by causing a fibrosis of the thrombus, a positive culture will never be obtained. It is only when the thrombus has undergone disintegration that infected particles will gain entrance to the general circulation and on culture of the blood a positive growth of bacteria will be obtained. To permit every sinus thrombosis to go on to this stage before operative intervention is undertaken, is not in accord with the best principles of surgery. One does not wait for an acute suppurative appendicitis to rupture before removing it surgically. So, in otology, one must not view a negative blood culture as ruling out the presence of an infected thrombus within the

sinus where the clinical symptoms point to its presence. Too much reliance has been placed upon the blood culture as a means of diagnosing sinus thrombosis. It has its value as a diagnostic measure but is not nearly as reliable in the greatest proportion of cases as the red cell and hemoglobin estimation and the white cell count. The findings at the primary operation on the mastoid process, coupled with the appearance of temperature, unilateral headache, etc., are also of far greater diagnostic value than the results of blood culture. In the last 18 cases of sinus thrombosis upon which I have operated, the blood culture was positive only in four of them. In two instances wherein I had obtained a positive blood culture of streptococcus hemolyticus in cases of sepsis subsequent to mastoid surgery, I excluded a sinus thrombosis from the clinical picture presented by the patients because of other factors. In both instances the patients recovered without resort to surgery on the blood vessel.

A positive blood culture is more apt to be found in cases of sinus thrombosis subsequent to the hemorrhagic mastoiditis. Here the thrombosis in the lateral sinus is the result of the direct growth of a smaller thrombus from one of the venules in the cellular walls into the larger vessel. The sinus plate is not eroded, no perisinal abscess is found and there is no involvement of the outer coat. The thrombus in this instance is infected from the outset and is more often of the mural than the occluding type. The blood stream, in passing, washes some of the organisms into the general system, and a positive blood culture is obtained much earlier than in the thrombosis following the acute coalescent mastoiditis.

In addition, a negative blood culture is to be expected where metastatic lesions are present. These lesions, particularly if they are located in the soft parts, act as fixation abscesses and clear the blood stream of infection. If the bacteria gain entrance to the pulmonary circulation, they are apt to lodge there and produce their characteristic lesion without ever reaching the general circulation.

Blood Sugar:

A blood sugar determination should always be part of the routine examination of any adult suffering from a mastoiditis. A diabetic who is under treatment will often show no sugar in the urine while an estimation of the blood sugar will reveal a hyperglycemia. Diabetes produces that type of ear lesion which has been termed the painless type of acute coalescent mastoiditis. An extensive necrosis of the mastoid process may exist and yet the patient have no pain nor tenderness. Where the otoscopic picture and the duration of the disease point to a definite acute surgical mastoiditis but where, because of the absence of pain and temperature, one is inclined to temporize, the finding of an increased blood sugar is a warning of the danger that diabetes presents. If such a case is permitted to proceed without surgical intervention, the pain and temperature will eventually appear concomitant with an intracranial lesion.

The Cerebrospinal Fluid:

Lumbar Tap: A lumbar puncture is indicated in every case of chronic suppurative otitis media before operation is undertaken on the mastoid process. It is indicated in every case of postoperative simple mastoidectomy wherein an intracranial lesion is suspected. The information to be gained at the time of the tap is the amount of intraspinal pressure and the presence of a block between the intracerebral and the intraspinal subarachnoid spaces. The latter can be determined by noting the rise in manometric pressure following jugular compression. If the spinal fluid is permitted to run into a mercury manometer, the pressure of the spinal fluid can be read. If pressure is then brought to bear over the internal jugular vein, first on one side and then on the other, a rise is noted in the intraspinal pressure where no obstruction exists between the intracranial space and the spinal column. Incidentally, Tobey and Ayres have utilized this procedure in the differential diagnosis of sinus thrombosis in cases of bilateral mastoiditis. The side whereon the thrombus is

present is obstructed and consequently compression of the internal jugular on that side will cause no rise in the intraspinal pressure. I have utilized this procedure to great advantage and found it to be an excellent diagnostic measure. It is not infallible, however. Where a thrombus is not of the obliterating type, compression of the jugular will permit a rise in intraspinal pressure. Then again, in one of my cases wherein I had completely obliterated the lateral sinus after incision and resection of a portion of the outer wall, but wherein the internal jugular vein had not been operated upon, compression of the internal jugular vein on the side of operation caused a rise in intracranial pressure equal to the rise obtained from compression of the opposite internal jugular vein. This case has been reported by me elsewhere.

In cases of acute labyrinthitis following suppurative otitic disease, the spinal fluid will serve as a diagnostic measure in determining whether or not the labyrinthitis is of the suppurative type. Where the spinal fluid shows no increase in the number of cells, has a normal chemistry and shows but a slight increase in pressure, the labyrinthitis is of the serous variety. On the other hand, where a large increase in cells is noted and the albumin is increased, a suppurative labyrinthitis is to be suspected. If the increase in cells is predominantly lymphocytic, one can continue to employ palliative measures. Where the increase is largely polynuclear, a threatened invasion of the subarachnoid space is indicated and operation is advisable. This is especially true if, on successive taps, a high lymphocyte count is found to be replaced by a high polynuclear count.

If examination of the spinal fluid shows a high cell count, lymphocytic or polymorphonuclear, increased albumin and globulin and increased pressure, but no bacterial growth, one is dealing with a sympathetic meningitis. This is always indicative of a threatened invasion of the subarachnoid space. It can be produced by a sinus thrombosis, brain abscess or suppurative labyrinthitis.

Where such a fluid is obtained, every measure must be employed to eradicate the focus of infection immediately, if a suppurative meningitis is to be averted.

One of the earliest findings in the spinal fluid in cases of suppurative meningitis due to a fermentative type of bacteria is a reduction in the percentage of glucose normally found in the spinal fluid. This is often demonstrable even before any organisms can be isolated. Where the copper reduction is absent or diminished, a suppurative meningitis can be diagnosed even though no bacteria can be isolated on smear or culture.

Bacteriology:

A culture of the pus from the middle ear is of no value unless taken very soon after the paracentesis or spontaneous rupture of the drum. Cultures taken after twelve hours or so will almost always show a mixed infection due to contamination. A culture of a chronic aural discharge is of no value at any time because the organism isolated is usually different from that which produced the lesion originally.

There are only four organisms which when isolated from an aural discharge influence the management of a case: The Klebs-Loeffler bacillus, the Vincent's bacillus and spirillum, the streptococcus mucosus capsulatus and the Friedländer's bacillus. The bacillus of diphtheria naturally indicates specific treatment with antitoxin. The Vincent's bacillus and spirillum necessitate salvarsan. The other two organisms depict an extremely dangerous type of lesion, insidious in character and asymptomatic in its course. Because of the very nature of the disease, it often goes on to intracranial invasion with no clinical evidence of its progress. Hence the finding of the streptococcus or the bacillus mucosus capsulatus indicates early complete surgery on the mastoid process in order to forestall intracranial invasion. It is true that a number of cases wherein these organisms have been found recover without any operative measures, but the seriousness of the mastoiditis

due to these bacteria must be considered from the standpoint of the high mortality presented.

Roentgenographic Examination:

Roentgenographic examination of the mastoid process is to be considered an accessory laboratory aid in otological diagnosis. The otologist should be able to interpret the findings on radiographic examination of the mastoid himself. One cannot expect the roentgenologist to make a clinical diagnosis from the plate; and the roentgenologist who does so is presumptuous unless he is specially trained in otologic pathology. An x-ray examination will yield anatomical data and will show whether or not cellular destruction is present. It will not label the case as surgical or non-surgical, acute or chronic, emergent or non-emergent. This the otologist alone can determine by a correlation of the clinical, laboratory and x-ray findings.

In every acute middle ear infection, a clouding of the mastoid process will be noted. This is due to the edema of the cellular lining which accompanies every acute middle ear suppuration. The same cloudiness will also be found in cases of external otitis accompanied by inflammation of the periosteum over the mastoid process. In the absence of any evidence of cellular destruction, the finding of a cloudiness of the mastoid process has no significance from a clinical standpoint.

Where the diminution in illumination is accompanied by evidences of cellular destruction in an acute middle ear infection, an acute coalescence of the mastoid cells is indicated. Here, however, the clinical appearance of the ear and the general systemic reaction will guide the surgeon in his determination as to whether or not the case is a surgical one. A certain amount of mastoidal destruction may exist and yet the case may recover without surgical intervention on the mastoid process. In such instances successive mastoid plates are valuable to check the progress of the destruction. In a patient who has had one or

more attacks of mastoiditis prior to the current acute attack, the finding of cellular destruction on the mastoid plate may be the result of the previous mastoid inflammations. Here, also, repeated x-ray examinations are valuable in determining whether there is an extension of the destruction.

In a case which has been under observation for a greater length of time than that usually required for the cessation of an acute aural suppuration, the finding of an extensive cellular development of the mastoid process with a small amount of destruction would tend to indicate a beginning chronicity and operative interference is usually advisable before complete destruction occurs. On the other hand, the same amount of destruction in a small mastoid process would indicate that the infection was being walled off and would recede of its own accord.

Very frequently, on a well-taken plate, an erosion of the sinus plate or tegmen can be noted by a break in the continuity of the shadow cast by these structures. In this way an epidural or perisinal abscess can be detected.

In the chronic purulent otitis media, a sclerosis of the mastoid process will be noted in every case. This is not to be interpreted as indicating a radical mastoid operation for it will be found in all cases—dangerous and non-dangerous—of chronic purulent otitis media.

In taking plates of cases with severe prostration, a septic temperature curve and a bacterial report of streptococcus hemolyticus, the negative finding of undestroyed intercellular bony walls is a clue to the fact that we are dealing with the hemorrhagic type of mastoiditis. In other words, failure to find bone destruction is substantiation of the diagnosis of hemorrhagic mastoiditis and usually indicates surgical intervention.

In discussing the laboratory aids at our disposal in the diagnosis of the non-suppurative otitic diseases, I am sorry that I will not be able to speak as definitely as I have of

their help in the suppurative otitis. Our main problem in this field is deafness and I feel that laboratory medicine will do more to help solve this problem than anything we may do in perfecting the testing of hearing. All our hearing tests have until now done nothing but confuse us. They have afforded us a classification of deafness which is largely erroneous and which, used as a basis for investigation, has misled us. Permit me to digress for a moment to make clear just what I mean. For example, a hearing test may yield results characteristic of a so-called catarrhal deafness and yet an acoustic neurofibroma is found to be the cause of the loss in hearing. A typical hearing test of a so-called otosclerosis is found to be due to syphilis of the central nervous system. These are actual occurrences from my practice and I could quote many others.

I have accordingly come to view deafness from a different standpoint—whether or not it is progressive in character. Some patients in whom a loss of hearing is present will never get worse, whereas others show a gradual progression of the loss in hearing acuity. Consequently, the division of deafness into progressive and unprogressive types permits a study of this condition from a new angle; namely, what causes the deafness to progress. That the cause may be different in each case is granted. Whether it is endocrine, toxic, infectious, the result of inherited latent characteristics or the product of faulty metabolism, is yet to be determined. In all probability each of these factors plays a role.

Blood Chemistry:

In studying these cases, Almour and I have found that most patients with progressive deafness show a derangement of the calcium content of the blood. In cases of long standing, a decrease in the calcium content is noted. In the more recent and active cases, an increase is more apt to be found. In conjunction with the calcium derangement, a great number of the cases show a high uric acid in the blood.

Just what the significance of this finding is, I do not know. We originally reported it as an observation and as an aid in the diagnosis of chronic progressive deafness While we have several theoretical explanations, we are as yet unable to account for it definitely. Nevertheless, I consider it a valuable diagnostic aid, in helping to differentiate the progressive from the stationary types of deafness.

Wassermann:

The Wassermann test should be a routine procedure in all cases of deafness. Syphilis does not always yield the typical acoustic reactions of a nerve deafness, either with the forks or with the audiometer. Where, on examination of the blood, a negative Wassermann is obtained but syphilis is strongly suspected from the clinical data, a spinal fluid Wassermann and colloidal gold test are advisable.

X-Ray:

In the diagnosis of deafness, too, the x-ray holds forth a possibility of help. Joseph C. Beck, in 1915, made a preliminary report of the finding of changes in the long bones in cases of otosclerosis. With a view to checking his findings and learning what possible lesions might be presented, I had Dr. Henry K. Taylor make a roentgenographic study of the long bones in several of my cases of deafness. In those cases wherein I had made a diagnosis of progressive deafness, Dr. Taylor reported changes in the periosteal coverings of some of the long bones. In two of the cases he found early evidences of cystic formation in the cranial vault indicative of an early Paget's disease. In those cases which were non-progressive in character, no abnormalities were found in the skeleton. Here also I am unable to explain the findings. I simply submit them for your study and future investigation.

The correct estimation of laboratory criteria removes many cases in otologic practice from groups to which preconceived routine therapy is applied. It permits an individualization of treatment according to the given case because it furnishes data so significant as to differentiate

conditions which appear clinically alike. Thus details of pathology are better comprehended, the management of cases becomes increasingly resourceful and results are more uniformly successful.

In conclusion, I desire to stress the importance of the role which bedside observation plays in all otological cases and to emphasize the need of accurate clinical observation. In the last analysis, it is good clinical judgment which furnishes the foundation upon which laboratory aids build in making diagnoses.

Section of Otology, May 11, 1928

UNUSUAL CASE OF CHRONIC MASTOIDITIS

JOHN HORN

A case of bilateral otitis media, purulenta chronica, chronic mastoiditis. Right ear with a fistula in the outer border of the posterior wall of the external auditory canal. Simple mastoid operation. Recovery.

Patient, a girl of fourteen, who gave a history of having had discharging ears for seven years. Examination, October 16, 1926, showed both canals filled with a foul smelling secretion; drum membranes lacking—no ossicles present. The outer border of the posterior wall of the right external auditory canal showed a fistula containing thin pus. This was carefully probed, the probe entering a cavity. A crucial incision was made through the opening and with a blunt curette some diseased bone removed. Simple mastoid operation three days later. Bone was found markedly sclerosed and deep layer of cells diseased; thorough curetting, especially in the region of fistula. Small area of sinus exposed during operation. Wound was drained with gauze strips and closed with catgut sutures. Uneventful recovery. Discharged from the hospital after three days.

Post-operative treatment of O.M.P.C., both ears with Unguentum Credé (Horn). Right ear dry after six weeks. Except for the fistulous opening there were no other symptoms. The left ear still has a slight colorless discharge. Hearing in both ears has improved.