EXTRADURAL INTRACRANIAL (MIDDLE FOSSA) APPROACH TO THE INTERNAL AUDITORY CANAL*

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In recent years, otologic surgeons have made an exponential advance in the surgical management and understanding of conduction deafness, largely as a result of rather highly developed microscopic techniques. Surgical procedures through the external auditory canal or mastoid do not provide a satisfactory access to the cochlea, labyrinth and their nerves. For this reason, surgical progress in the problem of perceptive loss of hearing has been limited. Classic posterior-fossa approaches limit visualization of the 7th and 8th nerves to the cerebellopontine cistern medial to the meatus of the internal auditory canal.

Approximately 3 years ago we learned from a series of dissections on cadavers that a modification of the extradural (Spiller-Frazier)7 rhizotomy approach to the middle fossa provided us with a theoretically safe and surprisingly adequate exposure of the internal auditory canal and its contents.

The purpose of this presentation is to describe this operation which, to date, has been performed by the authors and Dr. W. F. House‡ in 40 cases.

PROCEDURE

The operation has been a combined neurosurgical-otologic procedure. The preoperative neurological status and assessment of the surgical risk are the responsibility of the neurosurgeon. Preoperative hearing and vestibular testing are the responsibility of the otologic surgeon. The subtemporal craniotomy and extradural dissection of the greater superficial petrosal nerve are performed by the neurological surgeon. The removal of the petrous bone usually is done by the otologic surgeon.

1) A modification of the Spiller-Frazier extradural subtemporal decompression in the upright position is used.

2) The dura mater is dissected off of the superior surface of the temporal bone from the petrous ridge to the middle meningeal artery. Division of the middle meningeal artery seldom is required. The dissecting microscope (6X) is used to identify the greater superficial petrosal nerve as it exits from the hiatus of the facial canal. The efficiency of hemostasis resulting from coagulation of extradural bleeding points identified by the dissecting microscope is remarkable when compared to macroscopic experience. This nerve is separated carefully from the dura mater to permit insertion of self-retaining retractor to the petrous ridge.

3) Utilizing the point of exit and direction of the greater superficial petrosal nerve as a landmark, the surface of the petrous bone is thinned by the diamond drill enough to identify the facial canal containing the geniculate ganglion.

4) The facial nerve then is traced posteriorly toward the petrous ridge by continued removal of bone. Care is taken to leave a thin shell of bone over the dura mater of the internal auditory canal. The apex of the roof of the internal auditory canal is indicated by a faint blue line which serves as a directive guide for the complete unroofing of the internal auditory canal back to and including the petrous ridge. Careful attention to this guiding line is essential to prevent opening into the cochlea immediately anterior and the vestibular apparatus posterior to this line of dissection.

5) Following complete exposure of the roof of the internal auditory canal, the remaining thin layer of bone is flicked off by manual dissection. When a tumor of the angle is known to be present, additional exposure can be obtained by further removal of bone along the petrous ridge immedi-

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ately lateral and medial to the internal auditory canal.

6) The dura mater and arachnoid then are opened, revealing position of the 7th nerve uppermost and anterior in relation to the other nerves. The superior and inferior branches of the vestibular can be identified posterior to the cochlear portion of the 8th nerve and can be resected by elevation with a hook, thereby denervating the labyrinth.

7) If a neoplasm is encountered, the surface of the capsule is exposed; and a plane of cleavage is developed between the capsule and the 7th nerve. The capsule of the tumor then is opened and evacuated by suction and alligator forceps with progressive reduction of mass and resection of capsule.

8) The dural opening and the bony defect then are sealed with Gelfoam and routine closure in layers is accomplished.

Postoperative care largely has been uncomplicated and does not require more than the routine precautions used in any postoperative craniotomy. When the vestibular nerve has been resected, there is frequently a 48-hour period of vertigo with nausea.

RESULTS

Our initial experience comprised the 8 cases of acquired and congenital loss of hearing. The therapeutical rationale is not within the scope of this presentation and has been reported elsewhere. The results were disappointing; however, the absence of mortality and morbidity and the tolerance the patients exhibited for the procedure were encouraging. As our skill increased, commensurate with our experience, we learned that a considerable amount of surgical manipulation of the 7th and 8th nerves was permissible and that the vestibular components of the 8th nerve could be identified and resected while sparing the cochlear division.

Total resection of the superior and inferior branches of the vestibular division resulted in a complete remission of vertigo in each instance. The undesirable factors that caused qualification of the results in 6 cases (Table 1) were related to continuation of the non-vertiginous clinical components of Ménière’s syndrome.

The limited number of cases in each category of assorted neuropathies of the 7th nerve does not justify any conclusion regarding the effectiveness of the surgical procedure. However, this group does indicate an

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No.</th>
<th>Results</th>
<th>Complications</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceptive hearing loss (Congenital &amp; acquired)</td>
<td>8</td>
<td>Unsatisfactory</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ménière's syndrome</td>
<td>19</td>
<td>13 Excellent</td>
<td>1 Increased tinnitus</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 Fair</td>
<td>6 Discomfort, fullness</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Poor</td>
<td>1 Progressive postop. hearing loss</td>
<td>0</td>
</tr>
<tr>
<td>Seventh nerve neuropathy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herpetic neuritis (Ramsay Hunt)</td>
<td>1</td>
<td>Fair improvement</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Trauma (basal skull fx)</td>
<td>2</td>
<td>Fair improvement</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Greater superficial petrosal (infected adj. air cell)</td>
<td>1</td>
<td>Excellent relief eye pain, ear pain &amp; decreased lacrimation</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neurinoma</td>
<td>1</td>
<td>Complete tumor removal</td>
<td>C.S.F. otorrhea &amp; meningitis</td>
<td>0</td>
</tr>
<tr>
<td>Eighth nerve tumor</td>
<td>6</td>
<td>Excellent</td>
<td>All with mild facial weakness, none total</td>
<td>0</td>
</tr>
<tr>
<td>Neurinoma</td>
<td>1</td>
<td>Poor</td>
<td>Coronary occlusion &amp; massive pulmonary thromboembolism</td>
<td>1</td>
</tr>
<tr>
<td>Epidermoid</td>
<td>1</td>
<td>Excellent</td>
<td>No facial weakness</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td></td>
<td></td>
<td>1</td>
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</tbody>
</table>
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additional clinical spectrum that deserves further investigation.

Experience with the group of 8th-nerve tumors revealed that it is possible to accomplish total removal of small to moderate-size tumors of the angle with preservation of anatomic continuity of the 7th nerve, resulting in partial interruption of function. This group of cases has been quite recent, and the permanent result is not known. The validity of the von Békésy Type IV audiometer curve in indicating early retrocochlear involvement of the 8th nerve has been verified surgically thus far in our experience. The surgical procedure has been lengthy (6 to 8 hours) and certainly not easy from the surgeon’s standpoint. However, the remarkable paucity of even immediate postoperative neurological deficit is gratifying.

The fatal case is that of the following patient:

A 73-year-old woman was thought to have a massive tumor with papilledema, ataxia, vertigo, involvement of the 5th nerve and extensive erosion of the petrous bone. Although the patient’s immediate general health was good, she had advanced atherosclerosis of the coronary artery and was considered as a poor surgical risk.

The plan was to decompress the tumor with as minimal a surgical procedure as possible. The subtemporal method was selected with considerable preoperative reservation. The patient appeared to tolerate the procedure well; but, as the head dressing was being applied, she became apneic and failed to respond despite vigorous efforts for 3 days.

Postmortem examination revealed acute massive myocardial infarction and multiple bilateral pulmonary emboli. Blood was found in the subarachnoid space and within the capsule of the tumor but did not appear responsible for the patient’s sudden death.

It is our present opinion that this approach should not be used when the presence of a massive tumor is certain.

DISCUSSION

Increasing awareness of the potential significance of early symptoms related to disturbance of cochlear and vestibular mechanisms has led to the development and refinement of clinical diagnostic techniques such as von Békésy audiometry, Hallpike vestibulocaloric testing, and improved radiography of the temporal bone and its cisterns. However, in spite of these advances, preoperative clinical analysis does not permit a precise identification of the pathologic mechanism responsible, particularly in the early phase of the disease.

Total surgical removal without additional neurological deficit represents the ideal goal in the management of acoustic neuroma. Surgical removal of massive acoustic neuroma via classic techniques under the most ideal circumstances can be a precarious undertaking. Instances of complete removal of tumor with preservation of the 7th cranial nerve are rare. A small acoustic tumor within the internal auditory canal of the type described by Hardy and Crowe may not be visualized by exploration via the posterior fossa.

Pool and Pava found that deafness preceded all other symptoms of acoustic neuroma in 90 out of 122 cases by an average of 3.9 years. In only 16 cases were additional symptoms concurrent with the deafness. Therefore, if the goal is to be realized, surgical exploration must be accomplished when the tumor is small and when the differential diagnostic possibilities are not established completely.

In many instances, the clinical distinction between Ménière’s disease and an early acoustic neurinoma can be difficult, as illustrated by the following cases:

Case 1. J. H., a 36-year-old woman, for the past 9 years had experienced episodic systematized vertigo, and tinnitus with an associated perceptive loss of hearing. In 1957 no abnormalities were found on neurological examination, including lumbar puncture, roentgenograms of the skull and petrous bones, pneumoencephalogram and electroencephalogram. Patient continued to have intermittent attacks of incapacitating vertigo which increased in frequency and severity. Examination in February 1960 gave normal neurological findings. However, there was an increase in the loss of hearing, a reduced vestibuloculocoric response and definite enlargement of the right internal auditory canal, which was not present when compared with the roentgenograms taken in 1957.

Case 2. L.B., 57-year-old father of a physician, experienced progressive incapacitating vertigo of
about 2½ years' duration with a progressive increase in frequency and severity; insidious headache almost constant during 3 months, requiring 8 to 10 tablets of aspirin per day; associated with tinnitus and progressive loss of hearing in the left ear; a questionable caloric response; a subjective complaint of gradually increasing numbness of the left side of his face; an absent corneal reflex on the left and hypaesthesia in the ophthalmic division of the left trigeminal nerve. The results of two recent previous neurological studies, including spinal fluid, were reported as normal.

In both Cases 1 and 2, exploration of the internal auditory canal via the extradural middle-fossa approach was accomplished. No evidence of neoplasm was found in either case, and in both cases the superior and inferior branches of the vestibular nerves were resected. There was an uneventful postoperative course in both instances, and there has been a complete remission of all vertiginous attacks without any increase in either patient's loss of hearing.

SUMMARY

1) A technique for intracranial extradural (middle fossa) exploration of the internal auditory canal is described.

2) Initial experience suggests that this procedure provides a reasonably safe access to the internal auditory canal.

3) Clinical experience with this procedure in 40 cases of perceptive loss of hearing, neuropathy of the 7th nerve, Ménière's disease and acoustic tumor are reviewed briefly.

4) The indications, contraindications and applications of the specific surgical task to clinical problems remain to be established.

CONCLUSION

1) It is hoped that the procedure will provide us with the opportunity of studying the electrophysiology of perceptive loss of hearing in the human, and that this information will offer a guide to therapy.

2) Further clinical study and practical experience may indicate the value of this procedure in the therapy of acoustic neuroma and Ménière's disease.

3) Neurosurgical experience with microtechniques in this problem suggests its potential usefulness in many other areas.

REFERENCES


DISCUSSION

Dr. Charles G. Drake: I think this paper is another happy example of two surgical disciplines getting together over a common problem, and it represents a new step in the understanding of perceptive deafness. It is of particular interest to me because of an interest in Dr. Norman Dott's extratemporal graft for recovery of facial function, in which recovery of emotional expression seems worth the effort involved.

[Slide] In this case, the stump of the facial nerve, greatly elongated by the tumor, could be anastomosed directly to the distal end after removal of the posterior wall of the porus with a punch, and satisfactory recovery of facial function ensued.

Ordinarily this is not feasible, for the facial nerve proceeds directly away from the operator. Now, Dr. Kurze and Dr. Doyle have shown us that the medial portion, that is the intrapetrous part of the facial nerve, can be exposed by this approach. This offers the use of a much shorter graft or even direct repair which could be done as a secondary procedure, providing one left the stump of the facial nerve well marked before closing the posterior fossa. It remains to be seen what can be done with this method.

Dr. Kurze, I think, rightly cautions against the routine use of this approach in the surgical treatment of acoustic neuroma. It may be ideal when the tumor is small and confined chiefly to the porus. However, in large tumors, which seem to be the garden variety up north, the primary consideration is safe removal of the tumor, facial function being quite secondary. I wonder how well the dissection could be done alongside the brain stem, preserving carefully the 9th and 10th nerves, getting the blood supply to the tumor, and so on. It would seem best that until more experience is gained
with this method, the routine approach to tumors of the angle should continue to be through the posterior fossa.

This paper, however, represents another contribution to the surgical treatment of Ménière's disease. The otologists have entered this field with vigor, but their operations, although simple, destroy hearing completely. This is not important when no useful hearing remains, but differential section still should be considered in those few cases in which it is present. My own reservation with regard to this approach is the factor of time and the use of two teams, which seems rather arduous; but the technique is beautifully precise. Having been brought up by Dr. McKenzie, of course I favor his procedure which is simple and safe, although perhaps dividing a few cochlear fibers. The importance of differential section is emphasized in Dr. Barber's review of Dr. McKenzie's cases in which 2 out of 3 patients retained useful hearing for periods varying up to 10 years. The hydrops, of course, is relentless and sooner or later will destroy cochlear function, but even 2 or 3 years would be worth while.

Lastly, I might add that neurosurgeons may well be required to familiarize themselves with this technique for their inquisitive otologic colleagues who wish to take advantage of it.

I have one question. I would like to know about the incidence of postoperative leaking of spinal fluid through the petrous bone into the throat, and does palsy of the facial nerve ever occur as a result of this section.

I would like to congratulate the authors on a fine piece of work.

**Dr. Joseph Ogura:** I am greatly impressed by this combined service of cooperation. I think that the applicability of this operation is probably to lesions that are small and those that are within the internal acoustic meatus. It remains for the selection of the cases as to the methods that are applied. The tests for threshold and discrimination of speech and vestibular test should be of great value. I would doubt that this procedure is applicable in cases of perceptive loss of hearing since this is an end-organ or hair-cell disease. With lesions that are more medical, I think it would be a little more difficult to apply this method. To cite an instance, in the 1 case of herpetic disease, Ramsay-Hunt disease, this decompressive operation might not serve a useful purpose.

It is the Ménière's part here that I would really like to discuss. For one thing, I note that 19 of the 40 cases concerned Ménière's syndrome or, as we call it, hydrops of the labyrinth.

[Slide] Hallpike and Cairns brought to our attention that dilatation of the saccule of the labyrinth in endo-lymphatic hydrops is the pathology present in Ménière's disease.

[Slide] Here you see a good example of the endolymphatic duct dilated completely. Of course, as we now know, it is associated with changes in the balance of sodium and potassium in the labyrinth.

[Slide] In the diagnosis of this, the most important part is here, the discrimination score. This is a double system disease, which involves not only the vestibule but also the cochlea. So, even when the threshold is down, the most important thing is that the patient can hear. When one comes up to 76, this represents a finding of normal hearing, and when the discrimination drops down to 28 per cent, this definitely is outside of the range of hearing.

[Slide] I would like again to point out one thing: that is, with losses of threshold across here, with a 30 per cent discrimination, and let us suppose that the labyrinth were hypoxic or even out. This does not mean necessarily that one needs to destroy this labyrinth, because even with small doses of histamine, for example, in the case which is over a period of 3 months, one can change the discrimination score back to normal.

[Slide] The other important thing is the distortion of sound. At 4000 cycles, with the left ear being normal, this is what happens when there is hydrops of the labyrinth. Distortion is so marked that the involved ear cannot be matched against the normal sound of 4000 cycles.

[Slide] Here is a typical surgical candidate. Dimplacis is present over a wide range and cannot be matched. Since cochlear disease here is poor, unintelligible and confusing, and if there is vestibular disease, the best thing to do is a complete destruction. On the other hand, if there is no cochlear disease, then vestibular section would seem the procedure of choice. Before undertaking this one must weigh the clinical situation carefully. Medical management should be given first.

In conclusion, I would like to summarize by saying I think this is a magnificent piece of work, and I think this should be pursued more and studied more carefully.

**Dr. Theodore Kurze:** I would like to thank the discussers for their kind remarks. In answer to Dr. Drake's two questions, we did have 1 postoperative spinal-fluid otorrhea. That was in the neurinoma of the 7th nerve, listed in the assorted neuropathies of the 7th nerve. There was quite a bit of erosion of the fossa.

Although we used the neural covering of the 7th nerve as our primary landmark in doing the dissection, we have, to our surprise and delight, not had a significant change in the 7th nerve. When we first started the dissection, we were having some fibrillary twitchings of the nerve but we did not have any paralysis or weakness.