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Acoustic neuroma surgery and delayed facial palsy

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Abstract Delayed onset of facial palsy is possibly an underestimated but distressing complication of acoustic neuroma surgery. The incidence of this complication reported in the literature has varied from 11.7 to 41%.This study reviewed retrospectively 60 primary acoustic neuroma surgeries performed by a single neurotologist. The deelayed onset of facial dysfunction was defined according to the guidelines described by of Lalwani Butt, Jackler, Pitts and Jingling in 1995. They considered either a deterioration of facial function from normal to abnormal or an increased severity of the degree of facial paralysis, which was grouped using the House-Brackmann scale system. Fifteen of the 60 patients (25%) were found to have a deterioration of facial function. The incidence of delayed facial palsy was not influenced by age, sex or tumor size. The majority of the patients had a favorable prognosis. Only three patients had a grade III–IV facial function at 1 year. It is possible that these latter cases might have benefited from intraoperative meatal facial nerve decompression, as advocated by Sargent, Kartush and Graham.

Key words Acoustic neuroma surgery · Facial palsy · Complications

Introduction

Delayed onset of facial palsy is possibly an underestimated but distressing complication of acoustic neuroma surgery. The reported incidence has varied from 11.7 to 41% [1–6, 10, 13]. The object of the present study was to review retrospectively a series of acoustic neuroma tumors that were surgically removed in order to detail the incidence of delayed facial palsy at University "La Sapienza", Rome, and to assess the course of its recovery.

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Materials and methods

The medical charts of acoustic tumor patients who underwent surgery between 1990 and 1995 at the Second and Fourth ENT Clinics and Neurosurgical Department of University "La Sapienza" were reviewed. Each record was analyzed for sex and age of the patient as well as location and extent of tumor. Different surgical approaches were dictated by the clinical and radiological criteria, and complications noted. The postoperative course of facial function in each patient was assessed by a single author (G. Magliulo) assigning each course to one of the six grades proposed by House and Brackmann [4]. This study included only patients whose clinical chart indicated the status of the facial function immediately after awakening from anesthesia. Postoperatively, facial function was evaluated daily until discharge from the hospital, and thereafter a visual assessment was made at various times during the first postoperative year and finally 2 years following surgery.

In all, 60 patients comprised the study group. There were 39 females and 21 males ranging, from 19 to 74 years of age (mean, 45.7 years). All operative procedures included use of intraoperative electromyographic (EMG) monitoring with a Xomed nerve integrity monitor (NIM-2). During general anesthesia the patients received muscle relaxant drugs only at the time of intubation. No other paralytic agents were administered during surgery in order to avoid interference with evoked motor activity [7, 8]. After tumor removal, the facial nerve was stimulated electrically at the brainstem level and at the internal auditory canal. The minimal level of stimulus-inducing facial muscle contraction and evoking an amplitude of muscle responses was recorded in order to predict postoperative facial function. The method used was proposed by Berges et al. [2].

Twenty-six patients were operated on using a retrosigmoid approach. In 15 of these patients, tumor size was less than 1.5 cm, allowing hearing preservation to be attempted. The remaining patients underwent a translabyrinthine approach. In all patients, tumor was totally resected. All operations were performed by the same surgeon (G.Magliulo).

The guidelines proposed by Lalwani et al. [6] were used. Delayed facial nerve dysfunction was defined as a deterioration of one or more grades of the House-Brackmann scale system [4] after initial assessment in the immediate postoperative period.

Data were statistically evaluated using the analysis of variance and the correlation test. A value of $P < 0.05$ was considered to be significant.

Results

Fifteen of the 60 patients studied (25%) were found to have delayed onsets of facial nerve dysfunction. Eleven

Table 1 Initial facial function and the occurrence of delayed facial palsy in 15 patients using the House-Brackmann [4] classification

Initial facial function	(*n*)	Delayed facial palsy					
		Grade					
		I	II	III	IV	V	VI
I	5	–	2	1	–	–	2
II	8	–	–	2	4	1	1
III	1	–	–	–	1	–	–
IV	1	–	–	–	–	–	1
V	–	–	–	–	–	–	–
VI	–	–	–	–	–	–	–

Table 2 Distribution of the tumor size in patients with and without delayed facial palsy

Tumor size	Delayed facial palsy *n*	Without delayed facial palsy *n*
< 1 cm	7	16
1–3 cm	5	20
> 3 cm	3	9

Table 3 Distribution of the surgical approaches in patients with and without delayed facial palsy

Surgical approach	Delayed facial palsy *n*	Without delayed facial palsy *n*
Retrosigmoid approach	6	20
Translabyrinthine approach	9	25

Table 4 Facial function in patients with and without delayed facial palsy 1 year after tumor resection

Facial function at 1 year	Delayed facial palsy *n*	Without delayed facial palsy *n*
I	7	20
II	5	11
III	1	7
IV	2	3
V	–	2
VI	–	2

patients had an immediate and unchanging dysfunction and were excluded from further analysis.

Eight patients (53.3%) had worsening of facial function that evolved in the first 5 postoperative days. Five cases showed a deterioration within 6–13 days, while two patients had onset of facial dysfunction after 15 days. All of these delayed facial palsies were managed with corticosteroid therapy. The incidences of deteriorated facial function data are summarized in Table 1. Five patients showed a facial nerve compromise of more than four grades. A single grade deterioration occurred in four cases.

Table 5 Comparison between initial and facial function 1 year after surgery in patients with delayed facial palsy

Initial facial function	Delayed facial palsy					
	Grade at 1 year					
	I	II	III	IV	V	VI
II	2	–	–	–	–	–
III	2	1	–	–	–	–
IV	1	2	1	1	–	–
V	1	–	–	–	–	–
VI	1	2	–	1	–	–

Table 6 Initial grade of facial function according to the House-Brackmann [4] classification and the "*R*" predictive ratio of Berges et al. [12]

Initial facial funciton	Delayed facial palsy	
	R < 2	*R* > 2
II	2	–
III	3	–
IV	3	2
V	1	–
VI	2	2

Delayed onset of facial dysfunction was correlatable with the size of tumor (Table 2) and the surgical approach (Table 3). However, statistical analysis revealed no differences between patients that presented with versus patients that did not present with delayed facial palsy, so that data were combined from patients undergoing different surgical approaches. After 1 year, 80% of the patients with delayed palsy had normal (grade I) or near normal (grade II) function (Table 4). Only one patient had grade III function and two cases had grade IV function. A comparison between initial facial nerve functions and long-term function after delayed palsy is depicted in Table 5. The House-Brackmann [4] grade results showed that the majority of patients fell into the categories with excellent or good function. The two patients showing a final grade IV result initially presented with grade VI and IV function, respectively.

When determining the predictive value of intraoperative monitoring using the Berges et al. [2] ratio as based on stimulus intensity and muscle amplitude contraction (Table 6), the ratio is defined by the formula $R = R/R''$. R' is equal to the ratio between the minimal intensity (I) required to induce a response at the level of cerebellopontine angle (CPA) and the amplitude (A) of the muscle contraction ($R' = I(\text{CPA})/A(\text{CPA})$). In R'' the same measurements are taken at the internal auditory canal (IAC) ($R'' = I(\text{IAC})/A(\text{IAC})$). Fifty percent of the patients with $R > 2$ responses had less favorable (grade IV) results.

Discussion

In acoustic neuroma surgery, early postoperative facial function can be variable, remaining stable in some patients, while manifesting some degrees of gradual deterioration in others. Delayed onset of facial palsy can occur in

certain cases and be a clinical problem [1, 13]. This incidence has varied from 11.7% [10] to 41% [6]. Arriaga et al. [1] reviewed a series of 468 patients and found that the percentage of delayed facial palsy was 14.5% (68 patients), which may be an incidence closer to most practices. In reviewing various reports, the definitions of delayed facial palsy have differed. Rosenstock et al. [10] did not use the House Brackmann [4] classification and evaluated only subjects presenting with normal facial function that subsequently deteriorated to complete paralysis. Others such as Arriaga et al. [1] considered delayed facial palsy to be an evolution of an early grade I–IV palsy towards a poor or null function (grades V–VI) at discharge. Lalwani et al. [6] gave a more complete definition that included delayed palsy with minimal deterioration, also a single grade, with the period of observation extended to the first postoperative months. Lalwani's system was adopted by us to analyze our current series in an attempt to provide comparable information delayed facial palsy after acoustic neuroma surgery. Such palsies were found in 30.6% of our patients and had a generally favorable spontaneous recovery. In the series reported by Lalwani et al. [6], 90% of the patients recovered to grade I or II function. An earlier and immediate onset of palsy represents a reliable indicator for denervation. Lalwani's group [6] had similar outcomes with our findings paralleling their observations. Moderate (grades III and IV) function was finally achieved in only three patients.

Our overall findings showed that delayed facial palsy did not affect final facial function. However, certain patients did not recover adequately. At the present time, no definitive studies are available to identify these latter cases, nor to date have we been able to provide a reliable, successful treatment for delayed facial palsy. The possible pathogenetic mechanisms resulting in the development of a delayed facial weakness are still poorly understood. Based upon interval evolution of delayed dysfunction, Lalwani et al. [6] suggested that neural edema may be the cause of the deterioration in facial function observed in the early postoperative period (1–3 days), while a reactivation of a dormant (herpes virus) [3, 9, 12, 14, 15] could explain a delayed palsy that occurs later. In such a condition, prophylactic administration of antiviral agents (such as aciclovir) has been suggested as well as the admnistration of corticosteroids. However, some authors, as exemplified by Lalwani et al. [6], do not support the use of these agents due to the possible development of a potentially dangerous wound infection or the masking of fever. We are unable to address these questions at this time, especially since all of our cases were treated perioperatively with corticosterioids.

Another therapeutic option advocated by Kartush et al. [5] and Sargent et al. [11] involved surgical decompression of the meatal portion of the facial nerve to assure sufficient space for expansion of an edematous facial nerve that otherwise might be compressed in the bony fallopian canal. Our own surgical criteria for nerve injury include the macroscopic presence of edema or discoloration of the facial nerve and changes in electromyographic intraoperative monitoring (traction patterns) or poor facial muscle contraction in response to electrical stimulation of the nerve following tumor removal. However, when the incidence of delayed palsy was compared between patients who were or were not compressed, no differences were noted. Nonetheless, it was our impression that facial recovery was faster and more favorable in the patients who were decompressed.

We evaluated the predictive value of the mathematical ratio of Berges et al. [2]. It was interesting to us to find that 50% of the patients belonging to the group characterized by $R > 2$ exhibited a less acceptable facial function. This approach combined with a macroscopic evaluation of the facial nerve at the end of tumor removal might facilitate the identification of patients for surgical decompression of the facial nerve in the internal auditory meatus. Further study is underway to investigate this possibility.

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