NEUROSURGERY 40:4 | APRIL 1997 | 684-695
DOI: 10.1097/00006123-199704000-00006

Management of 1000 Vestibular Schwannomas (Acoustic Neuromas): The Facial Nerve-Preservation and Restitution of Function

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Abstract

OBJECTIVE

Although the rate of reported facial nerve preservation after surgery for vestibular schwannomas continuously increases, facial nerve paresis or paralysis is a frequent postsurgical sequelae of major concern. The major goal of this study was to define criteria for the right indication, timing, and type of therapy for patients with palsies despite anatomic nerve continuity and those with loss of anatomic continuity.

METHODS:

One thousand vestibular schwannomas were surgically treated at the Department of Neurosurgery at Nordstadt Hospital from 1978 to 1993. Of 979 cases of complete removal and 21 cases of deliberately partial removal, the facial nerve was anatomically preserved in 929 cases (93%). The rate of preservation is increasing, as is evidenced in the most recent cases, and preservation is supported by special electrophysiological monitoring. The facial nerve was anatomically severed in 60 cases (6%). It was anatomically lost in previous operations that were performed elsewhere in 11 cases (1%). In case of nerve discontinuity (42 cases), immediate nerve reconstruction by one of three available intracranial procedures (within the cerebellopontine angle, intracranial-intratemporal, intracranial-extracranial) was performed in the same surgical setting. In case of loss of the proximal facial nerve stump at the brain stem, early reanimation by combination with the hypoglossal nerve was achieved in most patients within weeks after tumor surgery. In a few patients with anatomic nerve continuity but absence of reinnervation for 10 to 12 months, a hypoglossal-facial combination was applied. All the patients with partial or with complete palsies were treated in a special follow-up program of regular controls and of modulation of physiotherapeutic treatment every 3 to 6 months.

RESULTS:

In intracranial nerve reconstruction at the cerebellopontine angle, 61 to 70% of patients regained complete eye closure and an overall result equivalent to House-Brackmann Grade 3. Hypoglossal-facial reanimation led to Grade 3 in 79%. The duration between the onset of paralysis and the reconstructive procedure is decisive for the quality of the outcome. These data are discussed in view of other treatment options and certain parameters influencing outcome.

CONCLUSIONS:

This management contains three major principles as follows: 1) preservation of facial nerve continuity in function by the aid of intraoperative monitoring, 2) early nerve reconstruction in case of lost continuity, and 3) scheduled follow-up program for all patients with incomplete or complete palsies.

Key words: Acoustic neuroma, Facial nerve, Facial nerve monitoring, Facial nerve reconstruction, Suboccipital approach, Vestibular schwannoma

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Received: November 09, 1995
Accepted: August 06, 1996

The handling of the facial nerve during the treatment of a vestibular schwannoma should aim for long-term satisfactory facial nerve function, avoiding functional and social compromise for the patient. This goal can be achieved by instituting two major principles: 1) increasing the rates of anatomic and functional nerve preservation, and 2) determining the right timing and indication of the adequate reconstructive procedure in case of nerve discontinuity or persistent paralysis despite nerve continuity. These surgical aspects need to be included in a thoughtful patient care program that guarantees close follow-up for clinical and electrophysiological controls and for modulation of physiotherapy.

Although many authors report increasing rates of nerve preservation([2, 6, 21, 58, 62]), patients with facial nerve paralysis are frequently neglected in many centers, because the microsurgical restitution of facial nerve function requires special training and experience ([24, 47–49, 51]). This situation seems

bizarre if we remember that the fundamental discoveries of the opportunities of facial nerve reconstruction were laid by pioneers like Koerte in 1903 ([26]), Bunnell in 1927([5]), Maxwell in 1951 ([33]), Dott in 1958 ([11]), Conley in 1961 ([8]), and Scaramella in 1971 ([59]), to mention only a few among many others. Based on their investigations, it is known that the facial nerve may be reconstructed along its anatomic course from the brain stem to its peripheral branches in the face. Further concerted studies by joint groups of neurosurgeons and otorhinolaryngologists developed the intracranial-intratemporal and the pure intracranial methods of reconstruction([12, 13, 47–50]) (L. Osterwald, personal communication). Moreover, the technique of combination with a donor nerve (so-called neurotization) is still valuable today.

PATIENTS AND METHODS

From 1978 to 1993, 1000 vestibular schwannomas (acoustic neuromas) were removed from 962 patients using the suboccipital approach. Eighty-two patients had evident Neurofibromatosis 2 (NF-2) with bilateral acoustic neuromas, 120 of which were operated on at our institution. Eight hundred and eighty patients were free of any NF-2 features and were treated for unilateral tumors ([55]).

Seventeen percent of the patients presented with facial paresis or palsy of various degrees ([31]). One and one-tenth percent of the total patients had lost their facial nerves in previous operations that were performed elsewhere and presented with House-Brackmann Grade 6 palsies([19]).

Tumor sizes were measured considering intra- and extrameatal tumor extension; large tumors were greater than 30 × 20 mm, and small tumors measured up to 30 × 20 mm. Tumor extension was described as follows: Class T1, purely intrameatal; T2, intra-extrameatal; T3a, filling the cerebellopontine cistern; T3b, reaching the brain stem; T4a, compressing the brain stem; and T4b, severely dislocating the brain stem and compressing the fourth ventricle ([54]). The management of the facial nerve was performed according to four treatment options: 1) nerve preservation, 2) nerve reconstruction, 3) nerve reanimation, and 4) clinical guidance and physiotherapeutic management.

Nerve preservation

Preservation of the facial nerve was attempted for all patients, except those in whom the nerve was lost during previous surgery, using a special microsurgical technique. After opening the internal auditory canal, the intrameatal tumor extension and the position of the seventh and eighth nerves are inspected and are controlled by their electrophysiological response (see below). The extrameatal tumor mass is reduced by tumor enucleation from inside, either by the Cavitron ultrasonic surgical aspirator or by the platelet-shaped knife. As soon as the pressure of the tumor on the adjacent structures is reduced, dissection of the tumor from the surrounding neural and vascular structures is performed by strictly gripping the tumor capsule and dissecting in the level of the arachnoid plane. Maximum safety to all relevant structures is thereby guaranteed, and the structures can be handled from all directions. (Over-)Stretching of neural structures in one direction for a long time is avoided, but changing the sites of microsurgical actions is regarded as favorable, enabling different nerve fibers to recover from temporary tension. The tumor-nerve border is prepared medially along the brain stem for identification of the proximal part of the facial nerve. The tumor-nerve border is prepared upward at the upper extension of the tumor. When gripping the tumor rest and pulling it medially and upward, the lowest and most lateral aspects of the facial nerve become visible. This part can usually be dissected free by preparation from the intrameatal portion laterally toward the extrameatal portion and by repeatedly moving the dissector in the right plane between the tumor and the nerves. Continuous electromyographic recording provides the feedback for the exact identification of all parts of the facial nerve; after complete tumor removal, nerve continuity can be confirmed by light touching or by electrical stimulation of the nerve from the brain stem to the intrameatal portion (see below).

Intraoperative neurophysiological monitoring is performed in the patient, who is anesthetized without or with light relaxant medication, by continuous electromyography transferred by loudspeakers. Bipolar recording is set up with needle electrodes at the eyebrow for the orbicularis oculi muscle and at the mouth angle for the orbicularis oris muscle. The filters are set to 20 Hz to 20 kHz, sensitivity is set at 20 to 50 uV, and the time base is 10 milliseconds to 2 seconds. Muscle activation is initiated by mechanical nerve stimulation through touching the arachnoid around the nerve with the forceps or by water irrigation ([30]). Electrical activation by 1 to 4 mA is applied in selected cases, such as those with decreasing reactivity to mechanical stimulation during the course of surgery or in case of difficult nerve identification.

Facial nerve reconstruction

In case of nerve discontinuity, immediate nerve reconstruction is performed in the same operation. If a proximal and a distal nerve stump are available within the cerebellopontine angle (CPA), direct end-to-end coaptation or reconstruction using a 5- to 20-mm nerve transplant is performed. If only the proximal nerve stump is available, the distal part of the facial nerve is to be found within the temporal bone by drilling it open and is fused using a 30- to 50-mm transplant and an intracranial-intratemporal technique ([12, 47–49]). If previous petrous bone trauma or mastoiditis disallow this method, intracranial-extracranial transplantation is used by fusing the proximal nerve stump, using an 80- to 150-mm transplant, to the distal part at the stylomastoid foramen ([11]).

Facial nerve reanimation

Facial nerve reanimation by a donor nerve, either the hypoglossal or the contralateral facial nerve, is indicated in case of loss or complete degeneration of the proximal facial nerve stump at the brain stem. This might occur during the resection of large tumors with complete nerve loss when a reanimation procedure is scheduled within the 1st weeks after tumor resection or, in some cases of anatomic nerve preservation, when the nerve is severed too strongly, the nerve might degenerate with paralysis persisting for 10 to 12 months. The nerve reanimation procedure should be scheduled not later than 1 year after the onset of the paralysis. Hypoglossal-facial nerve anastomosis(as presented by Koerte in 1903 [26]) is performed by combining the proximal part of the hypoglossal nerve (gained below the digastric muscle) with the distal segment of the facial nerve at the stylomastoid process; microscopic nerve adaptation is performed and stabilized by two 10-0 sutures. Facio-facial nerve anastomosis (as presented by Scaramella in 1971 [59] and Smith in 1971[61]) may be used in patients in whom no other cranial nerve can be sacrificed (i.e., in a patient with caudal cranial nerve palsy). Approximately one-third to one-half of the plexiform facial nerve branches on the healthy side of the face may be cut without discernible deficit. These branches can be connected, using nerve transplants, to the corresponding branches of the plegic side; because of the wide range of the individual innervation pattern, intraoperative electrophysiological identification of the branches and their functions may be helpful. Two-centimeter-long incisions lead to exposure of the zygomatic branches, the strongest among the facial branches, related to the orbicularis oculi as well as orbicularis oris muscles. As the reinnervation is generally fairly weak, additional plastic surgery, such as Pitanguy's oval skin resection at the nasolabial fold or temporalis or masseter muscle transfer, is usually necessary.

Clinical guidance and physiotherapeutic management: clinical follow-up

Outpatient clinical follow-up every 3 to 6 months includes electromyography until some reinnervation is documented and physiotherapeutic teaching. Depending on the degree of reinnervation or the possible onset of synkinesias, special instructions are given to intensify certain aspects of physiotherapy. A brief summary of instructions follows.

1) In case of complete paralysis, the patient needs to participate in a physiotherapy program for at least 3 years. The program consists of daily individual exercises that are performed six times for 5 to 10 minutes. During the first 1 to 2 years, the patient needs to be accompanied by a specialized physiotherapist for two to three weekly sessions. 2) Each exercise consists of the trial of an active movement and then a massage in the requested direction, as follows: a) lifting eyebrows without any help and striking eyebrows up with the fingertips, b) frowning and pushing eyebrows together, c) closing eyes and sliding down eyelids, d) wrinkling the nose and pushing cheeks together, e) blowing the cheeks and holding the mouth angles tight, f) forming a small "o" with the lips and pushing the mouth angles together, g) smiling and pulling mouth angles apart, and h) smiling strongly and pushing the healthy side toward the midline to facilitate some action on the sick side. 3) If eye closure is especially weak, it is combined with strong biting until it becomes complete. Then the patient tries very slowly to relax the masseter muscles while imagining closed eyes; after complete relaxation, the eyes are opened. This whole sequence is repeated three to five times. The same exercise is helpful in reducing synkinesias between eye closure and mouth angle. 4) If eye closure is weak in hypoglossal-facial combination, a similar exercise is performed. The eye is closed and supported by pressing the tongue against the teeth. The tongue is then very slowly loosened while the eye is imagined to be closed. Within weeks to months, independent eye closure without any tongue movement becomes possible. 5) In case of a weak mouth angle, the healthy side is pushed toward the midline and fixed with the fingertips. The sick mouth angle can then be trained more easily. 6) Any electrical stimulation of nerves or muscles must be avoided to minimize the development of contractures.

Clinical evaluation is based on the control of face symmetry at rest, symmetry during movement, separate movements of all three segments (forehead, eyes, mouth) of the face, and incidence of conjunctivitis and synkinesias. The results are correlated with the House-Brackmann scale.

RESULTS

Prior surgery

Fifty-six patients had undergone surgery at other hospitals. Twenty-four had undergone subtotal tumor resection, and 32 had undergone biopsies.

Surgical radicalness

In 979 cases, tumor removal was complete. In 21 cases, deliberate subtotal tumor removal was performed (10 cases for brain stem decompression and 11 cases of NF-2 for cochlear nerve decompression in the last hearing ear). The facial nerve was anatomically preserved in 929 cases (93%). Preservation rates ranged from 87 to 94% for large and small tumors, respectively (i.e.,>30- or <30-mm diameter) until 1988. In the most recent 200 cases of tumor resections using the CPA approach, preservation rates rose to 94%, independent of tumor size. Tumor extension was a further decisive factor. Preservation rates were as follows for the facial nerve: Class T1, 100%; T2, 96%; T3a, 98%; T3b, 94%; T4a, 86%; and T4b, 84%. Morphological aspects of the tumor influenced the preservation rates. In case of cystic tumor formation, the

anatomic preservation rate of the facial nerve was reduced from 93 to 88%.

Facial nerve function ([55]), graded according to the House-Brackmann scale within 2 weeks after surgery, was Grade 1 in 47%, Grade 2 in 12%, Grade 3 in 14%, Grade 4 in 6%, Grade 5 in 10%, and Grade 6 in 11% of the patients. The 11% with Grade 6 included 7% of the cases of facial nerve discontinuity and 4% of paralysis despite nerve continuity. Most of the patients with paralysis despite nerve continuity showed spontaneous reinnervation within the 1st postoperative year. Of the 1.7% who lacked any reinnervation for 10 to 12 months, 11 patients were treated by reanimation procedures whereas a few refused any nerve reconstruction and underwent plastic surgery. Facial nerve function of those with preserved anatomic continuity is also listed in Tables 1 and 2.

TABLE 1.

Facial Nerve Symptoms

Nerve	Symptom	Incidence Immediately before Surgery (%)	Incidence 2–8 wk after Surgery (%)
Seventh	Facial paresis	5.2 (Duration = 1.9 yr)	53
	Taste disturbances	2	1

TABLE 2.

Facial Nerve Signs

Nerve	Neurological Sign		Incidence Immediately before Surgery (%)	Incidence 2–8 wk after Surgery (%)	
				All 1000	929 with Nerve Continuity
Seventh	Facial spasm		0.6		
	Facial paresis		17	53	
	House-Brackmann	Grade 1	83	47	51
		Grade 2	8	12	13
		Grade 3	6	14	15
		Grade 4	0.7	6	6
		Grade 5	1	10	11
		Grade 6	1.3	11	4

The facial nerve was anatomically lost in previous operations that were performed elsewhere in 11 cases. In two cases, a nerve reanimation was achieved by hypoglossal-facial nerve combination, and in nine cases, plastic surgery had been performed.

TABLE 3.

Summary of Clinical Results after Facial Nerve Reconstruction and Reanimation[a]

Type of Reconstruction	Results (%)				
	Excellent HB 3+	Good HB 3	Fair HB 4	Bad HB 5 and 6	Too Early
23 Reconstructions in the CPA	4 (21)	10 (53)	4 (21)	1 (5)	4
16 Reconstructions from CPA to mastoid	4 (25)	7 (44)	3 (19)	1 (6)	
3 Reconstructions from CPA to F. stylomast.	1 (10)	6 (60)	2 (20)	1 (10)	
29 Hypoglossal-facial reanimations	5 (21)	18 (75)	1 (4)		5
2 Facio-facial reanimations		2			

[a] HB, House-Brackmann Grade; CPA, cerebellopontine angle; F. stylomast., facial nerve at the stylomastoid foramen. Follow-up at 12 to 120 months; further improvement is anticipated in some cases.

TABLE 4.

Results of 23 Intracranial Facial Nerve Reconstructions in the Cerebellopontine Angle[a]

Patient No.	Age (yr)	Sex	Paresis → Op (mo)	Lesion	Fixation	Op → Recovery (mo)	Follow-up (mo)	Forehead	Eye	Mouth	Tears	Summary
1	43	F	0	VS	Suture	4	94	++	+++	+++	?	Excellent (HB 3+)
2	37	F	0	VS	Glue	10	27	+	+++	++	+++	Excellent (HB 3+)
3	23	M	0	VS	Glue	7	21	+	+++	++	?	Excellent (HB 3+)
4	45	M	0	VS	Glue	4	18	+	+++	+++	+	Excellent (HB 3+)
5	52	F	0	VS	Suture	10	54	+	++	+	+	Good (HB 3)
6	57	M	0	VS	Glue	?	30	+	++	+	?	Good (HB 3)
7	18	F	0	VS	Glue	7	13	0	++	++	+	Good (HB 3)
8	46	F	0	VS	Glue	4	23	0	++	+++	+++	Good (HB 3)
9	31	F	0	VS	Glue	12	56	0	++	++	0	Good (HB 3)
10	54	M	0	VS	Suture	11	62	0	++	+	0	Good (HB 3)
11	25	F	0	VS	Glue	6	24	0	++	++	0	Good (HB 3)
12	57	M	0	VS	Glue	5	22					Good (HB 3)
13	50	F	0	VS	Glue	6	23	0	+	++	+	Good (HB 3)
14	52	F	0	VS	Suture and glue	6	28	0	+	+	0	Fair (HB 4)
15	57	F	0	VS	Glue	12	24	+	+	+	?	Fair (HB 4)
16	21	F	0	VS	Glue	12	56	0	+	+	0	Fair (HB 4)
17	56	M	0	VS	Suture and glue	?	56	0	+	+	+++	Fair (HB 4)
18	56	M	0	VS	Glue	6	23	+	+++	++	++	Good (HB 3)
19	38	F	0	VS	Glue	12	24					Bad (HB 5, NF-2)
20	19	M	0	VS	Glue	?	?					?
21	17	M	0	VS	Glue	?	?					?
22	61	F	0	VS	Glue	?	?					?
23	51	M	0	VS	Glue	?	?					?

[a] Op, operation; VS, vestibular schwannoma; ?, unknown; +, incomplete function; ++, complete function at reduced power; +++, complete powerful function; HB, House-Brackmann Grade; NF-2, Neurofibromatosis 2.

TABLE 5.

Results of 16 Intracranial-Intratemporal Reconstructions from the Cerebellopontine Angle to the Mastoid[a]

Patient No.	Age (yr)	Sex	Paresis → Op (mo)	Lesion	Graft Length	Op → Recovery (mo)	Follow-up (mo)	Forehead	Eye	Mouth	Tears	Summary
1	45	M	3	VS	5 cm		73	++	+++	++	?	Excellent (HB 3+)
2	49	M	0	VS	4 cm	7	85		+++	++	+−+	Excellent (HB 3+)
3	27	M	1	VS		9	123		+++	+−+	0	Excellent (HB 3+)
4	54	F	0	VS	5 cm	9	126	+	+++	+−+	+−+	Excellent (HB 3+)
5	30	F	12	VS + fn	6 cm		24		++	++	?	Good (HB 3)
6	40	F	0	VS	5 cm	12	120	0	+++	−	0	Good (HB 3)
7	35	M	0	VS	5 cm		41	++	++	++	?	Good (HB 3)
8	50	M	0	VS	4 cm		27	+	++	++	+−+	Good (HB 3)
9	53	F	4	VS	5 cm	9	99	0	++	−	0	Good (HB 3)
10	29	F	20	VS	5 cm	8	14	0	++	++	?	Good (HB 3)
11	51	M	8	VS	5 cm	12	26	+	++	++	?	Good (HB 3)
12	40	M	3	VS	4 cm	4	81	+	+	++	+−+	Fair (HB 4)
13	53	M	0	VS	4 cm	10	16	0	+	++	?	Fair (HB 4)
14	40	M	0	VS	4 cm				+	++	?	Fair (HB 4)
15	56	F	95	VS	4 cm		90	0	0	−	?	Bad (HB 5)
16	20	M	0	VS	5 cm	1						Died after 3 mo

[a] Op, operation; VS, vestibular schwannoma; fn, facial neuroma; ?, unknown; +, incomplete function; ++, complete function at reduced power; +++, complete powerful function; HB, House-Brackmann Grade.

TABLE 6.

Results of 29 Hypoglossal-Facial Reanimations after Resection of Primary Schwannomas, Residuals, or Recurrencies (and of 33 Reanimations after Tumor Surgery Performed Elsewhere)[a]

HF Reanimation after Tumor Resection (%)	Mean Interval Paralysis → Op (mo)	Result	Transferral Only for HF Reanimation after Tumor Resection Performed Elsewhere (%)	Mean Interval Paralysis → Op (mo)
5 (21)	7.8	Excellent (HB 3+)	3 (9)	15
18 (75)	8.4	Good (HB 3)	18 (55)	16.3
1 (4)	14	Fair (HB 4)	11 (33)	23.6
		Bad (HB 5)	1 (3)	18
5		Too early		

[a] HF, hypoglossal-facial; Op, operation; HB, House-Brackmann Grade.

The facial nerve was anatomically severed in 60 cases (Tables 3 through 5). In 42 cases, facial nerve discontinuity could be treated by intracranial reconstruction by a nerve graft. In 23 cases, the facial nerve was reconstructed in the CPA by a sural graft of 5 to 30 mm(Table 4 andFig. 1). In 16 cases, reconstruction was performed from the CPA to the mastoid segment (intracranial-intratemporal transplantation)(Table 5). In three cases, the method of intracranial-extracranial transplantation from the CPA to the external segment at the

stylomastoid portion, as presented by Dott([11]), was performed. In 18 cases, the facial nerve got lost at its origin at the brain stem, and the facial nerve was reanimated by a donor nerve (by the contralateral facial nerve in 2 cases and by the hypoglossal nerve in 16 cases) (Table 6 and Fig. 2).

FIGURE 1.

At resection of a vestibular schwannoma, facial nerve discontinuity was bridged by a 20-mm sural transplant for an intracranial reconstruction in this 56-year-old man. His initial paralysis with facial asymmetry (*A*), lack of frowning (*B*), lagophthalmus (*C*), and lack of any mouth control on the right(*D*) started to improve after 5 months, with a satisfactory result documented after 2 years (*E-I*). There is good facial symmetry at rest (*E*), during movement with good emotional expressivity(*F*), some recovery of frowning (*G*), complete eye closure (*H*), and some mouth angle control and some residual autoparalytic syndrome (*I*).

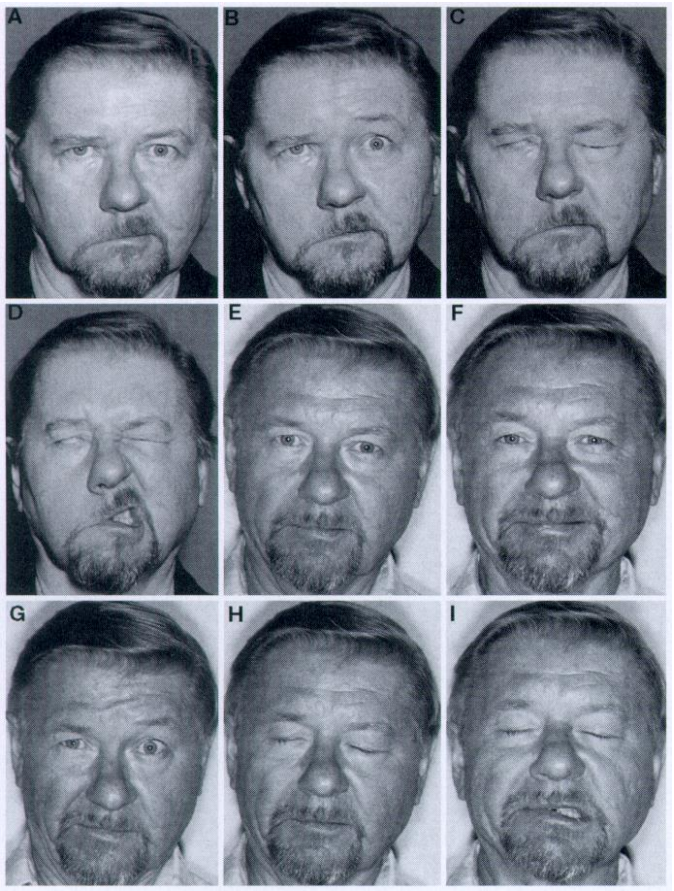
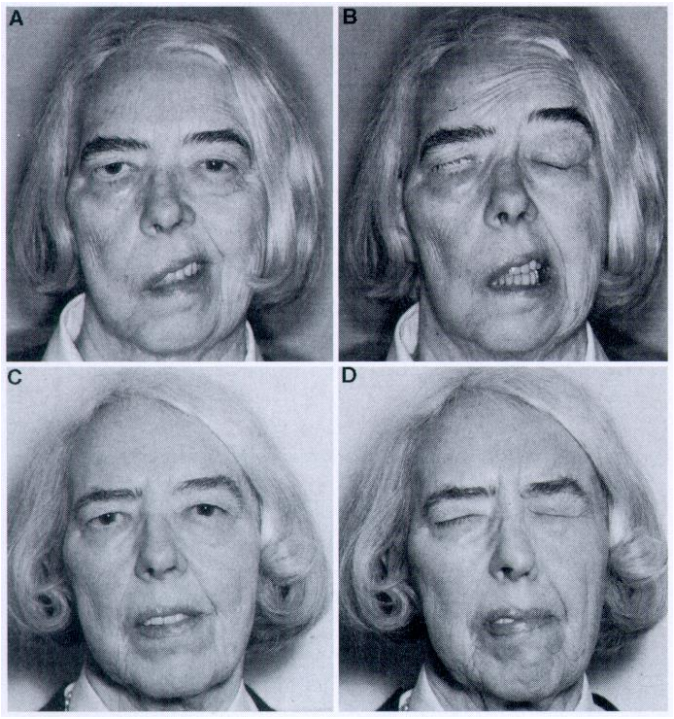


FIGURE 2.

In this 66-year-old woman, the facial nerve was anatomically preserved after the removal of a 35-mm schwannoma. Because postoperative paralysis persisted for 12 months with severe facial asymmetry (*A*) and lagophthalmus (*B*), facial nerve reanimation was achieved by combination with the hypoglossal nerve, with a good result (i.e., complete eye closure and almost no synkinesia after 2 yr)(*C* and *D*).



In summary, the facial nerve preservation rate was 93%, with 1.7% persistent paralysis and subsequent reconstruction by donor nerve reanimation but spontaneous recovery otherwise. Seven percent of the patients required immediate nerve reconstruction (1% after undergoing previous nerve lesion and partial tumor resection elsewhere and 6% after undergoing tumor resection at our institution). Facial nerve reconstruction (Table 3) at the CPA performed in 42 cases and evaluated in 38 cases led to satisfactory reinnervation in 69 to 74%, which is equivalent to House-Brackmann Grade 3 (Tables 4 and 5).

Facial nerve reanimation performed in 29 patients and evaluated in 24 led to good results in 96%. An additional 33 patients needed facial nerve reconstruction/reanimation after schwannoma resections performed elsewhere. Because some of these patients sought treatment much later than did those of our program, the quality of reinnervation was slightly less favorable(Table 6), with 64% achieving full eye closure.

DISCUSSION

Surgical approaches and their relevance with regard to facial nerve function

In the literature, results and complications are usually reported with special reference to the applied surgical approaches, such as the translabyrinthine, the middle fossa, and the

suboccipital/retrosigmoid (SO) approaches. Although the attitude of strictly insisting on one special approach has been abandoned, special advantages and disadvantages, such as the incidence of cerebrospinal fluid fistula, infection, and facial and cochlear nerve function, are still ascribed to each approach.

For the extended translabyrinthine-transotic approach, anatomic facial nerve preservation rates above 90% (94-95% are often reported)([3, 6, 44]) are achieved, with the necessity for hypoglossal-facial anastomosis in approximately 10%. The translabyrinthine approach is especially advocated in patients with deafness or poor chances of hearing preservation and in patients older than 70 years([46]) and is considered favorable because of the advantage of identification of the facial nerve proximally and medially and the ability to immediately repair the facial nerve ([4]) if it is severed during acoustic tumor removal. Most authors put special emphasis on the completeness of tumor removal ([46]).

Preservation rates are similar in the middle fossa and SO approaches, and the rates of early normal clinical function seem dependent on tumor size. Especially in small tumors, 70 to 80% normal function rates are reported([17, 34]). Glasscock et al.([15]), in 1993, reported on the SO and middle fossa approaches in 161 selected patients. They found a lower incidence of temporary facial nerve paresis using the SO approach.

The argument that the middle fossa or posterior fossa approaches, the only routes enabling hearing preservation (with few exceptions[35]), were more dangerous to the patient, especially to the facial nerve, than the translabyrinthine route was abandoned some time ago ([7, 16, 38, 39]). The translabyrinthine and SO approaches offer the best opportunities for preservation, as well as for reconstruction of the facial nerve([3, 7, 18–20]). Tos et al.([64]), in 1992, observed that suction is a damaging process during nerve preparation and emphasized that the most dangerous part for facial nerve severance is just medial to the porus ([52, 53, 64]).

In a mixed patient population with small and large tumors, anatomic preservation rates are above 90%, as reported by most authors. With the support of facial nerve monitoring, the rate of nerve preservation continues to increase. It is likely that there are always a few patients whose facial nerves are so elongated and damaged by the tumor that despite preservation of continuity, they will not recover but will undergo secondary degeneration and pose the indication for nerve reanimation after 10 to 12 months. Additionally, tumor biology plays an important role. Cystic tumor formation especially is associated with a reduced chance of facial nerve preservation([32, 55]).

Completeness of resection

A few authors still advocate leaving some residual tumor on the facial nerve in case of severe adhesion or apparent infiltration ([60, 65]). However, this imposes the likelihood of tumor regrowth within a few years and the necessity to perform subsequent surgery, during which the problem of the facial nerve will again present itself. Therefore, in the presented material of 1000 operations, the completeness of tumor resection at the facial nerve was a basic principle. If preservation of the nerve was not possible, the involved nerve portion was transsected and bridged by a nerve transplant to achieve two major goals (i.e., long-term freedom of any recurrence and long-term good facial function). An exception to this may be observed in some patients with NF-2 with multiple spinal and intracranial schwannomas. Tumors of several cranial nerves are encountered at surgery. Sometimes pearl-like changes with multiple tumors of the facial nerve along its course are seen while the nerve is clinically completely intact. In this situation, in which the tumorous changes do not offer a chance of useful curative nerve reconstruction, the tumor should be left in place ([57]).

Radiosurgery versus microsurgical resection

Radiosurgery is frequently indicated "for patients whose medical problems make surgery unacceptably dangerous" ([29]) and is designed to achieve "tumor control" in 80% to more than 90% of those patients([14, 29]). Facial nerve neuropathies frequently start to develop with some delay after a few months and may have an incidence of 30 to 33% and, rarely, of 17% ([41]). Mendenhall et al.([36]) presented a first report on Linear accelerator-based stereotactic radiosurgery; the results seem similar in that facial nerve paresis had an incidence of 19%. Hudgins([22]) analyzed results according to patients' preferences and therefore advocates radiosurgery for those who want to prevent any facial paresis, despite the recent data that indicate a high percentage of late trigeminal and facial nerve defects. In summary, radiosurgery offers an alternative for those patients with contraindications for narcosis or surgery. The rates for tumor control are reported to be at least 88%. Facial nerve palsies range from 17% immediately to 34% 0.5 to 1 year after surgery. When lower rates of facial nerve palsies were reported (~ 16%), the rates of tumor reduction were only 25 and 75% constant tumor size([25]). One major problem is the biological changes, especially the degeneration of the nerve secondary to the radiation for which no treatment options are available. It is likely that the compromise of feeding vessels and the direct radiation impact lead to this degeneration. In patients with NF-2, therefore, radiosurgery is not a good treatment alternative to surgery. In those patients, the residual capacity of the nerves is especially reduced because of the closer connection between the nerves and the tumor. In conclusion, young patients and those with NF-2 are not candidates because of the apparent degenerative changes induced in the cranial nerves. Long-term studies suggest that the late deterioration poses a more severe problem than the residual characteristics after recovery from facial paralysis or from facial nerve reconstruction.

The feasibility of electromyographic monitoring

As indicated, no or only temporary mild relaxation is used and controlled using recording hypothenar compound muscle action potentials with a relaxograph by our anesthesiologists without any adverse effects on the chances of monitoring. Although most colleagues use continuous electrical stimulation with their operative microinstruments ([28, 63]), this is not necessary with the set-up presented in this article, for which the response to mechanic stimulation by normal operative measures is recorded. Only when this diminishes or is very weak from the start (e.g., in partial paresis) is electrical stimulation applied.

Indication of nerve reconstruction versus primary plastic surgery

In case of a known nerve discontinuity, the reconstruction of the neural pathway is a logical and microsurgically feasible procedure. It also offers the best physiological response to the disadvantageous processes after nerve interruption. According to neuroanatomic studies, several degenerative processes occur after the interruption of a nerve. One of the most important processes is the degeneration of the nerve nuclei (i.e., of the facial nucleus within the brain stem [40]) and the subsequent degeneration and loss of plasticity and information in the facial motor cortex. The closer the interruption is located to the proximal nuclei (i.e., at the facial nerve origin at the brain stem), the faster these degenerative processes will occur. The only opportunity of counteracting this degeneration, and thereby the lasting facial disfigurement, is the early reconstruction of the facial pathway. The final result will be optimized from the cosmetic and the functional points of view. As long as some cortical and nuclear representation regain their connection with the facial muscles, vivid reinnervation with emotional expressivity will return. Therefore, pure plastic surgery will not mirror any vivid or emotional expression. Only nerve reconstructive measures projecting to and from the cortex can lead to innervation with mimic expression ([45]).

Timing is a decisive factor with regard to the quality of long-term outcome in nerve reconstruction as well as in nerve reanimation. This is a further consequence of the observations of nerve and muscle degenerations, and it is proven by clinical findings. Good results with complete eye closure were most likely achieved if the reconstruction was performed within the 1st year after onset of the palsy ([27, 51]).

Reanimation by hypoglossal-facial or by facio-facial combination?

In case of loss of the proximal facial nerve stump or in case of primary preserved nerve continuity and secondary degeneration up to the brain stem, a reanimation procedure can reactive the facial nerve. When compared with previous surgical trials with the accessory or phrenic nerves, surgery of the hypoglossal or the contralateral facial nerve produces far better results. From the physiological point of view, this is probably caused by the close connections of the facial and hypoglossal representations at motor cortex and brain stem levels; this is also reflected by the association of face and tongue movements in each healthy subject. Therefore, the refinement of facial movements after a reanimation procedure is especially subject to good physiotherapeutic exercises, and improvements may still be achieved 3 years after reconstruction. Comparing the hypoglossal and the contralateral facial nerve for reanimation procedures, one must anticipate a less powerful and a more delayed reinnervation with the contralateral facial nerve, and additional support by muscle transfer is therefore generally recommended([3, 9, 42]). The main indications for the less powerful facio-facial combination are in patients without functioning hypoglossal nerve or who are dependent on hypoglossal function. An alternative may be the partial use of the hypoglossal nerve ([1, 10]).

Nerve reanimation is definitely superior to pure plastic surgery, because the nerve reanimation will also bring about emotional expressivity. For these reasons, plastic surgery is helpful for patients with partial nerve palsies and its indication should be limited to them. Moreover, pure plastic surgery requires some repair and modification with time because of the elongation of fascia slings and lack of tone on the involved side of the face.

The effectiveness of nerve reconstruction

By intracranial nerve reconstruction and by hypoglossal-facial reanimation, more than 70% of treated patients achieve satisfactory results equivalent to Grade 3. This rate would be even higher if the patient population did not include several patients with intervals longer than 2 years between the onset of paralysis and the neurosurgical treatment. Although all the presented microsurgical measures are well established, they are mainly performed in centers for cranial base surgery or for nerve rehabilitation and their performance requires specific training and experience. Patients need to be transferred to a specific center before too many nerve nuclei and too many muscle fibers have undergone irreversible degeneration. Additional plastic surgery can be performed later, without any time limit.

Failures of nerve reconstruction?

Failures should not occur if the major principles of nerve microsurgery are applied (i.e., adequate indication, timing, technique, and postsurgical care). As mentioned above, the quality of the long-term results is greatly dependent on the early timing of surgery. Patients who are treated later than 1 or 2 years after the onset of palsy can regain facial activity, but some facial asymmetry at rest and especially during movement will persist and only weak or incomplete eye closure will be achieved, equivalent to Grade 4 (i.e., some lagophthalmus will persist and might need treatment by additional plastic surgery).

Moreover, microsurgical aspects have to be taken into consideration for optimal results. Special experience is necessary for the reliable evaluation of an adequate nerve stump, and precise microsurgical handling is mandatory to avoid tension, torquing, or incomplete nerve stump adaptation([51, 56]). A complete failure with virtually complete absence of recovery despite consideration of all listed aspects means that the adapted nerve endings were probably not vital enough. Reported less favorable results or complete failures have to be ascribed most likely to these technical difficulties ([23, 24]) and not to the method itself. The only exception might be considered in rare cases of NF in which tumorous nerve changes can always occur during nerve regeneration and can stop an ongoing process of reinnervation. We experienced such a situation in a female patient with NF-2 after she underwent surgery for the removal of her tumor at the temporal bone and face and nerve transplantation. After some reinnervation with initiation of eye closure within 1 year after nerve transplantation, secondary deterioration occurred and facial palsy increased. A malignant tumor regrowth in several of the tumors was noted.

Tarsorrhaphy is an effective method to overcome temporary problems of conjunctivitis and corneal ulcerations during the waiting period for reinnervation. Patients with negative Bell's sign, with severe muscle atrophy, and with trigeminal nerve deficits need special protection. In cooperation with the ophthalmologist, the indication for conventional lateral or medial tarsorrhaphy is determined. An alternative might be temporary adhesive eyelid weights that should not disturb eye opening and clear sight but prevent exsiccation ([37]). An hour-glass dressing is only a short-term solution. An additional trigeminal nerve deficit poses a serious problem, because this nerve is not to be reconstructed and the ability to use the reinnervated face is greatly dependent on sensation, as is known from the limbs. Physiotherapy with ice cooling activation might be helpful, but the final results will be always less favorable.

CONCLUSIONS

A basic goal of contemporary vestibular schwannoma therapy is the completeness of tumor resection along with preservation of facial nerve function. Anatomic facial nerve preservation in acoustic tumor resection is achieved at increasing rates by all authors. Independent of tumor size, overall preservation rates greater than 90% are reported. Functional facial nerve preservation is dependent on the microsurgical technique, which might be supported by facial electromyographic monitoring. The loss of facial nerve continuity at tumor resection poses a clear indication for nerve reconstruction. Depending on the exact site and on the extent of the neural defect, the mode of reconstruction must be decided on (either intracranial, intracranial-intratemporal, or intracranial-extracranial transplantation).

The loss of the proximal facial nerve stump poses a clear indication for a nerve reanimation procedure with the hypoglossal or with the contralateral facial nerve. All types of facial nerve reconstruction and reanimation are superior to any plastic surgery. The latter are indicated as supportive measures in incomplete palsies.

Despite the development and demonstrations of the above mentioned treatment options, patients with nerve discontinuity are frequently neglected and left without any treatment instead of being transferred to centers with special experience and operative and postoperative treatment programs. Facial palsy is a serious clinical condition frequently presented to clinicians of many specialties. The clinicians need to consult colleagues who are well familiar with the course of the palsy and its treatment options.

Combined trigeminal and facial nerve lesions pose a special problem to the patient because of the combined cosmetic and functional compromise and because impairment or loss of useful vision is sometimes caused by corneal scarring. Prophylactic temporary tarsorrhaphy during the period of facial nerve reinnervation is indicated.

For patients with partial facial paresis, specific physiotherapy is the first step of treatment. Additional correction using Pitanguy's plastic surgery, fascia lata sling, or temporal muscle transfer may be supportive. For each patient with facial paralysis, long-lasting cosmetic or functional problems can be avoided. Nerve reconstruction or nerve reanimation provides a reliable and satisfactory recovery, enabling a normal social and professional life.

ACKNOWLEDGMENT

We thank S. Brinkmann for preparation of the photographs.

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COMMENTS

We reported, in our discussion of the previous articles of this excellent series by Samii and Matthies, that it was possible to preserve the facial nerve in 97% in our recent series of 100 patients. At follow-up 1 year after surgery, the majority of the patients had no facial weakness, with the remainder having House-Brackmann Grade 3 or better and achieving at least complete eye closure. The three patients in whom the facial nerve could not be preserved underwent hypoglossal to facial nerve anastomosis. Our experience with intracranial facial nerve reconstruction in the cerebellopontine angle is limited to two patients, and it did not lead to a result superior to that seen with hypoglossal to facial nerve anastomosis. The hypoglossal to facial nerve anastomosis is completed during the early days or weeks after the removal of the tumor when it is known that the facial nerve cannot be preserved at surgery. We wait 11 or 12 months after the operation if there is facial paralysis but the nerve was preserved at the operation and may recover. Delays beyond that period are less likely to result in satisfactory functional reinnervation of the facial muscles. During the years, accessory to facial nerve anastomosis has been selected for a few patients who experienced disturbance of swallowing or in whom alteration of the voice was a major concern.

Our technique of facial nerve preservation is based on the use of anatomic landmarks and intraoperative facial nerve monitoring. In each operation, the facial nerve is identified both medial and lateral to the tumor before dissecting the tumor capsule from the intervening segment of nerve. The nerve is identified lateral to the tumor after removal of the posterior wall of the internal acoustic meatus as it passes above the transverse crest and behind the vertical crest in the anterosuperior quadrant of the meatus. It is identified at the brain stem as the tumor capsule is being removed from the brain stem based on three landmarks. The first is the pontomedullary sulcus, the sulcus separating the pons and medulla. The facial nerve exits the brain stem at the lateral end of the pontomedullary sulcus. The second landmark is related to a line drawn along the posterior margin of the inferior olive at the site where the glossopharyngeal, vagus, and accessory nerves enter the brain stem. These nerves are usually seen below the tumor during the early stages of tumor removal. The facial nerve arises from the brain stem, even when distorted by tumor, along this line 2 or 3 mm above where the glossopharyngeal enters the brain stem. The third landmark is related to the flocculus, which attaches to the brain stem along the margin of the lateral recess of the fourth ventricle, and the choroid plexus, which protrudes from the foramen of Luschka behind the IXth and Xth cranial nerves. The eight nerve, which is located just behind the facial nerve at the lateral end of the pontomedullary sulcus, joins the brain stem in front of the site of attachment to the flocculus to the margin of the foramen of Luschka and the lateral recess of the fourth ventricle. The choroid plexus protruding from the foramen of Luschka sits on the posterior surface of the ninth and tenth nerves, just behind and below where the facial nerve enters the brain stem. The use of these anatomic landmarks at the brain stem medial to the tumor and within the meatus lateral to the tumor, when combined with facial nerve monitoring, will assist in the goal of preserving the facial nerve during tumor removal. In our previous comments, we pointed out that anatomic preservation of the bundles of the eight nerve, even when they cannot be functionally preserved, acts as a protective barrier, frequently protecting the facial nerve in the interval between the brain stem and the meatus. Samii and Matthies present an excellent series of articles and make a magnificent contribution to neurosurgery.

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Samii and Matthies present their management strategy for and results of restoration of facial nerve function after surgery for acoustic neuromas. Their results emphasize the importance of early facial nerve reconstruction in instances of nerve discontinuity, achieving a "satisfactory result"(defined as House-Brackmann Grade 3 or better) in approximately 70% of 38 patients. In 62 patients treated with reanimation procedures, similar rates of improvement were noted. In their surgical management of acoustic neuromas, the authors emphasize preservation of facial nerve continuity and function, achieving anatomic preservation of the facial nerve in 93% of 1000 cases. A small number of patients (1.7%) did not demonstrate reinnervation at 10 to 12 months, despite maintenance of nerve continuity.

The authors conclude by emphasizing the availability of treatment options and the importance of close follow-up (including physiotherapy in many cases) in a center with

extensive experience in cranial base surgery. This is important with regard to the type of intervention and its timing. Their results support these conclusions and their approach.

However, the authors' point regarding the relative importance of complete tumor resection versus facial nerve preservation deserves comment. The authors suggest, under Discussion, that intentional sacrifice of the facial nerve during tumor resection and then reconstruction is preferable to careful radiographic and clinical follow-up with the possible need for further treatment. Section of the facial nerve seems a radical approach in the treatment of this disease at present for several reasons. First, the results presented for facial nerve reconstruction are encouraging, but recovery is not certain and is nearly always incomplete. Approximately 30% of the patients treated with reconstructive procedures in this series were left with Grade 4 or worse facial function. Second, the natural history of a small tumor remnant adherent to the facial nerve is variable, and no further treatment is required in some cases. Finally, although not directly comparable, the incidence of postradiosurgical "facial neuropathy" ranges from 10 to 32% ([1, 3]). Further, radiosurgical treatment of a small tumor residual or recurrence should be considered separately from primary radiosurgical treatment of acoustic tumors, because risk of deficit secondary to radiosurgical treatment decreases in tandem with target size ([2]). We agree that surgical resection of acoustic tumors is preferable to primary radiosurgical treatment but generally place anatomic preservation of the facial nerve over "total" tumor resection, preferring instead to use adjuvant radiosurgical treatment for a small volume residual tumor or recurrence.

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This article presents an excellent review of the problems and their solutions. The data is precise and well presented and describes the 15-year experience of a very experienced and talented neurosurgeon.

Although my numbers are slightly less than 50% of those presented by Samii and Matthies, my experience has generally been the same. On the other hand, I personally have found the reconstruction of the seventh nerve in the angle to be a very difficult and tedious procedure with which I have been singularly unsuccessful. As a consequence of that, I have abandoned it and have proceeded to hypoglossal facial anastomosis, the results of which have been previously reported in *Neurosurgery*.

With the excellent results of hypoglossal facial anastomosis, one can be more aggressive in removing the tumor, even at the expense of the seventh nerve. In my judgment, the biggest mistake is to injure the nerve and at the same time leave tumor. In general, if the seventh nerve has been stretched to the breaking point and very little viable nerve remains, I think that the nerve should be sacrificed and attention directed to removing the lesion. If this series of events happens, I usually wait 2 to 3 months to let the patient recover from the craniotomy before undergoing a hypoglossal facial anastomosis. In general, patients are very happy with the results of anastomosis.

I share the authors' viewpoint regarding plastic surgery. The hypoglossal facial anastomosis plus some minor ocular plastic surgery with gold weight in addition to the anastomosis usually produces excellent results.

In my judgment, this article presents a worthwhile compilation of data. It can be used as a gold standard.

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There is little question that partial or complete facial palsy is the major concern for patients considering surgery for vestibular schwannomas. The possibility of avoiding it is one of the reasons for the increase in the use of radiosurgery. In our experience, monitoring for facial nerve discharges during surgery is not always reliable. There are cases in which there seems to be no abnormal activity and yet there is diminished facial function postoperatively at least transiently.

Samii and Matthies discuss the management of those cases in which the facial nerve is lost during the surgical procedure. Elements of their management deserve emphasis. They recommend intracranial grafting or immediate anastomosis if possible and reasonably early hypoglossal-facial anastomosis if there is no evidence of reanimation within 10 to 12 months. In our experience, hypoglossal-facial anastomosis provides only modest facial function, especially with eye closure. Samii and Matthies suggest that two of three patients will achieve this with intracranial anastomosis but fewer with hypoglossal-facial. These data emphasize that useful facial recovery can be achieved, even in cases of large tumors, with appropriate techniques of aggressive nerve management.

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