Section of Otology
President—Donald Watson, F.R.C.S.

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Progress in the Treatment of Mastoid Infection and some of its Complications

PRESIDENT'S ADDRESS

By Donald Watson, F.R.C.S.

The discovery of antibiotics has simplified and, with the advent of others, will increasingly simplify the treatment of acute conditions of surgery.

In his Presidential Address two years ago Graham Brown (1945) said:

"I believe the opportunities for mastoid surgery are gradually decreasing in proportion as the prophylactic measures against the incidence and spread of aural disease increase. Indeed, the time may not be far distant when intracranial complications of otitic origin will be considered rare phenomena."

My introduction to mastoid surgery was in 1920 in Edinburgh, where I worked for two years under Mr. J. S. Fraser.

His method of treating the acute mastoid cavity, after a most thorough removal of all infected bone, was to cleanse it with hot hydrogen peroxide and to pack the wound with iodoform gauze, leaving an adequate drain at the lower end after closure of the rest of the wound. The pack was removed on the third day, the wound was repacked and continued to discharge more or less for three to four weeks before healing took place. This ideal was not always attained: often the wound broke down, when eusol syringing was carried out twice daily. An infected, broken-down wound meant prolonged treatment, sometimes of many months’ duration.

Going to Bradford in 1922, for four or five years I tried other methods then practised, such as the blood clot method—closure without drainage. This was very unsatisfactory, as the wound usually broke down. Then there was the method of leaving the wound almost entirely open, packing it, and allowing it to heal by granulation tissue from the bottom. This was a tedious method, but it certainly obliterated the cavity, and there were no recurrences. The subsequent depression over the mastoid is unsightly. Nevertheless, it is a method practised even to-day in certain clinics.

Next came the use of various antiseptics with packing. Of these T.C.P., first produced about 1922, was the most successful in my hands.

It was in 1928 that I discarded bipp and reverted to the Carrel-Dakin method of flushing the wound with eusol and drainage by rubber tubes. This method was employed almost exclusively until early in 1933. It was a safe method—took time to do the dressings, but obliterated the cavity. T.C.P. was used as a final lotion for dressing the wound.
In the February of 1933 a severe influenza epidemic occurred and, at its height, no less than 53 cases of acute mastoiditis were in hospital at the same time. Most of these were severely infected, some had complications, and the labour of dressing them was a tremendous strain on our nursing resources.

I decided to go back to using bipp in the liquid form, with a small gauze drain at the lower end of the wound. The bipp in the paste form used a few years previously was too solid and I am sure it caused the recurrences. The liquid bipp plus drainage for two or three days greatly lessened the number of recurrences.

This bipp method, published by Mr. Herbert Tilley (1919), Holt Diggle and Gilhespy (1921), supported by Macnab of Johannesburg and others, was the most successful in my hands and also in the hands of other surgeons, and I have used it until 1944. The great majority of cases were discharged, with the wound healed, in a fortnight. Packing the wound was dispensed with.

Secondary suture.—When there was osteomyelitis of the squamous temporal and of the occipital bone, it might be necessary to enlarge the mastoid wound by one or more radial incisions, backwards or upwards and backwards.

In such wounds and also in widespread and severely infected cases, a bipp pack left undisturbed for five or six days enabled one to do the secondary suture, which was so important. The wound must be closed in five to six days, otherwise, owing to shrinkage and curling of the flaps, it may be impossible.

Even with bipp sometimes a child’s wound would not heal. Occasionally I gave these tardy cases a weekly dose of tuberculin in a glass of milk, an hour or so before breakfast. This has a most beneficial effect on these slowly healing wounds.

To-day the universal practice is to dust the mastoid wound lightly with penicillin and sulphathiazole powder and close it, except perhaps for a small gauze drain at the lower end left in situ for a couple of days. Most cases are discharged healed by the twelfth day with a dry ear. In severe infections, the giving of sulphonamides and penicillin is an added safeguard. This modern treatment has shortened the stay in hospital tremendously. Whether the aditus remains open and recurrences are to be expected more frequently, I am doubtful, but the great saving of time and worry outweighs this possibility. Even secondary suture in mastoid surgery will, I believe, also become a relic of the past.

Lateral sinus thrombosis.—In these days of antibiotics and chemotherapy cases without cerebral complications should all survive.

With regard to ligature of the jugular vein: years ago, Mr. Ballance said there were occasions demanding ligature, and there were occasions where the local condition in the sinus could be dealt with efficiently without ligature. A more rational opinion one could not have on this problem. I was always prepared to ligate the vein if occasion arose, as I am firmly convinced of the efficacy of this procedure. Even with the aid of chemotherapy I should not hesitate to ligate the vein if the case had symptoms of pyæmia with infarction of the lung. Otherwise treatment of the local condition of the sinus is all that is necessary.

To obtain the best results in lateral sinus cases, surgeons have had to do the dressings themselves for many days, but we got good results, even as high as 80% recoveries in cases without cerebral complications.

The accompanying septicaemia is the danger in sinus thrombosis. Antitoxic sera of all kinds have been tried. The best, I believe, was the scarlatinal antitoxin. Quinine was also used. It did one good thing. Frequently a fixation abscess occurred at the site of the injection. One was always pleased to see this. These cases entail long and tedious treatment and careful observation.

I have opened 11 abscesses including an infected knee-joint and an ankle-joint in one boy, and he recovered with no disability except a stiff knee.
Lateral sinus thrombosis cases have given us many interesting struggles. Pus in the internal jugular vein was not a rare occurrence.

With the continuation of rigors or swinging temperature—a common occurrence—one has frequently traced the lateral sinus back to the torcular Herophili before effecting a cure, this process requiring three or more operations. Mr. J. S. Fraser always packed the interior of the open sinus with iodoform wool, but I understand some surgeons did not believe in this rational procedure.

I have had only one case of cavernous sinus thrombosis as a complication—a man aged 28. I am informed that after his recovery he lived for a further six months and died at home with symptoms of brain abscess.

Perhaps my most interesting case was one in which an abscess developed at the root of the neck, just between the clavicles. I waited before opening and packing this one. On removing the dressing two days later I was alarmed when I looked inside, as there was no reaction in the walls of the cavity and everything seemed to be pulsating. I repacked that cavity with bipp and left it. This child of 5 had an interesting reflex. Her lateral sinus had been opened almost to the torcular, and each day as I dressed her and touched the sinus wall she complained of pain on her nose—a reflex through the meningeal branches of the fifth nerve. She eventually recovered after having been on sulpha-thiazole continually for six weeks and having taken nearly 800 grammes.

The actual percentage of cure, in cases where lateral sinus thrombosis arises, is high, but in the future it may be expected, with the early use of chemotherapy, that this complication will be progressively more rare. Apart from the Schwartze or other original operation there should be next to no need of any added major operative procedure and, consequently, a greatly lessened stay in hospital.

*Meningitis* is a fairly common complication of mastoiditis. Its early diagnosis is important.

Progress in the treatment of meningitis is so intimately dependent upon early diagnosis that I shall now discuss the sign upon which early diagnosis depends. The picture of meningitis I need not detail.

The most important sign in my opinion is early stiffness of the neck. You know how easily anyone, and especially a young child, is able to flex the neck, put the chin on the chest, and then move the chin laterally—i.e. rotation. The slightest restriction of extreme flexion and rotation is demonstrable by comparison with a normal patient, and is the most valuable sign.

In a paper on meningitis, T. B. Layton (1935) laid great stress on this early stiffness of the neck. In spite of views to the contrary, I think this sign is always present if it is looked for in the manner which he described.

On one occasion I was called to a fever hospital to see a nurse. Three days previously she had a pimple just inside her nose, and as she was going to a dance that evening she pricked it. When I saw her, she was lying on her back, her head propped up by pillows, with violent headache and a high temperature. I took the pillows away and tested her neck. She could bend her neck easily up to the angle of rest on the pillows, but beyond that it was rigid. Diagnosis—meningitis. The doctor in charge said “nonsense, she can flex her head easily,” and so she could up to the angle of rest. We did a lumbar puncture: the C.S.F. was very cloudy—she died in 24 hours. I mention this as one of the pitfalls in testing the neck for stiffness.

Twenty-five years ago, meningitis was a deadly complication. About 80% or more of the cases died, and although one’s luck varied, the results were poor, or at any rate mixed, until the sulphonamides arrived. In 1929 I had three cases in succession who survived but this was just a flash in the pan.

My personal results improved after using the technique of T. B. Layton (1935), that is early operation to remove the focus of infection, with free removal of the
tegmen, keeping the wound open by means of stitching the flaps back, and flushing regularly with saline. As a modification, later I used hypertonic saline packs. Lumbar punctures were done daily, and sometimes twice daily. My figures improved to over 30% recovery with these methods. I also tried injecting the theca with various antiseptics, solganol and others, but it did not appear to affect the recovery rate.

In the early thirties, Neuman evolved an operation with a very wide removal of the tegmen tympani as far forward as the eustachian tube, elevation of the dura and exposure of the upper surface of the petrous, then wide removal of the tegmen antri. Ruttin (1934) using this method claims a 50% recovery. These were the best results published up to 1934. Sulphonamides increased the recovery rate greatly, up to 50%, and penicillin has improved it still further, until to-day the recovery rate is over 80% in uncomplicated cases.

**TABLE I.—BENIGN FORMS OF OTOGENIC MENINGITIS (MYGIN, 1922)**

<table>
<thead>
<tr>
<th>Meningitis (uncomplicated)</th>
<th>Total</th>
<th>Recoveries</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>115</td>
<td>38</td>
</tr>
<tr>
<td>&quot; plus sinusphlebitis</td>
<td></td>
<td>42</td>
<td>16</td>
</tr>
<tr>
<td>&quot; plus brain abscess*</td>
<td></td>
<td>21</td>
<td>4</td>
</tr>
<tr>
<td>&quot; plus subdural abscess</td>
<td></td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>&quot; plus subdural abscess plus brain abscess</td>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>&quot; plus sinusphlebitis plus brain abscess</td>
<td></td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>&quot; plus sinusphlebitis plus subdural abscess</td>
<td></td>
<td>7</td>
<td>1</td>
</tr>
</tbody>
</table>

210 59  
*Including encephalitis.  
= 28% recovery.

These are the best, and by far the most complete results published in the *J.L.O.* up to 1922. 28% over-all recovery.

My personal results for seven years after 1922 show a 19.6% recovery. This is just less than 20% against Mygind's 28% all cases.


<table>
<thead>
<tr>
<th>Operation</th>
<th>No. of cases</th>
<th>Previous otitis</th>
<th>Recovered</th>
<th>Died</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mastoidectomy before meningitis</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>Abscess</td>
</tr>
<tr>
<td>Mastoidectomy at height of meningitis</td>
<td>3 (?2)</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>Heart failure and inhalation pneumonia</td>
</tr>
<tr>
<td>Myringotomy at height of meningitis</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Mastoidectomy during convalescence</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>= 77.7% recovery, 7 out of 9</td>
</tr>
<tr>
<td>No operation</td>
<td>13</td>
<td>4</td>
<td>9</td>
<td>4</td>
<td>(i) delay in chemotherapy (ii) and (iii) abscess (iv) acute cerebral edema = 69% recovery, 9 out of 13</td>
</tr>
</tbody>
</table>

22 16 6  
= 72.72% recovery.

One patient (Case II) had two operations. Previous otitis = a previous history of infection of the ear.

This paper was the result of experimental research by Professor Cairns and his team. As an experiment it is a remarkable tribute to the power of present-day chemotherapy. I should like to draw attention to one or two facts in this table, but not in a critical vein.

It is interesting to note that in those cases: (a) Operated on (9 in all) only 2 died—77.7% recovery; (b) not operated on (13 in all) 4 died—69% recovery.

This is in spite of the fact that in 6 of (a) no pre-operative chemotherapy had been carried out.
Professor Cairns states that in the 13 cases operation could not have altered the course of the disease. This statement is debatable. He postulates two questions: (a) Is the brain abscess produced concomitantly with the meningitis? (b) Is it produced later?

Nobody can answer these questions definitely, but, if it is later, and we know it often is by direct extension from the mastoid through the tegmen, then early mastoidectomy might have saved the two brain abscess cases, and even the acute cerebral edema.

I have no knowledge, but I can quite appreciate the difficulty of aural surgeons advising operative interference during this experiment—evidently they were not consulted in some of the cases.

In this experiment it will be observed that brain abscess, even in the hands of a neurosurgeon, caused most of the deaths—3 out of 6.

<table>
<thead>
<tr>
<th>Date</th>
<th>Name</th>
<th>C.S. fluid</th>
<th>Organism</th>
<th>Treatment</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>13.1.44</td>
<td>J. C. B.</td>
<td>Cells + (5)</td>
<td>Streptococcal</td>
<td>Sulph.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>10.4.44</td>
<td>H. B.</td>
<td>Cells + (6)</td>
<td>Small coagulum</td>
<td>Sulph.</td>
<td>Mastoiditis and brain abscess</td>
</tr>
<tr>
<td>19.5.44</td>
<td>L. J.</td>
<td>(1)</td>
<td>No growth</td>
<td>Sulph.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>9.1.45</td>
<td>G. O.</td>
<td>Cells ++ (2)</td>
<td>No growth</td>
<td>Sulph.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>18.4.45</td>
<td>A. P.</td>
<td>Cells + (3)</td>
<td>Pneumococci</td>
<td>Sulph. and pen.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>18.9.45</td>
<td>V. N.</td>
<td>Cells ++ (5)</td>
<td>No growth</td>
<td>Sulph. and pen.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>29.12.45</td>
<td>M. B.</td>
<td>Turbid (3)</td>
<td>Streptococcal</td>
<td>Sulph. and pen.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>7.1.46</td>
<td>R. P.</td>
<td>Turbid cells</td>
<td>Streptococcal</td>
<td>Sulph. and pen. intrathecal</td>
<td>Meningitis</td>
</tr>
<tr>
<td>6.2.46</td>
<td>J. B.</td>
<td>Cells + (3)</td>
<td>No growth</td>
<td>Sulph. and pen.</td>
<td>Mastoiditis and meningitis; lateral sinus thrombosis ligature of vein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(died 14th)</td>
</tr>
<tr>
<td>12.2.46</td>
<td>G. B.</td>
<td>Cells ++ (1)</td>
<td>No growth</td>
<td>Sulph. and pen.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>4.9.46</td>
<td>W. S.</td>
<td>Turbid (2)</td>
<td>No growth</td>
<td>Pen.</td>
<td>Mastoiditis and meningitis (died 11th), edema of lungs, duodenal case</td>
</tr>
<tr>
<td>23.12.46</td>
<td>R. C.</td>
<td>Cells + (8)</td>
<td>No growth</td>
<td>Pen.</td>
<td>Mastoiditis and meningitis</td>
</tr>
<tr>
<td>19.8.47</td>
<td>M. F.</td>
<td>Clear (2)</td>
<td>No growth</td>
<td>Sulph. and pen.</td>
<td>Mastoiditis and meningitis</td>
</tr>
</tbody>
</table>

The figures in brackets indicate the number of lumbar punctures performed. Sulphathiazole was used in every case.

This table emphasizes the value of chemotherapy, which was given in all cases before operation for about twelve hours, and continued after operation until the T. had remained normal for two days. The recovery rate of 84.6% compared with my earlier records up to 1927 of 20% recovery rate needs no comment. All the cases except one (7.1.46. R. P.) had mastoidectomy performed.

Labyrinthitis.—The subdivisions of this disease usually given in a textbook are somewhat bewildering.

The circumscribed type with the fistula sign is definite, as is the diffuse purulent type, but the serous types are not so definite and clinically do not matter—if left alone they recover. Lastly, there is the latent diffuse type—probably with a dead labyrinth.

As a complication of acute mastoiditis, I have seen only one case of diffuse purulent labyrinthitis with its severe symptoms, a small boy of 6. He had an extremely severe mastoid infection with labyrinthitis, lateral sinus thrombosis and meningitis. He died twenty-four hours after admission. I had performed a Schwartz operation in the meantime, but his was a fulminating generalized infection. At the post-mortem the saccus endolymphaticus was a bag of pus—the only case I have seen. I am aware that, through the years, recoveries of a few cases of acute diffuse labyrinthitis have been reported in the journals. This rare complication of mastoiditis—primary acute diffuse purulent labyrinthitis—is almost certainly a blood-borne infection as is primary pan-ophthalmitis.

I shall return to serious labyrinthitis in acute mastoiditis. In the following remarks, I shall discuss two problems:
(1) The inadvisability of testing the labyrinth by caloric and rotation tests in cases of mastoiditis with labyrinthise symptoms, either acute or chronic.

(2) The relationship of labyrinthitis and meningitis. This problem appears to me to be more readily studied and understood in acute than in chronic mastoiditis.

The accepted views on labyrinthitis were clearly expressed by E. D. D. Davis (1928). He stated:

"The records of cases of meningitis which followed chronic suppuration reported by Logan-Turner, Fraser, Jenkins and myself have shown that the symptoms and signs of labyrinthitis were present in a very large proportion. Vertigo, tinnitus, nystagmus and a marked degree of deafness accompanied by headache and fever with an increase of cells in the cerebrospinal fluid call for a translabyrinthine drainage of the meninges without hesitation. There are certain signs of the spread of infection which forecast coming events. Labyrinthitis is such a sign, and it requires careful investigation and prompt action as soon as it becomes apparent."

Tweedie (1934), who knew as much as anyone of his generation about the physiology of the labyrinth, supports the above view. In a paper published in 1934 he stated that in inflammatory lesions of the labyrinth—as in inflammation of any other organ—the normal responses to stimuli were upset, and all sorts of contradictory results obtained. In his 7-point summary of the tests, in point 6 he stated "that an attempt should still be made to assess the inference of spontaneous nystagmus in cases of acute otitis media". Previously he states that it is in these acute cases where we require assistance in assessing the amount of labyrinthitis that we shrink from applying the tests, and that generally we restrict the use of the labyrinth tests to chronic inflammatory cases of otitis media to determine whether or not the labyrinth is still functional.

I am of opinion that the above views are entirely wrong, and if carried into practice increase the danger to life in the treatment of labyrinthitis whether associated with acute or chronic mastoiditis. During the four years 1922–25 I carried out the usual cold caloric and rotation tests (Cold water (65° F., 17° C.) and the Bárány chair) in all cases of chronic mastoiditis. I operated on 3 cases with signs of labyrinthitis, and all 3 cases died of meningitis. During these years my colleague had no deaths. On careful investigation I found that the difference between his technique and mine was that he did not test any cases of labyrinthitis, whereas I did both caloric and rotation tests. I think it was the late Mr. Albert Gray who said that the labyrinthine capsule had great powers of defence and was capable of looking after itself in the presence of aural infection. As a result of these practical and theoretical considerations, I decided to stop testing the labyrinthine function, and wherever symptoms suggestive of labyrinthine upset occurred, to put the patient at rest, sand-bagging the head. During the twenty-one years that have passed since then, though I have had a number of cases of circumscribed labyrinthitis with the fistula sign, I have had no deaths from labyrinthitis in chronic mastoid disease.

It is my emphatic opinion that cases of mastoid disease in which labyrinthitis is suspected should be left at rest and that this complication will disappear in about three weeks. Several reasons may be put forward for regarding the widespread practice of testing the functions of the labyrinth in those cases as both useless and dangerous.

The labyrinth is endowed by Nature with strong defensive powers against infection. It is separated from the middle ear by a thin layer of bone—the stapes in the oval window, and the round window—and has to resist infection from a vast number of cases of middle-ear suppuration and mastoiditis annually. I have diagnosed about 6 cases of petrositis, but none complicated by labyrinthitis; cases of Gradenigo’s syndrome without labyrinthitis; cases of herpes of the geniculate ganglion; all of which had vestibular upset, but all recovered. In chronic mastoiditis, I have had a number of cases of circumscribed labyrinthitis with the fistula sign. Since 1926 not
one has developed diffuse labyrinthitis. Surely all these points are evidence of its great resistance to infection.

The rarity of severe labyrinthine infection compared to the vast number of middle-ear infections bears witness to the efficiency of its defensive mechanism. Where it becomes mildly infected and Nature has succeeded in walling off the infection it is surely dangerous to subject the patient to a series of tests which, I believe, are well calculated to break down the natural barriers and produce diffuse labyrinthitis, just as perforation of an acutely inflamed appendix sets up dangerous peritonitis. Tests of functional activity suitable in physiological conditions may be fraught with danger in pathological conditions and should be avoided. If active surgical treatment is indicated for the mastoiditis it should be carried out, but the labyrinthine upset should be left undisturbed and allowed to settle.

There is an analogy between the eye and the ear. Both are developed from the ectoderm. Primary acute pan-ophthalmitis is rare. The lesser degrees of inflammation interfering with function are more common, similar to cases of serous labyrinthitis which, if left alone, and rested will recover.

To conclude this portion of my paper, I believe that the use of these physiological tests on the labyrinth is the direct cause of the many cases of diffuse purulent labyrinthitis in chronic aural suppuration and the reason of the many translabyrinthine operations. These operations, with their fatalities, are a blot on our escutcheon.

I wish now to return to serous labyrinthitis in acute mastoiditis and to discuss the second problem, i.e. the relationship between labyrinthitis and meningitis.

Apart from the single case of acute purulent labyrinthitis already mentioned, I have seen a number of cases of acute mastoiditis with labyrinthine symptoms. One of these cases, about 1933, caused me much worry, and also gave me the key to this problem. She had an acute mastoiditis with very marked signs of labyrinthitis plus meningitis and a temperature of 104° F. Her symptoms subsided after the Schwartz operation had been performed. This wound was left open and treated according to Layton’s technique. Lumbar puncture showed increased pressure in the C.S.F. with some increase of cells but no organisms. This patient not merely recovered but she regained her hearing. She could not have had a purulent labyrinthitis: it had been of a serous type, yet her labyrinthine symptoms had been so extreme that I diagnosed her as a fulminating case of acute diffuse purulent labyrinthitis plus meningitis, and gave her father, who was a doctor, a fatal prognosis.

In this case there were marked symptoms of labyrinthitis plus meningitis. These should be regarded as two manifestations or complications of the parent acute otitis media—twins if you like, but not Siamese twins—separate entities.

In many clinics of the Continent and elsewhere, if such a case of labyrinthitis plus meningitis with an increased cell count had occurred, labyrinthine drainage would have been done at once, on the supposition that the meningitis develops from the labyrinthitis and this, I think, is wrong. Nearly all are simply cases of serous labyrinthine upset—the meningitis is the danger. It must be treated and has nothing whatsoever to do with the labyrinthine condition. The labyrinthitis heretofore has been regarded as the danger causing the meningitis.

There is one other condition that does occur, that is induced purulent diffuse labyrinthitis induced by operative procedure or injury, and this applies equally to the eye. Labyrinthitis induced by operation may occur once in every surgeon’s life, but it should be only once, and to-day, with chemotherapy, the patient should live. When induced purulent diffuse labyrinthitis is produced, it infects the meninges. This sequence has frequently occurred, and is responsible for most of the work and written papers on labyrinthitis in the past. There has been no progress in any published material on labyrinthitis.


Summary

(1) *Infection.*—The labyrinth is very resistant to infection, and diffuse purulent labyrinthitis is rare and is, or should be, almost entirely a primary disease.

(2) *Tests.*—(a) Diffuse purulent labyrinthitis, following labyrinthine tests, I regard as induced.

(b) The practice of testing the labyrinth is due to a confusion of thought. Physiological tests of function have been used on the labyrinth when it is in a pathological state. They are not clinical tests, and should be discontinued.

(3) *Relationship of labyrinthitis to meningitis.*—(a) Even when labyrinthine symptoms are very severe, they may not be due to a purulent infection of the labyrinth. They are simply a labyrinthine upset—"serous labyrinthitis" if you like the term.

(b) This labyrinthine disorder will recover without any interference with the labyrinth, provided, of course, the patient recovers from the original mastoiditis and its other complications.

(c) Meningitis is not as a rule caused by labyrinthitis as many observers think. It is always a primary complication of mastoiditis.

(d) Labyrinthine upset may be associated with meningitis, but the two conditions are entirely distinct. This is the fundamental point in the understanding of this problem.

The complication of brain abscess has not been included. It is a difficult problem. There has been marked progress in its prevention since its parent, the acute mastoid, is becoming more scarce because of chemotherapy.

REFERENCES


Mr. E. D. D. Davis said that it was most important to decide when to operate on a case of acute mastoiditis. The operation should be delayed if possible until the mastoiditis was localized and suppuration was present. If the mastoid were opened too soon, healing was prolonged, and the inflammatory condition appeared to spread. On the other hand an abscess healed rapidly. If all the air cells were opened in the simple complete mastoid operation a drainage tube for forty-eight hours only was all that was necessary. It was the unopened suppurating air cell which caused trouble and not the lack of drainage. Packing the wound with gauze led to a surface slough and secondary sepsis and must be avoided, a drainage tube was far better.

When meningitis was threatened the wound was left open but not packed. Rigidity or slight stiffness of the neck was a too late sign. Very few cases of meningitis with rigidity of the neck recovered. The rigidity was caused by a collection of purulent fluid in the basal cistema and around the crura cerebri. The examination of the cerebrospinal fluid was the only way to make an early diagnosis. Cases of meningitis arising from a blood-borne infection died very rapidly as in the case the President had mentioned.

In 1922 it was agreed that an operation on the labyrinth for labyrinthitis should not be done before the labyrinthitis was localized. A threatened or established meningitis demanded operation.

Lund of Copenhagen and others relied on the examination of the cerebrospinal fluid. If the cell count increased it was an indication for operation. A large number of labyrinth operations had been done because of the fear of meningitis.

Children and young adults were more susceptible to meningitis than old people. The unexplained severe headache with a rise of temperature and pulse, the cellular mastoid pouring serous fluid with deep-seated pus in the cells behind the facial nerve, the mastoid following scarlet fever and measles
all pointed to the possibility of meningitis. Treatment by sulphonamides and penicillin had improved the prognosis of meningitis.

Mr. T. M. Boyle made a plea for the use of heparin in a small number of cases of lateral sinus thrombosis. He cited the case of a girl aged 15 who had the lateral sinus drained and continued to have rigors for about a week afterwards. She was having large doses of penicillin and sulphonamides. Then a general surgeon put her on heparin, and from that day she made a rapid recovery. He would not say that all such cases should have heparin, but those cases not responding to treatment. In another small group of cases of mastoiditis there were resistant organisms, _B. pyocyaneus_ being one of the offenders, and here he thought phenoxetol, a proprietary preparation, was of great value. He recalled a case which had had the mastoid opened about ten years ago, and later developed a recurrence: an attack of mastoiditis with a green discharge. It dried up but recurred on several occasions. The mastoid was opened and the drainage tube inserted and phenoxetol applied in the cavity. Within three days the discharge from the external meatus was sterile, and within six days the middle ear was dry.

Mr. W. H. Bradbeer said, with regard to latent labyrinthitis, that two patients in whom he suspected that the labyrinth was dead both developed meningitis following operation. One was a mild case and recovered. In the other a very severe meningitis developed, and eventually a cerebella abscess, but this case also recovered after opening the labyrinth and draining the abscess. If one was doing a radical mastoid operation with a dead labyrinth he had come to the conclusion that one should open the labyrinth as well, but after what the President had told them he was uncertain on that point. He thought they would still see chronic ears, and one came across children in school clinics with chronic otorrhoea and with an attic perforation.

Mr. F. McGuckin said he believed a cerebral abscess might arise at any stage of an otitis because of the possibility that a localized infection in a perivascular space might act as a preformed path. On the question of labyrinthitis he agreed almost unreservedly with the President’s plea, in so far as the complication was related to acute otitis.

Mr. F. W. Watkyn-Thomas said that a danger in using heparin was that the clotting time was altered for a considerable time afterwards, which might give trouble should further surgery be needed. The justification for the use of heparin in sinus thrombosis would be to make sure that the antibiotics came into contact with the organism; an organism in the clot was safe from anything in the way of penicillin or sulphonamide treatment.

On the question of translabyrinthine drainage, since the use of sulphonamides he had only seen two cases of meningitis which were undoubtedly due to labyrinthine infection—at any rate they had certainly followed upon a labyrinthitis. In these two cases he refrained from translabyrinthine drainage because he felt that, if the cerebro-spinal fluid was released, it would be more difficult to keep up the requisite sulphonamide concentration. Both patients recovered. The translabyrinthine operation had been the greatest advance in treatment in its day, but now it would rarely, if ever, be necessary.

Mr. Ogilvy Reid referred to the rarity of such a complication as facial paralysis in acute mastoiditis or acute otitis media. He had in mind one or two recent cases which illustrated the difficulties which chemotherapy had introduced in the treatment of mastoid conditions and the way in which the picture was obscured. One case had had massive doses of penicillin and was apparently doing very well when a facial paralysis developed. He operated on the case and though he found no lesion relating to the facial nerve yet the facial paralysis began to recover after the operation.

Mr. R. R. Simpson asked what would be the President’s attitude in the case of the labyrinth in the presence of otitis media with fistula signs? He himself did a radical mastoidectomy in such a case without interfering with the labyrinth at all, and so far he had never regretted it.

Mr. J. H. Ott felt sure that an adequate dosage was the essential in the use of the sulphonamides or penicillin, but it was equally important to tighten up the criteria of cure. These patients should not be let out of hospital unless the surgeon was satisfied, not only that the ear was dry but that the tympanic membrane had returned to normal and the hearing had been restored.

Mr. L. Graham Brown said that as a senior he felt that he had had a considerable experience of these cases during the past quarter of a century. The young practitioner only saw the question from his own recent experience, and naturally was a little perturbed as to what the treatment really ought to be. The time had now come when chemotherapy had taken a large part in the treatment of these affections. He wished to say with regard to chemotherapy that he used it only as an adjunct to
surgery. They were all aware that its application in the early stages of acute inflammation of the middle ear might be successful in relieving the condition, but also they knew the dangerous effects which might be brought about if it was used at a time when pus was formed in the middle ear or the mastoid bone or in the further complications of the disease. They were aware of the fatalities which occurred. Perhaps the mastoid operation would be followed by almost complete resolution, and then, some weeks afterwards, a brain abscess or meningitis occurred and the patient often died.

He had not operated on a case of labyrinthitis for many years—perhaps fifteen. Cases in which there were signs of labyrinthine irritation in chronic disease of the mastoid, which flared up, he had dealt with by performing a radical mastoid at the opportune time, providing there was no evidence of the disease spreading to the meninges. Such cases had invariably got well. In view of the statements that these cases were rapidly diminishing, it was strange that Mr. Simson Hall should have seen recently several cases of lateral sinus thrombosis. He himself had not seen one for two or three years, but, curiously enough, he had three cases of meningitis, and these were cases of chronic mastoiditis, all of which, after removal of the focus in the mastoid—going no further, and putting them on chemotherapy—had recovered. The most important thing was to learn how and when to use chemotherapy.

Mr. Terence Cawthorne said that the high light of the President’s Address was that he had had the courage years ago not to treat the labyrinth surgically in the early stages of an infection. Most of them had come to realize, now that they had recourse to chemotherapy and biotherapy, that early surgical treatment was wrong. He agreed with Mr. Layton who had suggested that chemotherapy should precede surgery even in chronic conditions where there was an acute flare-up.

Mr. H. S. Sharp said that in acute middle-ear disease he never gave chemotherapy unless he had first obtained drainage from the ear. Such cases were followed up very carefully by means of X-rays, and in adults, in addition to X-rays, by tests for deafness.

The President (Mr. Watson), in a brief reply, said that if there was any surgery to be done it must be done and done early, but on cases that had been on penicillin or sulphathiazole for three or four weeks there was no harm in operating if there was any doubt. Mr. Boyle had spoken of cases in which sulphathiazole and penicillin were given at the same time. That was an interesting point and one for discussion in the Section. He had seen two cases of meningitis in patients who had had too large doses of penicillin and sulphathiazole. The temperatures did not drop and he could not understand it. In one of the cases he stopped penicillin and the temperature dropped immediately, nothing else happened. In the other case he stopped sulphathiazole, with the same result. Some of these patients were “killed by kindness”. Too much in the way of antibiotics could be given. Whether heparin counteracted the effects he could not say.

There had been a great deal of confusion about the chronic mastoid. People talked about labyrinthitis after they had been, he was sure, testing the cases. He himself would not have anything to do with testing, and if anyone talked about labyrinthitis after having done caloric, rotation or electrical testing he thought it a sequel. The plea he would put forward was to stop physiological tests. He drew an analogy from the heart. There was a tolerance test for the heart by which one observed the heart’s function. This was carried out on a normal or near-normal heart. Would a clinician test a heart with valvular disease by means of these tolerance tests? Would he test a patient with a temperature of 102°F. whom he thought was developing endocarditis by asking him to run upstairs and down again? Of course he would not, but it was something like this that they had been doing in otology.