

Postparotidectomy Facial Nerve Paralysis: Possible Etiologic Factors and Results With Routine Facial Nerve Monitoring

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Objective: Analyze the incidence and factors responsible for postparotidectomy facial nerve paralysis when the surgery is performed with the routine use of facial nerve monitoring. **Study Design:** A prospective, nonrandomized study. **Methods:** Seventy consecutive patients underwent parotidectomy with intraoperative facial nerve monitoring. Two devices were used: a custom mechanical transducer and a commercial electromyograph-based apparatus. All patients were analyzed, including those with cancer and those with deliberate or accidental sectioning of facial nerve branches. The outcome variables were the motor facial nerve function according to the House-Brackmann grading scale (HB) at 1 week (temporary paralysis) and 6 to 12 months (definitive paralysis). Facial nerve grading was performed blindly from reviewing videotapes. **Results:** The overall incidence of facial paralysis (HB > 1) was 27% for temporary and 4% for permanent deficits. Most of the deficits were partial, most often concerning the marginal mandibular branch. Temporary deficits with HB scores of greater than 2 were only present in patients with parotid cancer or infection. Permanent deficits were present in three patients, including one patient with facial nerve sacrifice. Factors significantly associated with an increased incidence of temporary facial paralysis include the extent of parotidectomy, the intraoperative sectioning of facial nerve branches, the histopathology and the size of the lesion, and the duration of the operation. **Conclusions:** Despite a stringent accounting of postoperative facial nerve deficits, these data compare favorably to the literature with or without the use of monitoring. An overall incidence of 27% for temporary facial paralysis and 4% for permanent facial paralysis was found. Although the lack of a control group precludes definitive conclusions on the role of electromyograph-based facial nerve monitoring in routine parotidectomy, the authors found its use very

helpful. **Key Words:** Parotidectomy, facial nerve, monitoring, surgery, nerve, paralysis.

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INTRODUCTION

Since it was first reported by Thomas Carwardine¹ in 1907, facial nerve preservation during parotidectomy has become the standard rather than the exception. Although the early 20th century surgeons identified a peripheral facial branch that was followed to the main trunk,² it remained for Janes³ to describe the routine identification of the facial nerve trunk at the beginning of the procedure, before proceeding with an anterograde dissection of facial nerve branches. Nevertheless, 60 years since the technique of parotidectomy was standardized, the factors associated with postparotidectomy facial nerve paralysis are still poorly understood.

Ninety-five publications giving postparotidectomy facial nerve results on approximately 12,750 patients can be found in the literature.⁴ Although such data could be approached through a meta-analysis, the retrospective nature of the studies and, more importantly, the lack of a standard evaluation method for grading facial nerve deficits would render such comparison meaningless. In the majority of papers, postparotidectomy facial nerve function is simply stated as abnormal without clearly specifying the criteria used. Only three studies have used an accepted facial nerve grading scale: Arndt et al.⁵ used the Stennert scale, and Olsen and Daube⁶ and Wolf et al.⁷ used the House-Brackmann (HB) grading system.

Notwithstanding these shortcomings, previously described risk factors associated with increased facial nerve paralysis include: 1) more extensive surgery, i.e. more facial nerve deficits with total versus superficial parotidectomy,⁸⁻¹⁴ 2) previous parotid surgery, i.e. more paralysis in recurrent cases,^{10,11,13} 3) malignant tumors,^{12,13,15} 4) lesion size,^{12,13} and 5) inflammatory conditions.^{9,15-18} There is no general consensus, because several studies did not confirm these factors.¹⁹⁻²¹

Mra et al.¹⁹ studied several factors in relation to postoperative paralysis in 65 patients with benign lesions and found only age as a statistically significant factor,

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although the population size may have been small for a multivariate analysis. A similar conclusion was reached by Laccourreye et al.²¹ on a larger population of 229 patients with pleomorphic adenoma. In another multivariate analysis study, Terrell et al.²² found operating time and age as significant factors.

Recently, commercial electromyograph (EMG)-based apparatuses have become available for continuous intraoperative monitoring of facial nerve function and have been widely used in complicated otological and neurotological procedures, although the exact indications are still debated.²³ We report on a prospective study of postparotidectomy facial nerve outcome in cases in which such devices were used during parotidectomy.

METHODS

This was a prospective, nonrandomized study of patients undergoing parotidectomy. From April 1994 to April 1998 all patients undergoing parotid surgery at the Clinic of Otolaryngology Head and Neck Surgery of the University of Geneva were invited to participate in this trial. Besides the usual preoperative explanation and consent, all patients underwent a preoperative evaluation of facial nerve function.

In all patients parotidectomy was performed using standard surgical techniques and intraoperative facial nerve monitoring. In the first 35 patients a custom mechanical transducer was used, while, since April 1996, a commercial EMG-based nerve monitoring device was employed. The mechanical transducer was placed in the mouth between the cheek and the teeth. The apparatus transduced the mechanical pressure generated by the contraction of the buccinator, and possibly other midfacial muscles, into an audible alarm. This system provided feedback on stimulation of either the buccal branches or of the entire facial nerve. The EMG-based facial nerve monitor (Neurosign 100, Magstim Company Inc., Spring Gardens, UK) consists of a differential electromyographic recording on two channels. Because of the importance of eye and mouth closure in facial motor function, the orbicularis oculi and oris were monitored.²⁴ Qualitative auditory and semiquantitative visual feedback of the stimulated branches or nerve trunk is provided.

During the surgical procedure facial nerve handling was as atraumatic as possible. The number of facial nerve stimulations was limited to the fewest necessary, with the minimal amount of current. When the mechanical transducer was used, monopolar commercial stimulators were used with the lowest electrical current setting that generated a twitching response. For the EMG-based nerve monitor, the provided bipolar electrical stimulating probe was used at current levels less than 0.05 mA, sufficient to generate a sound from the monitoring device, but without producing facial muscle twitching.

The parotidectomy operations were classified into three types: superficial, total, and radical parotidectomy. While enucleation was never performed, some procedures classified as "superficial" parotidectomy could have been better named "lateral superficial" parotidectomy.¹⁸ Also, some procedures classified as "total" parotidectomy should be named "near-total" parotidectomy.¹⁸ These were all primary parotidectomies, because no revision surgery was done during this period.

Facial branches that were intentionally sacrificed or accidentally damaged were noted on an ad hoc drawing. In the analysis section of a facial nerve branch (usually small peripheral branches) in the context of a parotidectomy for a benign tumor was always classified as accidental. Deliberate sacrifice of the facial nerve or its branches was not an exclusion criterion. Therefore, all patients operated on were included in the data analysis.

Besides the type of parotidectomy and the possible section of any facial nerve branches, the other intraoperative variables examined included the duration of the procedure and the type of facial nerve monitoring device used. The histopathology of the removed parotid lesions was classified according to the World Health Organization classification.²⁵ The size of the lesion was estimated by the pathologist by measuring the macroscopic lesion within the parotidectomy specimen.

Facial motor function was evaluated before surgery and at 1 week and approximately 6 months after surgery. For patients with abnormal facial nerve function at 1 week, the evaluation was repeated at monthly intervals until normalization. Facial nerve function was graded according to the HB scale.²⁴ The method used was somewhat different from the usual clinical grading because subjects were videotaped during the facial nerve evaluation. For videotaping, subjects were sitting comfortably on a customized chair with a headrest, to provide for a head support and to minimize head movements. Facial landmarks, similar to those used by Burres,²⁶ were placed with a colored (blue) eyeliner pen. The videotapes were graded by authors who were not involved in the patient's care. Final objective grading of the facial motor function of these patients using digital techniques will be the subject of a future report.

The relationship between continuous data (age, duration of surgery, diameter of the tumor) and HB scores was analyzed with the Pearson correlation test. Average HB scores and other categorical data were compared with the exact Fisher test. The statistical algorithms of the SPSS 7.5 software (SPSS Inc., Chicago, IL) were used.

RESULTS

During the 4-year study period 73 patients underwent a parotidectomy. Two patients with a complete preoperative facial nerve paralysis (HB 6) were excluded and one patient refused to participate. In all 70 patients were enrolled. The population was composed of 40 males and 30 females for male-female ratio of 0.57. The average age was 50 ± 17 years, with a range of 12 to 83 years. The right side was involved in 37 cases and the left side in 33.

Superficial parotidectomy was performed in 43 cases (62%), total parotidectomy in 26 cases (37%), a radical parotidectomy in 1 case (1%). For the analysis total parotidectomy and radical parotidectomy were grouped together. A neck dissection was performed in six cases (7%), a supraomohyoid selective neck dissection in five cases, and a radical neck dissection in one case.

The pathological diagnosis was a benign process in 62 cases and a malignancy in 8 cases (Table I). Benign processes were divided into adenomas (48 cases), nonepithelial tumors (2 cases), tumor-like lesions (9 cases), and infections (3 cases). The adenoma group was composed of 37 pleomorphic adenomas (52.9%), 9 adenolymphomas, and 2 other monomorphic adenomas (1 basal cell and 1 canalicular). The tumor-like lesions group was composed of six cysts (three lymphoepithelial cysts, one lymphoepithelial cyst associated with AIDS, and two salivary duct cysts), and three intraparotid adenopathies. The infection group was composed of one parotid abscess, one parotid tuberculosis, and one chronic sialadenitis. The malignant lesions were divided into six carcinomas and two melanomas. The average size of the parotid lesions removed was 2.4 ± 1.2 cm. Lesions were classified according to size into

TABLE I.
Histology of Parotidectomy Specimens.

Histopathology	N	%
Total	70	100
Adenomas	48	68.6
Pleomorphic adenoma	37	52.9
Monomorphic adenoma	11	15.7
Adenolymphoma (Warthin's tumor)	9	12.9
Other monomorphic adenoma	2*	2.8
Carcinomas	6	8.6
Acinic cell	1	1.4
Mucoepidermoid carcinoma	2	2.9
Adenoid cystic carcinoma	1	1.4
Adenocarcinoma	1	1.4
Carcinoma ex-pleomorphic adenoma	1	1.4
Nonepithelial tumors	2†	2.9
Malignant lymphomas	0	
Secondary tumors (metastasis)	2‡	2.9
Tumor-like lesions	9§	12.9
Infections	3	4.3

*Other monomorphic adenomas: 1 basal cell and 1 canalicular.

†Nonepithelial tumors: 1 lipoma and 1 neurinoma.

‡Secondary tumors: 2 metastatic melanomas.

§Tumor-like lesions: 6 cysts, 3 parotid lymphadenopathies.

three groups: < 3 cm (n = 52; 74%), 3 to 5 cm (n = 13; 19%), and > 5 cm (n = 5; 7%).

Clinical evaluation using the HB scale showed that all 70 patients had normal preoperative facial nerve function. The average score on postoperative day 7 was 1.43 ± 0.90 . The facial function was normal (HB 1) in 51 patients (73%) and close to normal (HB 2) in 13 patients (18.6%). Long-term facial function was evaluated in 67 patients, because 3 patients with abnormal postoperative facial function had not reached the minimal follow-up delay of 6 months. The facial function was normal in 64 patients (96%), with 2 patients having a HB score of 2 (Table II). One of these patients had a 3.5-cm melanoma parotid lesion and had had several small peripheral branches cut. He died 8 months after surgery and therefore might have regained a normal facial function. The second patient had a 4.5-cm abscess of the lower portion of the gland and the

TABLE II.
Facial Function Scores of the Entire Population.

House-Brackmann Score	Postoperative day 7		Long-term (>6 mo)	
	N	%	N	%
1	51	72.9	64	95.5
2	13	18.6	2	3.0
3	4	5.7	0	0.0
4	0	0.0	0	0.0
5	1	1.4	1	1.5
6	1	1.4	0	0.0
Total	70	100.0	67	100

marginal mandibular nerve was inadvertently sectioned. Despite an epineural neuroorrhaphy the patient never regained normal long-term facial nerve function. The final patient with persistent facial paralysis had a high-grade adenocarcinoma and underwent a radical parotidectomy with facial nerve sacrifice and grafting.²⁷ Therefore, only patients who had undergone sectioning of a portion of the facial nerve had persistent deficits. The average score in the long-term population was 1.09 ± 0.51 . In view of the paucity of persistent facial nerve function deficits, the remaining facial nerve data will be limited to the postoperative data.

Patient Demographics and Postoperative Facial Function

No clear influence on postoperative HB score was obvious for patient sex or age, or for the side of the lesion.

Parotidectomy and Postoperative Facial Function

The 43 patients who underwent a superficial parotidectomy had an average score on postoperative day 7 of 1.21 ± 0.46 . The facial function was normal in 35 (81%) patients and close to normal (HB 2) in 7 (16%). The 27 patients who underwent a total parotidectomy had an average score on postoperative day 7 of 1.77 ± 1.28 . The facial function was normal in 16 patients (59%) and close to normal (HB 2) in 6 patients (22%). The difference in terms of HB scores between superficial and total parotidectomy is statistically significant ($P = .035$) (Fig. 1).

Role of Facial Nerve Branch Sectioning

In eight patients a portion of the facial nerve was cut during the procedure. These cases include the previously discussed facial nerve trunk sacrifice in the adenocarcinoma case, the inadvertent section of the marginal mandibular nerve, and six patients in whom small peripheral branches were sectioned. Only two of these patients had a normal postoperative HB score. The average postoperative HB score in patients with cut facial nerve branches is 2.63, compared to 1.27 for patients with intact facial nerve ($P < .01$). The average long-term HB score in patients with cut facial nerve branches is 1.63, compared with 1.02 for patients with an intact facial nerve ($P = .036$) (Fig. 2).

Histopathology and Postoperative Facial Function

The distribution of the postoperative HB scores according to the histopathology of the parotidectomy specimen is shown in Figure 3. The 37 patients with pleomorphic adenomas had either normal (HB 1: 28 patients [76%]) or near normal (HB 2: 9 patients [24%]) postoperative facial function. The 11 patients with monomorphic adenomas had either normal (HB 1: 10 patients [91%]) or near normal (HB 2: 1 patient [9%]) postoperative facial function. Both patients with benign nonepithelial tumors had normal (HB 1) postoperative facial function. The nine patients with tumor-like lesions had either normal (HB 1: 7 patients [78%]) or near normal (HB 2: 2 patients [22%]) postoperative facial function.

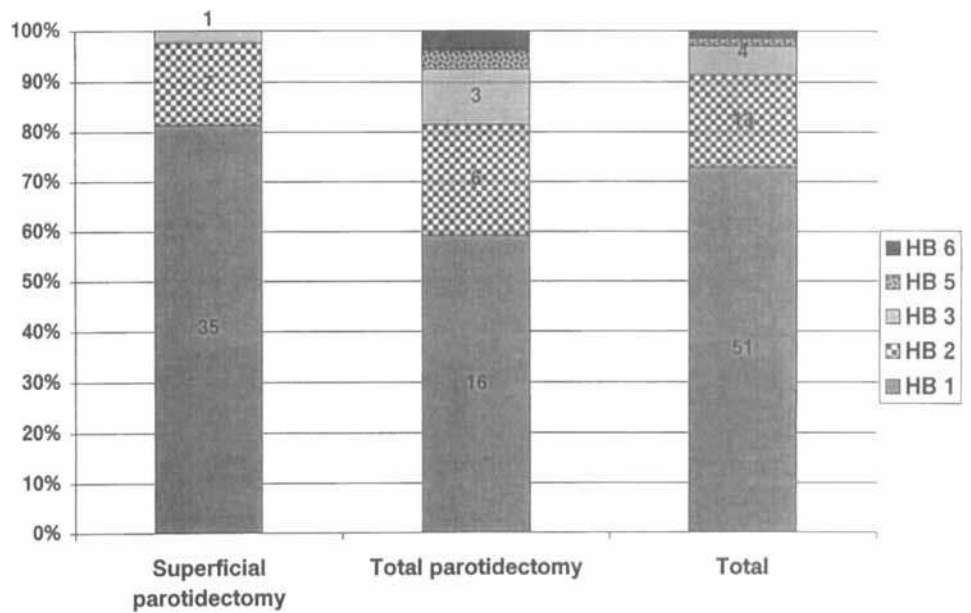


Fig. 1. Distribution of postoperative facial nerve grading scores (House-Brackmann [HB]) according to the type of parotidectomy performed.

Patients with infections ($n = 3$) had less optimal results: only one patient (33%) had a normal postoperative facial function, whereas two patients had a HB score of 3 (67%). One of these patients had a large lesion (5 cm) of tuberculosis involvement of the parotid. The second patient with a large intraparotid abscess was previously discussed. This case is the only noncancerous lesion with long-standing facial motor deficit.

Patients with primary ($n = 6$) or secondary ($n = 2$) parotid malignant lesions also fared less well than those with benign lesions. Only three patients (37.5%) had normal postoperative facial function, whereas the remaining five patients had HB scores of 3 or worse. One of these patients had a facial nerve sacrifice with a sural nerve graft for an adenocarcinoma.

Figure 4 shows the lesions grouped as benign tumors, infections, and malignant lesions. In the 59 patients with benign tumors the average postoperative score was 1.20; the facial function was normal in 47 patients (80%) and close to normal (HB 2) in 12 (20%). Therefore no important facial nerve deficit (HB > 2) was found in patients operated on for benign tumors. Patients with infections had an average postoperative score of 2.33 and those with cancer had an average postoperative 2.75. The difference in terms of HB scores between these 3 groups is significant ($P < .001$).

Lesion Size and Postoperative Facial Function

The 52 patients with lesions smaller than 3 cm had an average postoperative score of 1.29 ± 0.49 . The facial

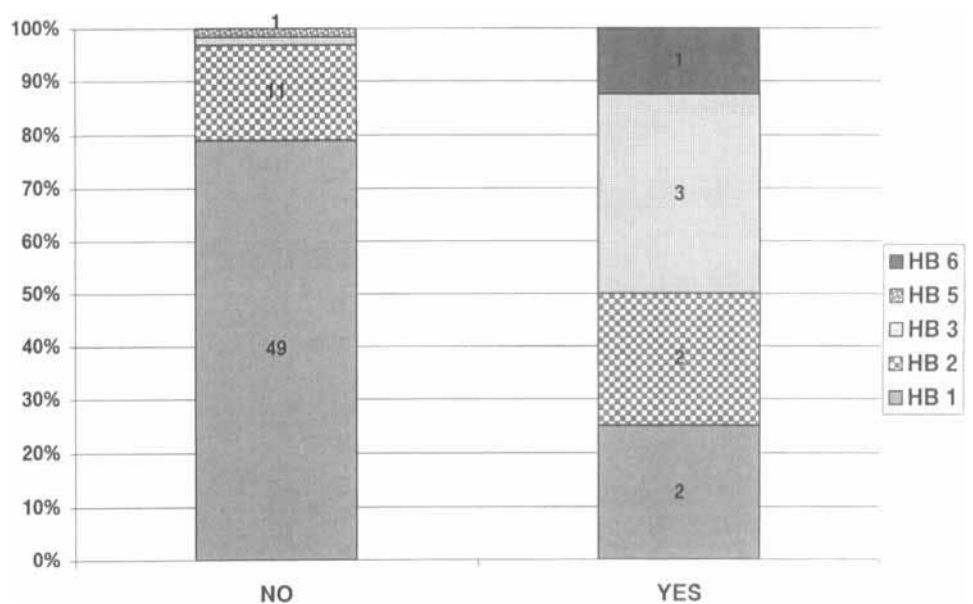


Fig. 2. Distribution of postoperative facial nerve grading scores (HB) according to whether a part of the facial nerve was sectioned.

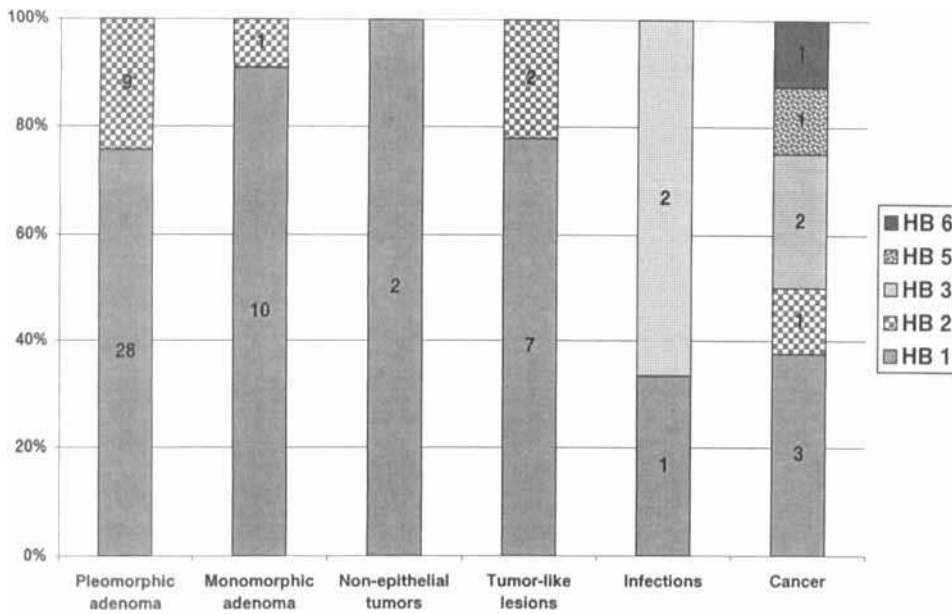


Fig. 3. Distribution of postoperative facial nerve grading scores (HB) according to the histopathology of the parotid lesion.

function was normal in 38 (73%) patients and close to normal (HB 2) in 13 (25%). The 13 patients with lesions between 3 and 5 cm had an average postoperative score of 1.61 ± 1.26 . The facial function was normal in 10 (77%) patients, the remaining 3 having HB scores worse than 3. In lesions bigger than 5 cm, the average postoperative score was 2.4 ± 2.2 . The correlation coefficient between lesion size and HB scores was significant ($P = .025$) (Fig. 5).

Parotidectomy Duration and Postoperative Facial Function

The average duration of the procedure was 148 ± 57 minutes. Patients with normal facial function had an average duration of 137 ± 50 minutes, patients with a HB score of 2 had an average duration of 147 ± 43 minutes, and patients with HB scores greater than 2 had an aver-

age duration of 240 ± 76 minutes. The duration of parotidectomy was well correlated with the postoperative HB score ($r = .52$; $P < .01$) (Fig. 6).

Intraoperative Monitoring Technique and Postoperative Facial Function

Patients monitored with the balloon mechanical transducer had an average postoperative score of 1.60 ± 1.17 . The facial function was normal in 24 of 35 patients (69%) and close to normal (HB 2) in 6 patients (17%). Patients monitored with the EMG-based device (Neurosign) had an average postoperative score of 1.26 ± 0.51 . The facial function was normal in 27 of 35 patients (77%), and close to normal (HB 2) in 7 patients (20%). The difference between the two types of monitoring devices did not reach statistical significance (Fig. 7).

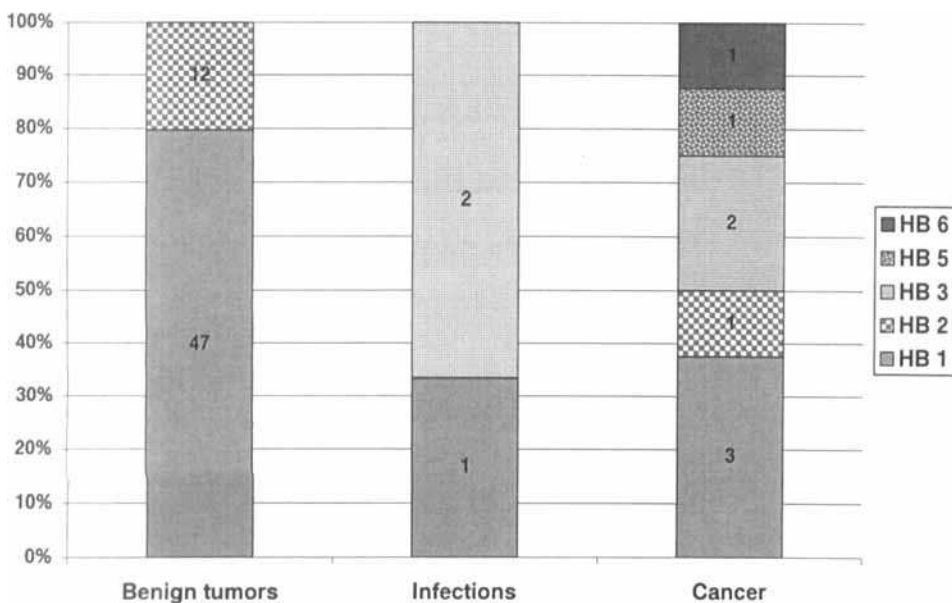


Fig. 4. Distribution of postoperative facial nerve grading scores (HB) according to the histopathology of the parotid lesions, grouped into benign lesions, infections, and cancer.

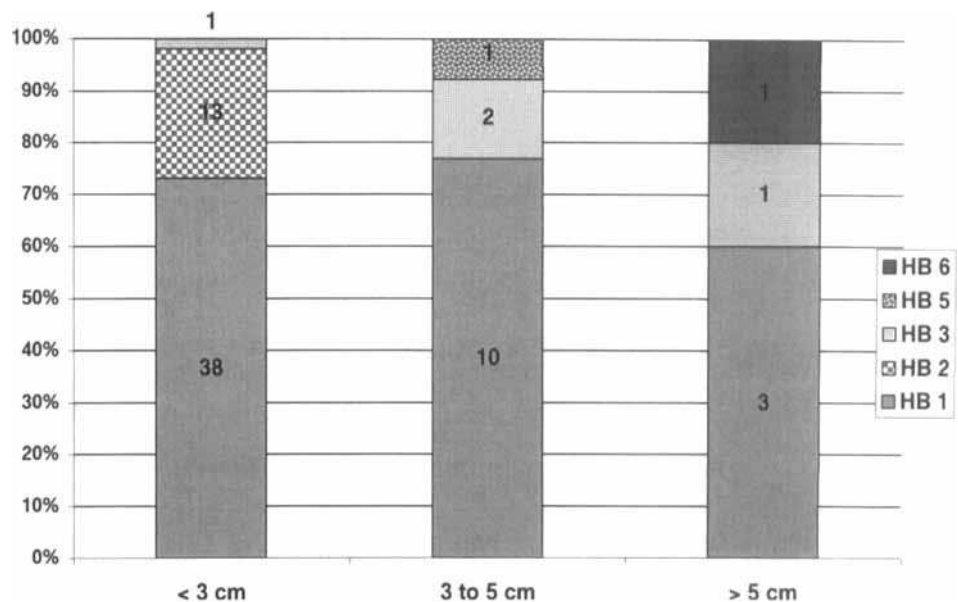


Fig. 5. Distribution of postoperative facial nerve grading scores (HB) according to the size of the parotid lesion removed.

Patients With Poor Postoperative Facial Function

Only six patients (8%) in this series had a postoperative (day 7) HB score worse than 2. Their main characteristics are shown in Table III. The majority of these patients had a total parotidectomy, a diagnosis of either infection or cancer, and large lesions.

DISCUSSION

Contrary to previous studies, the data presented were prospectively collected and there was no specific patient selection. In addition, facial motor function was evaluated according to an established grading system.²⁴ Furthermore, the evaluation of the facial data was performed from a videotape review and in a blinded fashion. These features make meaningful comparison of our data with previous publications difficult.

Overall, normal facial function (HB 1) was present in 73% of our patients on postoperative day 7 and in 96% on a prolonged follow-up of 6 months or more. Stated otherwise, 27% of patients had some form of postoperative facial deficit and 4% (including one patient with nerve sacrifice) had a long-term deficit. If the population is restricted to benign tumors, the incidences are respectively 20% for temporary deficits and 0% for permanent paralysis. In recent publications the incidence of temporary deficits was 18% for O'Brien et al.¹⁸ and Watanabe et al.,¹² 37% for Bron et al.,¹⁵ 46% for Mehle et al.,¹¹ 52% for Ruaux et al.,¹³ 62% for Terrell et al.,²² 65% for Laccourrye et al.,²¹ and 68% for Wolf et al.⁷ In the same publications the range of long-term deficits is from 0%^{7,12} to 19%.¹⁸

Whether the use of a routine continuous intraoperative EMG-based facial nerve monitoring has a significant impact

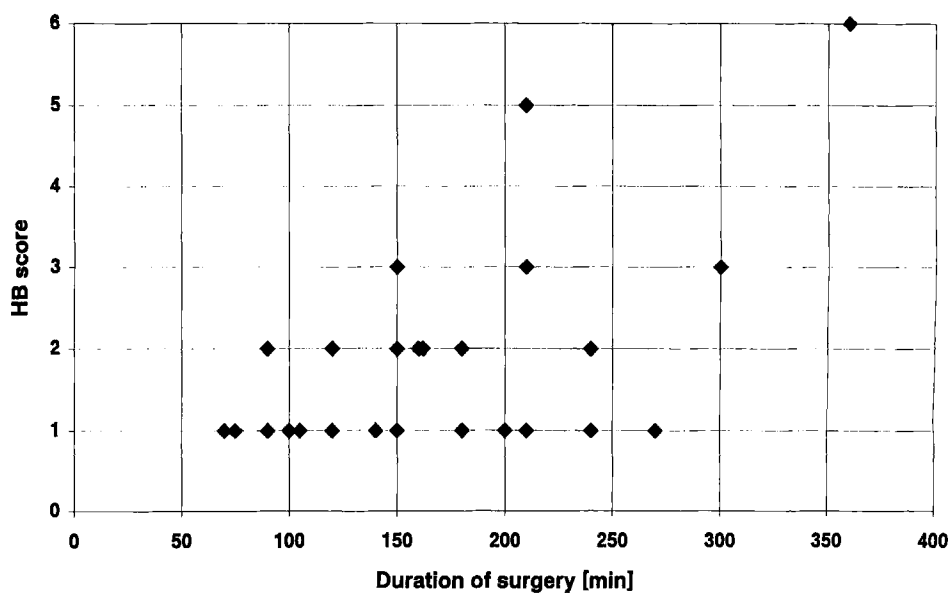


Fig. 6. X-Y plot of postoperative facial nerve grading scores (HB) versus the duration of parotidectomy.

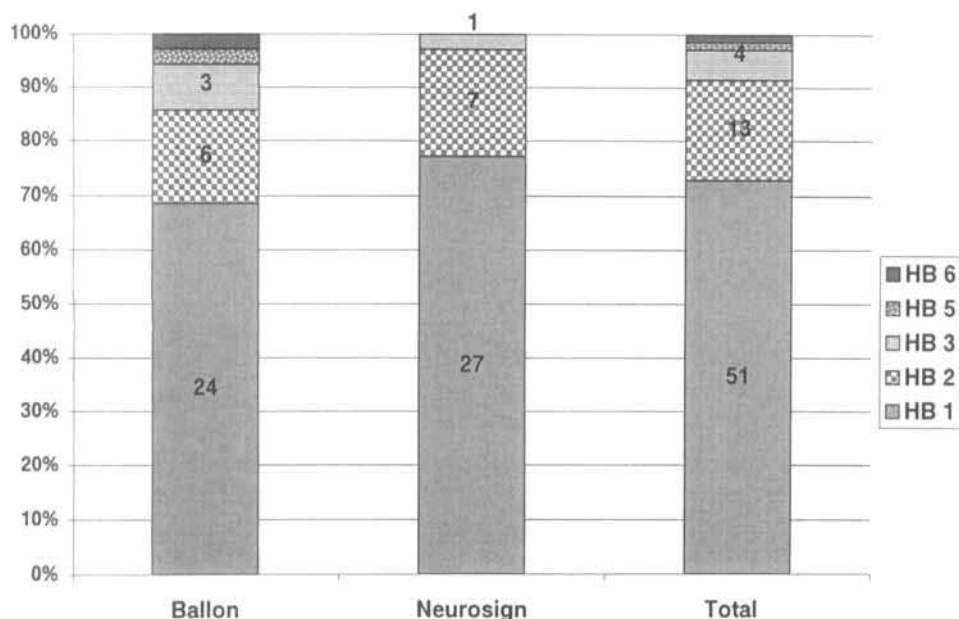


Fig. 7. Distribution of postoperative facial nerve grading scores (HB) according to the type of facial nerve monitoring device used.

on postoperative facial function remains debatable. Our study did not include a control population and therefore our data should not be used as an argument for the superiority of EMG-based facial monitoring in postparotidectomy facial outcome. Two retrospective, nonrandomized studies have compared EMG-monitoring with traditional parotidectomy. Wolf et al.⁷ found HB scores of greater than 1 in 69% of monitored versus 75% of unmonitored patients (no statistical analysis provided). Terrell et al.²² reported on abnormal facial function (author's scale of deficit grading) in 44% of monitored and 62% of unmonitored patients ($P = .04$). While these data are in favor of the routine use of EMG-based facial nerve monitoring, only a prospective, randomized study, after stratification for the risk factors discussed below, could settle the role of routine facial nerve monitoring in parotid surgery. Our opinion is that such a study might be unethical. When we experienced some technical problems with the EMG apparatus,²⁸ we rescheduled the patients until the equipment was repaired.

The factors associated with a higher incidence of a temporary facial nerve deficit include the extent of surgery (superficial vs. total parotidectomy), the sectioning of facial nerve branches during surgery, the histopathology,

the size of the lesion, and the duration of the operation. As discussed earlier, several of these factors have been previously reported, although their exact significance remains controversial. Obviously, these factors are interrelated and a multivariate analysis is a valid solution. Interestingly, two multivariate studies found patient's advanced age as the mean factor associated with postparotidectomy facial deficits.^{21,22} We were unable to confirm these findings and did not perform a multivariate analysis because of the relatively small size of our population.

Although the role of these factors deserves further study, most of them cannot be directly controlled by the surgeon. More interestingly, the exact physiopathological mechanisms of postsurgical nerve paralysis are still poorly understood. Patey logically classified postparotidectomy facial paralysis as secondary to: 1) deliberate sacrifice of the facial nerve or its branches, 2) inadvertent but recognized section of the facial nerve or branches, and 3) unclear causes while the anatomical integrity of the nerve is intact.⁹ In one of the rare experimental studies addressing this issue, Patey and Moffat⁸ found the rabbit facial nerve "quite resistant to direct mechanical trauma" and sensitive to cooling (although the temperature was not

TABLE III.
Main Characteristics in Patients With Postoperative House-Brackmann Score > 2.

Patient	Age (y)	Parotidectomy	Histopathology	Lesion Size (cm)	Monitoring Device
AL	68	Superficial	Infection (tuberculosis)	5.0	Balloon
BM*	57	Radical	Cancer (adenocarcinoma)	5.0	Balloon
LC	73	Total	Cancer (melanoma)	1.5	Neurosign
HR†	64	Total	Infection (abscess)	4.5	Balloon
RW	65	Total	Carcinoma ex-pleomorphic adenoma	3.5	Balloon
GSL	51	Total	Cancer (adenoid cystic)	4.5	Balloon

*Facial nerve sacrifice with neurorraphy.
†Marginal mandibular nerve section.

measured), and they speculated against nerve edema and for the possible role of nerve ischemia. The role of nerve ischemia as a direct etiology can probably be safely ruled out because numerous animal experimental studies have shown peripheral nerves to be quite ischemia-resistant. For example, rat sciatic nerves withstood 3 hours of complete lower limb anoxia before irreversible damage could be found in the compound action potential.²⁹ Cooling is also an unlikely etiology, although data on facial nerve temperature during parotidectomy have never been published, because nerve cooling to 20°C has repeatedly been shown to be unharmed.³⁰

Mechanical trauma can be separated into compression, crushing, and stretching (see Lundborg et al.³¹ for a review). Apparently, peripheral nerves can withstand compressions of approximately 100 mm Hg (13 kPa, 2 lbf/in²) before the nerve microcirculation becomes impaired, resulting in a metabolic conduction block.³¹ At higher and sustained pressures, focal demyelination takes place, which requires 6 to 12 weeks for a complete recovery.³² Again it seems unlikely that during a careful parotidectomy the facial nerve will be compressed with such high pressures. Crushing of peripheral nerves with surgical forceps reliably produces a mechanical deformation of the myelin sheaths, resulting in a segmental demyelination,³¹ which also requires 6 to 12 weeks for recovery. While crushing of facial nerve branches could occur, it seems to be a rare phenomenon in careful parotidectomy. The most probable mechanical factor involved is nerve stretching. Peripheral nerves have been found to follow a peculiar stress-strain curve with zones of straightening and elastic elongation, followed by mechanical rupture (see Grewal et al.³³ for a recent review). Earlier data showed that rupture occurs at 38% elongation, but more recent studies have demonstrated perineurium tears with disturbances of the intrafascicular homeostasis at elongations of 6%.³³ The resulting edema further impedes the microcirculation of the nerve and results in an unrecoverable loss of the compound action potential. During such trauma the nerve remains grossly normal. It is easy to imagine how such nerve stretching could happen during parotidectomy.

Other possible etiologies of nerve damage include heat damage from electrocoagulators (unlikely without massive nerve twitching), damage from overzealous nerve stimulation (unlikely in view of experimental and clinical data with various functional electric stimulating implants), and damage from neurotoxic substances placed in the surgical wound (unlikely). Therefore, experimental animal data point to nerve elongation as the most probable factor involved in anatomically intact facial nerves associated with postparotidectomy facial paralysis.

CONCLUSION

In this study of unselected patients the overall incidence of facial paralysis is 27% for temporary deficits and 4% for long-term deficits. Important temporary facial nerve deficits (HB > 2) were not found in patients undergoing parotidectomy for benign tumors. Permanent deficits were present only in patients who had a section of nerve branches. Factors associated with an increased in-

cidence of temporary facial paralysis include the extent of parotidectomy, the intraoperative sectioning of facial nerve branches, the histopathology and the size of the lesion, and the duration of the operation. We found the routine use of an EMG-based facial monitoring to be helpful during routine parotid surgery. A review of the pathophysiological factors possibly responsible for facial nerve deficits points to nerve stretching as the most probable etiology.

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