# *Lower cranial nerves function after surgical treatment of Fisch Class C and D tympanojugular paragangliomas*

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OTOLOGY

# Lower cranial nerves function after surgical treatment of Fisch Class C and D tympanojugular paragangliomas

**Andrea Bacciu · Marimar Medina · Hassen Ait Mimoune · Flavia D'Orazio · Enrico Pasanisi · Giorgio Peretti · Mario Sanna**

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**Abstract** The aim of this study was to report the postoperative lower cranial nerves (LCNs) function in patients undergoing surgery for tympanojugular paraganglioma (TJP) and to evaluate risk factors for postoperative LCN dysfunction. A retrospective case review of 122 patients having Fisch class C or D TJP, surgically treated from 1988 to 2012, was performed. The follow-up of the series ranged from 12 to 156 months (mean, 39.4 ± 32.6 months). The infratemporal type A approach was the most common surgical procedure. Gross total tumor removal was achieved in 86 % of cases. Seventy-two percent of the 54 patients with preoperative LCN deficit had intracranial tumor extension. Intraoperatively, LCNs had to be sacrificed in 63 cases (51.6 %) due to tumor infiltration. Sixty-six patients (54.09 %) developed a new deficit of one or more of the LCNs. Of those patients who developed new LCN deficits, 23 of them had intradural extension. Postoperative follow-up of at least 1 year showed that the LCN most commonly affected was the CN IX (50 %). Logistic regression analysis showed that intracranial transdural tumor extension was correlated with the higher risk of LCN sacrifice ($p < 0.05$). Despite the advances in skull base surgery, new postoperative LCN deficits still represent a challenge. The morbidity associated with resection of the LCNs is dependent on the tumor's size and intradural tumor extension. Though no recovery of LCN deficits may be expected, on long-term follow-up, patients usually compensate well for their LCNs loss.

**Keywords** Tympanojugular paraganglioma · Glomus tumor · Lower cranial nerve outcomes · Intradural extension · Lower cranial nerve palsy · Paraganglioma

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## Introduction

Tympanojugular paragangliomas (TJPs) account for approximately 0.6 % of all head and neck tumors and represent the most common tumor of the jugular foramen, constituting 80 % of all neoplasms at this site [1]. TJPs arise from the dome of the jugular bulb and are located within the jugular foramen. The jugular foramen can be considered the key structure in the lower skull base, being the site exit of cranial nerves IX to XI and of the major venous drainage of the brain.

The growth pattern of TJPs is slow and insidious, often invading and eroding the bony skull base, infiltrating the regional cranial nerves, and sometimes also transgressing the dural barrier [2]. The mainstay of treatment for these tumors is total surgical extirpation [2–7]. Other management options for patients with TJPs include fractionated radiotherapy, stereotactic radiation, observation with serial imaging, and a combination of these modalities [8]. The choice of treatment either with surgery or radiotherapy has been widely debated, being the main aim minimal morbidity.

In fact, the surgical management of TJPs is particularly challenging as a result of the complex anatomic location,

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the nearby major neurovascular structures, and the proximity of intracranial structures. In early studies the surgical attempt to remove these lesions was associated with high rates of morbidity and mortality [8]. Nowadays, surgical resection of TJPs is safer and better results in terms of surgical morbidity are achievable due to advances in preoperative interventional neuroradiology and refinements in skull base microsurgery.

However, due to the intimate relationship between LCNs and TJPs, resection of these lesions may produce multiple LCNs deficits with resultant dysphonia, dysphagia and aspiration [3, 9–13].

To our knowledge, there are very few studies focusing on LCN function after surgery for TJP and predictive factors of pars nervosa tumor involvement [5, 13]. The aim of this study was to report the pre- and postoperative LCN functions in patients undergoing surgery for Fisch class C and D TJPs. In addition, factors that were predictive of new postoperative LCN dysfunction were investigated.

**Table 1** Fisch classification of tympanojugular paragangliomas

| | |
|---|---|
| Class C | Tumors involving the carotid canal |
| C1 | Tumors destroying the jugular foramen and bulb with limited involvement of the vertical portion of the carotid canal |
| C2 | Tumors invading the vertical portion of the carotid canal |
| C3 | Tumors invading the horizontal portion of the carotid canal |
| C4 | Tumors reaching the anterior foramen lacerum |
| Class D | Intracranial extension |
| De1 | Tumors with up to 2 cm dural displacement |
| De2 | Tumors with more than 2 cm dural displacement |
| Di1 | Tumors with up to 2 cm intradural extension |
| Di2 | Tumors with more than 2 cm intradural extension |

Class D defines only the intracranial extension and should be reported as an addendum to the C stage

*De* extradural, *Di* intradural

## Methods

After institutional review board approval, the charts of 171 patients with a diagnosis of Fisch class C and D TJP that underwent surgical treatment during the period from April 1988 to April 2012 were examined retrospectively. Postoperatively, 23 patients were lost to the follow-up and 16 cases had less than 1-year follow-up; these cases were excluded from the study. Patients who had undergone either previous surgery (8 cases) or preoperative radiotherapy (2 cases) for treatment of their paraganglioma were also excluded. Thus, 122 patients became the subjects of this study. All patients underwent complete otological and neurological examinations, flexible fiber optic laryngoscopy, and hearing assessment by pure-tone audiometry. All patients were evaluated before surgery by high-resolution computed tomography (HRCT) with bone windows, gadolinium-enhanced magnetic resonance imaging (MRI), and four-vessel angiography with manual cross-compression testing. In addition, superselective tumor embolization using polyvinyl alcohol was performed 2–4 days before the surgery. The postoperative diagnostic work-up for evaluation of clinical signs correlated to CN IX and CN X deficits included flexible videoendoscopy with functional evaluation of swallowing (FEES), and sensory testing (FEEST). A pulmonary X-ray examination was obtained postoperatively only in patients showing signs of acute infection (i.e., high temperature, persistent cough, pathologic pulmonary auscultation or chest pain).

Tumors were staged according to Fisch classification (Table 1; Fig. 1) [14]. Pre- and postoperative facial nerve functions were graded according to the House–Brackmann (HB) scale [15]. Follow-up was defined as the period of time from the surgery to the most recent office visit.

The data were analyzed with a statistical software programme (SPSS Statistics for Windows version 20, Chicago, IL, USA). Continuous data are summarized as mean ± standard deviation. Categorical data are presented as frequencies and percentages. Differences between two categorical data were analyzed using the Chi-square test. Logistic regression analysis was carried out to identify predictive factors of postoperative LCN dysfunction. Presence of preoperative LCN deficits, presence of intracranial extradural tumor extension, and intracranial transdural tumor extension were evaluated as predictors of the involvement of LCNs. *P* values below 0.05 were considered significant.

## Results

### Demographic data

The relevant patient demographic characteristics and preoperative signs and symptoms evaluated in relation to Fisch classification are summarized in Table 2.

The study population consisted of 37 (30.3 %) men and 85 (69.7 %) women. Ages ranged from 16 to 67 years, with an average of 45.5 ± 12.3 years. Sixty-seven tumors (54.9 %) occurred on the left side, and 55 (45.1 %) were on the right side. Tumors were classified according to Fisch classification with 20 cases (16.4 %) in class C1, 68 cases (55.7 %) in class C2, 31 cases (25.4 %) in class C3, and 3 cases (2.5 %) in class C4. There were a total of 65 cases (53.3 %) with intracranial

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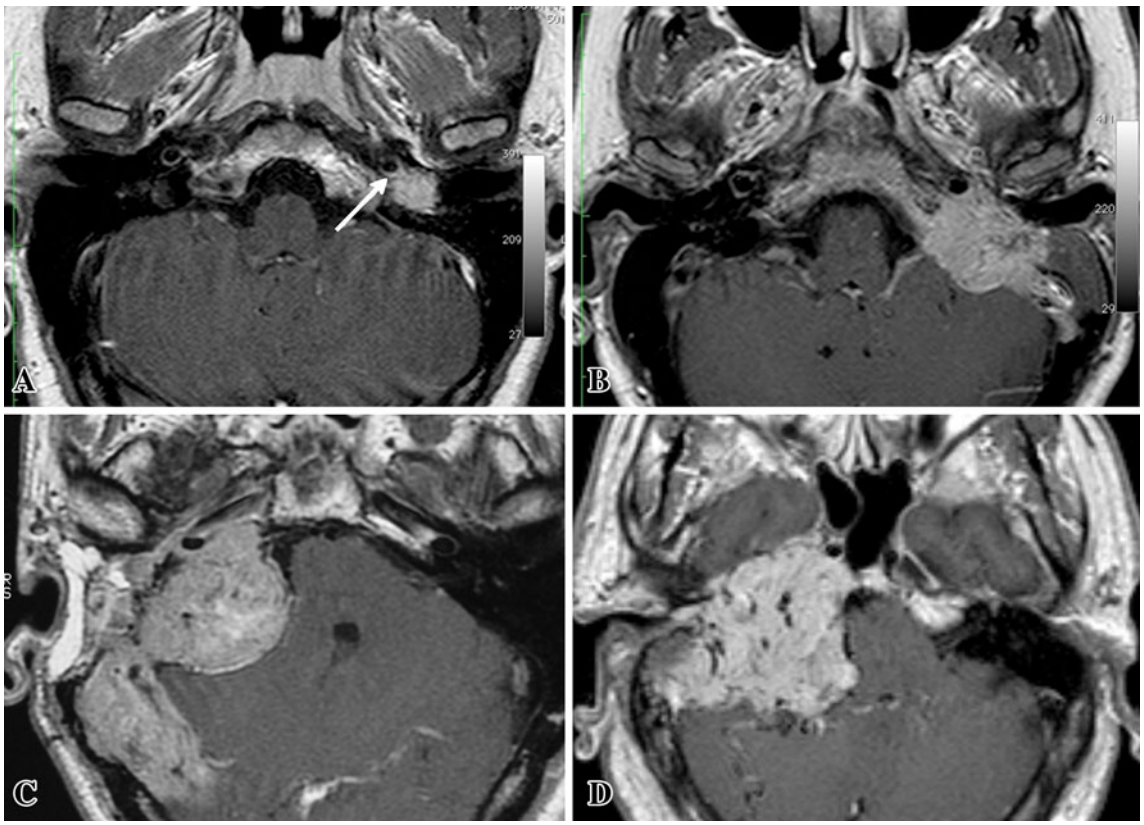


**Fig. 1** MRI illustrating the different classes of tympanojugular paragangliomas according to Fisch classification. **a** Axial MRI showing a Class C1 paraganglioma extending to but not eroding the vertical portion of the carotid canal (*arrow*). **b** Axial MRI showing a Class C2 tympanojugular paraganglioma engulfing the vertical portion of the carotid canal. **c** Axial MRI showing a Class C3 paraganglioma involving the horizontal portion of the carotid canal with intracranial extradural extension (De2). **d** Axial MRI demonstrating a Class C4 paraganglioma with clear involvement of all the horizontal portion of the intrapetrous carotid artery with extension to the cavernous sinus. Notice the intradural extension (Di2)

extension, 47 of which had intradural involvement. The incidence of intracranial extension was directly related to the tumor class, with class C1 tumors having the lowest incidence of intracranial involvement (0 %) and Class C4 the highest (100 %).

Distribution of tumors according to Fisch's classification is detailed in Table 3. The follow-up period ranged from 12 to 156 months (mean 39.4 ± 32.6 months). The most common otologic symptoms at the time of presentation included: pulsatile tinnitus (82.8 %), hearing loss (76.2 %), vertigo (17.2 %), and facial nerve dysfunction (15.5 %). Dysphonia (25.4 %) was the most common non-otologic presenting symptom, followed by dysphagia (9.8 %), weakness of sternocleidomastoid and trapezius muscles (2.4 %), and diplopia (2.4 %). A reddish middle ear mass behind the tympanic membrane was found in 119 patients (97.5 %). All patients had a combination of two or more symptoms at the time of diagnosis.

## Surgical procedure and facial nerve management

A single-stage procedure was adopted in 111 cases. The infratemporal type A approach (IFTA-A) was chosen in 109 patients. The fallopian bridge technique was used in 2 patients who presented with a Fisch class C1 TJP with predominantly posterior disease. A planned staged procedure was adopted using the IFTA-A as the initial surgery in 11 patients who had more than 2 cm intradural tumor extension; a petro-occipital trans-sigmoid approach was used for the planned second-stage intradural removal of the tumor in 5 cases, a transcochlear approach was used in 5 cases, and an extreme lateral approach in the remaining case. This staging strategy has been adopted to avoid the risk of having postoperative cerebrospinal fluid leak resulting from the wide neck exposure and to reduce intracranial tension during the second stage.

Table 4 summarizes the facial nerve outcomes based on facial nerve management. The facial nerve was left intact

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**Table 2** Demographic and clinical characteristics of the study patients

| Characteristic | Tumor Class | | | | Total |
|---|---|---|---|---|---|
| | C1 | C2 | C3 | C4 | |
| | 20 | 68 | 31 | 3 | 122 |
| Age (years) | | | | | |
| Mean ± SD | 50.5 ± 9.3 | 46.9 ± 2.5 | 43.5 ± 1.5 | 35 ± 12 | 45.5 ± 12.3 |
| Range | 29–63 | 28–67 | 17–66 | 25–49 | 16–67 |
| Sex (*n*) | | | | | |
| Males | 3 | 20 | 12 | 2 | 37 |
| Female | 17 | 48 | 19 | 1 | 85 |
| Symptom/sign | | | | | |
| Hearing loss | 12 | 53 | 26 | 2 | 93 |
| Tinnitus | 19 | 52 | 29 | 1 | 101 |
| Vertigo | 2 | 15 | 3 | 1 | 21 |
| Facial paresis | 1 | 9 | 9 | 0 | 19 |
| Dysphonia | 3 | 18 | 7 | 3 | 31 |
| Dysphagia | 0 | 9 | 3 | 0 | 12 |
| Weakness of SCM and trapezius | 0 | 1 | 2 | 0 | 3 |
| Diplopia | 0 | 0 | 2 | 1 | 3 |
| Tongue deviation | 0 | 0 | 0 | 3 | 3 |
| Bleeding from ear | 1 | 2 | 1 | 0 | 4 |
| Mass behind tympanic membrane | 17 | 68 | 31 | 3 | 119 |

*SCM* sternocleidomastoid muscle

in 2 (1.6 %) of the 122 patients using the fallopian bridge technique. Ninety-eight patients (80.3 %) underwent a permanent anterior transposition of the facial nerve. Anterior transposition of the facial nerve with segmental removal of the epineurium of the mastoid segment of the nerve due to tumor infiltration was performed in 6 patients (4.9 %). In 16 patients (13.1 %), the mastoid segment of the facial nerve was resected because of tumor infiltration, and the defect was repaired using a sural nerve graft in the same sitting.

## Lower cranial nerves status

Fifty-four patients (44.2 %) were found to be suffering from at least one CN deficit in the preoperative evaluation. Lower cranial nerve dysfunction was more common in patients with C4 tumors (66.7 %), followed by C3 (61.3 %), C2 (43.3 %), and C1 (20 %) ($p < 0.05$). The presence of preoperative LCN deficits was directly proportional to the size of tumor, with the lowest incidence of dysfunction in classes C1 and C2 and the worst results in classes C3 and C4 ($p < 0.05$). Figure 2 displays the percentages of patients with preoperative LCN deficits, postoperative new LCN deficits, and definitive LCN deficits at last follow-up according to tumor class.

Of the 54 patients with preoperative LCN deficits, 39 (72 %) had intracranial tumor extension whereas 15 cases (27.8 %) had no intracranial tumor extension. This difference was statistically significant ($p < 0.05$). Among the 39 patients with preoperative LCN deficits and intracranial tumor extension, 31 had intradural extension. Patients with intradural tumor extension had a higher incidence of preoperative LCN deficits when compared to patients with intracranial extradural extension ($p < 0.05$).

Intraoperatively, the LCNs could be preserved anatomically in 59 cases (48.4 %) and had to be sacrificed due to tumor infiltration in 63 cases (51.6 %). Specifically, the LCNs had to be resected in 17 of the 57 cases (29.8 %) without intracranial tumor extension, in 9 (50 %) of the 18 cases that had intracranial extradural extension, and in 37 (79 %) of the 47 cases that had intradural extension. Intradural extension was correlated with the highest risk of LCN sacrifice ($p < 0.05$).

After surgery, none of the patients recovered the function of the preoperatively paralyzed LCNs. Sixty-six patients (54.09 %) developed a new deficit of one or more of the LCNs. Of the 66 patients who developed new LCN deficits, 36 had intracranial extension, 23 of which were intradural. Table 5 summarizes the preoperative and definitive postoperative LCN deficits in relation to intracranial and intradural extension. Table 6 displays the new LCN deficits depending on age.

Postoperative follow-up of at least 1 year showed that the LCN most commonly affected was the CN IX (50 %),

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**Table 3** Distribution of tumors according to Fisch classification

| Class | No. of cases |
|---|---|
| C1 | 20 |
| C2 | 33 |
| C2De1 | 12 |
| C2De2 | 1 |
| C2Di1 | 14 |
| C2Di2 | 2 |
| C2De1Di1 | 3 |
| C2De2 Di1 | 3 |
| C3 | 4 |
| C3De1 | 5 |
| C3De2Di1 | 2 |
| C3De1Di2 | 1 |
| C3De2Di2 | 1 |
| C3Di1 | 12 |
| C3Di2 | 6 |
| C4 De1Di1 | 2 |
| C4Di2 | 1 |

**Table 4** Facial nerve outcomes based on facial nerve management

| Treatment of FN | No. of cases | Preoperative | At last follow-up |
|---|---|---|---|
| Left in place | 2 | 2 (I) | 1 (I) |
| | | | 1 (III) |
| Sural graft | 16 | 4 (I) | 12 (III) |
| | | 3 (II) | 2 (IV) |
| | | 1 (V) | 2 (VI) |
| | | 8 (VI) | |
| Anterior rerouting with resection of epineurium | 6 | 5 (II) | 1 (I) |
| | | 1 (III) | 1 (II) |
| | | | 2 (III) |
| | | | 2 (IV) |
| Anterior rerouting | 98 | 97 (I) | 23 (I) |
| | | 1 (IV) | 27 (II) |
| | | | 45 (III) |
| | | | 2 (IV) |
| | | | 1 (VI) |

*FN* facial nerve

followed by CN X (45.5 %), CN XI (31.5 %), and CN XII (22.8 %).

Logistic regression analysis showed that intracranial intradural tumor extension was a high-risk factor for involvement of LCNs (OR = 7.9; IC = 3.066–20,397). Presence of preoperative LCN deficits and presence of intracranial extradural tumor extension were not independent predictors of new postoperative LCN deficits.

## Complications

Perioperative complications are summarized in Table 7. There were no perioperative deaths. The most frequent perioperative complication was cerebrospinal fluid leak, which occurred in 5 cases (4 %). One patient (0.8 %) developed a new IX–XII palsy with respiratory distress and required tracheotomy (C2 Di1). A prophylactic nasogastric feeding tube was required in the immediate postoperative period for those patients who developed deficits of CNs IX and X. Oral intake was reintroduced on the third postoperative day in patients with preoperative paralysis and on the sixth postoperative day in the acute paralysis cases. There were 4 (3.3 %) patients who had varying degree of aspiration.

Feeding by percutaneous endoscopic gastrostomy (PEG) tube was used in one (0.8 %) of these patients after she developed pneumonitis in the postoperative period, from which she recovered with conservative treatment. The PEG was successfully removed after she developed good compensation of the contralateral vocal cord.

A vocal cord injection (Bioplastic Vox-Implant®) to medialize the abducted vocal cord was needed by 4 patients (3.3 %) with poor CN X function. Glottic competence was improved by performing type I medialization thyroplasty in 3 patients (2.5 %) with non-compensated swallowing disorders. Physical therapy was administered in patients with CN XI paralysis to prevent persistent shoulder dysfunction and pain. All the remaining patients were able to compensate well for their lower CN deficits with the help of speech and swallowing rehabilitation.

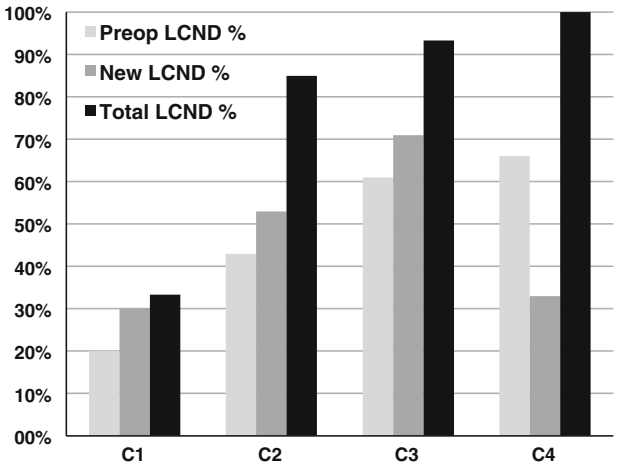


**Fig. 2** Preoperative, immediate postoperative (new deficit), and follow-up LCNDs according to Tumor Class

## Tumor removal/recurrences

Gross total tumor removal was achieved in 105 cases (86 %). Among them, 4 cases (3.8 %) developed tumor

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**Table 5** Preoperative and follow-up LCN deficits according to intracranial and intradural extension

| Cranial nerve | Extracranial ($n = 57$) | | Intracranial extradural ($n = 18$) | | Intracranial intradural ($n = 47$) | |
|---|---|---|---|---|---|---|
| | Preop | Postop | Preop | Postop | Preop | Postop |
| IX | 7 (12.3) | 20 (40) | 5 (27.8) | 7 (53.8) | 28 (59.6) | 14 (73.6) |
| X | 8 (14) | 17 (34.6) | 7 (38.9) | 6 (54.5) | 28 (59.6) | 13 (68.4) |
| XI | 7 (12.3) | 8 (16) | 3 (16.7) | 5 (33.3) | 17 (36.2) | 17 (56.6) |
| XII | 6 (10.5) | 8 (15.7) | 2 (11.1) | 5 (31.2) | 22 (46.8) | 8 (32) |

*DE* intradural extension, *Postoperative* at least 1 year after the initial procedure

recurrence. Seventeen cases (14 %) underwent subtotal tumor removal. Fifteen of these residual tumors were intradural, involving the cavernous sinus, posterior fossa dura, and the vertebral and basilar arteries. For extracranial residual tumors, partial resection was performed in 2 cases due to significant cardiac morbidities.

## Discussion

The treatment options for patients with TJPs include surgical resection, fractionated radiotherapy, stereotactic radiation, observation with serial imaging, and a combination of these modalities [8, 16, 17].

Wait and scan policy is based on the belief that TJPs are usually slow-growing lesions. This strategy might be recommended in selected cases, mainly those of elderly patients with intact LCNs function. Some authors propose stereotactic radiotherapy as a primary treatment for TJPs reporting results equivalent to surgery [8, 18–20]. We would agree with Patel et al. [21] that "… while radiation therapy may render glomus tumor stable, it is not curative, and in the younger patients with an additional life expectancy of at least 20 years, surgical extirpation of the tumor provides the best modality of cure". In our opinion, radiotherapy might be approved as an alternative primary treatment in elderly patients with tumor progression, in patients who are medically unfit for surgery, as well as in cases with recurrent or residual tumors at the level of the cavernous sinus or with engulfment of intradural vertebral and posterior-inferior cerebellar arteries. However, in-depth review of the experience with radiotherapy is beyond the scope of this article. In agreement with other authors [4, 21–27], we believe that surgical gross total resection of the tumor still remains the mainstay of treatment of TJPs, at least in young patients.

Sudden deficit of preoperative functioning LCNs is considered the most dreaded complication in TJP surgery and it might be followed by a number of undesiderable consequences such as dysphagia, aspiration, and dysphonia. The overall incidence of one or more new postoperative LCN deficits has been reported between 5 and 59 % [3–5, 7, 28–31]. In the largest series, new deficits were detected in 2.8–77.7 % for CN IX, in 1.3–58 % for CN X, in 0–39 % for CN XI, and in 2.9–27 % for CN XII [3–5, 7,

**Table 6** Distribution of new LCN deficits according to age groups and tumor class

| | No. of patients in each subgroup according to Fisch classification | No. of patients with new postoperative LCN deficits |
|---|---|---|
| <39 years | | |
| C1 | 3 | 2 (66.7 %) |
| C2 | 20 | 12 (60 %) |
| C3 | 11 | 7 (63.6 %) |
| C4 | 2 | 1 (50 %) |
| Total | 36 | 22 (61 %) |
| 40–59 years | | |
| C1 | 15 | 2 (13.3 %) |
| C2 | 35 | 18 (51.4 %) |
| C3 | 17 | 12 (70.58 %) |
| C4 | 1 | 1 (100 %) |
| Total | 68 | 33 (48.5 %) |
| ≥60 years | | |
| C1 | 2 | 2 (100 %) |
| C2 | 13 | 6 (46.2 %) |
| C3 | 3 | 3 (100 %) |
| C4 | – | – |
| Total | 18 | 11 (61 %) |

**Table 7** Perioperative complications in 122 cases

| Complication | No. of cases (%) |
|---|---|
| Meningitis | – |
| Aspiration | 4 (3.3) |
| Pneumonitis | 1 (0.8) |
| Respiratory distress | 1 (0.8) |
| Cerebrospinal fluid leak | 5 (4) |
| Wound infection | – |
| Death | – |

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**Table 8** Comparative review of the major TJP series in the last 20 years

| References | No of cases | Preoperative LCN deficit/New LCN deficit (%) | | | | TTR (%) | Recurrence (%) |
|---|---|---|---|---|---|---|---|
| | | IX | X | XI | XII | | |
| Green et al. [30] | 28 | 22/33.4 | 31/18.2 | 18/31.8 | 20/7 | 85 | NA |
| Moe et al. [7] | 119 | 34/77.7 | 40/46 | 27/39 | 31/27 | 80.7 | 1.6 |
| Gjuric and Bilic [3] | 49 | 20.4/38.4 | 26.5/52.7 | 2/14.2 | 12.2/32.5 | 81.6 | 16 |
| Jackson et al. [5] | 176 | 21/39 | 30/25 | 17/26 | 24/21 | 89.8 | 5.5 |
| Pareschi et al. [28] | 37 | 17.5/65 | 2/58 | 15/NA | 7.5/NA | 96 | 0 |
| Borba et al. [4] | 34 | 50/35.3 | 50/35.3 | 0/5.8 | NA | 91 | 5.8 |
| Makies et al. [31] | 75 | 6.6/2.8 | 2.6/1.3 | 2.6/0 | 9.3/2.9 | 78.7 | 0 |
| Present study | 122 | 32.8/50 | 35.2/45.5 | 27.1/31.5 | 24.6/22.8 | 86 | 3.8 |

*LCN* lower cranial nerve, *NA* not available, *TTR* total tumor removal

28, 30, 31]. Our results are comparable with the other major series reported in the literature (Table 8). Analysis of the table shows that only one study [31] reports excellent results that are not consistent with those reported in other series.

TJPs usually arise from the superolateral aspect of the bulb and during their growth they gradually fill the lateral compartment of the jugular foramen and medially displace the LCNs, which are covered and protected by the medial wall of the jugular bulb [32]. Thus, resection of early TJPs (Class C1–C2), where the lateral aspect of the bulb is involved and medial wall is not infiltrated makes preservation of the LCNs possible [32–35]. The technique of preservation of the antero-medial wall of the jugular bulb in order to preserve the LCNs (intrabulbar dissection technique) was first described by Gejrot [35] in 1965 and later popularized by Al-Mefty and Teixeira [36] and it is suitable for tumors that do not breach the wall of the jugular bulb.

Makek et al. [11] in their histopathologic study demonstrated that TJPs although being histologically benign have a local invasive behavior infiltrating adjacent neural structures. The degree of infiltration increases with the size of the tumor. The authors proposed the following pathophysiologic mechanism for neural infiltration: (1) the tumor grows toward the nerve; (2) the tumor comes in contact with the epineural tissues and its vessels; (3) the tumor breaks into the perineurium along the perivascular spaces of capillaries; and (4) the tumor reaches the endoneurium [11]. In a previous study, we observed that preservation of the LCNs is nearly impossible once the tumor has invaded the medial compartment of the jugular foramen [37]. Furthermore, it was noticed that the larger the intradural extension was, the more frequently the LCNs were involved [37]. We concluded that the presence of intracranial tumor extension had a positive correlation with the appearance of new LCN deficits underlining the importance of appropriate counseling of the patient with intracranial extension and clinically uninvolved LCNs. To our concern, it is important to distinguish between the types of intracranial extension. In our experience, the presence of intradural extension usually indicates infiltration of the LCNs. This can be explained by the observation that the most common route of the intradural spread is through the medial wall of the jugular bulb. The proximity of this structure to the LCNs makes their preservation in the setting of an infiltrated medial wall a disappointing endeavor. We have not found extradural extension to be indicative of LCN infiltration. The preservation of the LCNs in the extradural intracranial extension group of patients was similar to the group without intracranial extension, whereas it was significantly worse in the group with intradural extension ($p < 0.05$). This is in contrast with the results reported by others [38]. In the review of 119 patients with TJP, Jackson et al. [5] found that preoperative LCN deficits, intracranial transdural tumor extension, tumor classification, and a combination of these factors were predictors for both involvement of the pars nervosa and ultimate resection of all four LCNs. In our study, logistic regression analysis showed that only intracranial intradural tumor extension was a predictive factor of new postoperative LCN dysfunction. Preoperative LCN deficits and intracranial extradural tumor extension were not factors predictive of new postoperative LCN deficits.

Thus, if the goal is total removal, invasion of the medial wall of the jugular bulb by tumor requires its resection with a correspondingly high risk for LCN deficits. To avoid significant postoperative morbidity, the alternative in the setting of preoperatively functioning LCNs is to leave a small piece of tumor around the infiltrated nerves [31]. However, this can compromise the integrity of resection, especially in younger patients who may be better treated by a more aggressive resection in light of the fact that they tend to compensate better than older patients [37].

In fact, postoperative LCN deficits may represent a serious complication especially in the elderly [7]. When

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LCN deficits are present preoperatively, the decision to resect the involved nerves is simple. In contrast, the decision to sacrifice affected LCN in previously asymptomatic patients should be carefully balanced. In our opinion, an aggressive removal of the tumor with the risk of LCN dysfunction may be justified when the patient is young and wishes total tumor removal. The exception to this rule is the area of the cavernous sinus where we do not hesitate to leave behind a small remnant to preserve the III, IV, and VI CNs [2]. The presence of contralateral disease with the risk of bilateral LCN paralysis is another condition in which the conservative approach is recommended. In elderly patients with minimal clinical symptoms and in patients with preoperative poor general condition, a conservative approach in the form of observation with serial MRI scanning is preferred. If there is growth of the tumor or the neurologic status of the patient worsens, a partial resection of the tumor can be undertaken with a very high probability of preserving the preoperative LCN functional status.

It has been noted earlier that patients with pre-existing LCN deficits have a better prognosis in regard to postoperative compensation [39]. Interestingly, 5 of the 7 patients in our series requiring further intervention (vocal cord injection and medialization thyroplasty) had pre-existing paralysis of the IX and X CNs. Only 2 of our patients developing a new LCN palsy needed surgical rehabilitation to overcome their prolonged LCN dysfunction. These results compel us to disagree with the authors recommending early rehabilitative surgery after major skull base surgery [40]. In the current series, the majority of patients suffering postoperative cranial deficits compensate well on long-term follow-up with the help of speech and swallowing rehabilitation. Only in patients with persistent postoperative LCN dysfunction with poor compensation, we recommend rehabilitative surgical procedures, such as vocal cord injection and medialization thyroplasty. The goal for any vocal cord mobilization is to provide glottal competence, an effective cough for pulmonary protection and toilet, and support and volume voice. Moreover, the immediate glottal closure also decreases the need for tracheotomy performed for airway protection. In a quality of life study on 37 patients surgically treated for TJPs, Briner et al. [10] reported that 97 % of the patients considered the postoperative dysphagia tolerable and 86 % would choose to undergo surgery again.

In the immediate postoperative period, intensive speech pathology and physiotherapy are essential to facilitate rehabilitation. Usually, short-term nasogastric feeding is required for those patients who develop deficits of CNs IX and X. The nasogastric tube should be taken in place until swallowing tests demonstrate safe return to oral intake [31]. Percutaneous gastrostomy (PEG) tube insertion is used when swallowing difficulties persist for more than a few weeks [41]. Some authors [31, 42] suggest tracheotomy immediately after surgery if CN X is sacrificed. According to Sniezek et al. [12], we prefer to avoid tracheotomy and PEG immediately after surgery and wait until the postoperative period to adapt the optimal rehabilitative strategy. However, the presence of significant aspiration should be treated aggressively and early placement of a tracheostomy tube considered, although in our experience this was not required except in one patient who had a new IX–XII palsy with respiratory distress.

## Conclusion

Our results confirm the difficulties in preserving LCN function during TJP resection, especially in advanced lesions. In high vagal injury, particularly if combined with IX cranial nerve palsy, dysphagia and subsequent aspiration of food and fluids represent the most serious clinical problems. This must be taken into consideration during the preoperative planning. Sudden palsy of previously functioning LCNs in elderly patients would be met with serious morbidity. Surgery, although being the preferable mode of treatment in the vast majority of these tumors, is not always the most prudent choice. Watchful waiting should be, therefore, adopted in the elderly, and surgery only indicated in case of LCN deficits and/or impending neurologic complications.

Though no recovery of lower CN deficits may be expected, on long-term follow-up, patients usually compensate well for their lower CNs loss by the help of speech and swallowing rehabilitation. Physical therapy is recommended in patients with CN XI paralysis to prevent persistent shoulder dysfunction and pain.

**Conflict of interest** Authors declare no conflict of interests.

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