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Facial nerve function in carcinoma of the parotid gland

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ABSTRACT

Aim: To analyse, for patients with carcinoma of the parotid gland, the prognostic value for treatment outcome of the function of the facial nerve (NVII), and determining facial nerve dysfunction after treatment.
Methods and materials: In a retrospective study of the Dutch head and Neck cooperative group (NWHHT), data of 324 patients with parotid carcinoma were analysed. The function of N VII before treatment was intact in 77%, partially and completely impaired in 14% and 7%, respectively. Eighty-eight percent of the patients were treated surgically, and 77% of them were treated by a combination of postoperative radiotherapy. In 21% NVII was sacrificed, a reconstruction was performed in one of three.
Results: Independent risk factors for N VII dysfunction before treatment were tumour localisation, positive neck nodes at presentation, pain, increasing age, and perineural invasion. Regional, not local, control was significantly impaired for complete facial paralysis. N VII dysfunction was an independent factor for disease free survival, and was 69%, 37% and 13% for normal, partially and completely impaired function, respectively. After treatment 22% of the patients experienced a partial paralysis, and 13% of the patients experienced a complete parlysis of N VII.
Conclusion: For patients with parotid carcinoma, facial nerve function before treatment is a strong prognostic factor for disease free survival.

1. Introduction

In a retrospective study of the Dutch Head and Neck Oncology Co-operative Group (NWHHT), data concerning treatment of salivary gland carcinomas were analysed. Data were collected in the University hospital of Utrecht and were obtained from all the participating centres of the NWHHT (Free University hospital of Amsterdam, University Medical Centres of Groningen, Leiden, Maastricht (in conjunction with the Maastro clinic), Nijmegen, Rotterdam and Utrecht and the Dutch

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Cancer Institute, Antoni van Leeuwenhoek Hospital). In this retrospective study, salivary gland tumours of the parotid and submandibular glands, the minor salivary glands of the oral cavity and the pharynx/larynx were included. The study was co-ordinated by the first two authors. General results, prognostic factors for loco-regional control, distant metastases and survival were published recently.[1–3] For this article, we focussed on patients with a malignant tumour of the parotid gland, with emphasis on the function of the facial nerve (N VII) and prognostic factors related with this function. The likelihood of facial nerve dysfunction after treatment, based on pre-treatment N VII function and treatment performed, is also analysed.

2. Materials and methods

Data regarding 332 patients treated between 1985 and 1994 for a salivary gland carcinoma of the parotid gland were analysed. The mean follow-up of patients alive at last follow-up was 98 months. Data concerning facial nerve function were available in 324 patients. The tumour was located in the lateral lobe in 67%, in the medial lobe in 9%, and in both lobes in 21%. In 4% of the cases the location could not be redesigned in retrospective. Age ranged between 8 and 100 years, and the mean age was 60 years. Forty-five percent of the patients were female.

Clinical T-stage (UICC 1992) was 18%, 36%, 21%, 10% and 16%, for T1, T2, T3, T4 and T unknown, respectively. Clinical N-stage was N0 in 83%, N1 in 4%, N2 in 10%, N3 in 1% and N unknown in 1%. Including results accumulated from the neck dissection specimen (applied in 101 patients), and a single positive node found in the local resection specimen ($n = 8$), 26% was clinical/pN+. Distant metastases at first visit were seen in 8 patients.

Thirty percent of the patients complained of pain and in 11% the skin was invaded by tumour.

Distribution of histological subtype according to the WHO 1972 classification (WHO 1972) was acinic cell carcinoma in 17%, muco-epidermoid carcinoma in 16%, adenoid cystic carcinoma in 14%, adenocarcinoma not otherwise specified (NOS) in 25%, carcinoma ex pleomorphic adenoma in 9%, squamous cell carcinoma in 7%, undifferentiated carcinoma in 10% and others in 2%.

The function of the facial nerve before treatment was intact in 77% ($n = 255$), partially impaired in 14% ($n = 45$) and complete paralysis was noted in 7% ($n = 24$).

Treatment consisted of surgery alone in 11%, surgery combined with postoperative radiotherapy in 77%, radiotherapy alone in 8%, chemotherapy alone in 2 patients and chemotherapy followed by surgery and radiotherapy in another 2 patients, no treatment was given to 11 patients (3%). In Table 1 correlation between facial nerve function and treatment delivered is given.

For local surgical treatment local excision was performed in 5%, superficial parotidectomy in 30%, total parotidectomy with preservation of the facial nerve in 43% and with sacrifice of the facial nerve in 21% (see Table 1). In two patients, debulking was performed followed by postoperative radiotherapy. In 10/25 patients with preoperative normal facial nerve function reconstruction of the facial nerve was attempted, for partial and complete facial nerve dysfunction in 6/24 and 3/11, respectively.

In the primary radiotherapy and no-treatment group 79% had T3–T4 disease, 29% had clinically positive neck nodes, 16% of these patients had distant metastases at first visit, and only 48% had normal function of the facial nerve.

2.1. Statistics

For univariate analysis the SPSS/PC, version 10.1 program was used. Statistical significance was calculated using the χ^2 test and Mann–Whitney test. For actuarial curves the method of Kaplan–Meier was used, with the log rank test for computing statistical significance. For multivariate analysis, the Cox step-by-step proportional hazards regression was used.

3. Results

3.1. Correlation between facial nerve function before treatment and patient and tumour characteristics (see also Table 2)

There was no significant correlation between sex, duration of complaints, histological subtype (WHO 1972 classification) and facial nerve function before treatment. Age marginally correlated with facial nerve function.

Table 1 – Correlation between treatment and facial nerve function *before* treatment

	Surgery ($n = 37$)	Surgery + RT ($n = 247$)	Radiotherapy ($n = 25$)	No treatment/others ($n = 15$)
All patients (324)				
Intact N VII ($n = 255$)	12%	79%	5%	4%
Partial paralysis ($n = 45$)	13%	73%	11%	2%
Complete paralysis ($n = 24$)		50%	33%	17%
	Local excision ($n = 15$)	Superficial parotidectomy ($n = 86$)	Total parotidectomy N VII pre-served ($n = 123$)	Total parotidectomy, N VII sacrificed ($n = 61$)
Selection surgery performed, type of surgery (284)				
Intact N VII ($n = 234$)	6%	34%	49%	11%
Partial paralysis ($n = 39$)	–	13%	23%	64%
Complete paralysis ($n = 12$)	–	–	8%	92%

Table 2 – Correlation between facial nerve function *before* treatment and tumour and patients characteristics (*p*, probability, ns, not significant)

	Intact	Partial paralysis	Complete paralysis	*p*
Mean age (years)	59	66	62	0.06
% Female	46	44	38	ns
Median duration of symptoms (months)	6	6	6	ns
% With pain	26	54	58	<0.001
% With tumour invasion of skin	8	20	27	0.004
% T1–T2–T3–T4	24–44–23–9	12–42–29–17	9–27–27–36	0.005
% Clinical/pN+	22	44	35	0.001
Localization				
% Lateral lobe	76	33	29	
% Medial lobe	6	20	17	<0.001
% Lat. + med. lobe	15	40	50	
% Unknown	3	7	4	
Histological subtype				
% Acinic	18	16	8	
% Muco-epidermoid	16	18	13	
% Adenoid cystic	13	18	17	
% Adenocarc. NOS	24	24	46	ns
% Carc.ex pl.adenoma	9	7	4	
% Squamouscell ca.	7	9	8	
% Undifferentiated ca.	11	9	4	

The percentage of pain and clinical tumour involvement of the skin was about twice for facial nerve dysfunction compared to normal function.

T- and N-stage correlated significantly with facial nerve function. Tumour location predicts facial nerve function significantly with predominance for normal function for tumours located in the lateral lobe and dysfunction for tumours located in the medial lobe or both lobes.

In 284 patients data were obtained from the resection specimen. Facial nerve function before resection did not correlate significantly with bone invasion, status of the resection margin, histological invasion of the skin, and vascular invasion. Perineural invasion, however, was strongly correlated ($p < 0.001$) with pre-treatment facial nerve function, respectively, 22%, 56%, and 83% for normal, impaired, and complete paralysis of the facial nerve.

In 95 patients a neck dissection was performed. Pre-treatment facial nerve function did not correlate with extranodal disease, number of neck nodes, and only marginally with status of the 'neck' resection margin.

With logistic regression analysis independent variables for yes or no normal facial nerve function were analysed. In the model the *univariate* significant variables tumour localisation, T- and N-stage, age, pain and skin invasion were incorporated. *Independent* prognostic factors were localisation, with a high risk of dysfunction for tumours originating from the medial lobe, N-stage, pain and age (see Table 3). Perineural invasion, as obtained from the resection specimen, was the only independent correlating factor for nerve facial function.

3.2. *Local-regional control*

Absolute local control rate for pre-treatment intact, partially impaired and complete paralysis of the facial nerve was 84%, 73% and 54%, respectively ($p = 0.001$). Neck node control was 91% for normal, 89% for partially impaired facial nerve

Table 3 – Results of *multivariate* analysis and facial nerve dysfunction *before* treatment, RR, relative risk, CI, confidential interval

	Risk factor	RR (CI)
Risk on pre-treatment facial nerve dysfunction		
Tumour localisation:	Medial *versus* lateral lobe	8.4 (2.8–25.7)
	Bi-lobe *versus* lateral lobe	6.3 (2.8–14)
N-stage	N_+ *versus* N_0	2.6 (1.2–5.6)
Pain	Yes *versus* no pain	2.6 (1.3–5.2)
Age	Yearly	3% (0.7–5.4%)
Perineural invasion	Yes *versus* no perineural invasion	4.2 (1.7–10.3)
Pre-treatment facial nerve dysfunction as a prognostic factor		
Neck node recurrence	Partially *versus* normal function	1.7 (1.0–2.9)
	Complete *versus* normal function	2.5 (1.2–5.1)
Disease free survival	Complete *versus* normal function	4.8 (1.8–12.9)

function, and 65% for complete paralysis of the facial nerve ($p = 0.001$).

For the selection of patients treated with surgery with or without postoperative radiotherapy absolute local control was significant by better after combined treatment compared to surgery alone, 93% and 77% for intact N VII ($p = 0.003$), 88% and 17% for partially impaired n VII function ($p < 0.001$), respectively, (Fig. 1). Local control was 93% for local excision, 88% for superficial parotidectomy, and 83% and 92%, respectively, for total parotidectomy with or without preserving of the facial nerve. In Table 4 absolute local control rates depending on treatment modality are shown (combining local excision, superficial parotidectomy and N VII preserving total parotidectomy as N VII preserving procedures). Actuarial local control rates were equal for preserving or sacrificing N VII, but significantly better for combined therapy compared to surgery alone ($p = 0.0001$ for preserving N VII, and $p = 0.008$ for sacrificing N VII, see Fig. 2).

Independent prognostic factors for local control were treatment applied ($p < 0.0001$), age ($p = 0.03$, younger patients fared better) and clinical T-stage. Postoperative radiotherapy increased local control ($p < 0.001$). Bone invasion ($p = 0.001$)

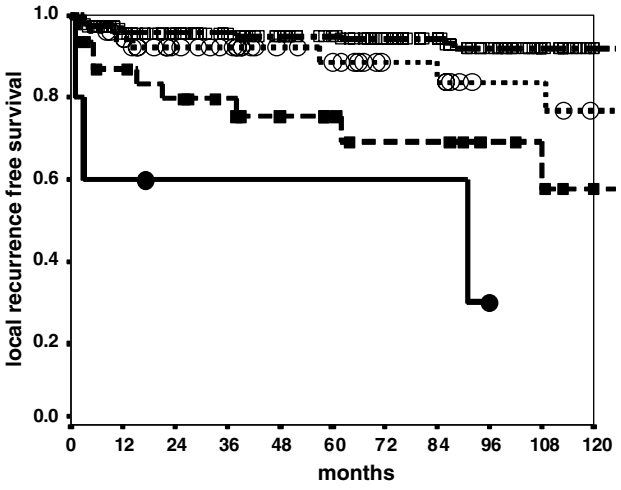

Fig. 1 – Local recurrence free survival and type of therapy, (□) S + RT N VII p., (○) S + RT N VII s., (■) S alone N VII p., (●) S alone N VII s., S, surgery, RT, radiotherapy, p., preserved., s., sacrificed.

Table 4 – Correlation between facial nerve function before treatment, conservation or sacrificing the facial nerve during operation and local recurrence

	N VII conservation	N VII sacrificed
Surgery alone		
Pre-treatment intact N VII	6/28 = 21%	1/2
Partially impaired	3/3	2/3
Complete paralysis	–	–
Surgery + postoperative RT		
Pre-treatment intact N VII	11/180 = 6%	3/22 = 14%
Partially impaired	1/12 = 8%	3/21 = 14%
Complete paralysis	0/1	1/11 = 9%

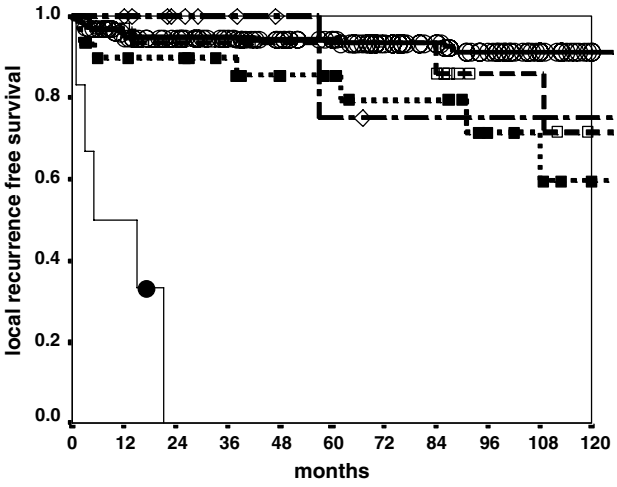

Fig. 2 – Local recurrence free survival for preoperative N VII function and therapy. (○) S + RT, intact N VII, (◇) S + RT, partial p. (□) S + RT, complete p., (■) S Alone intact N VII, (●) S alone, partial p., S, surgery, RT, radiotherapy, p, paralysis.

and status of the resection margins ($p = 0.05$) were additional independent prognostic factors ($p = 0.001$). Type of surgical intervention and pre-treatment facial nerve function were *no* independent prognostic factors for local control.

Neck node control correlated *independently* with N-stage, treatment and function of the facial nerve, with a p-value of 0.002 for completely paralysed facial nerve (see also Table 3).

3.3. *Distant metastases free survival*

Distant metastases were seen in 23%, 39% and 57%, respectively, for normal, partially impaired and complete by paralysed facial nerve ($p = 0.001$). Function of N VII before treatment was *no* independent prognostic variable for distant metastases free survival. Besides T- and N-stage, sex and histological classification were independent variables, with perineural invasion as additional variable, obtained from the local resection specimen.

3.4. *Disease free survival*

Percentage disease free survival was 69, 37 and 13, respectively, for normal, partially impaired and completely paralysed facial nerve ($p < 0.001$), see also Fig. 3.

In univariate analysis, age, sex, pain, clinical N- and T-stage, localisation (medial/lateral lobe or both), clinical invasion of the skin, treatment and function of the facial nerve correlated significantly with disease free survival. In multivariate analysis, clinical T-stage ($p = 0.004$), treatment ($p < 0.001$) and pre-treatment function of the facial nerve ($p = 0.03$) were independent variables (see also Table 3). In the subgroup of patients treated by surgery with or without postoperative radiotherapy, the variables bone invasion, status of the resection margins, pathological skin invasion, perineural and vascular invasion were significantly correlated with disease free survival in an univariate analysis. Bone invasion and pathological skin invasion were, however, the only independent additional prognostic variables.

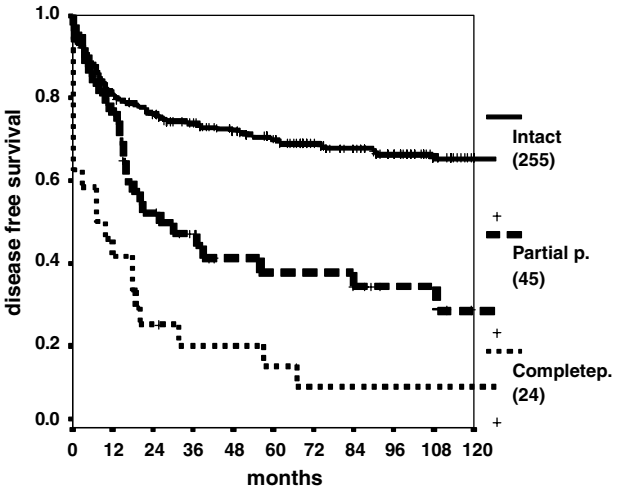

Fig. 3 – Disease free survival for N VII function at presentation, p., paralysis, $p < 0.0001$.

3.5. *Facial nerve function after treatment*

In Table 5 the correlation between surgical procedure, according to facial nerve function, before treatment and the definitive facial nerve function in the follow-up is shown. Facial nerve function in the follow-up after surgery was unknown in 2%. Intact N VII in the follow-up after surgery was noted in 61%, partial paralysis in 22% and complete paralysis in 17%. There was no association between permanent facial nerve dysfunction and age or delay between surgery and postoperative radiotherapy. Patients with tumours of the medial lobe, especially if extending to the lateral lobe, were at risk for permanent facial nerve dysfunction, even if the function of N VII was normal before treatment.

Despite the attempt to preserve the N VII in patients with normal N VII function before treatment, partial and complete loss of function were seen in 15% and 4%, respectively, after treatment. Reconstruction of the facial nerve during surgery was attempted in only 19/60 patients. Reconstruction of the facial nerve resulted in partial maintenance of the function of N VII in 74% (14/19), compared to 22% (9/41) for no attempt to reconstruct the nerve ($p < 0.001$). Complete normalisation of facial nerve function after reconstruction was not shown. A possible negative role of postoperative radiotherapy on permanent facial nerve function after reconstruction was not seen. However, since 85% of the patients receiving a reconstruction were treated with postoperative radiotherapy, this factor could not be properly analysed.

4. Discussion

In the Netherlands, most patients with malignant salivary gland tumours are treated in the centres of the NWHHT. The follow-up in these centres is precise, and in the study the mean follow-up was 98 months. Although this concerns a retrospective study, detailed information about the facial nerve function was only lacking in 2%. Since most possible prognostic factors were well registered, the correlation between these factors and pre-treatment function of the facial nerve could be analysed adequately. In two out of three cases the tumour was confined to the superficial lobe and in 9% it was confined to the medial lobe only, which conform data from the literature.[4,5] However, in most publications concerning parotid gland carcinoma data about the exact anatomic distribution of the tumour in the parotid gland are lacking.

The mean age of 60 years as found in our study is somewhat higher compared to that found in other publications (range 50–60 years).[6–10] Published male to female ratio for parotid gland cancer is almost equal with a slight predominance for the male race, like in our study.[5,8,11–13]

The distribution of T-stage may vary according to the classification used. Carinci *et al.*[14] in their studies showed that the percentage of T4 patients increased from 10% in the UICC T-classification of 1987 to 33% in 1997 T-classification, mainly caused by the inclusion of the facial nerve involvement in the T4 category. Using the UICC 1992 classification we found a T3–T4 tumour stage in about 1/3 of our cases similiar to the findings of Vander Poorten *et al.*[11]

Table 5 – Correlation between facial function *before* and *after* treatment depending on therapy

	N VII conserv. th.	N VII sacr./ no recon.	N VII sacr./ recon.
Preoperative intact N VII			
N VII in FU: intact	81% ($n = 168$)	–	–
Partial paralysis	15% ($n = 31$)	47% ($n = 7$)	80% ($n = 8$)
Complete paralysis	4% ($n = 8$)	53% ($n = 8$)	20% ($n = 2$)
Preoperative partial paralysis of N VII			
N VII in FU: intact	15% ($n = 2$)	–	–
Partial paralysis	69% ($n = 9$)	11% ($n = 2$)	83% ($n = 5$)
Complete paralysis	15% ($n = 2$)	89% ($n = 16$)	17% ($n = 1$)
Preoperative complete paralysis of N VII			
N VII in FU: intact	–	–	–
Partial paralysis	–	–	1
Complete paralysis	1	8	2

conserv., conservation; th., therapy; sacr., sacrificed; recon., reconstruction; FU, follow-up.

Patient data concerning the percentage of positive nodes at presentation vary from 13% to 28%,[5,9,11–13,15,16] and were 17% in our study. Inclusion of the pathological nodes, as found in the neck dissection specimen, increased the percentage of the positive neck nodes to 26%.

In the literature, the distribution of the histological type shows a wide variation. Mucoepidermoid carcinomas accounted for 50% of major salivary gland subtypes according to a study made by Spiro.[7] However, in more recent publications this percentage of mucoepidermoid carcinoma is 25% or less, similiar to our findings.[4,6,8,9,15] The wide variation in distribution of histological subtypes may be due to the different opinions among pathologists. The prognostic importance of the histological subtype may be limited, as shown in our study.[2] The histological subtype was a significant factor for distant metastases only.

4.1. *Pre-treatment facial nerve function*

In our study, facial nerve impairment was shown in 21%, with a partial loss of function noted in 14% and complete loss of facial function noted in 7%. In the literature, the subdivision between partial impairment and total paralysis of nerve VII is not always mentioned. Some authors only mention paralysis, with a variation between 12% and 35%,[17–21] and others mention nerve VII involvement or impairment, with a variation between 12% and 35%.[4,8,12,15,22]

In general, our percentage of pre-treatment facial nerve dysfunction compares favourably with data from the literature.

In the literature, data about risk factors for pre treatment N VII function are scarce. In our multivariate analyses of prognostic factors concerning pre-treatment impairment of the function of the facial nerve, five independent prognostic factors were found. It was obvious that the location of the primary tumour was an important prognostic factor, showing the highest risk of facial nerve involvement for tumours of the medial lobe.The second factor was age showing a higher risk of facial nerve impairment with rising age. Pain was also an independent prognostic factor, correlated with risk of facial nerve weakness.

Clinical pathological N-stage was strongly correlated with risk of facial nerve weakness. In a study by Frankenthaler *et al.*[5] facial nerve impairment was not only predictive for clinically evident positive nodes (8% for normal facial nerve function versus 33% for facial nerve involvement), but was also predictive for occult neck disease.

Regis de Brito Santos *et al.*[16] also found this high predominance between neck metastases and facial nerve involvement in an univariate analysis, however not in a multivariate analysis.

Skin invasion and perineural invasion, as seen in the histological specimen, were the last two independent prognostic factors for pre-treatment facial nerve involvement.

Vander Poorten *et al.*[11] found that perineural invasion was also strongly correlated with pre-treatment facial nerve involvement.

Prognostic significance of pre-treatment facial nerve weakness and treatment outcome:

The published data on predictive value of pre-treatment N VII on treatment outcome varies widely in the literature. In multivariate analysis pre-treatment N VII function is often not incorporated, or published data are based on univariate analysis only. In our study, in univariate analysis, pre-treatment N VII involvement was prognostic for locoregional control, distant metastases, as well as disease free survival. In multivariate analysis, this was only true for disease free survival (partial and complete dysfunction) and regional control (complete paralysis). The relative risk in disease free survival of 2.5 for complete paralysis V. normal function is comparable with the results of Vander Poorten *et al.*[11] and Malata *et al.*[23] Others[9,13,17] did not find this unfavourable prognosis.

Since disease free survival combined locoregional control and distant metastases, we also examined the separate items. The relative risk on regional recurrence was 4.8 for complete paralysis compared to normal function of the N VII. To our knowledge, this was not mentioned by others in the literature. In the study of Frankenthaler *et al.*[5], pre-treatment N VII function was only a prognostic factor in univariate analysis. N VII function influenced local control in a study of North *et al.*[19] with a hazard ratio of 11. Unlike our results, Gallo *et al.*[8] and North *et al.*[19] showed a significant, independent, correlation between pre-treatment N VII function and distant metastases.

4.2. *Facial nerve function after treatment*

One of the main issues in the treatment of malignant tumours of the parotid gland is at least partial preservation of the facial nerve. Spiro *et al.*[18] showed a decrease in extended surgery in the course of years, resulting in a decrease of sacrifice of the facial nerve. In our study, permanent partial and complete paralysis were seen in 22% and 17%, respectively, comparable with some published results.[5,21,23] In our study, the risk of permanent facial nerve dysfunction correlated, as expected, with pre-treatment facial nerve function. Deep sided tumours in the parotid glands were at highest risk for dysfunction, which conform data with the data of Bron *et al.*[24] Age and delay between surgery and radiotherapy were, unlike the findings of Brown *et al.*[10] no prognostic factors for permanent facial nerve dysfunction.

The relatively high percentage of permanent facial nerve dysfunction in our study is caused by the sacrifice of the facial nerve in 21% and the low percentage of reconstruction in 31%. As is shown, for patients with pre-treatment intact or partially impaired facial nerve function conservation of the facial nerve, if combined with postoperative radiotherapy, did not result in increased local recurrence rates compared to patients in whom the facial nerve was sacrificed (Fig. 1). Postoperative radiotherapy has been shown to improve locoregional control, not only in salivary gland cancer in general,[2] but also in this study of parotid gland cancer. In other words, aggressive surgery does not improve disease free survival, as shown by others.[14,25] So, nerve sparing surgery with postoperative radiotherapy is the preferable treatment.

In our study, permanent partial and complete facial nerve damage after conservative surgery were seen in 15% and 4%, respectively. This may be caused by a mechanical trauma, nerve stretching, oedema, diminished circulation, duration of the operation, size of the lesion and histopathology.[26] Postoperative radiotherapy did not influence facial nerve function. Since most patients received postoperative radiotherapy after

reconstruction of the nerve, we could not analyse the role of radiotherapy in these cases. However, Reddy *et al.*[27] and Brown *et al.*[10] found no influence of radiotherapy on facial nerve function after reconstruction. Recovery after facial nerve reconstruction may be disappointing.[24] Nerve grafting is mostly used in a minority of cases[5,28] comparable with the 31% found in our study. Functional outcome, although retrospectively analysed, was significantly better compared to no reconstruction performed (Table 5). In most cases only partial recovery was noted. Good functional recovery may be seen for the orbital branch, results for the mandible branch are more disappointing.[28] For the benefit of quality of life, immediate facial nerve reconstruction is, if technically feasible, advised.

Conflict of interest statement

None declared.

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