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ORIGINAL ARTICLE

FACIAL NERVE DYSFUNCTION FOLLOWING CONSERVATIVE PRIMARY PAROTID SURGERY

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Aim: *The aim of this study is to analyse the incidence and highlight the risk factors associated with facial nerve dysfunction after conservative primary parotidectomy.*
Methods: *The study included 41 patients, who were initially with normal facial nerve function and had been treated by conservative primary parotidectomy (42 procedures) for parotid neoplasms in Sohag University Hospital, during the period from March 2002 to March 2005. Facial nerve function was assessed on admission before surgery; and then at one day, one month and six months following the parotidectomy. Extent of the surgery, size of the parotid neoplasm, and histopathological type of the neoplasm were correlated with the incidence of postoperative facial nerve dysfunction.*
Results: *The rate of postoperative facial nerve dysfunction was 35.7% in the first post-operative day, 19% and 4.8% at one month and six months, respectively. Cases treated with total parotidectomy with or without neck dissection showed poorer facial nerve function ($p < 0.001$), ($p < 0.01$), and ($p < 0.04$) at one day, one month, and 6months, respectively. Overall, neoplasms with size ≥ 5 cm had a higher prevalence of facial nerve paresis ($p < 0.03$ at one day, and 0.04 at one month postoperative). Patients with malignant parotid neoplasms had more tendency to develop facial nerve dysfunction ($p < 0.02$), ($p < 0.001$), and ($p < 0.03$) at one day, one month, and 6months, respectively.*
Conclusion: *In our study, the following were associated with higher risk of facial nerve dysfunction: extensive surgery; large sized neoplasms; parotid cancer, when treated with total parotidectomy or combined with neck dissection; chronic sialadenitis; and vascular malformation.*

Keywords: *Facial nerve function, parotid cancer, parotidectomy.*

INTRODUCTION

Surgeons exert much effort to minimise the risk of facial nerve dysfunction following parotid gland surgery, especially for primary parotidectomy. Despite this, the incidence of transient facial nerve paresis ranging from 18% to 68% and long term dysfunction from 0% to 19% has been reported.[1-4]

Contemporary parotid surgical philosophy dictates that if the facial nerve is functioning normally preoperatively then it should be preserved intraoperatively. The reasoning behind this approach is that facial nerve sacrifice may do very little to improve oncologic safety, while adding very significantly to morbidity and not obviating the need for postoperative radiotherapy. A caveat to this philosophy, however, is that every attempt should be made to achieve clear surgical margins.[5]

Smaller tumors, less than 3 cm in size according to Woods[6] or 4 cm according to Renehan et al.,[7] may be managed by limited parotid resection, preserving the facial nerve unless direct infiltration is seen at operation. Larger tumors warrant more aggressive surgery, and the bulk and degree of local infiltration of larger cancers are likely to necessitate sacrifice of the nerve even if it is functioning normally.[5]

In the literature, conservative parotidectomies with facial nerve dissection have been classified according to the extent of gland resection. Limited superficial parotidectomy (enucleation, lumpectomy) implies resection of a superficial parotid neoplasm away from the normal parotid tissue with or without limited facial nerve dissection.[2,3]

Superficial parotidectomy requires dissection of the facial nerve and its branches from the superficial lobe of the parotid and includes resection of the superficial (lateral) parotid tissue away from facial nerve.[8,9] Total parotidectomy consists of dissection and removing all the parotid tissue lateral (superficial) and medial (deep) to the facial nerve and its branches.[2] It has been reported that the more extensive the surgical procedure the more common the rate of facial nerve paresis and this rate again increased with malignant parotid neoplasms.[1,3]

Facial nerve palsy after parotid gland surgery has significant functional and emotional impact on patients. Often patients, especially those with complete paralysis, complain of troubles with mastication, drooling, poor eye closure, xerophthalmia, and the most distressing disorder is the social isolation due to the obvious cosmetic deformity.

Facial nerve dysfunction after conservative parotidectomy is still poorly understood and debatable. Many of these studies were retrospective and, more importantly, the lack of a standard evaluation method for grading facial nerve deficits. In the majority of publications, facial nerve dysfunction is simply stated as abnormal without clearly specifying the criteria used. Only few have used an acceptable facial nerve grading scales.[10] Contrary to previous studies, data presented were prospectively collected. In addition, facial motor function was evaluated according to an established grading system. The aim of this study is to analyse the incidence and highlight the factors associated with facial nerve dysfunction after conservative parotidectomies.

PATIENTS AND METHODS

This study included 42 parotidectomies in 41 patients (one case had been treated with bilateral superficial parotidectomy), 22 men and 19 women with age range of 1-77 years and mean age of 52 ± 27.4 years. All cases had been treated during the period from March 2002 to March 2005, at Sohag University Hospital. The patient series included those, who were initially with normal facial nerve function and had been treated by conservative primary parotidectomy for benign, benign-like neoplasm, or malignant neoplasm of the parotid gland. Patients, who had pretreatment facial nerve affection, or those, who had radical parotidectomy were excluded from the study.

Preoperative clinical evaluation was thoroughly performed supplemented with ultrasound scan in all patients. Computed tomography scanning was done in selected cases (large, fixed neoplasms, or when malignancy was suspected) with fine needle aspiration cytology. Routine search for metastasis was done, when it indicated.

The patients' details were retrieved from the patients' notes, which had been collected prospectively on admission and during their follow-up review. These included age and sex of the patients, extent of surgery, tumour size (according to the gross size of the neoplasm as measured by the histopathologist (< 5 cm or ≥ 5 cm), type of the pathology according to the 1991 international guidelines from the World Health Organisation (WHO) classification of salivary neoplasms (benign neoplasms, benign like neoplasms, and malignant neoplasms). [11] The removed parotid neoplasm, in all cases, was trimmed, paraffin embedded, then 5 micron tissue sections were made for histopathological evaluation. Some histological types with intraoperative and postoperative photos were shown in figures.[1-3]

Surgical procedure: Identification, exposure, and protection of the facial nerve will help in providing wide, safe and satisfactory excision of the parotid neoplasm with low morbidity and acceptable functional outcome.[12] The standard preauricular and cervical surgical approach was adopted and extended temporally or cervically, whenever indicated. After elevation of the skin flaps in a plain superficial to the parotid capsule, the lower pole of the parotid was mobilized from the sternomastoid muscle down to the posterior belly of the diagastric muscle. This would make the field more accessible and help in the next step of facial nerve identification, which traced usually a few millimeters and less than one cm below and medial to the pointing arrow of the bony tragus.[13] Once the main trunk of the facial nerve had been identified, we adopted the policy of insert, elevate and spread with mosquito artery forceps, and cut with fine scissors.[14] In cases when total parotidectomy was needed, after freeing the superficial part from the facial nerve and its branches, the same technique was used to free the nerve from the deep part of the parotid. At this step, the main nerve and its branches should be protected and great caution should be taken to ligate the facial vein and the superficial temporal vessels.

When parotidectomy was combined with neck dissection, the neck surgery was usually performed first and included level I to level V. This is according to standerdised neck dissection terminology.[15] Dissection of the parotid gland was performed after dissection of level II leaving removal of the submandibular triangle contents and thorough exposure of the marginal mandibular branch to its end. Our policy was to avoid sacrificing this branch during the

procedure unless if this would compromise the oncology effectiveness of the procedure. Intra-operative facial nerve monitoring or stimulation was not performed routinely in this study. Suction drainage was used in all cases.

Facial nerve function: Facial nerve function was assessed on admission before surgery, and then at one day, one month, and six months following parotidectomy. The House-Brackmann grading system (HB) was used to evaluate the facial nerve function.[16] It includes 6 grades, of which grade I is normal, while grade II-VI are abnormal and the higher the grade the higher the dysfunction. It is a well-known scale and used worldwide.[13,17,18] Its main advantages is that its evaluation criteria are clearly defined at each grade and include sequelae, such as synkinesis, contracture, and spasm.

Statistical analysis: Descriptive data were expressed as mean ± standard deviation for continuous variables while number and percent for categorical variables. Comparison between groups by chi-square test was used and level of significance was accepted at $p < 0.05$.

RESULTS

Patient characteristics: The eligible cases of this study were 42 parotidectomies in 41 patients. Superficial parotidectomy was performed in the majority of cases (66.7%), most of which was in benign or benign like neoplasms. Parotidectomy with neck dissection was performed for only 5 cases (11.9%). All neck dissections were modified radical Table 1.

Lesions were classified according to size into 2 groups: 18 cases (42%) < 5 cm, and 24 cases (58%) ≥ 5 cm.

The pathological diagnosis was benign in 29 cases (69%), most of which was benign pleomorphic adenoma. Malignant neoplasms of the parotid included the remaining 13 cases (31%) Table 2.

Incidence and extent of facial nerve dysfunction: The incidence of postoperative facial nerve dysfunction was found in 15 cases (35.7%) at the first post-operative day, 8 (19%) and 2 cases (4.8%) at one month and six months, respectively. Partial nerve affection in the form of single or double nerve branch was the predominant feature (65% of the affected patients). The most commonly affected branch was the marginal mandibular. It was encountered in 12 (28.7%), 6 (14.3%) and 2 (4.8%) cases at the first day, first month, and six months postoperative, respectively.

The majority of cases with facial nerve dysfunction according to HB system were grade II or III (9/15 cases). Most of these 9 cases improved by the first month and no one remains with persistent paresis after 3 months. The remaining 6 cases with grade IV - VI had prolonged recovery. Of these, 4 cases recovered at 6 months, while the remaining 2 had persistent paresis Table 3.

Risk factors of facial nerve dysfunction: Risk factors affecting facial nerve function is outlined in Table 4.

Both age and sex showed no differences in the postoperative function of the facial nerve.

Total parotidectomy with or without neck dissection versus superficial parotidectomy had a higher facial nerve dysfunction ($p < 0.001$), ($p < 0.01$), and ($p < 0.04$) at one day, one month, and 6 months, respectively. None of the cases treated with superficial parotidectomy for malignant neoplasms developed facial nerve weakness, while the four affected patients had vascular malformation and chronic sialadenitis.

Lesions with size of ≥ 5 cm showed more incidence of facial nerve dysfunction and this was statistically significant at one day ($p < 0.03$) and one month ($p < 0.05$) postoperative. Facial nerve recovery was slow, and the permanent facial weakness was observed in only two cases.

The difference between groups of cases with malignant neoplasms and those with benign or benign like neoplasms was statistically significant ($p < 0.02$), ($p < 0.001$), and ($p < 0.03$) at one day, one month, and 6months, respectively. The highest incidence of postoperative facial weakness was associated with malignant parotid tumours, which had been treated with total parotidectomy with neck dissection (100%). All facial nerve branches were affected and the recovery was slow in comparison with the other groups. There was only permanent weakness of the lower lip in two of these cases due to sacrifice of the marginal branch of the facial nerve intra-operatively for oncologic safety.

Table 1. Surgical procedures employed for different parotid neoplasms.

Pthological Type	Superficial parotidectomy (%)	Total parotidectomy (%)	Total parotidectomy with neck dissection (%)	Total number (%)
Benign or benign like parotid neoplasms	25 (59.5%)	4 (9.5%)	-	29 (69%)
Malignant parotid neoplasms	3 (7.2%)	5 (11.9%)	5 (11.9%)	13 (31%)
Total	28 (66.7%)	9 (21.4%)	5 (11.9%)	42 (100%)

Table 2. Histopathological types of parotid neoplasms.

Histopathology	Number of cases (%)
Benign and benign- like neoplasms:	**29 (69%)**
1. Pleomorphic adenoma	18 (42.9%)
2. Adenolymphoma	1 (2.4%)
3. Sialadenitis	5 (11.9%)
4. Tuberculous lymphadenitis	1 (2.4%)
5. Vascular malformation	2 (4.8%)
6. Benign simple cyst	2 (4.8%)
Malignant neoplasms:	**13 (31%)**
1. Adenocarcinoma	4 (9.5%)
2. Mucoepidermoid carcinoma	4 (9.5%)
3. Adenoid cystic carcinoma	2 (4.8%)
4. Salivary duct carcinoma	1 (2.4%)
5. Basal cell carcinoma	1 (2.4%)
6. Mucous secreting adenocarcinoma	1 (2.4%)

Table 3. Postoperative grades of facial nerve function according to HB grading score.

House-Brackmann Score	One day postoperative (%)	One month postoperative (%)	Six month postoperative (%)
I (normal)	27 (64.3%)	34 (81%)	40 (95.2%)
II	7(16.7%)	2 (4.8%)	0 (0%)
III	2 (4.8%)	1 (2.4%)	0 (0%)
IV	1 (2.4%)	1 (2.4%)	1 (2.4%)
V	4 (9.5%)	2 (4.8%)	1 (2.4%)
VI	1 (2.4%)	2 (4.8%)	0 (0%)
Total	42 (100%)	42 (100%)	42 (100%)

Table 4. Risk factors and their relation to postoperative facial nerve dysfunction.

Risk factor (number of patients)	No of patients (%) with weakness at one day post operative	No of patients (%)with weakness at one month post operative	No of patients (%)with weakness at 6 months post operative
Age groups:			
< 50 years (19)	6 (31,6%)	3 (15.8%)	1 (5.3%)
>50 (22)	9 (40. 9%)	5 (22.7%)	1 (4.5%)
P-value	NS	NS	NS
Sex groups:			
Men (22)	8 (36.3%)	4 (18.2%)	1 (4.5%)
Women(19)	7 (36.8%)	4 (21.1%)	1 (5.3%)
P-value	NS	NS	NS
Extent of surgery:			
Superficial parotidectomy (28)	4 (14.3%)	2 (7.1%)	0 (0%)
Total parotidectomy with or without neck dissection (14)	11 (78.6%)	6 (42.9%)	2 (14.3%)
P-value	< 0.001	< 0.01	< 0.04
Size of tumour:			
<5 cm (18)	3 (16.7%)	1 (5.5%)	0 (0%)
≥5 cm (24)	12 (50%)	7 (29.1%)	2 (8.3%)
P-value	< 0.03	< 0.04	NS
Histopathological type:			
Benign and benign like neoplasms (29)	7 (24.1%)	2 (6.9%)	0/29 (0%)
Malignant neoplasm (13)	8 (61.5%)	6 (46.2%)	2 (16.4%)
P-value	< 0.02	< 0.001	< 0.03
Total	15/42 (35.7%)	8/42 (19%)	2/42 (4.8%)

NS = not significant

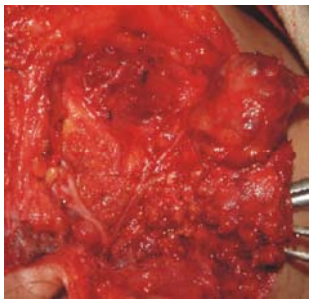

Fig 1a. Pleomorphic adenoma, at the end of superficial parotidectomy, and facial nerve branches had been identified and preserved.

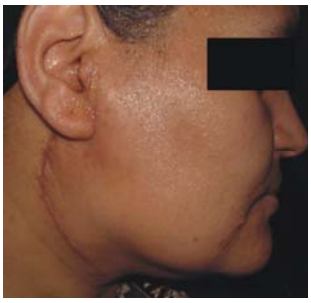

Fig 1b. Seven days post-surgery.

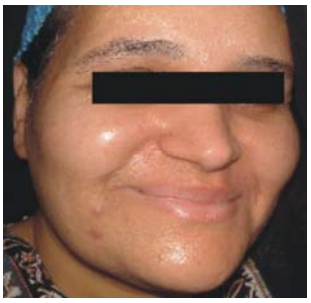

Fig 1c. Seven days post-surgery, with intact facial nerve function.

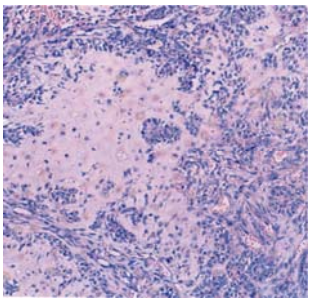

Fig 1d. Histopathology, pleomorphic adenoma- X200.

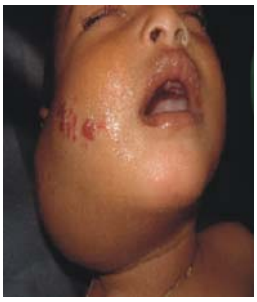

Fig 2a. Huge Vascular malformation of the right parotid, pre-operative.

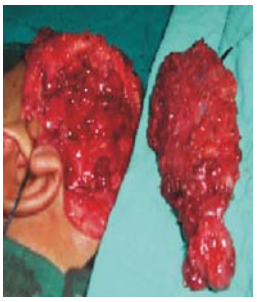

Fig 2b. At the end of superficial parotidectomy, with near total excision of the vascular neoplasm.

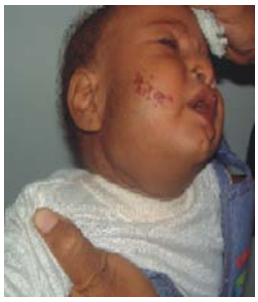

Fig 2c. Three weeks postoperative with grade III weakness of the marginal mandibular branch.

Fig 2d. Three month post-operative, with recovery of the marginal mandibular nerve.

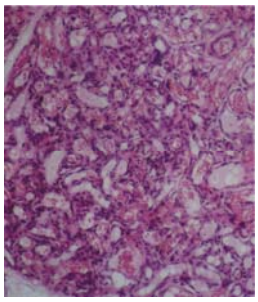

Fig 2e. Histopathology of vascular malformation X200.

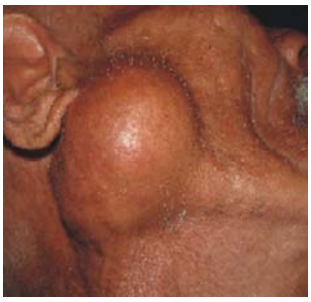

Fig 3a. Mucoepidermoid carcinoma of the right parotid, preoperative.

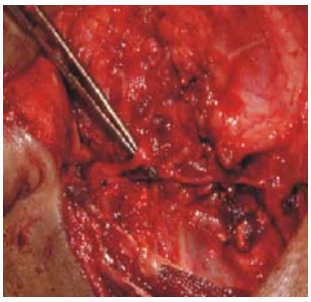

Fig 3b. At the end of total conservative parotidectomy and dissection and preservation of the facial nerve and its branches.

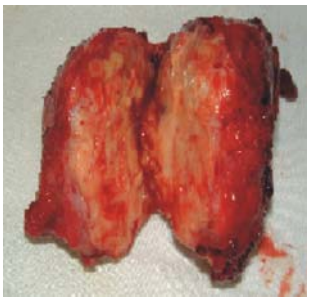

Fig 3c. Cut section of the tumour with diameter > 5 cm.

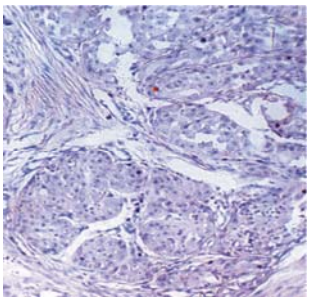

Fig 3d. Histopathology of Mucoepidermoid carcinoma X200.

DISCUSSION

Overall, facial nerve dysfunction was present in 35.7% of our patients in the first postoperative day and in 19% in the first one month. The incidence of long-term facial nerve dysfunction was found only in 4.8% (the two patients who had marginal mandibular branch sacrifice during neck dissection). In recent publications the incidence of temporary deficits was 18% for O;Brien et al.,[1] and Wtanabe et al.,[19] 27% for Ellingson et al.,[17] 35% for Ramadan,[20] 37% for Bron et al.,[3] 42.7% for Gaillard et al.,[21] 46% for Mehle et al.,[22] 62% for Terrell et al.,[4] and 68% for Wolf et al.[23] In the same publications, the range of long term deficits ranged from 0% for Wolf et al;[23] Gaillard et al.,[21] and Watanabe et al.,[19] 4% for Mehle et al.,[22] and Laccoyrreye et al.,[2] 5.5% for Ramadan,[20] to 19% in the study of O'Brien et al.[1] However, population standards, and type of surgery performed have been different, making comparative studies difficult. Our results are comparable with middle of these studies such as Brone et al.,[3] Ellingson et al.,[17] and Gaillard et al.[21] Our results are less favorable in comparison with some other studies. For example Dulguerov et al.,[24] had a figure of 27% temporary facial nerve weakness. The most probable reason of these better results was the early presentation of the cases of this study since the tumour size of > 5 cm was only in 5.7% of the total cases.

Nine out of fifteen of our cases of facial nerve dysfunction were grade II or III and involving single or double facial nerve branch, mainly the marginal mandibular branch. This was similar to other similar study.[21]

Age and sex of patients were not a risk factor in postoperative facial nerve dysfunction in our cases. Most of the published studies[2,17,25] agreed with our resultss, but there was a published study in which patients' advanced age was associated with higher postparotidectomy facial deficits.[4]

Factors associated with a higher incidence of temporary facial nerve deficit included: extent of parotidectomy whether neck dissection was included or not, size of the neoplasm, and type of the pathology. This has been addressed by some authors.[1,3,24] Some other studies identified other variables as a risk factor for facial nerve weakness following parotid surgery such as; recurrent parotid surgery, operating time, and close contact of the tumour to the facial nerve.[4,21]

In our study the most important factor affecting the short and long term postoperative facial nerve function was the extent of surgery. Total parotidectomy carried a higher risk of facial nerve weakness. This risk rose from 66.7% to 100% when neck dissection included. This was also associated with slow recovery and the only two cases with permanent weakness were due to deliberate sacrifice of the marginal mandibular branch. Bron and O'Brien[3] in a similar study reported an incidence of 100% and 83% of temporary facial nerve weakness in patients treated for malignant parotid with total parotidectomy and any form of parotidectomy with neck dissection, respectively. Bron and O'Brien[3] reported very slow recovery and high number of cases with permanent marginal mandibular branch {10 out of 23 (43%)}. The most likely important reasons are the more aggressive surgical attitude adopted to treat these malignant neoplasms, the difficulty to identify and dissect the nerve, and the more sizable and consequently the more needed dissection of the facial nerve and its branches from the tumour.

The level of morbidity suggests that elective parotidectomy and neck dissection should only be performed if there is a proven clinical benefit. In therapeutic settings, every attempt, if oncologically safe, should be made to minimise risk of the marginal mandibular branch affection. This is particularly when elevating the upper cervical flap, and during clearing the submandibular triangle.

Superficial parotidectomy was associated with the least functional impairment of the facial nerve even those with malignant parotid. Despite cases with sialadenitis and arteriovenous malformation was treated by superficial parotidectomy, they had the highest incidence of facial nerve dysfunction among this group of patients. Surgery for chronic sialadenitis is well known with its difficulties.[3,21] We observed that difficulties in finding and dissecting the facial nerve and its branches in both arteriovenous malformation and sialadenitis. In the former, the vascularity and in the later the fibrosis contributes to the difficulties of parotid surgery.

Dissection of the facial nerve, even with great care could result in significant facial nerve weakness. There have been many theories trying to explain facial nerve dysfunction after its anatomical preservation in parotid surgery. This may be due to mechanical trauma such as compression, crushing, and stretching during the operative procedure. Some advocate that it may be due to ischaemic injury as a result of facial nerve dissection from its surroundings. Dulguerov et al.,[24] concluded that nerve stretching may be the most probable etiology of facial nerve dysfunction following anatomical preservation of the facial nerve in parotid surgery.

Whether the use of a routine continuous intra-operative facial nerve monitoring has a significant impact on postoperative facial nerve function remains debatable. Some authors[23] found no significant benefits of this technique, while others[4] found that it was significantly better. Even though, use of this technique would add to the time and cost of the surgery and more importantly is that some technical difficulties of the monitoring apparatus may arise during the procedure.

In conclusion Despite, facial nerve weakness is a problem in conservative primary parotid gland surgery, it is mild, partial, and usually temporary. The risk of facial nerve dysfunction was proportional to the extent of parotid surgery, type and size of parotid pathology. The risk of temporary facial nerve weakness increased in surgical treatment of chronic sialadenitis, arteriovenous malformation, and cancer parotid when treated with total parotidectomy and this risk further increased when it was combined with neck dissection. Malignant parotid could be treated with less risk to the facial nerve in small superficially situating tumours and this highlights the importance of early intervention in such condition.

REFERENCES

1. O'Brien CJ, Malka VB, Mijajilovic M. Evaluation of 242 consecutive parotidectomies performed for benign and malignant disease. Aust N Z J Surg. 1993;63:870-7.

2. Laccoyrreye H, Laccoyrreye C, Cauchois R, Jouffre V, Minard M, Brasnu D. Total conservative parotidectomy for primary benign pleomorphic adenoma of the parotid gland: A 25-year experience with 229 patients. Laryngoscope. 1994;104:1487-94.

3. Brone LP, O'Brien CJ. Facial nerve function after parotidectomy. Arch Otolaryngol Head Neck Surg. 1997;123:1091-6.

4. Terrell JE, Kileny PR, Yian C, Escalmado RM, Bradford CR, Pillsbury MS, Wolf GT. Clinical outcome of continuous facial nerve monitoring during primary parotidectomy. Arch Otolaryngol Head Neck Surg. 1997;123:1081-7.

5. O'Brien C, Adams J. Surgical management of the facial nerve in the presence of malignancy about the face. 2001;9:90-4.

6. Woods JE. The facial nerve in parotid malignancy. Am J Surg. 1983;146:493–6.

7. Renehan AG, Gleave EN, Slevin NJ. Clinico-pathologic and treatment-related factors influencing survival in parotid cancer. Br J Cancer. 1999;8:1296–1300.

8. Rea JL. Partial parotidectomies: morbidity and benign tumour recurrence rates in a series of 94 cases. Laryngoscope. 2000;110:924-7.

9. Witt RL. The significance of the margin in parotid surgery for pleomorphic adenoma. Laryngoscope. 2002;112:2141-54.

10. Arndt F, Bock M, Draf W. Parotidectomie: Funktionnelle und Asthetische Ergebnisse. HNO. 1991;39:271-7.

11. World Health Organisation. International Histological Classification of Tumours, 2nd ed. London: Springer.1991.

12. Kabash M, El-Hwaig MT, Mohammed GH, Omar A E-L. Technical aspects of identification and exposure of the nerves during surgery of major salivary glands: Value and neurological assessment. South Valley Med J. 2000;4:307-24.

13. Cannon CR, Replogle WH, Schenk MP. Facial nerve in parotidectomy: a topographical analysis. Laryngoscope. 2004;114:2034--7.

14. Watkinson JC, Gaze MN, Wilson JA. Tumours of the major salivary glands. In Still and Maran's Head and Neck Surgery (4th edn). Buttermouth Heinemann: Oxford, Auckland, Boston, Johannesburg, Melbourne, New Delhi. 2000;441-458.

15. Robbins KT, Clayman G, Levine PA, Medina J, Sessions R, Shaha A, et al. Neck dissection classification update: revisions proposed by The American Head Neck Society and the American Academy of Otolaryngology-Head and Neck Surgery. Arch Otolaryngol. Head Neck Surg. 2002;128:751-8.

16. Houes JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg. 1985;93:146-7.

17. Ellingson TW, Cohen JL, Andersen P. The impact of malignant disease on facial nerve function after parotidectomy, Laryngoscope. 2003;113:1299-303.

18. Kimata Y, Sakuraba M, Hishinuma S, Ebihara SHayashi R, Asakage T. Free vascular nerve grafting for immediate facial nerve reconstruction. Laryngoscope. 2005:115:313-36.

19. Watanabe Y, Ishikawa M, Shojaku H, Mizukoshi K. Facial nerve palsy as a complication of parotid gland surgery and its preservation. Acta Otolaryngologica. 1993;504:137-9.

20. Ramadan MM. Facial nerve morbidity following parotid surgery. Suez Canal Univ Med J. 2003;6:29-34.

21. Gaillard C, Perie S, Susini B, St Guily JL. Facial nerve dysfunction after parotidectomy: The role of local factors, Laryngoscope. 2005;115:287-91.

22. Mehle ME, Krause DH, Wood BG, Benninger MS, Eliachar I, Levine HL, et al. Facial nerve morbidity following parotid surgery for benign disease: The Cleveland Clinic Foundation experience. Laryngoscope. 1993;103:386-8.

23. Wolf SR, Schneider B, Suchy B, Eichhorn B. Intraoperatives Fazialisnervmonitoring in der parotischirurgie. Head Neck Oncol. 1995;43:294-8.

24. Dulguerov p, Marchal F, Lehmann W. Postoperative facial nerve paralysis: Possible etiologic factors and results with routine facial nerve monitoring. Laryngoscope. 1999;109:754-62.

25. Witt RT. Facial nerve function after partial superficial parotidectomy: An 11-year review (1987-1997). Otolaryngol. Head Neck Surg. 1999;121:210-13.