THE NEUROSURGICAL ASPECTS OF SEVENTH NERVE NEURILEMMOMA*

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In 1935, Altmann, in a paper on neurogenic tumors of the descending part of the facial nerve, described the characteristic symptoms of this condition. He reviewed the cases reported by Schmid and Schroeder, and included as one of the cases in his own paper a patient reported previously by Grossmann and Leidler. It is not necessary to go into the details of the symptomatology in each of the 4 cases presented by Altmann, or of the 2 cases reported previously, but it is interesting to outline the symptom complex that Altmann attributed to these tumors as they developed within the facial canal. The first symptom in all cases was a facial paralysis. In some instances it developed gradually, progressing in a step-like fashion from lower to upper branches of the nerve. In other cases it appeared suddenly, as does Bell’s palsy. In general, this paralysis, once developed, was persistent and unchanged, but occasionally a partial remission was seen.

In the cases in which taste was tested there was much variation. In 1 it was totally absent, in 2 it was diminished and in 1 it was undisturbed. Obviously this depended upon the site of origin of the tumor, for if the lesion began distal to the branching off of the chorda tympani, taste could be retained. If it began proximal to this point, taste was lost, and if the chorda tympani was only involved by pressure as the tumor grew, taste would be progressively altered.

A careful examination of the ear usually disclosed a mass of tumor in the external auditory canal. It seemed to arise from the posterior wall, and would obstruct the canal, either partially or completely. Although in the early stages the mass was covered by intact epithelium, later there was loss of epithelium, and ulceration, because of retained matter within the canal. In some cases the drum was intact. Eventually, however, as the middle ear was filled by tumor, the mass might erode through the drum. This almost always led to secondary infection with chronic otitis media. Indeed, in most of the cases in the literature, it was this sequence of events that led to the eventual diagnosis. Because of the persistent infection, mastoidectomy was done and the tumor was discovered during the surgical procedure.

In the beginning, hearing was normal. Later, it would be lost, either because the tumor obstructed the canal, filled the middle ear, or contributed to the persistent secondary infection. Exactly the same situation applied

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as regards reaction to caloric stimulation. Altmann concluded that these tumors could cause intracranial complications by either the extension of tumor into the cranial cavity, or the development of meningitis or brain abscess, secondary to the induced infection. He felt that the only satisfactory treatment was surgery, for in 1 of his cases irradiation was useless, and this was to be expected, for neurinomas as a class had not responded to radiotherapy. He also determined that the facial paralysis would remain unless the lesion could be removed at an early stage before total atrophy of the facial muscles had developed.

In 1953 I saw for the first time a patient having such a lesion.

Case 1. P.R., a white male aged 48, a resident of Indiana, was seen on May 18, 1953. In 1940 a peripheral facial paralysis began to develop which came on gradually and first involved the superior portion of the face, with decreased to finally absent blinking, and smoothing out of the forehead. Eventually the paralysis became complete. In 1942 he was seen at the Mayo Clinic, but nothing of significance was found except for the right 7th nerve palsy. The Department of Otolaryngology reported “negative” studies, and there was no involvement of the 5th nerve. The presumptive etiology was pressure neuritis, caused by the patient’s long established habit of sleeping with the right shoulder wedged into the right side of his face. A change of sleeping posture was recommended. He carried on his work as an automobile salesman without difficulty except for gradual loss of hearing in the right ear, which began in 1950 and was complete by 1952.

In December 1952, while applying warm compresses to the exposed right eye, he was conscious of “whirling spots of bright colors,” and that night during sleep he had a generalized convulsion. When he regained consciousness he was in an oxygen tent in a local hospital. Since that time, in spite of anticonvulsant medication, he had had five other convulsions, and during the last attack he dislocated his left shoulder. In January 1953, while talking to a client, he suddenly got up without any reason and left the client. The next thing he remembered was being in his car driving home. He had an uncontrollable desire to void, was incontinent of urine, and arrived home with wet clothes but no other reasonable excuse for being there. His wife also volunteered that for several months he had been moody, irritable, careless in business and in dress, and uninterested in his home.

Examination. He had no pressure signs and no involvement of the 5th nerve. There was an obviously long-standing paralysis of the 7th nerve on the right with eversion of the lower lid and pronounced sagging of the right face (Fig. 1). There were no significant motor, sensory or reflex changes, and there was no evidence of cerebellar dysfunction. He had several small café-au-lait spots on his back but no palpable neuromata.

Audiometer tests showed slight remnants of hearing in the right ear, and normal hearing in the left. There was no reaction to caloric stimulation on the right except very slight vertigo after 4 minutes of 68°F water. His visual fields were normal.

The electroencephalogram was interpreted as mixed fast and slow dysrhythmia, nonfocal, consistent with convulsive disorder. Radioactive isotope localization was said to indicate a right cerebellopontine angle tumor. However, roentgenograms of the skull showed a slight displacement of the pineal to the left, and a thin, curvilinear calcification extending up into the middle fossa, suggesting a large globular mass in
this area (Fig. 2). Until the roentgenograms were seen, in spite of the absence of involvement of the 5th nerve, an angle tumor was suspected. The films, however, raised the possibility of two lesions, so angiography was done (Fig. 3). The right middle cerebral artery was displaced anteriorly, superiorly, and medi ally.

Operation. On April 23, 1953 the middle fossa was explored. The temporal bone was so thin that it was fractured with a periosteal elevator. A low flap was reflected. The very tight dura mater was controlled by ventricular puncture and release of spinal fluid. The Sylvian fissure was found pushed upward for at least 3 to 4 cm. A very thin layer of temporal cortex covered a tennis ball-sized tumor, of firm, rubbery consistency. The interior of the tumor was yellowish in color with an occasional small cyst. Around the periphery of the tumor were several areas of encapsulated yellow fluid in the arachnoid such as is seen over an 8th nerve tumor. With intracapsular enucleation, the tumor was gradually collapsed down to a point of firm attachment along the midportion of the petrous bone. The medial margin of the tentorium was split, but there was no evidence of tumor in the cerebellopontine angle, so the dura mater around the point of attachment was sectioned, and the tumor was lifted out of a cavity which was thought to be the inner ear. With a chisel, the bone around the edge of the area of tumor extension was removed and

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**Fig. 1. Case 1.** Long-standing facial paralysis with muscular atrophy and eversion of lower lid.

**Fig. 2. Case 1.** Roentgenogram showing thin curvilinear calcific line suggesting mass in middle fossa (retouched).
Fig. 3. Case 1. Angiograms showing displacement of middle cerebral artery anteriorly, superiorly, and medially.

the site was covered with Gelfoam. The dural flap was resutured, no decompression being done.

Postoperative course was uneventful and the patient was discharged with the diagnosis of verified neurilemmoma of the right middle fossa, 7th or 8th nerve tumor.

Pathologic Report. The tumor weighed 25 gm. Microscopic sections "show a tumor formed by intermingled bundles of fusiform cells (Fig. 4). Verocay's bodies are present. There are a large number of foam cells in the tumor (Fig. 5). The underlying bone shows fibrosis of the marrow cavities and invasion by the tumor. No mitotic figures are seen."

Subsequent Course. He continues to have an occasional convulsion, always nocturnal, but he takes only 1 gr. of phenobarbital daily. He still has normal facial sensation and a deaf right ear, but there are no other abnormal neurological findings except for the unchanged facial paralysis. Until November 1958 he was working regularly and feeling quite well. At that time, however, a routine roentgenogram of the chest disclosed a mass in the upper lobe of the right lung. He was re-admitted to Barnes Hospital and a pneumonectomy was done by Dr. Thomas Burford. His postoperative convalescence was uneventful, and at the present time his condition is good. The tumor removed was bronchogenic carcinoma.

In searching for details of a similar case, I found a paper published in 1936 by Tremble and Penfield, on the operative exposure of the facial canal with removal of a tumor of the greater superficial petrosal nerve.

Their patient was a 42-year-old man who had had facial paralysis of 5 years' duration. The paralysis had become complete over a period of 1 year. He had been seen and treated at the New York Neurological Institute with electrical stimulation. Two years after onset of the facial paralysis, tinnitus developed, and gradually the hearing in the left ear became reduced.

The outstanding findings on neurological examination were paralysis of the left
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Fig. 4. Case 1. Photomicrograph of tumor in the region where it extended into bone. The cells have elongated, slightly irregular nuclei which are arranged parallel to one another and form palisades, so common to these tumors. Hematoxylin and eosin, X220.

Fig. 5. Case 1. This photomicrograph demonstrates the large foamy cells with eccentric nuclei which were present in the tumor. Their cytoplasm was filled with sudanophilic material. Numerous large pale cells of this type interspersed among elongated cells similar to those shown in Fig. 4 made up the histologic pattern of a major portion of the tumor. Hematoxylin and eosin, X400.
face, of a peripheral type, with occasional involuntary twitchings around the corner of the mouth. Studies made by Dr. Erickson, who was then the resident on that service, showed that taste was decreased over the left anterior part of the tongue. The parotid secretion was normal, but there was decreased secretion from the submaxillary and sublingual glands, and decreased lacrimal secretion on the left. The Weber's sign was referred to the left, hearing was decreased on the left, more for high tones, and caloric stimulation gave a sluggish reaction on the left.

At operation, using a radical mastoid approach, the facial canal was opened. Four mm. from the bend, a tumor of the nerve which had grown into the middle fossa through the bone to the dura mater, apparently following the canal of the greater superficial petrosal nerve, was found. As much of this tumor as possible was removed but the dura mater was not disturbed because of potential infection. The Eustachian tube was curetted so that it would close if re-operation became necessary.

From a pathological standpoint this tumor was diagnosed as perineurial fibroblastoma and was thought to be the first case of a perineurial fibroblastoma arising from the greater superficial petrosal nerve.

The point was stressed in the article that in cases of facial nerve paralysis clinical tests should aid in deciding the exact point of damage to the nerve, and it was thought that in this instance there was no question but that the chorda tympani was involved.

In 1950, in the proceedings of the Staff Meetings of the Mayo Clinic, Love9 reported the following case.

A 16-year-old girl had had deafness of the right ear for 8 years, and facial paralysis for 2 years. No statement was made as to the completeness of the deafness or facial paralysis, and no caloric reactions were reported. The physical findings were described as being normal, so obviously no tumor was seen in the ear. The roentgenograms showed a destructive expanding lesion in the tip of the petrous apex, this finding suggesting an intrapetrous tumor.

Because of the possibility of an angle tumor, however, a right suboccipital craniotomy was done first, with negative results. The middle fossa was then explored, using an incision as for trigeminal neuralgia. The exposure, however, was farther posterior, and the middle meningeal artery was not ligated. An expansion of the medial one-half to one-third of the medial portion of the petrous bone was seen, and the roof of this expanded area was removed with a chisel. A firm fibrous neuroma was exposed, and it was completely removed, the tumor being entirely within the petrous bone. Neither the 7th or 8th nerve was seen.

There was, however, no improvement in the facial paralysis, so a spinofacial anastomosis was done subsequently, and, after several months, improvement was noted.

It was presumed by Love that this was an 8th nerve tumor, but it was unique in his experience, and no other case was known in which a neurofibroma had arisen in the petrous portion of the temporal bone. As a preoperative diagnostic possibility, Love had considered an intratemporal epidermoid, for Jefferson and Smalley7 in 1938 had reported 6 cases of progressive facial palsy caused by an epidermoid lodged within the temporal bone. In
each case, paralysis had taken several months to develop, and in that respect
was totally unlike Bell’s palsy. However, the intact drum, absence of pain,
and lack of sepsis had in some cases led others to consider obscure neuritic
processes as the cause of the facial paralysis. In all cases there was a definite
change in the bone radiologically, with a clearly defined cyst-like space with
an even capsule of condensed bone, usually seen best in the Towne view.
All of these were true embryonic epidermoids and not the cholesteatoma so
frequently seen with infections of the middle ear.

With these facts in mind, the second patient, G.L., was evaluated.

Case 2. A white male, also from Indiana, an insurance agent aged 44, was ad-
mitted May 3, 1958. He had served in the Navy during World War II and had from
time to time been exposed to the noise of five-inch guns. Occasionally he had been
temporarily deafened by this noise. In 1946, after discharge, weakness of the right
side of his face gradually developed, and in 1947 he went to the Mayo Clinic. At
that time he had a right facial paresis, decreased taste on the right anterior portion
of his tongue, decreased hearing in the right ear, and no other abnormal neurological
findings. Roentgenograms of his skull were negative, and no specific diagnosis was
made.

His condition remained essentially unchanged until 1950 when he was struck
over the right side of his face with a baseball bat. His jaw was fractured, the cornea
was lacerated, and this produced permanent visual impairment. In 1953 he had
several dizzy spells, and again returned to the Mayo Clinic. There had been no ap-
parent progression, and the diagnosis of a cerebellopontine-angle lesion could not be
made, so he was again sent home. There was no recurrence of dizziness. He con-
tinued to work regularly until 9 days prior to admission, when he had a nocturnal
convulsion with incontinence. This was followed by a short period of combative be-
havior, and he was hospitalized. Spinal puncture done in the local hospital showed
increased spinal fluid pressure and an increase in spinal fluid protein, so he was
transferred to Barnes Hospital.

Examination revealed a dilated and irregular right pupil with a corneal scar (the
result of an old injury), a long-standing peripheral facial paralysis on the right (Fig.
6), and a deaf right ear. There were no other abnormal findings. Audiometry showed
a totally deaf right ear, but caloric tests showed reactions on both sides. Dr. T. E.
Walsh found a mass in the middle ear, bulging against the posterior edge of the
tympanic membrane. Rinne’s test was negative on the right. Spinal puncture showed
clear fluid with a pressure of 190 mm. of water, and a total protein of 143 mg. per
cent.

The electroencephalogram was reported as follows: “Record establishes convul-
sive character of past seizures, we believe, but contributes little to the localization
of the disorder; however random slow spikes that appear suggest organic etiology.”

Roentgenograms of the petrous bone of the skull were reported as negative. It
was the consensus of opinion that this man had a 7th nerve tumor which had eroded
through the bone and extended into the middle fossa.

Ventriculography was done on May 8, 1958 (Fig. 7), the ventricles being of nor-
mal size but displaced $\frac{1}{2}$ cm. to the left. The anterior portion of the temporal horn
was elevated; there was a curvilinear collection of air beneath the temporal horn,
and this was interpreted as being air over a tumor arising from the floor of the middle
fossa (Fig. 8).
Fig. 6. Case 2. Facial paralysis of long duration.

Fig. 7. Case 2. Ventriculogram showing displacement of ventricles to the left, and air over tumor.

Fig. 8. Case 2. Elevation of temporal horn on the right with air over tumor beneath temporal horn.

1st Operation. Immediate craniotomy was carried out. A low temporal flap was reflected, pressure being controlled by removal of ventricular and spinal fluid, and a large smooth tumor, easily separated from the brain, was removed from the middle fossa. It was yellowish in color, contained a few cysts, and was firmly attached to about the midportion of the petrous portion of the temporal bone. The dura mater was split around the point of attachment, and some adjacent dura mater which was invaded by tumor was removed, and additional tumor was curetted out of the cavity which was thought to be the middle ear. For radiographic identification, a clip was placed in the opening in the bone, and the wound was closed.
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Course. Except for several convulsive seizures in the immediate postoperative period, his course was satisfactory. He was discharged on May 21, 1958.

Pathologic Report. The tumor weighed approximately 10 gm. and was reported as neurilemmoma of typical appearance (Figs. 9, 10 and 11).

Course. The patient did well and was able to resume the major part of his regular duties. During the middle of July, he was re-admitted to Barnes Hospital.

Fig. 9. Case 2. Photomicrograph of neurinoma of 7th nerve. The histologic picture is that of bands of cells with eosinophilic cytoplasm and a tendency to palisading of the elongated nuclei. Some of the cells are arranged in loose whorls, as is seen in the upper portion of the figure. Hematoxylin and eosin, ×150.

2nd Operation. On July 15, 1958, using an endaural approach, the mastoid was opened by Dr. Walsh. In the antrum tumor could be seen extending from the middle ear. The tumor filled the middle ear, so the drum, malleus, and the small remnant of the incus were removed. No remnant of facial nerve could be seen. Just anterior to the usual location of the geniculate ganglion a large hole in the bone contained tumor. When this mass of tumor was removed, the silver clip, previously placed there, was found to be attached to it. All of the tumor was removed from the middle ear and the mastoid, and Oxycel was packed into the dehiscence in the middle fossa. The rest of the cavity was packed with iodoform gauze. It was Dr. Walsh's opinion that this tumor had arisen from the facial nerve, and that the entire nerve had been replaced by tumor in its intratympanic course.

Pathologic Report. Sections of the tumor were identical with those removed from the intracranial cavity.

Course. Following this procedure, the patient was placed on antibiotics because of the drainage of cerebrospinal fluid from the ear. With some misgivings, he was discharged on the 11th postoperative day to the care of a competent otologist for dressings and supervision.
Fig. 10. Case 2. Electron micrograph of characteristic cells in same tumor as shown in Fig. 9. The cells have prominent nuclei (N) of irregular outline. The cytoplasm is profuse and has a characteristic arrangement of complicated folding and interdigitation. A distinct basement membrane is usually evident (arrows). These cells are considered to be neoplastic Schwann cells. The presence of a basement membrane and the complexity of the cytoplasmic indentations set them apart from fibroblasts. ×8000.

Exactly 1 month later he was re-admitted to Barnes Hospital with fever and stiff neck. His spinal fluid contained 200 cells, 90 per cent of which were polymorphonuclear. He was put on intensive antibiotic coverage; spinal fluid cultures were reported as negative. When the packing gauze was removed from the ear, the drainage stopped, but he became drowsy. Roentgenograms were made (Fig. 12) and these showed a large spontaneous pneumocephalus with a fluid level.
Fig. 11. Case 2. Electron micrograph of one of the rare fibroblasts present in the tumor shown in Figs. 9 and 10. It is an elongated cell with an ovoid uniform nucleus. The cytoplasmic outline is smooth. A basement membrane is absent. Scattered collagen fibrils are evident. At the upper right is a part of the cytoplasm of an adjacent neoplastic Schwann cell. X8000.

3rd Operation. On Sept. 6, 1958, the bone flap was again elevated. About 50 cc. of air and 40 cc. of fluid were aspirated from a cavity in the temporal lobe. The cortex over this cavity was only a few mm. in thickness, and when it was opened, there was a point of adherence in the midportion of the petrous bone. The edge of the bone was freed, and an attempt was made to mobilize and suture the dura mater over this opening. This was impossible, however, so the opening in the bone was occluded with crushed temporal muscle, a piece of temporal fascia was incised and sutured to the dura mater over the muscle, and the wound was closed.

Fig. 12. Case 2. Spontaneous pneumocephalus with fluid level.
Postoperative Course. His recovery was prompt and smooth with no further drainage. There was radiologic evidence of clearing of the pneumocephalus.

Pathologic Report. The tissue removed was reported as focal fibrosis with no evidence of tumor.

Subsequent Course. The patient has resumed his former activities, is feeling quite well, but is still taking anticonvulsant medication. He has had no more convulsions and is working regularly.

Actually the first case of neurinoma of the facial nerve was reported by Schmid in 1930. This was a 16-year-old girl who had a long-standing facial paralysis and a mass of tumor on the posterior wall of the external auditory canal. Mastoid exploration revealed a cavity the size of a bean which was filled with masses of granulation-like tumor. The facial nerve was exposed in the descending portion at the bottom of the cavity, and the pathological diagnosis on the tumor removed was neurinoma arising from the sheath of the facial nerve.

In 1931 Schroeder reported the case of a 25-year-old male who had had a facial paralysis for 8 years with chronic purulent otitis media for 4 years. He had had two mastoid operations, and at the second operation tissue removed was found to be neurinoma. At the third operation a large mass of tumor filling the middle ear and mastoid cavity was found and removed, and the patient was given roentgen-ray treatment. One year later, a small fistulous opening was injected with Lipiodol and the fistula was said to extend into the frontal lobe of the brain. The deafness and facial paralysis persisted. Further history is given on this patient in an interesting footnote in a case report of a 7th nerve tumor by Kettel in 1946. He stated that the patient reported by Schroeder in 1931 was still alive but was unable to work because of "fits of fainting and fury." On four occasions he had been in mental hospitals, but the fistulous opening had finally closed and the facial paralysis was still complete.

In 1936 Greifenstein reported a 7th nerve neurilemmoma discovered at autopsy on a patient who had died following operation for a pituitary tumor. There was no preceding history of facial paralysis or deafness. In 1937 Altmann reported an additional case and again reviewed the clinical syndrome. He advocated preliminary biopsy in all cases, and pointed out that the tumors are of slow growth but could, by expansion and erosion, give rise to middle-ear or intracranial complications. An additional case was reported during that year by Ohnishi.

In 1939 Williams and Pastore reported the first case in American literature:

A 59-year-old man had a left facial paralysis which had developed, over a period of 1 month, 4 years previously. Five months before he appeared at the Mayo Clinic pre-auricular and mastoid pain had developed with increasing deafness. His local otologist removed a "polyp" from the external auditory canal, but the symptoms were not relieved and the polyp recurred.

On examination he was found to have a complete left facial paralysis with
atrophy of the facial muscles. The left ear was deaf and a polypoid mass filled the external auditory canal. There were no other abnormal neurological findings. Roentgenograms were reported as showing an area of irregular destruction of the bone in the left mastoid in an area overlying the lateral sinus.

The polypoid mass in the external canal was biopsied and reported as "inflammatory ulcer and granulation tissue." A radical mastoidectomy was done, and a yellowish tumor extending into the facial canal was found and removed. The patient was given 12,000 mg. hours of radium. The facial paralysis persisted.

Additional cases have been reported by Rejtö\textsuperscript{13} and by Vysotskaya,\textsuperscript{18} although these are not available to me.

In 1943 the literature was reviewed and an additional case was reported by Roberts.\textsuperscript{14} His case was unusual in that the facial paralysis had been present for 30 years, deafness for 20 years, and there had been purulent drainage for 15 years. The patient, a male, was operated upon actually because of the long-standing infection, but when the mastoidectomy was done, a neurilemmoma which had eroded bone and become attached to the dura mater was found and removed. Recovery was good except for the persistent facial paralysis and deafness.

Additional cases have been reported by Bogdasarian,\textsuperscript{3} Kettel,\textsuperscript{8} and Lundgren.\textsuperscript{10}

In 1953 Collins and Thomson\textsuperscript{4} reported a case in which a tumor was excised, the tumor extending from 4 mm. proximal to the bifurcation of the nerve in the parotid gland to within 3 mm. of the genu. The bony roof of the canal was opened and the tumor was removed, and a graft was sutured to the divided ends of the nerve. The graft was successful, facial function reappearing in 8 months, and the ultimate result was excellent, as shown by photographs.

The most recent report is by Pou,\textsuperscript{12} who recorded 2 cases. One is most unusual, for this patient had had an acoustic neurilemmoma removed 6 years previously with a persistent facial paralysis since that time. The 7th nerve tumor presented within the auditory canal and in the neck. The tumor, which filled the mastoid, middle ear, and extended into the neck in the region of the parotid gland, was removed. The facial paralysis was unaltered, but the ear and a fistulous opening in the neck ceased to drain.

It seems almost unbelievable that the subject of 7th nerve neurilemmoma has received such scant mention in neurologic or neurosurgical literature. It is a relatively rare tumor but a total of at least 22 cases are recorded in otologic publications, and the possibility of intracranial extension has been recognized. In its early stages it is certainly a problem for the otologist, but neurologists should recognize it as a diagnostic possibility, for only then will the early cases be referred for surgery so that complete cure can be obtained. Cure should consist not only of eradication of tumor, but this should be accomplished at a stage early enough to permit the facial paralysis to be corrected. In some cases this may be done by nerve graft, in others by nerve anastomosis, but in the late stages, when total atrophy of
muscles has occurred, neither will be corrective. I think it would be advisable to urge exploration by a competent otologic surgeon in all cases of facial palsy which persists for as long as 6 months, and this is even more imperative in patients in whom the paralysis comes on gradually. It is, therefore, to be hoped that all cases will be diagnosed and the tumor removed while the lesion is still confined to the middle ear, or middle ear and mastoid. If, however, as in the 2 cases here reported, such a diagnosis is not made, one should be suspicious when the patient with a long-standing facial paralysis begins to have either convulsive seizures or change in personality. Since the lesion is very slowly growing and because there is considerable available space in the middle fossa, pressure signs will occur late in the course of the disease. If such a lesion be suspected, the diagnosis may be confirmed by either angiography or ventriculography, but if the latter procedure is used, films to outline the temporal horns may be very valuable.

SUMMARY

Two cases of middle-fossa extension of 7th nerve neurilemmoma are reported. A peripheral facial paralysis, usually of gradual development, is the first symptom of such a tumor. Subsequently loss of hearing and secondary infection may occur. Intracranial complications may result from extension of tumor or extension of infection. Such a lesion should be suspected when the patient with a long-standing facial paralysis has development of either convulsions or change in personality. Ideally, the lesion should be diagnosed and treated at a stage early enough to forestall intracranial complications and to permit the correction of the facial paralysis.

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REFERENCES