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Acoustic Neuromas

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INDEX

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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

Visitors Welcome

March 1 - March 6, 1948

No. 192

Monday, March 1

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns' Quarters, U. H.

9:15 - Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.

10:00 - 12:00 Neurology Ward Rounds; A. P. Baker and Staff; Station 50, U. H.

11:00 - 11:50 Physical Medicine Conference; Parkinsonism; Joe Brown; E-101, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Staff; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; M-435, U. H.

12:30 - 1:20 Pathology Seminar; Apparatus and the Hypertensive Atrophy. John Des Prez; 104 I. A.

12:30 - 1:30 Physiology Seminar; Pulmonary Vascular Bed Capacity; M. B. Visscher, 214 M. H.

12:30 - 1:50 Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U.H.

4:00 - 5:00 School of Public Health Seminar; Poliomyelitis Studies; A. J. Gilliam, University of Michigan; 113 MeS.

4:15 - 5:15 Pediatric Seminar; Occurrence, Nature and Metabolism of Folic Acid; R. J. Salmon; 6th Floor Seminar Room, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy and H. M. Stauffer and Staffs; M-515, U. H.

Tuesday, March 2

8:30 - 10:20 Surgery Seminar; Lyle Hay; Small Conference Room, Bldg. I, Veterans' Hospital.

9:00 - 9:50 Roentgenology Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Robert Hebbel; Veterans' Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans' Hospital.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans' Hospital.

4:00 - 5:30 Surgery-Physiology Conference; O. H. Wangensteen and M. L. Visscher; Eustis Amphitheater, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

5:00 - 5:50 Roentgenology Diagnosis Conference; Oscar Lipschultz and Staff of General Hospital; M-515, U. H.

Wednesday, March 3

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans' Hospital.

11:00 - 11:50 Pathology-Medicine-Surgery Conference; Subject to be announced; E. T. Bell, O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

4:00 - 5:00 Infectious Disease Rounds; Todd Amphitheater, General Hospital, Veterans' Hospital.

Thursday, March 4

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Walter Walker and H. M. Stauffer; M-515, U. H.

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans' Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U. H.

12:00 - 12:50 Physiological Chemistry Seminar; Histochemical Aspects of the Mast Cell with Special Reference to Alkaline Phosphatase and Cytochrome Oxidase; Marco Rabinovitz; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

4:00 - 4:50 Bacteriology S eminar; Heat Resistance of Bacterial Spores; Arnold Lund; 214 M. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 5:50 Roentgenology Seminar; Emphysematous Cholecystitis; Jack Friedman; M-515, U. H.

Friday, March 5

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans' Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, A. V. Stoesser and Staffs; Minneapolis General Hospital.

11:30 - 12:50 University of Minnesota Hospitals General Staff Meeting; Surgical Treatment of Noncalculous Obstruction at the Uretero-pelvic Junction; C. D. Creevy and Brian McGroarty; New Powell Hall Amphitheater.

12:00 - 1:00 Surgery LiteratureConference; Clarence Dennis and Staff; Minneapolis General Hospital, Small Class Room.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

Saturday, March 6

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor West Wing, U. H.

8:00 - 9:30 Psychiatry and Neurology Grand Rounds; Staff; Veterans' Hospital.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, and Staff; Todd Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-515, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; M-515, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - 12:20 Anatomy Seminar; The vaginal smear as an index of hormone secretion; Marthella Frantz: The innervation of the lung; Roger M. Berg; 226 I. A.

II. ACOUSTIC NEUROMAS

William T. Peyton
C. Kent Olson

The treatment of acoustic neuromas has undergone a gradual evolution since the first attempt was made by McBurney[33] in 1891 to remove a cerebellopontine-angle tumor. Acoustic neuromas are usually one of the most easily diagnosed tumors of the cranial cavity. Nevertheless, patients with these tumors still come for surgery in the final stages of their untreated evolution. In these late stages not only is the risk of removal enhanced but the residual sequellae are more severe. It, therefore, seems proper to devote this staff meeting to a discussion of cases of acoustic neuroma. Included are those cases encountered since 1941 at which time we began to totally, rather than subtotally, remove these tumors.

Frequency

Unilateral acoustic neuromas are said[7] to constitute eight to ten per cent of all intracranial tumors. In Cushing's[9] 2023 intracranial tumors, there were 176 acoustic neuromas (8.7%). It is probable that even this is greater than the true incidence because Cushing's special interest in acoustic tumors was well known at least since 1917 when his monograph[10] was published. Dandy[43] apparently operated upon 154 cases of acoustic neuromata. They have constituted much less than ten per cent of intracranial tumors in this clinic, where since January, 1941 we have seen twenty verified acoustic neuromas.

Bilateral acoustic neuromas are even more rare than the unilateral and are prone to occur in young people. They are then frequently associated with generalized neurofibromatosis (von Rechlinghaussen). Six of the 154 (4%) acoustic neuromas reported in Dandy's material[43] were bilateral. Frykolm[21] described six cases of bilateral acoustic neuromata operated upon by Olivecrona during a period in which 241 unilateral neuromata were encountered in the same clinic (2.4%). Frykolm found only thirteen other bilateral cases in the literature which had been operated upon. In our series of twenty cases, there is one in which bilateral tumors were verified and one in which it is presumed that there were bilateral tumors but verified only on the one side which was operated upon.

Gross Pathology

Acoustic neuromas occur in the cerebellopontine-angle and apparently originate in most, if not in all, instances on that portion of the 8th nerve which lies in the internal auditory canal. All of the twelve small tumors which have been reported as chance post-mortem findings were so situated[10,25]. Six of these asymptomatic tumors were discovered by Hardy and Crowe[25] in 250 unselected cases in which serial sections of the temporal bone were made. They were too small and too deep in the internal auditory canal to be found at autopsy. Four of these 6 tumors involved the vestibular portion of the nerve, one involved the cochlear portion of the 8th nerve, and the other one seemed to arise from the dura. All were deep in the canal close to or directly involving Scarpa's ganglion. No case of small tumors involving only that part of the nerve between the internal auditory meatus and the pons was found in the literature, but Dr. A. B. Baker[3] states that he has observed in routine autopsies two small tumors in this location. When large enough to bring the patient to operation acoustic neuroma always extend into the porus acusticus and when this large, it is impossible to determine their site of origin.

Acoustic neuromas are subarachnoid in location and expand in the lateral pontine cistern. There is almost always a few cubic centimeters of encysted fluid in the lateral part of this cistern overlying the tumor, sometimes this cyst becomes quite large, but it is extremely rare that the cyst becomes as large as one reported by List in which there was 100 cc. of fluid. Eventually in the terminal stages, acoustic neuromas may obstruct the circulation of the cerebrospinal fluid in the region of the foramen ovale of Pacchioni (tentorial notch). At this site the fluid passes

caudally through the aqueduct and after circulating in the posterior fossa the fluid then passes back upwards through either the cisterna pontis, ventral to the pons, or the cistern of the great vein of the cerebrum (cisterna ambiens), dorsal to the pons. Eventually displacement of the brain stem and cerebellum, by the tumor, or even extension of the tumor through the tentorial notch into the middle cranial fossa occludes all of these cysterna about the pons[50,17,30,5]

This b.ock of the return flow of fluid through the tentorial notch by angle tumors apparently occurs without obstruction of the aqueduct or at least earlier than aqueduct obstruction and accounts for the fact that the cisterna cerebello-medullaris is seldom compressed but is often enlarged[31].

Acoustic neuromas grow slowly. The average duration of 8th nerve symptoms before operation was 3 years and 10 months in Cushing's 176 operated cases[31]. It is not very uncommon for the history of tinnitus and decreased hearing to extend back 10 years or more.

Due to the slow growth and displacement of surrounding structures, acoustic neuromas sometimes attain surprisingly large size when the small space in which they originate is considered. Weight is not frequently determined because they are removed piecemeal with currett and sucker. Revilla[43] in a series of 154 tumors removed at operation found the largest one which was eighed to be 70 grams of tissue but one weighing 154 grams was reported by Sachs[45]. To obtain such a size they must grow slowly, displace the pons and medulla, and project themselves into every available crevice including an extension through the foramen ovale of Pacchioni. They also become embedded in and adherent to the cerebellum and pons. Arterial branches from the basilar artery may grow into the medial side of the tumor and spread over its capsule. The capsule, especially on its deeper side, is quite vesicular but the interior of the tumor is relatively avascular.

Microscopic Findings

There has been much difference of opinion concerning the nature of acoustic neuromas, especially as to whether or not they are ectodermal or mesodermal in origin. It is not the purpose of this report to enter into this discussion but let it suffice to say that the best evidence seems to be in favor of their taking origin from the perineurium rather than from the sheath of Schwann. Hence, they are probably derived from mesodermal elements.

Histological examination reveals acoustic neuromata to be comprised of elongated fibroblastic cells within a matrix of collagen. The fibroblasts are arranged in bundles. Cut section of these bundles may show the fibroblastic nuclei to be arranged in parallel rows, the typical pallisade appearance of neuromas. The bundles of fibroblasts are usually in an orderly arrangement but occasionally they are extremely intertwined and intermixed producing a very heterogenous appearance. There may be areas of large clear cells (foamy cells) which result from fatty degeneration within the tumor.

Radiographic Changes

Skull roentgenograms of patients with acoustic neuromas in late stages, as they usually are when they come to surgery, show evidence of increased intracranial pressure. In addition, and of especial interest, is the appearance of the petrous pyramid, for acoustic tumors may produce erosions of both the porus acousticus and the pyramid.

When Henschen[26,27] realized that these tumors had their origin in the depth of the internal auditory canal, and by growth expanded the canal, he attempted to visualize the dilated porus acusticus in roentgenograms. The first case in which he thought he was able to demonstrate enlargement came to postmortem a few weeks later in 1911 and the porus was much larger than he saw it. About this same time Cushing[10] also began to attempt visualization of the eroded porus acusticus. In these early attempts to visualize the porus acusticus the position of the patient's head in which the exposures were made was not

proper and the external auditory canal was mistaken for the internal so that the results were very unsatisfactory. Inefficient mechanical equipment also contributed to these unsatisfactory results.

It was not until 1926 when Chambers demonstrated destruction of the petrous bone in a verified case of acoustic nerve tumor that much was contributed to the diagnosis by roentgenograms of the temporal bone. Chambers showed this destruction by a posterior projection in which the petrous bones are identified in profile. Subsequently, this case in which erosion was demonstrated by Chambers plus two more were reported by Towne[49] and this has become known as the Towne position. This position shows best the destruction of the medial part of the superior border and posterior surface of the pyramid, but the internal auditory meatus in this projection is obscured by other confusing shadows. The porus acusticus or internal auditory meatus is best shown by an anterior-posterior position with the head placed at such an angle that the shadow of the pyramids is cast through the orbits (Camp[7]). In this position the internal auditory canal runs approximately perpendicular to the plane of the projection although the posterior surface of the petrous portion of the temporal bone is at an angle of approximately 45 degrees to this plane. The Stenvers position gives a satisfactory but foreshortened internal auditory canal.

When one does get good detail as a result of improved technique, it is well to remember that the porus may vary from 2.5 to 11 mm. in diameter and even between the two sides variations of as much as 2.5 mm. may occur[7].

Erosion of the petrous portion of the temporal bone or porus is very important confirmatory evidence when otherwise one is in doubt about the presence of an angle tumor. Although it may not be of much value in determining the type of tumor, this is of lesser importance than the decision of whether or not to explore this region. Unfortunately when the tumor is small and the diagnosis is in doubt, erosion is not apt to be demonstrable.

Dyke[12] states that the erosions of the pyramid are present in about 50 per cent of patients with acoustic neuroma. Roentgenologic examination was made in 130 cases of verified tumor in Olivecrona's clinic and 106 or 81 percent were found to have some degree of enlargement of the porus acusticus with or without destruction of the pyramid[38]. Five years later this same material was again reported by Lysholm[32] but was now expanded to 236 cases and 90 per cent of them showed bone changes.

Acoustic neuromas are said to never[12] or almost never[4] calcify. We have recently seen calcification in roentgenograms of a very chronic tumor. Its presence was verified at operation.

Symptoms and Signs

Detailed enumeration of the symptoms and neurological findings which are available in any textbook of neurology will not be repeated here. In the typical case there is a chronological progression of symptoms and signs about as one would expect from involvement of the 8th nerve, compression of contiguous structures, and eventually obstruction of the cerebrospinal fluid pathways at the tentorial notch. The sequence of symptoms and signs are variable, but in general follow a pattern of 8th nerve involvement, 5th. and 7th nerve symptoms and signs, cerebellar compression, obstruction of cerebrospinal fluid and finally medullary failure.

Hardy and Crowe[25] concluded after finding 6 small asymptomatic tumors within the internal auditory canal that they do not become symptomatic until compression is produced within the internal auditory canal. It is surprising that occasionally even in large tumors there is little or no loss of hearing and even more often no demonstrable loss of vestibular function[38,41] which may be due to inability to recognize minor loss of function by the tests available. Also tinnitus commonly regarded as a most frequent and early sign is not always present. In 42 verified acoustic neuromas, tinnitus was reported by Olson and Horrax[41] as absent in 16

cases or 38 per cent. In these same 42 cases nystagmus was present in every case but even this finding is sometimes absent as Nielsen[38] found nystagmus in only 125 of his 130 cases (96%).

Order of Frequency of Cranial Nerved Involved in 130 Cases of Acoustic Neuroma (Nielsen)

Nerves Involved	No.of Cases	Per Cent
1. Acoustic (cochlear division)	129	99.2
2. Acoustic (vestibular division)	118	90.8
3. Trigeminal	92	70.8
4. Optic (diminished vision)	72	55.4
5. Facial	71	54.6
6. Abducens	16	12.3
7. Glossopharyngeal and vagus	7	5.4

Acoustic neuromas have long been considered[11] to be easily diagnosed tumors, and are, when symptoms and signs progress in the usual manner, yet there are many exceptions when there are unusual symptoms and signs, especially symptoms and signs resulting from compression of the brain stem which depend upon the direction of growth, rapidity of growth, and consistency of the tumor.

Such a case in which the proper diagnosis was suspected during life and confirmed by autopsy is illustrated by the following brief resumé of a case record.

- SIXTY-SIX YEAR OLD MAN WITH STAGGERING GAIT, DIZZINESS, DISTURBED MENTATION, NYSTAGMUS, BILATERAL LOSS OF FIFTH AND EIGHTH NERVE FUNCTION - FIFTH MORE IMPAIRED ON LEFT AND EIGHTH NERVE MORE IMPAIRED ON THE RIGHT. AUTOPSY ACOUSTIC NEUROMA ON LEFT.

On July 22, 1942 this 66 year old retired salesman was admitted because of staggering gait and dizziness.

The history was that a year previously he began to have dizzy spells and in the last 6 months, he had staggering gait, falling to the right, difficulty with speech and swallowing. He had tinnitus in the right ear for one to two years.

Examination revealed the mentation to be slow and he seemed to have some difficulty in understanding questions and commands. He had a staggering gait. The Rhomberg test was positive. There was nystagmus on extreme lateral gaze with quick component to the right. There was decreased sensation in the 5th nerve bilaterally, more on the left. Audiogram showed moderate loss of hearing for high tones in both ears, but more on the right side. Caloric tests showed normal vestibular response. The spinal fluid pressure was measured as 108 mm. and the spinal fluid had a protein content of 129 milligrams per cent. The skull roentgenograms were negative. An encephalogram was attempted but was unsatisfactory because the ventricles did not fill with air. Diagnosis of a cerebellopontine angle tumor was suggested but we were unable to determine on which side it might be. We even suggested exploration of both angles because of this uncertainty. He was finally discharged with a diagnosis of pontine and medullary involvement probably from a tumor.

He went elsewhere for examination and after very complete studies were done, it was decided that he possibly had a frontal lobe tumor and ventriculogram was suggested but not performed.

He was admitted here again on the 19th of October, 1942. During the interval since his discharge on the 9th of August, his symptoms had progressed. He was incontinent of urine. He had pain in the right occipital region with radiation to the right temporal region. The findings were essentially the same as on the previous admission, hut his mental deterioration was definitely increased. On November 4 a ventriculogram demonstrated some dilatation of the lateral ventricles and a slight elevation of the floor of the third ventricle. The interpretation was an internal hydrocephalus with elevation of the floor of the third ventricle which was thought to be due to tumor of the cerebellum or midline tumor of the brain stem. Because it was concluded that this was a tumor of the brain stem and,

therefore, inoperable, no further operation was performed. The patient's condition was not immediately changed by the ventriculogram. Five days after the ventriculogram he developed pulmonary complications and died.

An acoustic neuroma, 6x3x3 cm. in size and on the left side, plus acute cystitis and bronchopneumonia were found on autopsy. There was slight dilatation of the lateral and third ventricle but no other reason was found for his disturbed cerebration.

Comment: There was bilateral 8th and 5th nerve involvement which would suggest a pontine lesion rather than a unilateral cerebellopontine-angle lesion. There was mental deterioration without increased intracranial pressure which, no doubt, led to the diagnosis of frontal lobe tumor.

Even on review of the record it is difficult to find a preponderance of evidence in the history and neurological findings for a lesion on the left side, but the ventriculogram, if properly interpreted, should have led to a proper localization of the lesion because now on review of the films it is noted that the aqueduct and 3rd ventricle were displaced slightly to the right and the floor of the 3rd ventricle was pushed upwards which findings could only be due to a left cerebellopontine-angle tumor[31, 30, 17].

Contralateral pontine symptoms, such as occurred in this case, may be produced by displacement of the pons by an angle tumor. Hamby[24] reported two cases of trigeminal neuralgia due to contralateral tumors of the posterior fossa.

Differential Diagnosis

In differential diagnosis one must especially consider other lesions which occur in the cerebellopontine-angle and lesions of the pons and medulla. Some of them are degenerative or inflammatory and, therefore, not surgical. To illustrate how frequently the cerebellopontine-angle is explored for lesions other than acoustic neuromas the 9 operations of this type which we performed during the period covered by this report, January 1941 to January, 1948, are given in brief form below. The postoperative diagnosis in these 9 cases were 3 meningiomas and one granuloma in the cerebellopontine-angle, two tumors of the pons and medulla, one abnormal artery impinging on the 8th nerve, one trigeminal neuralgia, and one negative exploration.

Case I

This 21-year old woman noted intermittent tinnitus in the right ear beginning in January, 1941. Four months later she first noticed diminished hearing in the right ear, then headaches and stiff neck. These symptoms were soon followed by ataxia and weakness of the right side of the face. Examination revealed bilateral papilledema, decreased right corneal reflex, paresis of the right 7th and 8th nerves, and hyperactive deep reflexes.

A tentative diagnosis was made of a right cerebellopontine-angle tumor, either meningioma or acoustic neuroma. Suboccipital exploration in August, 1941 revealed a meningioma arising from the floor of the right posterior cranial fossa. It was subtotally removed. Her postoperative convalescence was uneventful.

Case 2

This 54-year old woman admitted December, 1941 had intermittent severe headaches since 1938. She had been deaf in her right ear since childhood. For 3 months prior to hospital admission she had noticed difficulty in gait and had been hyper-irritable. Examination revealed biateral papilledema, decreased right corneal reflex, total loss of vestibular and partial loss of cochlear function of the right 8th nerve, increased deep reflexes on the right, and a positive Rhomberg sign. Roentgenograms revealed erosion of the petrous portion of the temporal bone on the right.

A diagnosis was made of a cerebellopontine-angle tumor in the right, probably meningioma rather than acoustic neuroma because of the lack of history of tinnitus or vertigo and the appearance of the skull erosion. A suboccipi-

tal craniectomy in December, 1941 revealed a meningioma in the right cerebellopontine-angle. It arose from the petrous pyramid. It was totally removed.

Case 3

This 12-year old girl had recurrent episodes of headache, vomiting, and abdominal pain beginning in August, 1942. Two months later she noted vertigo, tinnitus on the right, diplopia, pain behind the right eye, and a weakness of the right side of the face. Examination in July, 1943 revealed a decreased right corneal reflex, right 6th and 7th nerve paresis, loss of both the cochlear and vestibular functions of the right 8th nerve, right 9th nerve paresis, decreased deep reflexes, and ataxia.

A diagnosis was made of a brain stem neoplasm but because of the unilateral cranial nerve involvement it was felt advisable to explore the cerebellopontine-angle. Exploration August, 1943 revealed no abnormality. She expired 4 days later. Autopsy revealed a mass of varices surrounded by minute hemorrhages and areas of gliosis located in the brain stem.

Case 4

This 50-year old woman gave a history of decreased hearing and tinnitus since December, 1943. There were no further complaints until March, 1944 when she noticed loss of skill in hand movements, staggering gait, and numbness on the right side of her face. Examination in April, 1944 revealed a decreased right corneal reflex, paresis of the right 7th nerve, paralysis of the right 8th nerve, bilaterally positive Babinski signs, and a left hemihypaesthesia.

It was thought that she had a brain stem neoplasm but because of the unilateral cranial nerve and long tract involvement a cerebellopontine-angle tumor could not be ruled out. A suboccipital craniectomy in May, 1944 revealed a glioma (glioblastoma multiforme) of the brain stem. She expired one week later.

Case 5

This forty-eight year old man began to have a buzzing noise in his right ear in 1934. In 1943 he noticed vertigo, frontal headaches, and decreased hearing in his left ear. Examination in May, 1944 revealed decreased left corneal reflex, decreased hearing bilaterally, hyperactive left knee jerk, and decreased left abdominal reflex. Roentgenograms revealed an area of decreased density in the medial portion of the lrft petrous pyramid.

A tentative diagnosis was made of a left cerebellopontine-angle tumor. On May 23, 1944 this region was explored, but no abnormality found. On the assumption that this was a pontine glioma he was given deep roentgen therapy. He was working and without complaints two and one-half years later.

Case 6

This 61-year old man first noticed deafness and tinnitus in the right ear in August, 1933. Nine months later he noticed paraesthesias in the right side of his face and a right facial paralysis. Examination revealed bilateral papilledema and paralysis of the V, VI, VII, VIII, IX, and XII nerves on the right.

A diagnosis of a cerebellopontine-angle tumor was made and this region was explored on May 21, 1945. A meningioma arising from the right side of the clivus and medial part of the petrous pyramid was found and subtotally removed. His convalescence was uneventful.

Case 7

This 68-year old man had left maxillary trigeminal neuralgia. He also had tinnitus and loss of hearing in the left ear for 20 years. Examination revealed loss of cochlear and vestibular functions of the left 8th nerve.

The possibility of an acoustic neuroma was considered. Therefore, the left cerebellopontine-angle was explored, no abnormality cound, so the root of the left trigeminal nerve was severed. He was relieved.

Case 8

. . . This 41-year old woman had attacks of tinnitus, vertigo, vomiting, and gradual loss of hearing in the right ear over a 14-year period. Examination on admission revealed a decreased right corneal reflex, past pointing to the right, nystagmus, and complete loss of both auditory and vestibular functions of the right ear.

A tentative diagnosis was made of a left acoustic neuroma. The left cerebellopontine-angle was explored, June 19, 1945. An abnormal artery curving around between the temporal bone and the 8th nerve was found and severed. The 8th nerve was also severed. She was relieved.

Case 9

T his 54-year old woman noted in 1944 a right facial paralysis that lasted for one month. In May, 1946 she noticed diplopia, dizziness, hoarseness, right facial and left leg weakness. Examination in March, 1947 revealed paralysis of the last 7 cranial nerves on the right.

A tentative diagnosis was made of a right cerebellopontine-angle tumor. Suboccipital craniectomy revealed a granulomatous tumor mass (probably tuberculosis) over the floor of the right posterior fossa.

Treatment

Surgical removal partial or complete is now recognized as the only proper type of treatment. Irradiation has been tried[1] and was still advised in the form of radon seeds in "extensive inoperable" cases in 1941[2].

Surgery in the treatment of acoustic neuromas has undergone a gradual evolution from a very crude finger enucleation in the beginning to partial removal by intracapsular enucleation and finally total removal which seems to be more or less the operation of preference with neurosurgeons today. This gradual evolution of surgery for acoustic neuromata can only be appreciated by a review of some of the literature from the beginning of surgery for acoustic neuromas up to the present time.

The first attempt to surgically remove a tumor of the cerebellopontil-angle was made by McBurney in 1891[33]. He gives in detail his technique for exposure of the cerebellum over an area of one and one-half inches in diameter with a chisel and mallet. There was great pressure, the cerebellum herniated when the dura was opened, but he says it was quite easy to introduce the finger for some distance into the skull on all sides of this protruding cerebellar hemisphere. No tumor was found. It was necessary to shave off the somewhat injured excess cerebellum in order to close the wound, as he says even this gentle manipulation practiced, had somewhat injured the delicate surface convolutions so that this removal was done with the less compunctinn. The patient died 12 days after operation and fell out of bed 6 days after operation, which, it is inferred, caused or at least contributed to the fatal issue.

At this time, 1891, and for a number of years thereafted, the signs for cerebellar localization were not well understood[35] and surgery for brain tumors was at best very unsatisfactory. Most operations were for tumors of the cerebrum in the region of the motor cortex where according to Tooth[48] and Bergmann[4a], the danger of operation was lowest. Krause[14] also considered acoustic neuroma to yield the poorest results of all brain tumors. Therefore, it is not surprising that operations on the cerebellum were considered to be hazardous, disheartening, or even unwise and according to Starr[46] it was not until about 1905 that surgeons again became less reluctant to operate for tumors of the cerebellum. Yet, one finds an occasional case report of operations for tumors of the cerebellum that were acoustic neuromas, at least an angle tumor as near as one can tell from the history, neurological changes and specimen removed at operation or autopsy. Frazier[19] in 1905 collected from the literature 116 cases of operation upon the cerebellum. The operative mortality even in this selected group (those considered worthy of publication) was

42 per cent. It is impossible to determine how many of these were cerebellopontine-angle tumors but Fraenkel and Hunt[18] in the previous year were able to collect in addition to McBurney's case, 5 others all of which presented clinical and pathological characteristics of acoustic neuroma. In addition they reported two cases of their own both of which died a few hours after operation.

Horsely[47] who operated upon more brain tumors than anyone else in this period but never published a complete report on all his cases apparently operated upon 9 cases of acoustic neuroma prior to 1904 with 3 recoveries and 6 deaths immediately after operation.

The otologists in Germany considered the results of these cerebellar operations for acoustic neuromata so unsatisfactory that they began to consider an approach with which they were more or less familiar through the petrous pyramid or labyrinth. This operation was at first merely suggested[42], later performed on a cadaver[4] and finally 4 cases were operated upon[36] but this small and deep exposure with destruction of the 7th nerve did not prove satisfactory and it was abandoned.

Cushing operated upon his first case of acoustic neuroma in January, 1906 and in the same year began a fundamental revision in the previous operative procedure when he performed an intracapsular enucleation of the growth. Finally in 1917 after operating on 30 cases, he published his monograph, Tumors of the Nervus Acousticus[9] in which the subtotal intracapsular enucleation with "cross bow" bilateral cerebellar exposure was advocated as the best procedure in treatment of these tumors. This was the first real advance in the treatment of these tumors, became the accepted type of operation for acoustic neuromas and still is the procedure of choice by many if not most neurosurgeons. Dandy[13] in 1922 reported an operation for total removal which consisted of an extension of Cushing's intracapsular removal to include total removal of the capsule after the content had first been removed with a curette. Again in 1925 Dandy[14] reported this operation with some improvements and finally in 1934[15] he described this operation again as he now performed it through a unilateral cerebellar exposure, and very careful dissection of the capsule from the brain stem. For some time after he introduced his total removal, he occasionally removed the capsule by finger enucleation after it had been evacuated, but he was now convinced that the capsule should be removed only by most careful dissection.

Olivecrona[21] began to perform Dandy's radical removal in 1931 and in 1934[39] reported an improved technique for total removal of acoustic neuromas. Beginning with Dandy's operation for total removal he had gradually made minor changes in this technique while operating upon 30 patients. His operative mortality was the same as it had been with intracapsular enucleation, and there was much less disability in the survivors. He gave up intracapsular enucleation and adopted this operation because with intracapsular enucleation most of those surviving operation were very much disabled for work and one-half of these survivors were dead of recurrences within $3\frac{1}{2}$ years; also secondary operations for recurrences were very unsatisfactory with an operative mortality of 50 per cent and extreme disability in those surviving. In contrast Cushing's postoperative mortality in 47 recurrent cases was only 3 or 7 percent following a second operation but with a third operation it rose to 44 per cent[31].

Olivecrona[40] again in 1939 reviewed his experience with incomplete and complete operative removal of acoustic tumors. His detailed results will be given later in table form but let it suffice to say here that these results give additional evidence in favor of the total removal in all cases where this is possible.

Horrax and Poppen[28] in 1939 reported that they had adopted the operation of total removal of acoustic neuromas and in the previous 4 years carried it out in every previously unoperated case.

Cairns[5] in 1939 expressed a prefer-

ence for incomplete removal but stated that this must be extensive enough to relieve the obstruction in the cisterna. Adson and Baker[2] in 1941 found it rarely possible to totally remove acoustic neuromas because they saw most patients only when in the late stages of the disease, when many adhesions had developed about the tumor. They were convinced from comparative mortality and morbidity results of radical enucleation and conservative intracapsular enucleation that the latter was the more suitable in the average case.

The present status of surgery for acoustic neuroma was presumably expressed in 1946[8] thus: "the decision as to the exact technique of removal depends on the size of the lesion and the experience of the surgeon. Complete removal is the procedure of choice if it can be accomplished with a minimum of surgical risk. This can be done if the tumor is relatively small, but large lesions are best treated by intracapsular enucleation, although occasionally they too can be completely removed11.

Results of Surgery for Acoustic Neuromata

It is not surprising that the mortality was high and the morbidity in surviving patients severe in the early finger enucleations of angle tumors. Most of these operations were presumably performed through a small exposure of the cerebellar hemisphere a little larger than necessary to admit the finger. The herniating cerebellum was traumatized by forceful displacement or transected to expose the tumor, the finger introduced and the tumor evulsed from its attachments to the structures in the cerebellopontine-angle. The brain stem was injured and packing was apparently frequently necessary for otherwise uncontrollable hemorrhage. It is well known that hemorrhage about the brain stem is poorly tolerated. Two-stage operations were most frequently performed, the first stage consisted of removal of bone or perhaps more often removal of bone and opening the dura, but even after this first stage immediate death was not uncommon.

Decompression operations have not been found to be of much value in angle tumors. but suboccipital decompression may give much relief in tumors of the cerebellum and 4th centricle. The obstruction in the cisterna at the tentorial notch which is produced by angle tumors, is not relieved by decompression. Cushing[31] prior to 1926 performed only suboccipital decompression in 13 cases with very unsatisfactory results.

The high mortality of finger enucleation is evident in the following tabulation of mortality as reported by the authors from their own experience and not from collected cases in the literature which are always weighted by an excess of favorable cases.

Mortality was immediately reduced by the intracapsular enucleation of Cushing and with increasing experience and modifications in technique it was progressively reduced until in Cushing's last 76 cases his mortality was only 8 per cent. But during this period general improvements in neurosurgical technique too numerous to describe here, were made. A few of the more important are, wide exposure (bilateral cerebellar through cross bow incision, 1905), release of intracranial pressure by tapping the lateral ventricle during operation, introduced in 1903, became routine by 1910, use of suction instead of sponges to evacuate blood and fluid from the wound, silver slips to occlude bleeding vessels (1909), and late in this period (1926) the high frequency cautery was introduced in neurosurgery. Removal of the lateral 3rd of the cerebellar hemisphere (uncapping of the cerebellum) was introduced by Frazier in 1905[19] but was not generally adopted because means for control of hemorrhage in brain surgery were not available at that time. It was adopted by Cushing in 1928 and carried out in the last 45 patients he operated upon for acoustic tumors, and with this improved exposure the cerebellar symptoms after operation were less severe, a more complete removal of the upper pole to reestablish the flow of cerebrospinal fluid in the cysterna was possible and the mortality was reduced. Dandy also very enthusiastically adopted this uncapping of the cerebellum

Results of Finger Enucleation of Angle Tumors

Author and Date of Report	No. Cases	Tumor Removed			Exploration			Decompression		
		No.	Died	Percent	No.	Died	Percent	No.	Died	Percent
Stewart J.Holmes[47] Surgeon Horsley	9	9	6	67						
Tooth 1913[48] National Hospital Cases	36	24	17	71	1	1	-	11	10	90.9
V. Eiselsberg and Ranzi 1913[16]	17	15	11	73				2	2	-
Fumola 1915[22] Surgeon Kruase	30	30	26	87						
*Revilla[43] 1947 Surgeon Dandy	6	6	4	67						

*This is taken from Dandy's material including his early cases. Also another fourteen were removed by combination of intracapsular and finger enucleation with seven deaths.

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when he introduced his operation of unilateral exposure in 1934, but Olivecrona[38] finds it unnecessary.

Mortality was reduced but morbidity remained high after intracapsular enucleation so that few were able to lead a useful life for long after operation. At least in some of the earlier cases in Cushing's intracapsular enucleation he did not remove enough tumor, especially the upper pole to reestablish the flow of cerebrospinal fluid making it necessary to reoperate upon the patient again in the immediate postoperative period for the removal of more tumor. Twenty-six of his patients had secondary operations of this type.

Davidoff concluded from the results in 40 of Cushing's cases operated on between 1924 and 1927 that short of total removal the duration of life and degree of improvement varied directly with the amount of tumor removed. List[31] also found that outside of total removal those lived longest who had almost total removal. There are exceptions, however, in which some of the longest survivals follow incomplete removal because of the slow growth of the tumor. This long survival in the exceptional case with incomplete removal has been noted also by both List[31] and Olivecrona[40].

Intracapsular Enucleation

Surgeon	Date	No. Cases	Died	Percent	Died Later	Avg. life of those dying later
Cushing	1917	29	6	21		
Cushing	1932	176	26	15	34	3 yrs. 1 mo.*
Cushing	last 76 cases	76	6	8		
Olivecrona	1934	34	9	28	12(36)	$3\frac{1}{2}$ yrs.
Olivecrona	1940	55	14	26	5(9)	

*Only 125 were followed and those living had survived an average of 4 yrs. 10 mo.

Total Removal

Surgeon	No. Cases	Died (%)	Seventh N. Preserved
Dandy	117	16(14)	9 cases
Dandy in last eight years	62	4(7)	
Olivecrona	75	14(19)	
Horrax & Poppen	19	2(11)	

All authors reporting total removals[31,40,43] have noted that recurrence is very rare after "total" removal, and Olivecrona finds much less disability due to cerebellar dysfunction. If, therefore, a comparative mortality can be obtained with total removal the only disadvantage in the total removal is paralysis of the 7th nerve which seldom follows intracapsular removal but almost invariably followed total removal as this operation was first performed. It is true that there were occasional cases in which total removal was performed with preservation of the 7th nerve[6] but until recently no systematic attempt has been made to preserve the facial nerve. Beginning in January, 1937 Olivecrona[38] attempted to save the 7th nerve in every case and up to June, 1939 he had done 23 complete extirpations with anatomical preservation of the nerve in 15 or 65 per cent. In all except one of these cases there was a paralysis of the facial nerve immediately after operation but some recovered in 3 months to one year. According to Frykolm[21] who reported on this same material after 5 years' follow-up only 50 per cent of the nerves anatomically preserved recovered good function but most of the remainder of those anatomically preserved had reduced function and nerve anastomosis was carried out in only 10 per cent.

The University Hospitals material consisting of 20 cases is reviewed in the following tables. These cases have been treated too recently to make follow-up studies worth while. We are especially interested in the mortality in the total removals since this operation seems to us to be the preferable procedure in most cases, even though an occasional one, especially those with extensive erosion of the petrous pyramid may always have to be subtotally removed. With increased experience it should be possible to save the facial nerve in more cases and to do so it may be necessary, as recently suggested[38], to employ local anaesthesia so contractions in the face can be more easily produced when the nerve is endangered. It has been our custom to perform hypoglosso facial anastomosis in those cases in which the facial nerve is destroyed, but we have been able to save the 7th nerve in 3 of the 14 cases, and 2 of those saved have good 7th nerve function.

The senior author has done these 14 removals in every case he has operated upon since this procedure was begun. One death was due to meningitis which could equally as well follow a less radical operation; another was due to total removal to include 2 tumors which had eroded entirely through the temporal bone. It was impossible to totally remove the tumor but in attempting to do so the medulla was injured. The fatal issue in the 3rd case was possibly also due to injury of the brain stem but even after autopsy this was not clear.

Summary

Acoustic neuromata are as a rule easily recognized and can be removed with a minimum mortality and morbidity if operated upon before they become adherent to structures in the angle, eroded petrous pyramid, and projected through the tentorial notch. Lost vision is not restored. But the majority of them still come for operation late in the evolution of the disease.

The history of the surgical treatment of acoustic neuromata can be divided in-

UNIVERSITY OF MINNESOTA 1941 to 1948
20 Acoustic Neuromas

Six	
Male	7
Female	13

Side	
Right	9
Left	9
Bilateral	2

Classical Symptoms

	N. V	N. VIII			Cerebellum	Pressure Signs		
	pain or numb	Hearing loss	Tinnitus	Vertigo	(unsteady gait)	Headache	Vomiting	Vision
Present	6	18	9	11	14	13	4	10
Normal	7	0	3	1	1	4	8	5
No Note	7	2	8	5	5	3	8	5

Summary of Physical Signs

	Papilledema	N.V. Hypesthesia		N.VII Paresis	N.VIII Loss		Cerebellum	
		Corneal	Face		Auditory	Vestibular	Nystagmus	Incoordination
Positive	13	16	10	9	19	16	13	16
Normal	6	4	10	11	1	1	6	4
Not Recorded	1					3	1	

	Number	Deaths	VII N. Saved
Total	14	2(14%)	3
Intracapsular	4		
Reoperation	1	1	
No Angle Surgery	2	2	

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to three stages: 1. Finger enucleation through a small unilateral cerebellar exposure, with extremely high mortality, 67 to 87 per cent; 2. Intracapsular enucleation of Cushing with very much reduced postoperative mortality, 8 to 28 per cent, but recurrence in approximately 50 per cent of the survivors within 4 or 5 years; 3. Total removal of Dandy with a postoperative mortality, 7 to 19 per cent which is similar to intracapsular enucleation. The extent to which this operation has been adopted by neurosurgeons is still uncertain since those who have expressed an opinion are not consistently in favor of it for the average case.

With experience it would appear that total removal could be done in most cases with a mortality very similar to that of intracapsular enucleation and preservation of the facial nerve in many, but the exceptional case will probably continue to be best treated by intracapsular enucleation.

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