

Postoperative rehabilitation strategies and facial nerve recovery after cerebellopontine angle tumor surgery

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Abstract

Facial nerve injury remains a challenging complication of Cerebellopontine Angle (CPA) tumor surgery, often resulting in prolonged functional impairment. The present study aimed to examine the association between different postoperative rehabilitation strategies and long-term clinical and electrophysiological recovery following facial nerve reconstruction. We conducted a single-center retrospective cohort study including adult patients who underwent CPA tumor resection with intraoperatively confirmed facial nerve injury requiring reconstructive intervention between 2018 and 2023. Based on rehabilitation approaches applied in routine clinical practice, patients were classified into three groups: standard postoperative therapy, standard therapy combined with Transcutaneous Electrical Nerve Stimulation (TENS), and a multimodal rehabilitation strategy integrating TENS with targeted pharmacological support. Clinical follow-up extended to 12 months. Facial nerve function was evaluated using the House–Brackmann scale, Yanagihara system, Sunnybrook Facial Grading System, and Facial Disability Index, alongside surface and needle electromyographic parameters. Multivariable analyses were performed to account for demographic, tumor-related, surgical, and baseline functional factors. All groups demonstrated progressive improvement in facial nerve function over the follow-up period. Patients receiving rehabilitation protocols that included TENS exhibited greater improvements across clinical grading scales and electrophysiological measures compared with those receiving standard therapy alone. The multimodal rehabilitation group showed the largest magnitude of functional recovery, reflected by improved facial symmetry, voluntary movement, patient-reported physical and social function, and more favorable electromyographic patterns. These associations remained consistent after adjustment for potential confounders. In this retrospective cohort, postoperative rehabilitation strategies incorporating electrical stimulation were associated with enhanced functional and electrophysiological recovery after facial nerve reconstruction. Multimodal rehabilitation approaches may offer added translational value in clinical neurorehabilitation following CPA tumor surgery. Prospective studies are warranted to further refine and validate optimized rehabilitation protocols.

Key Words: facial nerve injury, cerebellopontine angle tumors, postoperative rehabilitation, electromyography, retrospective cohort study.

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Facial nerve injury remains one of the most disabling complications following cerebellopontine angle tumor surgery, frequently resulting in facial asymmetry, impaired voluntary movement, synkinesis, and long-term functional and psychosocial consequences.^{1,2} Despite advances in microsurgical techniques and intraoperative monitoring, postoperative facial nerve dysfunction continues to affect

a substantial proportion of patients, particularly those with large tumors, adherent capsules, or complex nerve anatomy.^{3,4} Restoration of facial nerve function therefore depends not only on surgical reconstruction but also on effective postoperative neuromuscular rehabilitation strategies.

Postoperative facial nerve recovery is a complex biolog-

ical process involving axonal regeneration, remyelination, neuromuscular junction reorganization, and central nervous system plasticity.⁵ Clinical recovery is typically slow and incomplete, and functional outcomes vary widely among patients. Standard postoperative management commonly includes corticosteroids to reduce neural edema and inflammation, B-group vitamins to support nerve metabolism, and structured physiotherapy aimed at maintaining muscle tone and preventing contracture.^{2,6} However, these approaches alone often provide limited functional restoration, particularly in cases of severe nerve injury or delayed regeneration.

Electrical stimulation has gained increasing attention as an adjunctive rehabilitation modality for peripheral facial nerve injury. Experimental and clinical studies have demonstrated that neuromuscular electrical stimulation can enhance axonal sprouting, improve muscle excitability, and facilitate functional reinnervation when applied during the appropriate recovery window.^{5,7} Clinical trials in Bell palsy and postoperative facial paralysis have reported faster and more pronounced recovery when electrical stimulation is combined with conventional therapy compared with standard rehabilitation alone.⁸⁻¹⁰ Electromyographic monitoring further allows objective assessment of neuromuscular recovery and provides insight into underlying regeneration processes.

In addition to physical stimulation, pharmacological support plays a critical role in optimizing the neural micro-environment during recovery. Corticosteroids remain a cornerstone of early management due to their anti-inflammatory effects and ability to reduce perineural edema, thereby improving nerve conduction and regeneration conditions.^{2,4} Neurotrophic and neurometabolic agents, including nucleotides and B-vitamins, have been shown to support axonal metabolism, enhance myelin repair, and improve functional outcomes in peripheral nerve injuries.^{6,11} Agents targeting vestibular and microcirculatory disturbances, such as cinnarizine-based combinations and diosmin-containing compounds, may further contribute to symptom relief and neural recovery by improving regional blood flow and reducing secondary ischemic stress.⁴

Emerging translational research also highlights the potential of antioxidant and neuroprotective compounds in peripheral nerve regeneration. Experimental studies have demonstrated that reducing oxidative stress and inflammatory cascades can significantly enhance axonal recovery and functional outcomes after nerve injury.¹²⁻¹⁴ Although such agents are not yet integrated into routine clinical protocols, these findings support the rationale for multimodal rehabilitation strategies that combine physical stimulation with targeted pharmacological interventions to address multiple biological pathways involved in nerve repair.

Despite growing evidence supporting both electrical stimulation and pharmacological therapy, few clinical studies have systematically evaluated their combined effect on postoperative facial nerve recovery following reconstructive surgery. Variability in study design, outcome measures, and rehabilitation protocols has limited the development of standardized postoperative management

strategies.⁵ Moreover, objective electrophysiological outcomes are often underreported, despite their value in elucidating mechanisms of neuromuscular recovery.

Although multimodal postoperative rehabilitation strategies following facial nerve injury are increasingly used, comparative real-world evidence remains limited.⁸⁻¹⁰ In particular, few studies have evaluated the association between routinely implemented rehabilitation combinations and long-term clinical and electrophysiological outcomes using retrospective cohort designs.^{9,15} Addressing this gap may help inform pragmatic rehabilitation decision-making in settings where randomized trials are not feasible.¹⁶

The aim of this retrospective cohort study was to compare three postoperative management strategies following facial nerve reconstruction after cerebellopontine angle tumor surgery: standard postoperative therapy alone, standard therapy combined with transcutaneous electrical nerve stimulation, and a multimodal approach integrating electrical stimulation with targeted pharmacological treatment. By combining validated clinical grading scales with electromyographic assessment, this study seeks to clarify the contribution of multimodal rehabilitation to functional recovery and to provide translational evidence supporting optimized postoperative management of facial nerve injury.

Materials and Methods

Study design and participants

This was a single-center retrospective cohort study conducted at the “Armenia” Medical Center. We reviewed medical records of patients treated between 2018 and 2023 who underwent CPA tumor resection and had intraoperatively confirmed facial nerve injury requiring reconstructive intervention. Data were extracted from operative reports, inpatient charts, rehabilitation records, and follow-up visits, using a standardized data-collection form. Eligible patients were identified from surgical logs and neurosurgery/ENT operative registries. Follow-up assessments at 3 and 12 months were abstracted from scheduled postoperative visits. This manuscript is reported in accordance with the STROBE guideline for observational cohort studies.

Patients were categorized into three groups based on the postoperative rehabilitation protocol implemented in routine care: i) standard therapy; ii) standard therapy + TENS; iii) multimodal rehabilitation (TENS + targeted pharmacotherapy). Group assignment reflected treating-team practice and protocol availability during the study period rather than random allocation. Time zero (baseline) was defined as the date of initiation of postoperative rehabilitation following facial nerve reconstruction.

Patients were included in the retrospective cohort if medical records documented age between 18 and 65 years, a histologically confirmed diagnosis of vestibular schwannoma, meningioma, or epidermoid tumor, unilateral tumor localization, and the development of acute facial nerve neuropathy following CPA tumor resection.

HB, Yanagihara, SFGS, and FDI were documented as part

of routine postoperative follow-up at baseline (postoperative initiation), 3 months, and 12 months. EMG parameters were obtained from the institutional neurophysiology database using standard operating procedures. In the present study, the term facial nerve injury refers to intraoperatively confirmed structural or functional disruption of the facial nerve requiring reconstructive intervention, rather than postoperative clinical severity alone. Surgical decision-making was based primarily on intraoperative findings and neurophysiological evidence, not solely on early postoperative facial grading scores. Indications for facial nerve reconstruction, including hypoglossal-facial nerve anastomosis, comprised the following scenarios: complete anatomical transection of the facial nerve; segmental nerve loss or irreversible structural damage precluding direct end-to-end repair; severe nerve ischemia or crush injury with absent or non-conductive intraoperative electromyographic responses; and loss of proximal nerve excitability indicating a high risk of delayed or incomplete spontaneous recovery.

Importantly, early postoperative facial function did not reliably reflect the extent of nerve injury, as transient conduction, partial axonal continuity, or compensatory muscle activation may result in House-Brackmann grades II-III or Yanagihara scores above 20 during the immediate postoperative period, despite substantial intraoperative nerve damage. In such cases, intraoperative EMG monitoring demonstrated absent or critically reduced compound muscle action potentials, prolonged latency, or loss of proximal nerve stimulation response, indicating poor regenerative potential without surgical reconstruction. Consequently, patients with apparently mild early postoperative palsy were included when intraoperative findings

supported a high likelihood of progressive deterioration, delayed paralysis, or incomplete recovery if reconstruction was not performed.

Hypoglossal-facial nerve anastomosis was therefore undertaken not as a response to clinical grading severity alone, but as a preventive and restorative strategy in patients with confirmed intraoperative nerve compromise. This approach reflects established surgical practice in complex cerebellopontine angle tumor surgery, where delayed facial paralysis and late functional decline are well documented. All 127 patients met at least one predefined intraoperative or neurophysiological criterion for facial nerve reconstruction, ensuring homogeneity of surgical indication and allowing meaningful interpretation of postoperative recovery trajectories within a reconstructed cohort (Table 1).

Exclusion criteria were non-fulfillment of inclusion criteria, diagnosis of neurofibromatosis type II, acute intracranial hemorrhage, myocardial infarction, active infectious disease, decompensated chronic conditions such as severe cardiovascular disease or uncontrolled diabetes mellitus, severe psychiatric disorders including schizophrenia or bipolar affective disorder, and lack of histopathologic confirmation of tumor type.

Comorbid conditions were frequently documented in the study cohort. The most commonly recorded comorbidities included arterial hypertension, gastrointestinal pathology, and type 2 diabetes mellitus. Less frequent conditions comprised atrial fibrillation, atherosclerosis, and elevated body mass index (BMI >25 kg/m²). According to operative reports, the extent of tumor resection varied across groups, with total removal achieved in 13.63%, 14.63%, and 19.05% of patients in Groups 1, 2, and 3, respectively;

Table 1. Surgical indications for facial nerve reconstruction in the study cohort (n=127).

Indication category	Intraoperative criteria	Rationale for reconstruction
Complete facial nerve transection	Visual confirmation of nerve discontinuity	No possibility of spontaneous regeneration
Segmental nerve loss or severe structural damage	Loss of nerve continuity or non-repairable segment	Direct repair not feasible
Severe ischemic or crush injury	Pale nerve, loss of elasticity, absent proximal stimulation	High risk of delayed degeneration
Absent or critically reduced intraoperative EMG response	No compound muscle action potentials or markedly prolonged latency	Poor regenerative prognosis
Progressive intraoperative loss of nerve excitability	Deterioration during tumor dissection	Anticipated delayed facial paralysis
Apparent mild early postoperative palsy with intraoperative evidence of injury	HB II-III or Yanagihara >20 with intraoperative EMG failure	Clinical grading underestimates injury severity

subtotal resection (<5% residual) and partial resection (>5% residual) were more common. Clinical and neurological findings were abstracted retrospectively from preoperative and postoperative medical records, including documentation of medical history, physical examination, and standard neurological assessments of tendon reflexes, muscle tone, muscle strength, balance, and sensory function, performed to exclude additional neurological deficits. Facial nerve function was evaluated using established grading instruments recorded in routine clinical follow-up, including the House-Brackmann scale,¹⁷ the Yanagihara system,⁹ the Facial Disability Index (FDI),¹⁸ and the Sunnybrook Facial Grading System (SFGS),¹⁰ each applied in accordance with published protocols. The House-Brackmann scale grades facial function from I (normal) to VI (total paralysis), the Yanagihara system scores ten facial movements from 0 to 4 with standardized severity categories, the FDI provides patient-reported physical and social function scores, and the SFGS integrates resting symmetry, voluntary movement, and synkinesis into a composite 100-point score. Adherence to rehabilitation protocols was not formally quantified, completion of TENS courses and pharmacological treatment was inferred from documented protocol delivery in rehabilitation records.

Tumor characteristics

Tumor characteristics were abstracted retrospectively from preoperative imaging reports and operative records and classified according to the KOOS¹⁹ and Samii²⁰ systems. Based on KOOS staging, 49.6% of tumors were classified as grade IV. Mean tumor size was 38.16 ± 7.21 mm in Group 1, 36.93 ± 5.86 mm in Group 2, and 36.29 ± 4.31 mm in Group 3. Tumor margins were described as indistinct in 65.9%, 65.8%, and 69.0% of cases in Groups 1, 2, and 3, respectively. Histopathological examination identified vestibular schwannoma as the predominant diagnosis, accounting for 75.0%, 75.6%, and 78.6% of tumors in Groups 1, 2, and 3, respectively, while meningioma and epidermoid tumors were less frequent. Tumor capsule adhesion to adjacent structures was documented as complete in approximately half of cases, partial in about one quarter, and absent in the remaining patients. The anatomical relationship of the facial nerve to the tumor capsule was most commonly anterior, followed by posterior and lateral positions.

Surgical intervention and postoperative care

Hypoglossal-facial nerve anastomosis was performed selectively in patients with intraoperatively confirmed facial nerve injury associated with a low likelihood of spontaneous functional recovery. The decision was based on predefined anatomical and neurophysiological criteria, including complete anatomical transection of the facial nerve, segmental nerve loss or irreversible structural damage precluding direct end-to-end repair, and severe ischemic or crush injury with absent or non-conductive intraoperative electromyographic responses. In addition, hypoglossal-facial anastomosis was undertaken in cases

demonstrating progressive loss of facial nerve excitability during tumor dissection or absent proximal stimulation, indicating poor regenerative potential. Importantly, surgical decision-making was guided by intraoperative findings and neurophysiological monitoring rather than early postoperative facial grading alone, as transient postoperative facial function may underestimate the severity of underlying nerve injury.

Surgical treatment involved hypoglossal-facial anastomosis under general anesthesia. The patient was placed supine with the head rotated to the contralateral side. An incision according to Le Fort was made, and subplatysmal flaps elevated. The sternocleidomastoid muscle was retracted posteriorly, and the facial nerve was identified at the stylomastoid foramen. The hypoglossal nerve was dissected distally and mobilized to allow tension-free epineural end-to-end anastomosis with the facial nerve, secured with 8-0 nylon sutures and reinforced with fibrin glue. The digastric muscle was preserved to provide an additional protective layer. A drain was placed and removed on the first postoperative day.

Postoperative management varied according to study group. Group 1 received standard postoperative therapy (Prednisone 60 mg orally daily for 5 days, then taper by 10 mg every day over 5 days (Duration: 10 days); Vitamin B6 (Pyridoxine) 50-100 mg orally (daily, duration: 30 days); Furosemide (Lasix) (optional, if facial swelling/edema suspected) 20 mg orally, once daily in the morning (Duration: 5-7 days)). Group 2 received standard therapy plus transcutaneous electrical nerve stimulation (TENS), which involved 30 Hz impulses of 2-6 mA intensity and 100 μ s duration applied for 10 minutes per session to both distal and proximal segments of the injured facial nerve, as well as to motor points, for 10 consecutive daily sessions per course, repeated at 3-month intervals. Group 3 received combined therapy consisting of TENS and pharmacologic treatment: dexamethasone 20 mg/day for 3 days, tapered by 4 mg every 3 days; Nucleo CMF-forte 61 mg/day intramuscularly for 6 days followed by oral administration twice daily for 9-10 days; Arlevert (40 mg cinnarizine + 20 mg dimenhydrinate) twice or three times daily for 20 days; and Venosmin (500 mg diosmin) twice daily for 7 days, then 1.000 mg once daily for a total of 20 days. Dexamethasone was omitted in subsequent treatment courses.

In this study, the term reinnervation refers exclusively to surgically achieved facial nerve reinnervation following hypoglossal-facial nerve anastomosis and does not imply spontaneous nerve recovery (Figure 1). All patients in Group 1, as well as in Groups 2 and 3, underwent facial nerve reconstruction prior to initiation of postoperative therapy. Accordingly, standard postoperative therapy following reinnervation denotes routine postoperative medical management initiated after confirmed surgical facial nerve reinnervation and does not include patients managed expectantly for spontaneous reinnervation alone.

Patients were allocated to three postoperative management groups to test a stepwise clinical question relevant to translational neuromuscular rehabilitation after facial nerve reconstruction (Supplementary Table 1). Group I

represented the reference standard of routine postoperative care used in our center, including anti-inflammatory therapy and neurotropic support, reflecting real-world baseline management. Group II isolated the incremental effect of adding transcutaneous electrical nerve stimulation to the same standard care, allowing evaluation of whether neuromuscular activation contributes additional benefit beyond usual therapy. Group III evaluated a pragmatic multimodal rehabilitation package in which electrical stimulation was combined with a targeted pharmacological regimen intended to optimize the biological and symptomatic environment for nerve recovery, addressing inflammation, neurometabolic support, and symptom-limiting factors that may reduce rehabilitation participation. This three-arm structure was selected to distinguish between standard management, the additive effect of electrical stimulation, and the clinical impact of a comprehensive multimodal strategy that reflects common translational practice when single-modality approaches are insufficient. The study was not designed to isolate individual drug effects within Group III, but to compare clinically implementable postoperative protocols as bundled strategies.

Transcutaneous electrical nerve stimulation

In the postoperative period, patients with facial nerve neuropathy (groups 2 and 3) underwent a combined rehabilitation program consisting of facial nerve electrostimulation and pharmacological therapy. Electro-stimulation was initiated on the 11th day of pharmacological treatment. The affected facial region was continuously stimulated for 10 minutes using biphasic rectangular pulses at a frequency of 30 Hz, with the current intensity gradually increased from 2 mA to 6 mA in discrete increments. Stimulation was applied to both the distal segment of the injured facial nerve (mastoid process exit point) and

the proximal segment (site of separation of the greater petrosal nerve and chorda tympani from the main trunk), as well as to identified motor points. Each stimulation site was activated for 5-10 seconds per cycle. The pulse duration was 100 μ s. Stimulation was delivered with a clinically approved TENS (BTL-4000 Premium physiotherapy unit, BTL Industries Ltd., United Kingdom), and was performed in 3 treatment courses, each consisting of 10 daily sessions, with 3-month intervals between courses. The selected TENS parameters (30 Hz frequency, 2-6 mA intensity, 100 μ s pulse width, and short-duration sessions) were based on prior clinical studies and consensus recommendations indicating favorable balance between neuromuscular activation and safety in facial nerve rehabilitation, particularly following surgical reconstruction.

Electromyography

Electromyographic (EMG) studies were performed bilaterally on the main branches of the facial nerve (BIOPAC Systems Inc., USA). M-responses were recorded from the frontal belly of the occipitofrontalis, orbicularis oculi, and orbicularis oris muscles. EMG was performed with the patient under controlled neuromuscular blockade using short-acting agents, with relaxation monitored via TOF-Guard. Electrical stimulation was applied to the branches of the facial nerve, and responses were analyzed for latency, amplitude, waveform, and duration. Both spontaneous EMG and triggered EMG were used intraoperatively for facial nerve monitoring. Intraoperative electromyography was used exclusively for real-time facial nerve monitoring and surgical decision-making during tumor resection and reconstruction. Postoperative EMG assessments were performed separately during follow-up visits to evaluate neuromuscular reinnervation and recovery and were not used to guide intraoperative management.

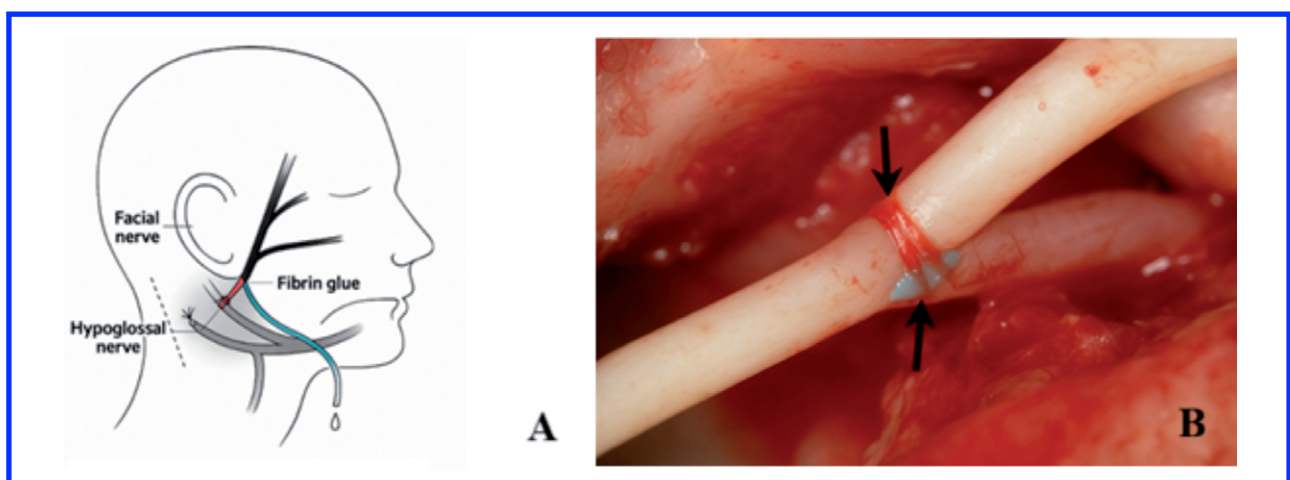


Figure 1. Schematic representation of hypoglossal-facial nerve anastomosis. (A) The hypoglossal nerve is dissected distally and mobilized to achieve a tension-free, epineural end-to-end anastomosis with the facial nerve. The coaptation is secured with 8-0 nylon sutures and reinforced with fibrin glue. (B) Intraoperative view of hypoglossal-facial nerve anastomosis. Black arrows indicate the site of epineural end-to-end coaptation secured with sutures and fibrin glue.

Statistical analysis

Statistical analysis was conducted with SPSS version 23. Normality was tested with the Kolmogorov-Smirnov test. Intergroup comparisons were made using Student's t test for independent samples, with variance homogeneity assessed by Fisher's F test. Paired t tests were used to compare pre- and postoperative values within groups. Categorical variables were analyzed using the chi-square test. A p value of less than 0.05 was considered statistically significant.

Because treatment allocation was nonrandom, we assessed baseline balance across groups and adjusted for confounding using multivariable regression models. Candidate confounders included age, sex, tumor size, histopathology, extent of resection, presence/absence of intraoperative EMG monitoring, and baseline facial function severity. Adjusted between-group differences in 12-month outcomes were estimated with linear regression (continuous scales) and logistic regression (binary symptom outcomes). Missing data were infrequent (<5% for all variables) and were handled using complete-case analysis. Prior to multivariable modeling, collinearity among candidate covariates was assessed using variance inflation factors, with all values remaining below commonly accepted thresholds. For linear regression models, assumptions of normality, linearity, and homoscedasticity of residuals were evaluated through visual inspection of residual plots and Q-Q plots, and no major violations were observed.

Results

Study population and group classification

A total of 127 eligible patients were included. Based on the postoperative rehabilitation protocol delivered in routine care, 44 received standard therapy, 41 received standard therapy plus TENS, and 42 received multimodal rehabilitation (TENS plus targeted pharmacotherapy). Baseline sociodemographic characteristics of the study groups are summarized in Table 2, demonstrating broadly comparable distributions across age, sex, education, and socioeconomic variables. Patients were identified from surgical logs; exclusions were applied per predefined criteria.

Baseline sociodemographic characteristics of the study groups

The general sociodemographic profile of participants is presented in Table 2. The study population included 53.5% participants younger than 50 years, with a nearly equal distribution of men and women (49.6% male). The most common educational level was higher education (44.1%), and the majority resided in urban areas (62.2%). Approximately half of participants were married or cohabiting (48.0%), and 61.2% had at least one child. Employment rate was 66.1%, and 39.4% had health insurance coverage. Household income at the time of survey was most frequently in the range of 121,000-320,000 AMD (47.2%) or 321,000-600,000 AMD (37.0%).

Baseline clinical and comorbidity profile

Comorbid conditions were common in the study cohort. Arterial hypertension, gastrointestinal pathology, and type 2 diabetes mellitus were the most frequent (Table 3). Other comorbidities included atrial fibrillation, atherosclerosis, and elevated body mass index (BMI >25 kg/m²). Distribution of patients according to the extent of tumor resection showed that total removal was achieved in 13.63%, 14.63%, and 19.05% of Groups 1, 2, and 3, respectively. Subtotal resection (<5% residual) and partial resection (>5% residual) were more frequent (Table 4).

Clinical evaluation included detailed medical history and physical examination. Neurological examination assessed tendon reflexes, muscle tone, muscle strength, balance, and sensory function, with the aim of excluding other neurological deficits. Facial muscle function was evaluated by established grading systems: the House-Brackmann scale,¹⁷ the Yanagihara system,⁹ the Facial Disability Index (FDI),¹⁸ and the Sunnybrook Facial Grading System (SFGS).¹⁰ Each system was applied according to published protocols. The House-Brackmann scale classified function from grade I (normal) to grade VI (total paralysis). The Yanagihara system rated 10 facial movements from 0 (total paralysis) to 4 (normal movement), with total scores interpreted as normal (40 points), mild dysfunction (32-38), moderate dysfunction (24-30), moderately severe dysfunction (16-22), severe dysfunction (8-14), or total paralysis (0-6). The FDI assessed physical and social function via patient self-report, producing separate scores for social/well-being function and physical function. The SFGS combined assessments of resting symmetry, voluntary movement symmetry, and synkinesis into a 100-point score.

Tumor characteristics and surgical features

Tumor characteristics were assessed preoperatively using both the KOOS¹⁹ and Samii²⁰ classifications (Table 5). According to KOOS staging, 49.6% of tumors corresponded to grade IV. The average tumor size was 38.16 ± 7.21 mm in Group 1, 36.93 ± 5.86 mm in Group 2, and 36.29 ± 4.31 mm in Group 3. Tumor boundaries were indistinct in 65.9%, 65.8%, and 69.0% of patients in Groups 1, 2, and 3, respectively. Histopathological analysis demonstrated that vestibular schwannoma was the most frequent diagnosis, occurring in 75.0%, 75.6%, and 78.6% of patients in Groups 1, 2, and 3, respectively. Meningioma and epidermoid tumors were less frequent. Tumor capsule adhesion to surrounding structures was complete in approximately half the cases, partial in about a quarter, and absent in the remainder. With regard to the position of the facial nerve relative to the tumor capsule, anterior location was most frequent, followed by posterior and lateral positions. Tumor classification followed both KOOS and Samii systems. The KOOS classification stages tumors from I (intracanalicular, ≤10 mm CPA component) to IV (CPA component >30 mm with brainstem compression).¹ The Samii classification categorizes tumor extent from T1 (intrameatal) to T4b (gross brainstem and fourth ventricle deformation).²⁰

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Preoperative and postoperative imaging consisted of contrast-enhanced Computed Tomography (CT) scans (Tomoscan SR 5000, Philips) using intravenous Omnipaque. Imaging served to define tumor localization, size, and boundaries. Preoperative imaging was performed using contrast-enhanced CT due to institutional protocols and

scanner availability during the study period. Although Magnetic Resonance Imaging (MRI) with dedicated skull base and internal auditory canal sequences is the guideline-recommended modality for diagnosis and characterization of cerebellopontine angle tumors because of its superior soft-tissue contrast and multiplanar assessment

Table 2. Sociodemographic profile of study participants.

Variable	Group 1 (n=44, %)	Group 2 (n=41, %)	Group 3 (n=42, %)	Total (n=127, %)
Age				
18-35 years	9 (20.45)	8 (19.5)	12 (28.57)	29 (22.8)
36-50 years	14 (31.8)	13 (31.1)	12 (28.57)	39 (30.7)
51-65 years	21 (47.7)	20 (48.8)	18 (42.9)	59 (46.5)
Sex				
Male	21 (47.73)	21 (51.22)	21 (50)	63 (49.6)
Female	23 (52.27)	20 (48.78)	21 (50)	64 (50.4)
Education				
Primary	6 (13.64)	5 (12.20)	5 (11.90)	16 (12.6)
Secondary vocational	11 (25)	10 (24.40)	11 (26.20)	32 (25.2)
Higher	18 (40.91)	19 (46.34)	19 (45.24)	56 (44.09)
Academic degree	9 (20.45)	7 (17.07)	7 (16.67)	23 (18.11)
Residence				
Urban	27 (61.4)	25 (61.0)	27 (64.3)	79 (62.2)
Rural	17 (38.6)	16 (39.0)	15 (35.7)	48 (37.8)
Marital status				
Married/cohabiting	21 (47.73)	19 (46.34)	21 (50)	61 (48.03)
Single	12 (27.27)	13 (31.71)	11 (26.20)	36 (28.35)
Divorced/separated	6 (13.64)	5 (12.20)	5 (11.90)	16 (12.60)
Widowed	5 (11.36)	4 (9.76)	5 (11.90)	14 (11.02)
Children				
Yes	26 (59.09)	25 (60.98)	24 (57.14)	75 (59.1)
No	18 (40.91)	16 (39.02)	18 (42.86)	52 (40.9)
Employment				
Employed	27 (61.36)	29 (70.73)	28 (66.67)	84 (66.14)
Unemployed	17 (38.64)	12 (29.27)	14 (33.33)	43 (33.86)
Insurance				
Yes	17 (38.64)	17 (41.46)	16 (38.10)	50 (39.37)
No	27 (61.36)	24 (58.54)	26 (61.90)	77 (60.63)
Income				
<120,000 AMD (<\$300)	4 (9.09)	3 (7.32)	3 (7.14)	10 (7.87)
121,000-320,000 AMD (\$300-800)	20 (45.45)	20 (48.78)	20 (47.62)	60 (47.24)
321,000-600,000 AMD (\$800-1,500)	16 (36.36)	15 (36.59)	16 (38.10)	47 (37.01)
>600,000 AMD (>\$1,500)	4 (9.09)	3 (7.32)	3 (7.14)	10 (7.87)

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of cranial nerve and brainstem relationships, particularly in vestibular schwannoma, and is emphasized in both the European Association of Neuro-Oncology (EANO) and Congress of Neurological Surgeons (CNS) imaging rec-

ommendations, MRI was not universally available for all patients in this cohort. MRI remains the imaging standard where available and should be utilized when feasible to optimize diagnostic accuracy and surgical planning.

Table 3. Comorbidity profile of study participants.

Comorbidity	Group 1 (n=44, %)	Group 2 (n=41, %)	Group 3 (n=42, %)
Arterial hypertension	20 (45.45)	22 (53.65)	28 (48.8)
Atrial fibrillation	6 (13.63)	8 (19.51)	5 (11.9)
Atherosclerosis	6 (13.63)	4 (9.76)	7 (15.9)
Prior cerebrovascular accident	27 (61.36)	20 (48.78)	26 (61.9)
Type 2 diabetes mellitus	21 (47.72)	25 (60.98)	22 (52.38)
BMI > 25 kg/m ²	7 (15.9)	6 (14.6)	6 (14.3)
Gastrointestinal disease	13 (29.55)	14 (34.15)	20 (47.62)

Table 4. Tumor profile according to histopathology, capsule adhesion, and facial nerve location.

Parameter	Group 1 (n=44, %)	Group 2 (n=41, %)	Group 3 (n=42, %)
Histopathological type			
Neurinoma	33 (75.0)	31 (75.6)	33 (78.57)
Meningioma	9 (20.45)	8 (19.51)	6 (14.29)
Epidermoid	2 (4.55)	2 (4.88)	3 (7.14)
Capsule adhesion			
Complete adhesion	21 (47.72)	21 (51.22)	20 (47.61)
Partial adhesion	12 (27.27)	10 (24.39)	12 (28.57)
No adhesion	11 (25.0)	10 (24.39)	10 (23.8)
Facial nerve position relative to tumor capsule			
Anterior surface	22 (50.0)	20 (28.1)	21 (36.6)
Posterior surface	11 (25.0)	11 (26.52)	10 (23.8)
Lateral surface	11 (25.0)	10 (24.39)	11 (26.19)

Table 5. Types of tumor resection performed in the study cohort.

Resection type	Group 1 (n=44, %)	Group 2 (n=41, %)	Group 3 (n=42, %)
Total removal	6 (13.63)	6 (14.63)	8 (19.05)
Subtotal (<5% residual)	14 (31.8)	13 (31.71)	12 (28.57)
Partial (>5% residual)	24 (54.55)	22 (53.66)	22 (52.38)

Longitudinal changes in clinical symptoms

Clinical symptom analysis (Supplementary Table 2) demonstrated a gradual regression of facial muscle weakness across all treatment groups. At baseline, nearly all patients exhibited muscle weakness (97.7% in Group I, 95.1% in Group II, and 95.2% in Group III). After 12 months, this symptom persisted in 59.1% of Group I, 51.2% of Group II, and only 26.2% of Group III patients, indicating a significantly greater improvement with the modified treatment and electrical stimulation protocol. Other clinical manifestations, including involuntary twitching, impaired taste sensation, ocular dryness, and mastoid pain, also regressed more markedly in Group III compared with Groups I and II. For example, involuntary twitching persisted in 19.0% of Group III patients at 12 months, compared with 29.6% in Group I and 26.8% in Group II.

Functional recovery assessed by clinical grading scales

Functional recovery was further supported by improvements in standardized scales (Table 6). House-Brackmann (HB) grades 2-3 decreased steadily in all groups, but Group III showed the most favorable reduction, reaching a mean of 0.89 ± 0.19 at 12 months, compared with 1.38 ± 0.57 in Group I and 1.32 ± 0.74 in Group II. Similarly, Yanagihara and Sunnybrook Facial Grading System (SBFGS) scores showed the greatest gains in Group III (38.3 ± 2.79 and 98.1 ± 4.39 , respectively), nearly doubling the improvements observed in Group I. Patient-reported outcomes reflected this trend, with the Facial Disability Index (FDI) revealing superior physical and social function recovery in Group III at 12 months.

Statistical analysis of intragroup changes (Table 7) confirmed these observations. The highest t-values across nearly all scales were observed in Group III, particularly for SFGS ($t = 55.92$, $p < 0.0001$) and FDI physical function ($t = 37.24$, $p < 0.0001$), underscoring the robustness of improvement in this group. In contrast, Groups I and II showed significant but more modest changes, and in some cases, no significant improvement (e.g., HB grade 4-5 asymmetry in Group I).

Electrophysiological outcomes

Electrophysiological data (Table 4) also corroborated these findings. In Group III, M-response distal latency improved from 6.31 ± 1.06 ms at baseline to 3.64 ± 0.28 ms at 12 months, and M-response threshold decreased from 24.4 ± 3.21 mA to 9.6 ± 3.28 mA, reflecting restored nerve excitability. Spontaneous EMG activity nearly disappeared in Group III (0.7 ± 0.05 at 12 months), whereas residual activity persisted in the other groups.

A summary of intergroup comparisons of EMG parameters is presented in Figure 2. Group III demonstrated the highest t-values across M-response threshold and spontaneous activity, confirming superior electrophysiological recovery compared with Groups I and II. This aligns with the clinical and functional outcomes, highlighting the efficacy of the modified protocol combined with electrical stimulation in promoting nerve regeneration and functional recovery. T-values are presented to illustrate the magnitude of within-group change over time rather than to infer causality between treatment strategies.

After multivariable adjustment, the association between postoperative rehabilitation strategy and 12-month facial nerve recovery outcomes remained statistically significant.

Table 6. Dynamics of the regression of assessment scale scores in patients with intraoperative injury of the facial nerve, evaluated before treatment and at 3 and 12 months post-treatment.

		Group I (n=44) Standard treatment protocol			Group II (n=41) Standard treatment protocol and electrical stimulation			Group III (n=42) Modified treatment protocol and electrical stimulation		
		Before treatment	3 months after treatment	12 months after treatment	Before treatment	3 months after treatment	12 months after treatment	Before treatment	3 months after treatment	12 months after treatment
HB	2-3	2.74±0.48	1.69±0.52	1.38±0.57	2.81±0.52	1.76±0.77	1.32±0.74	2.7±0.64	1.29±0.21	0.89±0.19
	4-5	4.39±1.76	3.72±0.81	3.59±3.71	4.26±1.0	4.15±2.92	3.9±1.88	4.21±1.89	1.93±0.37	1.38±0.54
Yanagihara		23.5±4.17	27.0±3.11	29.1±0.98	24.3±4.12	31.2±3.36	32.0±2.79	24.1±4.12	32.2±3.38	38.3±2.79
SBFGS (aggregate value)		27.69±7.66	41.45±6.61	59.76±4.32	29.0±7.57	50.21±8.0	62.12±6.98	28.0 ±6.84	80.22±4.17	98.13±4.39
FDI	PF	38.10±3.24	44.32±3.03	52.51±2.36	38.7±3.79	47.4±2.57	50.1±2.97	37.9±4.29	54.2±3.28	69.9±3.55
	SF	66.1±5.71	69.1±4.21	75.1±3.68	66.2±5.32	79.2±4.32	80.2±4.04	66.7±4.41	84.1±3.74	92.5±3.19

Table 7. EMG results before and after therapy.

	Group I (n=44) Standard treatment protocol			Group II (n=41) Standard treatment protocol and electrical stimulation			Group III (n=42) Modified treatment protocol and electrical stimulation		
	Before treatment	3 months after treatment	12 months after treatment	Before treatment	3 months after treatment	12 months after treatment	Before treatment	3 months after treatment	12 months after treatment
M-response distal latency index, in ms	6.34±0.93	6.19±0.35	5.3±0.71	6.46±0.88	6.04±0.78	4.78±0.75	6.31±1.06	4.09±0.41	3.64±0.28
Amplitude, mV	0.81±0.24	0.87±0.11	1.52±0.12	0.84±0.14	1.09±0.05	1.49±0.04	0.82±0.13	1.21±0.36	1.28±0.24
M-response threshold, mA	24.1±4.19	21.7±6.57	20.2±4.89	23.9±3.32	19.6±2.24	17.3±2.36	24.4±3.21	19.0±2.14	9.6±3.28
Spontaneous EMG activity	3.0±1.38	2.7±1.17	1.9±0.65	3.29±1.44	2.3±0.85	1.2±0.06	3.16±0.39	1.8±0.11	0.7±0.05
Neurogenic motor unit potentials (MUPs)	+	+	+	+	+	+	+	+	-

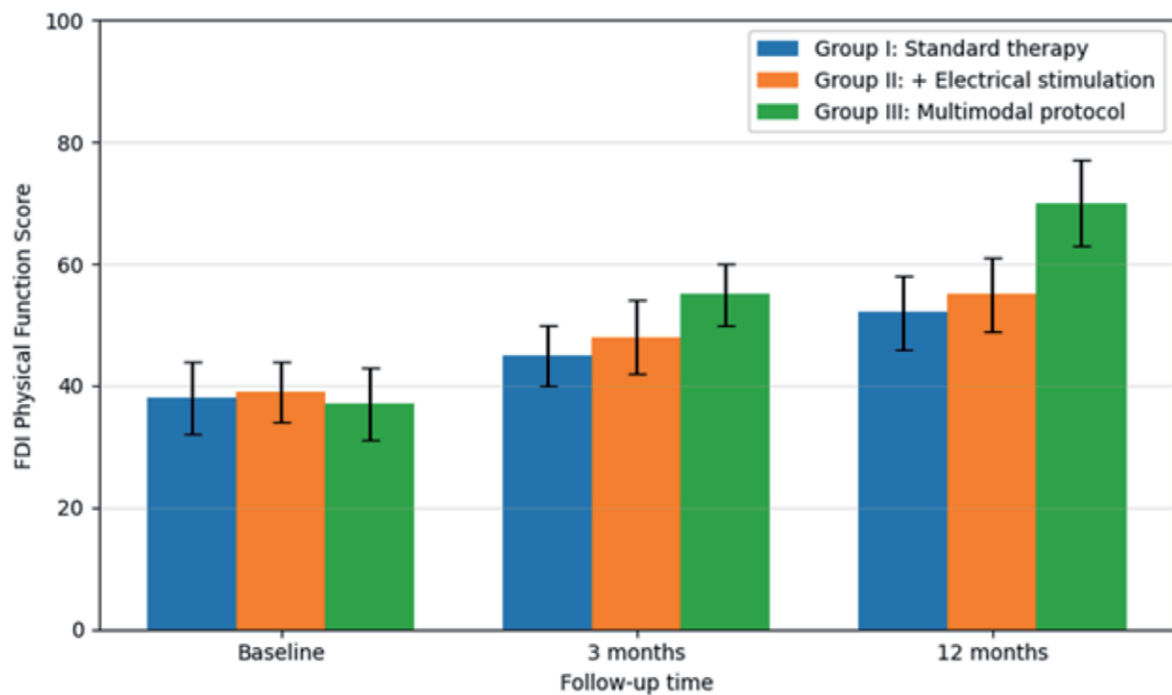


Figure 2. Facial Disability Index physical function (FDI-PF) scores at baseline, 3 months, and 12 months in patients receiving standard postoperative therapy (Group I), standard therapy plus transcutaneous electrical nerve stimulation (Group II), and multimodal rehabilitation including electrical stimulation and pharmacological support (Group III). Bars represent mean ± standard deviation.

In models adjusted for age, sex, tumor size, histopathology, extent of tumor resection, presence of intraoperative EMG monitoring, and baseline facial nerve function severity, patients managed with the multimodal rehabilitation protocol (Group III) demonstrated significantly greater improvements in House–Brackmann grade, Yanagihara score, Sunnybrook Facial Grading System score, and Facial Disability Index physical and social subscales compared with both standard therapy alone (Group I) and standard therapy plus TENS (Group II) (all adjusted $p < 0.01$). Similarly, adjusted analyses of electrophysiological outcomes showed larger reductions in M-response latency and threshold, as well as greater suppression of spontaneous EMG activity in Group III at 12 months. Adjustment for potential confounders did not materially alter the direction or magnitude of the observed between-group differences, supporting the robustness of the unadjusted findings.

Intergroup comparisons of clinical and electrophysiological recovery

Intergroup comparisons demonstrated differential recovery trajectories across the three postoperative management strategies over the 12-month follow-up period (Figures 2, 3 and 4). As illustrated in Figure 3, Sunny-

brook Facial Grading System scores improved progressively in all groups, with the greatest magnitude of change observed in the multimodal rehabilitation group. The largest absolute improvement was observed in Group III, which approached near-normal facial function by the final assessment. Group II exhibited greater gains than Group I at both follow-up time points, while Group I showed more gradual improvement over time. Figure 2 illustrates corresponding changes in patient-reported physical function measured by the Facial Disability Index (FDI-PF). Mean FDI-PF scores improved in all groups across follow-up, with Group III demonstrating the highest scores at both 3 and 12 months, followed by Group II and Group I, respectively. Electrophysiological comparisons at 12 months are summarized in Figure 4, which presents t-values for within-group changes in EMG parameters relative to baseline. Group III exhibited the largest magnitude of change across all electrophysiological measures, particularly for M-response threshold and spontaneous EMG activity, whereas Groups II and I showed progressively smaller changes. Collectively, these intergroup comparisons indicate a graded pattern of clinical and electrophysiological recovery across treatment arms, with the most pronounced changes consistently observed in the multimodal rehabilitation group.

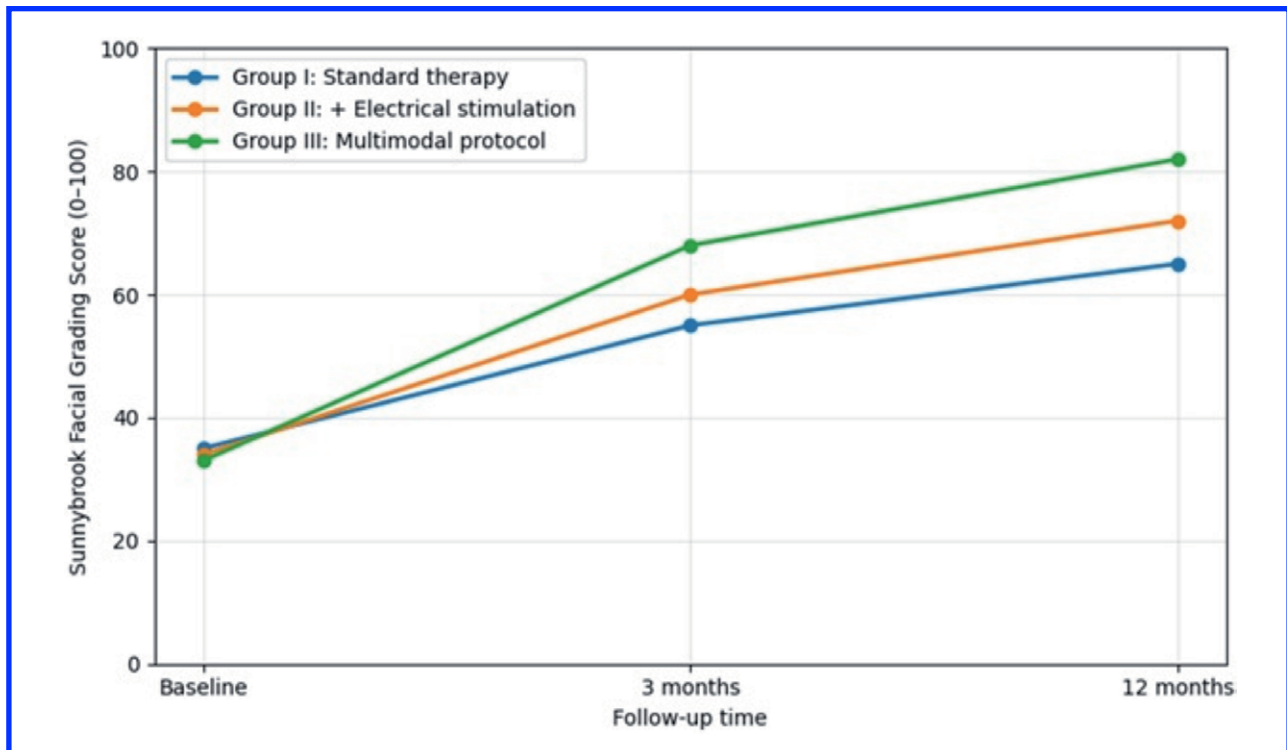


Figure 3. Mean Sunnybrook Facial Grading System scores (0-100) at baseline, 3 months, and 12 months following facial nerve reconstruction in patients receiving standard postoperative therapy (Group I), standard therapy plus transcutaneous electrical nerve stimulation (Group II), and multimodal rehabilitation including electrical stimulation and pharmacological support (Group III). Higher scores indicate better facial function. Values are presented as group means.

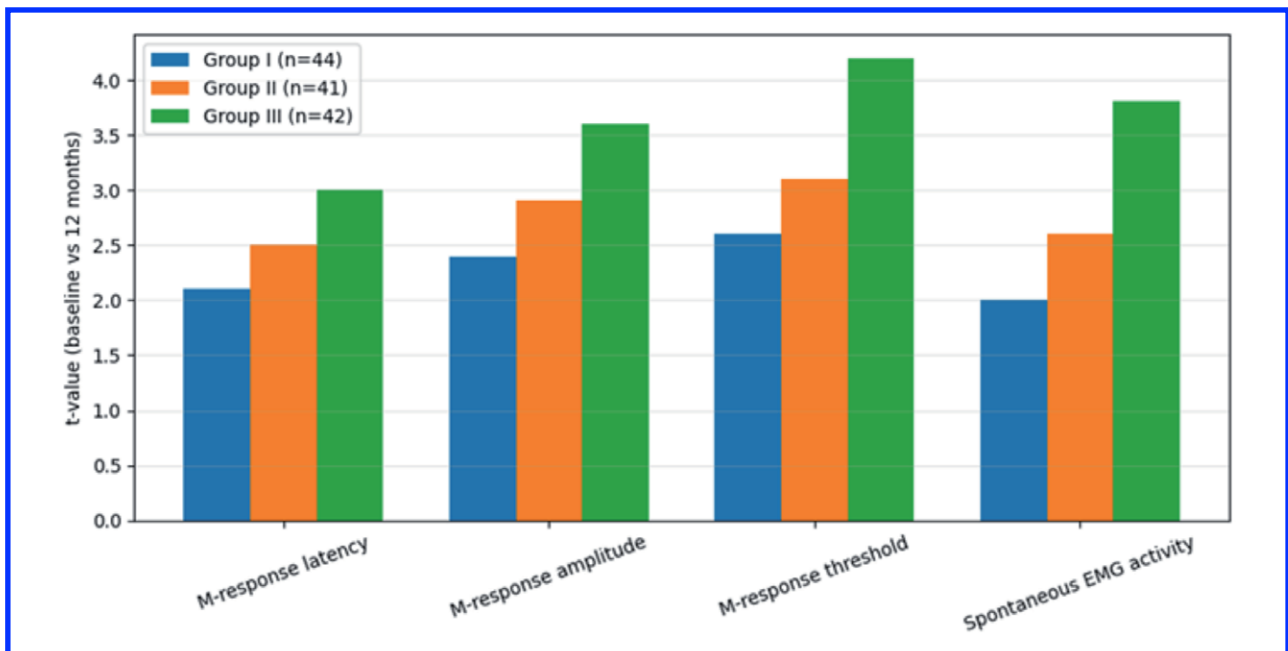


Figure 4. Changes in electromyographic (EMG) parameters at 12 months of follow-up across treatment groups. Bars represent t-values for within-group comparisons between baseline and 12 months in Group I (n=44), Group II (n=41), and Group III (n=42) for M-response distal latency, M-response amplitude, M-response threshold, and spontaneous EMG activity. Higher t-values indicate greater magnitude of change over time within each group.

Discussion

The present study evaluated functional recovery after intraoperative facial nerve injury in patients undergoing cerebellopontine angle tumor resection, with particular emphasis on comparing three postoperative rehabilitation strategies: standard therapy, standard therapy combined with TENS, and a multimodal approach incorporating TENS with targeted pharmacotherapy. The findings underscore several important aspects: the relevance of perioperative risk factors, the clinical trajectory of facial nerve recovery, and demonstrated larger magnitude of improvement in functional restoration. Given the retrospective design and nonrandom allocation of postoperative rehabilitation strategies, findings should be interpreted as associations. Residual confounding due to case complexity, rehabilitation adherence, or protocol availability may persist despite adjustment.

Our results demonstrated that larger tumor size (>3 cm), complete tumor removal, absence of EMG monitoring, longer preoperative symptom duration, and certain histopathological types (meningioma and epidermoid tumors) were significantly associated with higher rates of intraoperative facial nerve injury. These findings are consistent with prior literature showing that tumor size and adhesion to the facial nerve capsule significantly increase manipulation-induced injury risk during resection.¹ The lack of significant association between patient age, surgical approach, tumor-nerve adhesion, and injury frequency aligns with reports suggesting that surgeon experience and intraoperative monitoring techniques may be more critical determinants than demographic factors.²

The strong association between absence of EMG monitoring and increased injury frequency corroborates multiple meta-analyses highlighting intraoperative neurophysiological monitoring as a protective factor.²¹⁻²⁵ This reinforces the necessity of integrating EMG into standard surgical protocols for CPA tumor resection.

At baseline, the most prevalent symptom was difficulty with food and liquid intake due to facial muscle weakness, affecting over 95% of patients. Hypoacusis was the second most common symptom (>80% across groups), with minimal improvement postoperatively, indicating that auditory dysfunction may be less responsive to facial nerve-targeted rehabilitation and more related to cochlear nerve injury or surgical manipulation.⁴ Symptoms such as eye dryness, retroauricular discomfort, tearing, and gustatory disturbances showed progressive resolution over the 12-month follow-up, particularly in the multimodal therapy group.

The persistence of hypoacusis despite significant facial functional gains highlights the multifactorial nature of CPA tumor surgery sequelae and the need for targeted auditory rehabilitation strategies.⁶

Objective functional assessments using HB, Yanagihara, SBFGS, and FDI scales consistently demonstrated significant improvements in all groups. However, the magnitude of recovery was greatest in Group III, which received combined pharmacotherapy and TENS. In patients with mild facial asymmetry (HB grades 2-3), Group III achieved a 67.4% reduction in HB score over 12 months, compared with 49.6% and 53.0% in Groups I and II, respectively. Improvements were even more pronounced in severe asym-

metry cases (HB grades 4-5), with Group III achieving a 67.3% reduction versus <20% in Groups I and II.

The Yanagihara score in Group III increased by 58.96%, significantly outperforming the other groups. The SBFGS improvement in Group III (250.46%) was more than double that of the other two groups, reflecting enhanced facial symmetry and reduced synkinesis. The FDI physical function subscale gains were similarly largest in Group III (84.3%), indicating better patient-perceived functional capacity.

These results align with prior studies reporting that electrical stimulation enhances axonal regeneration, muscle excitability, and synaptic reorganization after facial nerve injury.⁵ Pharmacological interventions targeting neuroprotection and nerve metabolism, such as corticosteroids, B vitamins, and antioxidants, have independently shown moderate efficacy.² The combination likely produces synergistic effects by simultaneously promoting nerve repair and optimizing neuromuscular activation.

The superior outcomes in the combined therapy group may be explained by complementary mechanisms: TENS modulates neural excitability, promotes Schwann cell proliferation, and stimulates neurotrophic factor release, while pharmacotherapy mitigates inflammation, oxidative stress, and supports metabolic processes essential for axonal repair. Additionally, repetitive afferent input via stimulation may reduce cortical reorganization maladaptations that perpetuate asymmetry.^{4,18}

Across 12 months of follow-up, all three postoperative strategies were associated with improvement on clinician-rated scales and electromyographic parameters, consistent with progressive reinnervation and neuromuscular adaptation after reconstruction. Between-group comparisons favored the multimodal protocol that combined electrical stimulation with pharmacological support; however, these findings should be interpreted as evidence of an association between the bundled rehabilitation strategy and superior recovery rather than proof of superiority of any single component. In particular, because Group III incorporated multiple pharmacological agents concurrently with electrical stimulation, the present study cannot determine which elements were necessary or sufficient for the observed effect, nor can it exclude synergistic interactions as a contributing mechanism. In addition, although multivariable adjustment was applied to reduce confounding, the single-center setting and the inability to blind participants and therapists introduce potential performance and expectation effects, especially for patient-reported outcomes. Therefore, the results are best viewed as supportive translational evidence that structured neuromuscular stimulation, when delivered within a comprehensive postoperative rehabilitation framework, may enhance recovery trajectories after facial nerve reconstruction. Confirmation in multicenter trials with component-specific or factorial designs is warranted to quantify independent effects, define optimal dosing and timing, and establish generalizability.

Notably, the addition of TENS to standard therapy (Group I) also yielded superior results compared to pharmacotherapy alone (Group II) for certain outcomes, suggesting that

active neuromuscular stimulation may be more impactful than systemic pharmacological support when used in isolation.

Several limitations should be considered. The retrospective, nonrandomized design introduces potential residual confounding despite multivariable adjustment, and the bundled nature of the multimodal intervention precludes attribution of effects to individual components. The single-center setting may limit external validity, and blinding of patients and therapists was not feasible, particularly for patient-reported outcomes. Rehabilitation adherence was inferred from documented protocol delivery rather than objectively quantified, which may have contributed to outcome variability.

The decision to initiate multimodal rehabilitation, including transcutaneous electrical nerve stimulation, in the early postoperative period reflects a proactive intervention strategy grounded in peripheral nerve and neuromuscular physiology. Early activation of denervated or partially denervated facial muscles is hypothesized to mitigate rapid muscle atrophy, preserve neuromuscular junction integrity, and maintain muscle fiber excitability during the critical window preceding axonal regeneration. In parallel, early afferent input may modulate maladaptive central plasticity, limiting cortical reorganization patterns that are associated with synkinesis and persistent facial asymmetry. This approach contrasts with a traditional “wait-and-see” strategy that relies on spontaneous recovery but risks irreversible muscular and central changes during prolonged inactivity. In the present study, rehabilitation was initiated within a comparable early postoperative window across patients who underwent surgical facial nerve reconstruction, regardless of presumed injury severity, because all included cases met predefined intraoperative anatomical or neurophysiological criteria indicating a low likelihood of reliable spontaneous recovery. Importantly, stimulation parameters were deliberately conservative, and treatment was initiated only after surgical stabilization, minimizing the risk of adverse effects in cases with partial axonal continuity. Although the skull-base surgery literature offers limited consensus on the optimal timing of postoperative facial rehabilitation, available clinical and translational evidence increasingly supports early neuromuscular engagement following confirmed nerve injury or reconstruction. Thus, the timing adopted in this study represents a biologically driven and clinically pragmatic strategy aimed at optimizing the regenerative environment rather than accelerating recovery in isolation.

The study did not incorporate objective electrophysiological follow-up (e.g., electroneurography or blink reflex latency) postoperatively, which might have provided additional mechanistic insight. Additionally, long-term durability beyond 12 months remains unverified; late-onset synkinesis or recurrence of asymmetry is possible. Pharmacotherapy protocols were standardized but not individualized to biomarker profiles, which could be a future optimization strategy. A key limitation of the present study is that the multimodal intervention applied in Group III combined several pharmacological agents with trans-

cutaneous electrical nerve stimulation, which precludes determination of the independent contribution of each component to the observed functional recovery. The study was intentionally designed as a pragmatic evaluation of a comprehensive postoperative rehabilitation protocol rather than as a component-specific efficacy trial. Nevertheless, the concurrent use of anti-inflammatory, neurometabolic, microcirculatory, and neuromuscular activation strategies reflects the multifactorial biology of peripheral nerve regeneration, which involves overlapping inflammatory, metabolic, vascular, and electrophysiological mechanisms. From a translational perspective, it is biologically plausible that these interventions act in a complementary and potentially potentiating manner, whereby optimization of the neural microenvironment may enhance the responsiveness of regenerating axons and denervated muscles to electrical stimulation and rehabilitation. Similar synergistic principles have been proposed in experimental and clinical studies of peripheral nerve injury and neuromuscular recovery, supporting the rationale for combined rather than isolated interventions.

These results advocate for routine integration of multimodal rehabilitation, including electrical stimulation and pharmacotherapy, into postoperative management of CPA tumor patients with acute facial nerve injury. Implementation of intraoperative EMG monitoring should be universal, given its strong protective association.

Future research should investigate optimal stimulation parameters, timing of initiation, and the role of regenerative biologics (e.g., stem cell-derived neurotrophic factors, melanin, MSH) in further enhancing outcomes. Multicenter randomized controlled trials with long-term follow-up are essential to confirm the generalizability and durability of these findings. Generalizability of these findings may be influenced by institutional practice patterns. Centers without routine hypoglossal-facial nerve anastomosis or with limited access to structured neuromuscular rehabilitation resources may observe different recovery trajectories. Nevertheless, the observed associations support the broader principle that early, structured neuromuscular stimulation integrated into postoperative care may enhance facial nerve recovery, even when adapted to local surgical and rehabilitation capacities.

Conclusions

This study evaluated postoperative recovery following facial nerve reconstruction using a structured comparison of standard care, electrical stimulation-augmented therapy, and a multimodal rehabilitation protocol. Across a 12-month follow-up, all treatment strategies were associated with progressive improvements in clinical facial function scores and electromyographic parameters, consistent with ongoing reinnervation and neuromuscular adaptation after reconstruction. Patients managed with protocols incorporating transcutaneous electrical nerve stimulation demonstrated greater longitudinal changes in both functional and electrophysiological outcomes compared with standard therapy alone. The multimodal rehabilitation approach, which combined electrical stim-

ulation with targeted pharmacological support, was associated with the largest magnitude of change; however, these findings should be interpreted as reflecting the effect of a bundled postoperative strategy rather than the efficacy of individual components. Importantly, the present study focuses exclusively on facial nerve recovery and does not address auditory or vestibular outcomes. Overall, the results provide supportive translational evidence that structured neuromuscular stimulation, when integrated into comprehensive postoperative rehabilitation, may enhance recovery trajectories following facial nerve reconstruction. Further multicenter studies employing component-specific or factorial designs are needed to clarify independent treatment effects and to optimize postoperative rehabilitation protocols.

List of abbreviations

AMD, Armenian Dram
 BMI, Body Mass Index
 CPA, Cerebellopontine Angle
 CT, Computed Tomography
 EMG, Electromyography
 ES, Electrical Stimulation
 FDI, Facial Disability Index
 HB, House-Brackmann (scale)
 KOOS, Koos Classification (for CPA tumors)
 MRI, Magnetic Resonance Imaging
 MSH, Melanocyte-Stimulating Hormone
 MUPs, Motor Unit Potentials
 PNF, Proprioceptive Neuromuscular Facilitation
 SFGS, Sunnybrook Facial Grading System
 SPSS, Statistical Package for the Social Sciences
 TENS, Transcutaneous Electrical Nerve Stimulation
 TOF, Train-of-Four (neuromuscular blockade monitoring)
 CNS, Congress of Neurological Surgeons
 EANO, European Association of Neuro-Oncology

Authors' contributions

Sargis Yeghnyan conceived the study, contributed to study design, coordinated data collection, and drafted the initial manuscript. Tigran Petrosyan contributed to study design, methodological oversight, and critical revision of the manuscript for important intellectual content. Andriy Smolanka contributed to surgical data interpretation and critically revised the manuscript. Koryun Vahanyan performed electrophysiological assessments and contributed to data interpretation. Ruben Fanarjyan participated in data acquisition and clinical assessments. Mamikon Yeghnyan contributed to data collection, patient follow-up, and manuscript revision. All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Conflict of interest

The authors declare that they have no conflicts of interest related to this study.

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Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to institutional regulations but are available from the corresponding author on reasonable request.

Ethical approval

The institutional ethics committee of Yerevan State Medical University approved this retrospective chart-review study (approval No N 5/5 16.12.2021). Because the study used existing clinical data with no direct patient contact and minimal risk, the requirement for informed consent was waived in accordance with local regulations. All pharmacological interventions administered as part of postoperative management were prescribed within standard clinical practice and in accordance with institutional treatment guidelines.

Informed consent

The requirement for informed consent was waived for the study population due to the retrospective nature of the study.

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Online supplementary material:

Table 1. Postoperative management and rehabilitation protocols.

Table 2. Dynamics of clinical symptom resolution in patients with intraoperative facial nerve injury prior to treatment, and at 3- and 12-month follow-up.