The facial nerve in medial acoustic neuromas

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Object. Functional results after surgery for acoustic neuromas that have little or no growth within the internal auditory canal are controversial, because these medial tumors can grow to a considerable size within the cerebellopontine angle (CPA) before symptoms occur.

Methods. A prospective study was designed to evaluate the surgical implications of the course of the facial nerve within the CPA on medial acoustic neuromas. This study included a consecutive series of 22 patients with medial acoustic neuromas (mean size 32 mm, range 17–52 mm) who underwent surgery via a suboccipitotemporal approach between 1997 and 2001. All patients underwent pre- and postoperative magnetic resonance imaging and preoperative electromyography (EMG). Evaluation was based on continuous intraoperative EMG monitoring and video recordings of the procedure. All patients were reevaluated at a mean of 19 months (6–50 months) postsurgery.

Preoperative evaluation of facial nerve function revealed House–Brackmann Grade I in six, Grade II in 14, and Grade III in two patients. During surgery a distinct splitting of the nerve at the root exit zone through its intracisternal course was seen in eight patients and documented by selective electrical stimulation. The facial nerve was separated into a smaller portion that ran cranially and parallel to the trigeminal nerve, and a larger portion on the anterior tumor surface. Both components joined anterior to the porus without major spreading of the nerve bundle. In two cases the nerve was found on the posterior surface of the cranial tumor. In one case the facial nerve entered the porus of the canal at its lower part, obtaining the expected anatomical position proximally within the middle portion of the canal. An anterior cranial, middle (five cases each), or caudal course (two cases) was seen in the remaining patients. After surgery, facial nerve function deteriorated in most cases; on follow-up evaluation House–Brackmann Grade I was found in 11, Grades II and III in 10, and Grade V in one patient.

Conclusions. The facial nerve requires special attention in surgery for medial acoustic neuromas, because an atypical course of the nerve can be expected in the majority of cases. A split course of the nerve was found in 36% of the cases presented. Meticulous use of intraoperative facial nerve stimulation and continuous monitoring ensures facial nerve integrity and offers good functional results in patients with medial acoustic neuromas.

KEY WORDS • medial acoustic neuroma • internal auditory canal • facial nerve • intraoperative electrophysiological monitoring

Clinical Material and Methods

This prospective study involved a consecutive series of 22 patients with medial acoustic neuromas who underwent surgery via a suboccipitotemporal approach between 1997 and 2001. Histological diagnosis was confirmed in all cases. All patients fulfilled the neuroimaging criteria for a medial acoustic neuroma. Tumor growth inside the canal did not exceed 2 or 3 mm lateral to the porus, based on axial and coronal T1-weighted MR images obtained with Gd enhancement. In all patients the lateral part of the canal was free of tumor (Figs. 1 and 2). Patients underwent preoperative electrophysiological facial nerve and audiometric studies, including evaluations of pure tone average and SDS. Intraoperative monitoring was performed using continuous facial nerve EMG based on three-channel recordings of the muscles orbicularis oris, musculus nasalis, and musculus orbicularis oculi, the details of which have been published recently. Brainstem auditory evoked potentials were monitored in patients in whom hearing preservation was attempted.

All patients underwent surgery via a suboccipitotemporal approach, after being placed in a modified prone position,
except for one patient with a giant tumor who underwent surgery in a semisitting position. Osteoplastic trepanation was performed in four patients. The IAC was opened in all but four cases.

The surgical procedure was videotaped for detailed off-line analysis of the course of the facial nerve. All patients were reevaluated between 6 months and 4 years postsurgery (mean 19 months), including audiometric testing and MR imaging. Facial nerve EMG was performed in selected cases.

The mean age of the nine men and 13 women at time of surgery was 50 years (range 30–78 years). The mean extra- and intrameatal tumor size was 30 mm in axial diameter, ranging from 18 to 50 mm. Intrameatal extension of tumor as documented with axial and coronal MR imaging ranged from 0 to 3 mm, with a mean extension of 2 mm. All patients except one underwent primary surgery; 16 years earlier, this patient had undergone surgery for a medial acoustic neuroma, which was subtotally resected. Facial nerve function was graded using the House–Brackmann scale, with Grade I in six, Grade II in 14, and Grade III in two patients. Patients with preoperative evidence of facial nerve neuropathy only on EMG monitoring were categorized as Grade II (Table 1).

Hearing levels were classified according to the guidelines of the Committee on Hearing and Equilibrium for the Evaluation of Hearing Preservation in Acoustic Neuroma of the American Academy of Otolaryngology–Head and Neck Surgery Foundation. Nineteen patients had cochlear nerve function preoperatively (Class A in seven, Class B in four, Class C in two, and Class D in six). Other cranial nerve deficits included a fifth cranial nerve hypesthesia in eight patients, lower cranial nerve deficits in three patients, and a sixth nerve paresis in one patient. One patient suffered from a trigeminal neuralgia.

**Results**

Tumor removal was complete in all but six patients. In four patients small capsule remnants adherent to the facial nerve (maximum 2 × 3 mm) were left behind, which could not be confirmed on follow-up contrast-enhanced MR images in two cases. In one patient with a giant 5-cm tumor the IAC was not opened, leaving a small tumor remnant lateral to the porus (Fig. 2 lower right). In a 78-year-old patient suffering from trigeminal neuralgia, a considerable tumor capsule remained on the brainstem and on the facial nerve. There were no deaths in the study participants. Postoperative complications occurred in four patients (meningitis in two patients, pneumonia in one). In one patient a severe ataxia developed 1 week postsurgery, which could be attributed to a venous infarct within the cerebellar peduncle as documented on MR imaging. The patient’s symptoms have improved; however, there is still a moderate disability. 3 years after the surgical procedure. Review of the videotape revealed no evidence of damage to the middle

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**TABLE 1**

Facial nerve outcome in 22 patients with medial acoustic neuromas

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<tr>
<th>HB Grade</th>
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* Early postoperative functional fluctuation in nine patients is indicated by arrows (a 2 over the arrowhead means that there were two patients). All patients were reevaluated and follow up was performed at a mean of 19 months after surgery. Abbreviation: HB = House–Brackmann.

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Fig. 1. Axial (upper) and coronal (lower) T₁-weighted Gd-enhanced MR images demonstrating a medial acoustic neuroma with no extension into the IAC.

Fig. 2. Coronal and axial MR images demonstrating a large medial acoustic neuroma before (upper) and after surgery (lower), with excellent preservation of seventh (House–Brackmann Grade 1) and eighth (Class A) cranial nerve function. Arrow (upper right) indicates intrameatal tumor portion. The posterior lip of the meatus was not removed, leaving a small remnant of the tumor at the porus (arrow, lower right). Over a 4-year period this remnant has shown no evidence of growth.
cerebellar peduncular vein during surgery. In three patients temporary lumbar drainage was used for cerebrospinal fluid fistulas, and one patient required insertion of a lumboperitoneal shunt.

**Facial Nerve Function**

In the majority of patients facial nerve function deteriorated immediately after surgery (Table 1). In four patients House–Brackmann Grade I was documented immediately after the procedure, one patient was classified in Grade II, six in Grade III, eight in Grade IV, two in Grade V, and one patient had a complete paresis (Grade VI). Nine patients experienced postoperative fluctuation of facial nerve function. Function improved in five patients, four of whom underwent vasoactive therapy consisting of hydroxyethyl starch and nimodipine (two patients went from Grade IV to Grade III, two went from Grade III to Grade II, and one went from Grade IV to Grade II). In four patients a deterioration was noted. Of these, three patients received no vasoactive therapy; one received only nimodipine. In two a complete paresis developed (from Grade IV to Grade VI), in one patient a deterioration from Grade II to Grade IV occurred, and in the fourth one a change from Grade III to Grade IV was documented. On follow-up review, the majority of patients had normal facial nerve function (Grade I in 11 patients) or a discrete dysfunction (Grade II in six). Four patients had Grade III paresis and one had a Grade V deficit. There were no patients with complete and persistent facial nerve paralysis (Grade VI) on long-term follow-up (Table 1).

**Course of the Facial Nerve**

In 12 patients the facial nerve was identified at the anterior tumor capsule (Fig. 3). In five the nerve crossed on the middle anterior tumor surface (Fig. 3 upper right); two tumors originated from the inferior and three from the superior vestibular nerve. In five patients the nerve was identified on the anterior cranial tumor pole, in three cases immediately posterior to and in two cases anterior to the trigeminal nerve (Fig. 3 lower left). The tumor originated from the inferior vestibular nerve in three cases, and in two cases the tumor origin could not be identified. In two patients the nerve crossed the CPA on the lower anterior part of the tumor capsule (Fig. 3 upper left); in one of them the superior vestibular nerve was identified as the tumor origin. In two patients the facial nerve was identified on the posterior cranial pole of the tumor (Fig. 4). From the RExZ these nerves ran parallel to the brainstem in a cranial direction toward the RExZ of the trigeminal nerve, and then turned in an almost rectangular fashion toward the posterior cranial surface of the tumor. In both cases the inferior vestibular nerve was thought to be the site of tumor origin.

A distinct splitting of the facial nerve from the RExZ to the porus was seen in the remaining eight patients, and this was documented by selective electrical stimulation and three-channel EMG recordings (Figs. 3 and 6). In all patients the smaller portion of the nerve could be identified running parallel to the brainstem toward the cranial pole of the tumor, crossing the CPA on the cranial anterior part of the tumor capsule either posterior or anterior to the tri-
geminal nerve, and rejoining the major portion of the nerve immediately anterior to the porus (Fig. 5 upper left). On electrical stimulation, responses either from the musculus orbicularis oris alone or in addition from the musculus nasalis could be elicited. In no case could all three branches be stimulated (Fig. 6 upper left). The major portion of the nerve in these patients was located on the middle part of the anterior tumor capsule (Fig. 5 upper right, Fig. 6 lower right). Stimulation revealed EMG activity in all three facial nerve branches (Fig. 6 upper right). The separation of the two portions in all cases was found immediately distal to the RExZ of the facial nerve. There was no noticeable spreading of either portion. The area of realignment anterior to the porus showed the most adhesions between nerve and capsule and proved difficult to dissect (Fig. 5 upper right). In six of the eight cases the inferior vestibular nerve was identified as the tumor origin. In one case the facial nerve entered the meatus at the caudal ventral margin of the porus, finding its anatomical position in the middle segment of the meatus. In this case tumor origin was difficult to assess from the videotape. In the other patient, who had a giant tumor, the IAC was not opened.

Other Cranial Nerves

Preservation of cochlear nerve function was attempted in all 19 patients who had preoperative hearing, regardless of their hearing classification. At discharge 17 patients had lost their hearing completely. In three patients hearing preservation with an SDS of less than 50% (Class D) was documented, and two patients had excellent hearing (Class A). On follow-up review, 15 patients remained deaf (Class D). A total of four patients had an SDS below 50% (Class D), and three patients had good or excellent hearing outcome, with Class B hearing in one and Class A in two. It is noteworthy that both patients who recovered from deafness had received postoperative vasoactive treatment consisting of nimodipine and hydroxyethyl starch.15 Trigeminal and abducent nerve deficits resolved in all patients but three, with an improved but residual hypesthesia in the third branch. The trigeminal neuralgia and lower cranial nerve deficits resolved completely.

Discussion

Medial acoustic neuromas were first described and systematically investigated by Tos, et al.,16 in 1992. This tumor subtype is defined by a primarily extrameatal growth into the CPA, with little or no extension into the internal acoustic meatus. The lateral part of the meatus and the fundus is by definition free of tumor (Figs. 1 and 2). In a series of 400 acoustic neuromas up to 12% (48 lesions) were considered to be medial tumors, with a normal IAC in one third, porus enlargement in 20%, and a funnel-shaped canal in almost 50% of cases.

Outcome with respect to seventh and eighth cranial nerve
function is considered unfavorable because of tumor size. Because of the intracisternal growth pattern, medial-type tumors can reach considerable size before specific symptoms occur. Often these tumors become symptomatic, with other cranial nerve or cerebellar symptoms. Tumor size at diagnosis is significantly larger compared with nonmedial tumors, and 91.7% of tumors were either large (26–40 mm in diameter on axial imaging) or giant (> 40 mm). Preoperative facial nerve function is impaired in 10%, and facial nerve function as well as the degree of hearing loss in the series published by Tos, et al., did not significantly vary compared with nonmedial tumors. The facial nerve outcome is comparable with nonmedial tumors when taking lesion size into consideration. Tumor size also was the major argument for not attempting a hearing preservation procedure, because the majority of patients with reasonable preoperative hearing (hearing loss within the 40-dB range) had large tumors. A translabyrinthine procedure was performed in all patients. Complications, including a 2% mortality rate, were also attributed to tumor size.

Inamasu, et al., have limited the term “medial” to tumors with no enlargement of the IAC, and in their series of 466 patients, have seen 1.3% (six patients) in whom the tumors fulfilled the criteria of an exclusive intracisternal growth pattern. Their data analysis revealed similar results when compared with the series of Tos, et al., with respect to tumor size, preoperative symptoms, and results. Hearing preservation was possible in only one patient, despite the fact that at least some hearing was present in all six patients before the surgical procedure. Inamasu, et al., used an enlarged middle cranial fossa approach. Facial nerve outcome was considered worse than in nonmedial tumors, with good facial nerve function (House–Brackmann Grades I and II) in 67%, compared with 86% in nonmedial tumors.

As did Tos, et al., Inamasu and coworkers attributed the unfavorable results in seventh and eighth cranial nerve function in medial acoustic neuromas to the larger tumor size at the time of diagnosis compared with sizes of nonmedial tumors.

Only recently, Snyder, et al., have published a case report of a 3.5-cm medial acoustic neuroma that they treated with a two-stage suboccipitalateral approach, with excellent results in both the cochlear and the facial nerve, suggesting that even in medial type tumors every effort should be made to preserve function.

Internal Auditory Canal

The less restrictive definition of a fundus free of tumor proposed by Tos, et al., was used for the current series of 22 consecutive patients, because preoperative neuroimaging features of the IAC depend greatly on exact patient positioning. Tumor growth into the IAC was documented in all patients. We have actually seen one residual lesion in the case of a giant tumor for which the IAC was not opened be-
cause during the procedure we had the impression of complete removal (Fig. 2 lower right). We caution that, regarding management of the IAC in medial acoustic neuromas, we have always seen some intrameatal tumor extension in these cases, and therefore we have found that drilling of the posterior lip of the canal is mandatory, especially because intrameatal contrast uptake may be different from that seen in extrameatal tumors.9

The Facial Nerve

The most striking finding was the atypical course of the facial nerve in 10 of the 22 cases. Whereas the cochlear nerve bundle, as far as identified, was always located around the lower tumor pole, the course of the facial nerve differed from what is known in nonmedial type tumors. In nonmedial tumors, the facial nerve can be identified in the anterior tumor capsule in 78%, with a predominance in the middle and cranial section in 73% of the cases.13 In our series a typical anterior course was documented in 12 patients (55%; Fig. 3). In 10 (45%) of the 22 patients an anterior cranial (Fig. 3 lower left) or anterior middle position (Fig. 3 upper right) of the nerve was encountered. A course in the posterior tumor capsule was seen twice, accounting for 9% of all patients (Fig. 4). In the series of more than 1000 acoustic neuromas that was published by Sampath, et al.,13 a posterior course was seen in only 2% of the cases.

A split nerve course was seen—documented on videotape and electrically verified—in eight cases (Figs. 5 and 6). In these patients the major branch of the nerve could always be located on the anterior middle tumor capsule (Fig. 5 upper right). The smaller branch separated at the RExZ, running parallel to the brainstem toward the cranial pole of the tumor, crossing the CPA on the cranial exterior tumor capsule either posterior or anterior to the trigeminal nerve, and it rejoined the major portion of the nerve immediately anterior to the porus (Fig. 5 upper right, Fig. 6 lower right). In all cases of a split nerve the identification was documented by selective stimulation (Fig. 6 upper). Our multichannel recording setup proved essential for selective identification of facial nerve fibers in these cases. The course of the trigeminal nerve adjacent to the smaller facial nerve portion, and the positive stimulation effect at two different sites in the upper and middle anterior tumor surface may cause misinterpretation of the signals. The EMG responses obtained from the small, often not visible, facial nerve component in the upper CPA could be mistaken for motor responses from the trigeminal nerve and thus mislead the surgeon (Fig. 5 upper right). This is particularly true when a standard monopolar stimulation and recording setup are used, as described by Moller and Jannetta,7 in conjunction with loudspeaker-based EMG systems. We found it helpful to use three channels for facial nerve EMG (musculus orbicularis oculi, musculus orbicularis oris, and musculus nasalis) and a pseudobipolar recording setup with insulated needle electrodes 5 mm apart. Bipolar stimulation is more specific and the current does not spread to the surrounding tissue.

Fig. 6. Electrical stimulation of split nerves in which minimal stimulation intensity (30 Hz, 0.05 mA) is used, demonstrating selective responses of facial muscles. Stimulation of the minor portion of the nerve shows selective responses from the musculus orbicularis oris (m. orb. oris, upper left). An electrical stimulator seen on the minor portion (arrow, lower left) corresponds to the EMG traces (upper left). After lifting of the minor portion, the major branch becomes visible (black arrow, lower right). Corresponding EMG traces (upper right).
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The selective responses of facial nerve branches after bipolar constant current stimulation also support other clinical findings, which propose a topographical arrangement of facial nerve fibers within the course of the nerve through the CPA. From anatomical investigations it is known that these fibers represent the intermediate nerve. The split nerves documented in this series resemble the case illustration of a Grade 3 neuroma by Koos, et al. However, it is not clear whether their interpretation was based on a negative electrical stimulation. The intermediate nerve itself is rarely identified and preserved during removal of larger acoustic neuromas and is usually not specifically addressed. From anatomical investigations it is known that the intermediate nerve cannot be separated from the eighth cranial nerve complex through its course within the CPA in 22% of patients, and in 35% it is adherent through most of its intracisternal course. The description of the intracisternal portion of the intermediate nerve in the series of 73 specimens reported by Rhoton, et al., does not compare with our findings. A position of the smaller branch cranial to the REZ of the remaining larger facial nerve portion favors a division of the motor facial nerve fibers themselves rather than the presence of the intermediate nerve. Rhoton, et al., report a root exit of the intermediate nerve together with the eighth cranial nerve fibers. They also state that its junction with the facial nerve fibers can usually be found 5 mm proximal to the fundus of the IAC. This was not observed in any of our eight cases with a split course of the facial nerve, in which the junction was always located proximal to the porus (Figs. 5 upper right and 6 lower right). In addition, Rhoton, et al., found more than one root outlet fascicle in 50% of the specimens. Nevertheless, anatomical distortion caused by the tumor has to be taken into consideration. The electrophysiological results with selective EMG responses from the two separate branches of the facial nerve at lowest stimulation intensities (0.05 mA) obtained using the bipolar constant current method, clearly point to two separate bundles of the facial nerve (Fig. 6 upper).

In contrast with nonmedial tumors, in which a clear relationship between tumor size and postoperative facial nerve function is established, this could not be verified for medi al tumors. In two tumors smaller than 20 mm in axial diameter, House–Brackmann Grades I and III were documented. In nine patients with 20 to 29–mm tumors, Grades I and II were seen in four patients each, and Grade III in one patient. The majority of patients harbored large acoustic neuromas. In seven patients with tumor sizes between 30 and 39 mm, Grade I was documented in three cases, and Grades II and III were seen in two patients each. In four patients the tumor size exceeded 40 mm: of these, in two patients no facial nerve deficit (Grade I) was documented, and Grades II and V were seen in the two remaining patients. There was no influence of intrameatal tumor extension on these results.

Tumor Origin

The tumor origin could be documented in 17 cases; whereas in five cases, among them all four cases in which the meatus was not opened, the tumor origin remained unclarified. In the majority of cases the tumor appeared to arise from the inferior vestibular nerve (13 patients, 59%) although in two the interpretation of the videotape remained somewhat speculative. In four cases the superior vestibular nerve could be clearly identified as the site of tumor origin.

With respect to the position of the facial nerve an inferior vestibular nerve origin was found in both cases in which the facial nerve coursed on the posterior cranial capsule. In six of eight patients with a split facial nerve, the tumor appeared to arise from the inferior vestibular nerve (Fig. 5 lower right). A location on the cranial anterior capsule (five patients) was never associated with a superior vestibular nerve origin, whereas it was seen three times in association with a middle anterior course (five patients). Twice the tumor appeared to arise from the inferior vestibular nerve. A superior vestibular nerve origin was seen in one of these two cases, in which a facial nerve course on the lower anterior tumor capsule was observed. In the other case the tumor origin could not be identified.

Conclusions

There is definite evidence for a clinical subtype of a medial intracisternal acoustic neuroma and its surgical significance. These tumors, which primarily grow within the CPA, have a distinct impact on the course of the facial nerve, which has to be taken into consideration during surgical removal. In 36% of patients the facial nerve split into two separate components, with maximal adherence to the tumor capsule at the junction of the two components anterior and ventral to the porus. In two cases a cranial posterior displacement of the nerve was seen. We have never encountered a substantial spreading of the nerve anterior to the porus as described in nonmedial tumors. Continuous EMG monitoring with constant feedback to the surgeon, frequent use of direct stimulation, and a multichannel recording setup contributes to facial nerve preservation in surgery for medial acoustic neuromas. Despite the unfavorable results described in the literature, excellent functional results can be achieved.

Dedication

Dedicated to Rudolf Fahlbusch M.D., Chairman of the Department of Neurosurgery at the University of Erlangen-Nuremberg, Germany, on the occasion of his 60th birthday.

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