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Neurophysiological Evaluation of the Facial Nerve in Parry-Romberg Syndrome: A Proposed Preoperative Protocol Before Nerve Transfer

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Abstract

Parry-Romberg syndrome (PRS) is a rare disorder of unknown etiology characterized by unilateral atrophy of facial subcutaneous and deep tissues. Although the trigeminal and facial nerves may be involved, evidence is limited, and neurophysiological abnormalities are not routinely incorporated into presurgical evaluation. This study aimed to describe clinical and neurophysiological findings in these nerves among adults with unilateral PRS previously treated with autologous fat grafting alone. A cross-sectional descriptive study included 10 adults with unilateral PRS who underwent clinical examination, facial nerve neurography, electromyography, and blink reflex testing. Disease severity was classified using the Guerrerosantos scale. Eight participants (80%) were women; mean age was 30.8 ± 14.4 years. Facial neurography showed axonal degeneration in 7 of the patients (70%), while all demonstrated a chronic neuropathic EMG pattern. Guerrerosantos types 1 and 2 were most frequent (30% each). Blink reflexes on the affected side were normal in eight patients (80%) and abnormal in two (20%). Trigeminal motor and sensory function were preserved in most patients. These findings confirm predominant chronic facial nerve axonal degeneration with largely preserved trigeminal function. Preoperative neurophysiological assessment of facial nerve viability should be considered when planning dynamic reconstruction, including nerve transfer.

Introduction

Parry-Romberg syndrome (PRS), or progressive hemifacial atrophy, is an acquired craniofacial disorder whose exact pathophysiology remains incompletely understood.[1] The disease usually presents unilaterally, often respecting the midline, and progression may continue for several years before reaching a quiescent phase.[2]

Trigeminal and facial nerve involvement has been hypothesized as part of the underlying pathophysiological mechanism of PRS. However, these hypotheses have rarely been systematically evaluated using clinical neurophysiological techniques [3], and their surgical implications have been almost entirely unexplored.

Facial electromyography (EMG) and nerve conduction studies have been shown to detect denervation and myopathic changes in facial muscles, supporting the trophoneurosis hypothesis as an underlying mechanism in at least a subset of patients.[3] However, neural involvement in PRS is markedly heterogeneous: whereas some cases demonstrate documented dysfunction of the ipsilateral facial nerve, other patients with clinically comparable presentations show neurophysiological testing and intraepidermal nerve fiber density within normal ranges, with no differences between the affected and unaffected sides, suggesting that not all cases are accompanied by demonstrable structural damage to the trigeminofacial system.[3, 4]

The neurological injury variability has direct implications for surgical planning [5]. In PRS the reconstruction requires procedures involving neurorrhaphy such as anastomosis of a donor nerve to branches of the facial nerve[6], however the decision to proceed is typically guided by clinical and imaging findings alone, without objective preoperative confirmation of the functional status of the facial nerve.[7] This practice contrasts with other areas of facial nerve surgery, where preoperative electroneurography is routinely used to estimate the degree of subclinical neural degeneration and to guide surgical prognosis.[8]

Given that PRS does not follow a uniform pattern of neural involvement [1], and that the success of nerve transfer or grafting depends critically on the functional integrity of both donor and recipient nerves. [5], we propose that a systematic preoperative neurophysiological evaluation of the facial and trigeminal nerves be performed before any definitive surgical decision involving neurorrhaphy. Accordingly, this study provides a comprehensive clinical and neurophysiological characterization of facial and trigeminal nerve involvement in adults with unilateral PRS, aiming to enable objective identification of patients with a viable neural substrate for successful nerve transfer, avoid interventions with a low likelihood of functional reinnervation in those with established neural degeneration, and provide a basis for candidate selection grounded in functional rather than purely clinical or imaging evidence.

Results

Ten patients met the inclusion criteria, of whom 8 were women (80%). The mean age of the participants was 30.8 ± 14.4 years. The mean age at diagnosis was 12 ± 6.5 years, with a mean disease duration of 18.8 ± 15.3 years. The right side was the most affected (70%). The socio-demographic characteristics are summarized in Table 1. Two patients presented clinical neurological findings, manifested as epilepsy, and all lacked systemic non-neurological manifestations. Details of clinical manifestations and neurological examination are shown in Table 2. Regarding previous surgical treatments, all ten patients had undergone autologous fat grafting as static volume reconstruction before the present evaluation; none had received dynamic reconstruction such as free functional muscle transfer.

Table 1
Characteristics of the participants.

Variables	***n* = 10**
Sex (Female/Male)	8/2
Age in years	30.8 ± 14.4
BMI (kg/m^2)	25.2 ± 3.9
Race	10 Hispanic
Comorbidities (Absent/Present)	7/3
Facial reconstructive surgery (Yes)	10
Years at PRS diagnosis	12±6.5
Disease duration (years)	18.8±15.3
Values are expressed as mean ± standard deviation (SD). Abbreviations: BMI, Body mass index; PRS, Parry Romberg Syndrome.	

Table 2
Clinical manifestations and physical exploration.

Patient	Affected side	Neurological manifestations associated	Strength of mandibular muscles MRC Scale	Strength facial muscles House-Brackmann scale	External receptive sensitivity 3 branches of the V (1 to 10)	Proprioceptive sensitivity (vibratory) 3 branches of the V Seconds	Proprioceptive sensitivity (thermoalgesia) 3 branches of the V
1	Left	Absent	5	1	7	7	Absent
2	Right	Present (Epilepsy)	5	1	7	8	Present
3	Left	Present (Epilepsy)	3	5	5	10	Present
4	Left	Absent	5	1	0	0	Absent
5	Right	Absent	4	4	9	9	Present
6	Right	Absent	4	4	7	8	Present
7	Right	Absent	5	3	9	10	Present
8	Right	Absent	4	3	7	7	Present
9	Right	Absent	1	1	8	8	Present
10	Right	Absent	1	1	8	8	Present

Abbreviations: MRC, Medical Research Council.

Clinical Neurophysiological findings:

Blink reflex testing on the affected side was normal in eight patients (80%) and abnormal in two (20%): patient 2 showed an absent R2 contralateral response, and patient 3 showed absent ipsilateral responses on stimulation of the affected side. Facial neurography demonstrated axonal involvement in seven patients (70%), with amplitude reductions on the affected side ranging from approximately 20% to 96% relative to the unaffected side; the remaining three patients (30%) had quantitatively normal neurography. Facial EMG demonstrated a chronic neuropathic pattern on the affected side in all ten patients (100%), with signs of denervation and reinnervation; aberrant reinnervation manifested as hemifacial synkinesis was observed in five patients (50%). Based on the Guerrerosantos scale, PRS severity was distributed as follows: Type 1 in three patients (30%), Type 2 in three (30%), Type 3 in two (20%), Type 4 in two (20%). Individual values are detailed in Table 3, with full quantitative neurography and blink reflex latencies. Representative findings from one of our patients are illustrated in Table 4, Fig. 1 **and** Fig. 2.

Table 3
Neurophysiological profile of the affected side of the patients.

Patient	Affected side	Disease duration (years)	Guerrero Santos scale	Neuronography VII Facial nerve axonal loss *	BR on affected side	Aberrant reinnervation (synkinesis)
1	L	13	2	20–44%	Normal	Yes
2	R	7	2	~ 50% Nasalis only	Abnormal	Yes
3	L	47	4	86–96%	Abnormal	No
4	L	9	1	Normal ENoG	Normal	No
5	R	11	3	63–94%	Normal	Yes
6	R	26	4	Discrete axonal involvement (nasalis only; no quantitative value reported)	Normal	Yes
7	R	22	3	Normal ENoG	Normal	No
8	R	10	2	30%	Normal	No
9	R	5	1	Normal ENoG	Normal	No
10	L	7	1	50%	Normal	Yes

Abbreviations: BR, blink reflex; ENoG, electroneuronography (facial nerve neurography); L, left; PRS, Parry-Romberg syndrome; R, right.

* Facial nerve axonal loss corresponds to the percentage reduction in compound muscle action potential amplitude on ipsilateral facial neurography relative to the unaffected side, as reported in the original neurophysiological study of each patient. Ranges reflect the per-muscle values documented in each report (nasalis, orbicularis oculi, and/or orbicularis oris). "Normal ENoG" indicates patients whose quantitative neurography did not demonstrate significant amplitude asymmetry; all such patients nonetheless showed a chronic neuropathic pattern on needle electromyography of the ipsilateral facial muscles.

Table 4
Summary of findings in EMG performed on Patient # 3, a 59-year-old female with left-sided symptoms.

Side	Muscle	INS	FIB	POS	FASC	AMP	DUR	POL	REIN	RECRT	PATT	ERN
Left	Nasalis	Nml	Nml	Nml	Nml	2500	< 10	Many	1+	Nml	-1	Max
Left	Frontalis	Dism	Nml	Nml	Nml			None	Nml	Nml	-1	Max
Left	Orbic Oculi	Dism	Nml	Nml	Nml	1800	< 10	None	Nml	Nml	-2	Max
Left	Orbic Oris	Nml	Nml	Nml	Nml	1500	< 10	None	Nml	Nml	-2	Max

Abbreviations: INS: Insertion activity, FIB: Fibrillations, POS: Positive sharp waves, FASC: Fasciculations. AMP: Amplitude, DUR: Duration, POL: Polyphasic, REIN; Re-innervation, RECRT: Recruitment, PATT: Pattern (recruitment pattern on maximal effort); ERN: Effort (Max = maximal muscle contraction).

Discussion

Our findings demonstrate a pattern of neuropathic involvement in PRS, with chronic axonal degeneration of the facial nerve identified in 70% of patients and largely preserved trigeminal function, highlighting the diagnostic value of electromyography and nerve conduction studies in these patients.

The diagnosis of PRS is well established based on the topography and morphology of facial atrophy, supported by a complete medical history and adequate physical examination, with particular attention to facial asymmetry involving the maxillary, malar, and orbital regions.[9] Cases are usually described as a young patient with right-sided facial atrophic asymmetry due to PRS with discrete ipsilateral deviation of the labial commissure, enophthalmos, and periorbital hyperpigmentation of the affected side.[10]

The definitive treatment for the aesthetic and functional deformities caused by PRS is surgical reconstruction. Procedures include autologous fat grafting and dermal-fat grafts, and, in severe cases, free functional muscle transfer (FFMT), typically using the gracilis or masseter muscle combined with cross-facial nerve grafting (CFNG) or masseteric nerve coaptation [11, 12]. The principle underlying these procedures is that a transferred muscle becomes reinnervated through a donor nerve, restoring facial symmetry and voluntary movement; the success of this reinnervation depends critically on the availability of an intact and functional facial nerve, or an adequate motor nerve substitute, at the recipient site[13]. However, despite the central role of the facial nerve in determining surgical outcomes, its functional status is rarely assessed systematically before surgery in PRS. If the facial nerve is severely degenerated, standard surgical strategies that rely on it as the motor source for FFMT will predictably fail [14].

Only three case reports and a previous adult series have described the use of clinical neurophysiological techniques in PRS without any other neurological manifestation [3, 15–17]. One report involved a 26-year-old woman with PRS on the right side, where BR testing showed prolonged R2 responses after stimulation on the left side but normal limits after stimulation on the right; left trigeminal somatosensory evoked potentials exhibited slightly longer latencies compared to the opposite side. However, EMG examination of the left orbicularis oculi muscle and neurography of both facial nerves were within normal limits [16]. This contrasts with our findings, in which chronic axonal degeneration of the facial nerve was present in approximately 70% of patients despite normal BR responses. Similarly, Verma et al. found that facial nerve stimulation at the anterior ear on both sides revealed only minor differences in latency and amplitude on motor facial neuroconduction studies, and that the R2–R1 interval of the BR showed no significant difference between sides (27.95 ms left vs. 24.20 ms right).[17]. In contrast, a case involving a 14-year-old male showed R1 and R2 latencies within normal reference values, but an absent R'2 component on the opposite side [15]; needle EMG in this patient revealed abnormalities consistent with peripheral neuropathy affecting the right facial and hypoglossal nerves, which supports and relates to the findings of our study.

Few clinical neurophysiological studies in isolated patients have suggested mechanisms of damage in both the trigeminal and facial nerves through BR abnormalities [3], generally favoring a central rather than peripheral localization. Lonchampt et al. proposed that the absence of R2 and R'2 responses upon right supraorbital stimulation may indicate chronic denervation of the facial nerve, which could also explain the reduced amplitude of the right R1 response compared to the left side; however, since a normal contralateral R'2 discharge would be expected in that scenario but was not recorded, the authors raised the possibility of a central disorder instead.[15] Consistent with this, Aramideh et al. proposed that a dysfunction localized to the right brainstem, between the facial and hypoglossal nerve nuclei, could explain the absence of late responses (R2 and R'2) due to a functional interruption of the long reflex arcs[18]. Our findings of chronic distal axonal degeneration with normal BR responses localize the lesion peripherally, offering an alternative interpretation to these earlier central hypotheses.

It has also been suggested that the trigeminal nerve may be involved in the pathological process of PRS given the distribution of atrophy along its innervation territories, occasionally presenting as trigeminal neuralgia; however, such cases remain rare.[4] In one case, a 50-year-old woman with facial pain underwent evaluation of the trigeminal pathway, including masticatory muscle EMG, trigeminal reflexes (blink reflex and masseter inhibitory reflex), and laser-evoked potentials for Aδ and C fibers; all neurophysiological data were within normal ranges, with no differences between the affected and unaffected sides.[4] Similarly, a 37-year-old patient with left hemifacial pain and PRS showed normal neuroconduction and facial EMG of the orbicularis oris, masseter, and frontalis muscles.[19] Two additional case reports of PRS with HFS, in

patients aged 57 and 34 years, showed facial nerve EMG findings consistent only with HFS, with normal masseter neuroconduction in both cases.[9, 20]

From a surgical standpoint, our findings may have practical implications for reconstructive planning in PRS. Dynamic reconstructive strategies, such as FFMT with CFNG or masseteric coaptation, depend critically on the axonal capacity of donor and recipient nerves, and the degree of post-transfer facial movement has been shown to correlate with donor motor nerve axonal density.[11–13]

The chronic distal axonal degeneration of the facial nerve documented in our cohort, together with normal BR responses localizing the lesion peripherally rather than centrally, suggests that this segment may behave as a suboptimal recipient or motor source in such procedures, an aspect that, to our knowledge, has not been systematically addressed in the PRS reconstructive literature. Consistent with this, the preserved trigeminal motor function observed in our cohort supports the potential use of the masseteric nerve, a trigeminal motor branch widely used as a donor in facial reanimation for its high axonal load and proximity to the facial nerve, which appears to remain anatomically and functionally available in PRS.[14] Preoperative electromyographic assessment of donor and target nerves has already been incorporated into selection protocols for facial reanimation in other clinical contexts; a similar approach could provide useful information for surgical planning in PRS, particularly when the choice of motor source for dynamic reconstruction is being weighed. We emphasize that this rationale is hypothesis-generating: all ten patients in our cohort had received only static volume reconstruction, and none had undergone dynamic reconstruction at the time of evaluation.

The main limitations of this study are its cross-sectional descriptive design, the modest sample size, the absence of histopathological data and the lack of long-term postoperative outcome data; the surgical rationale proposed should therefore be interpreted as hypothesis-generating rather than as evidence that preoperative neurophysiological assessment improves reconstructive results. Within these limitations, the consistent finding of distal facial nerve axonal degeneration with preserved trigeminal function across our cohort suggests that neurophysiological evaluation may be a useful addition to the clinical workup of PRS, particularly in patients who are candidates for dynamic reconstructive procedures.

For this reason, we propose that neurophysiological findings should be correlated with surgical outcomes as part of the preoperative evaluation. Future studies applying this approach, together with novel electrophysiological techniques and advanced imaging, will be crucial to further elucidate the complex interplay between neural dysfunction and progressive facial atrophy.

Materials and Methods

We conducted a cross-sectional and descriptive study at the Department of Clinical Neurophysiology from the National Institute of Neurology and Neurosurgery in Mexico City, Mexico. The study was approved by the Research Committee of the National Institute of Neurology and Neurosurgery Manuel Velasco Suárez (Protocol No. 100/23, with an extension approved on March 20, 2024). All methods were performed in accordance with relevant guidelines and regulations (Declaration of Helsinki). We included adult patients older than 18 years, of any sex, who met the diagnostic criteria for PRS according to Guerrerosantos classification.[21] Written informed consent was obtained from all participants prior to undergoing clinical examination and neurophysiological testing.

We collected the socio-demographic and clinical characteristics such as age, sex, weight, height, age at diagnosis, time of diagnosis, race, comorbidities, and previous surgical treatments. All the patients underwent clinical examinations, brain 1.5 or 3 Tesla MRI (including T1-weighted, T2-weighted, FLAIR, and contrast-enhanced T1-weighted imaging, supplemented by high-resolution 3D sequences for detailed assessment of cortical and subcortical atrophy), and neurography and electromyographic facial tests, with blink reflex explorations as follows:

Trigeminal and Facial Neurological Physical Examination

Trigeminal nerve Neurological examination

The fifth cranial nerve was evaluated in both its sensory and motor portions. Using a swab, each of the branches (V1, V2, and V3) on the affected side was stimulated by touch and compared with the healthy side. The patient was then asked to score the similarity of the stimulus on a scale of 1 to 10; this same exercise was performed during the thermoalgesic tests. Proprioception was assessed by comparing each of the branches on the affected side against the healthy side using a 128 Hz tuning fork, counting the seconds the patient felt the vibration on each side. A cotton swab was also used to evaluate the corneal reflex.

Facial and trigeminal motor nerve neurological examination

For the physical examination of the trigeminal motor nerve, we employed a comparative technique of masseter muscle strength against resistance of the affected side to the unaffected side, with a maximum score of 5 on the Medical Research Council (MRC) scale.[22] Additionally, we assessed the unilateral facial musculature using the House-Brackmann Facial Paralysis Scale (Grade I: Normal function in all regions, Grade II: Mild dysfunction, Grade III: Moderate dysfunction, Grade IV: Moderately severe dysfunction, Grade V: Severe dysfunction, and Grade VI: Total paralysis)[23]

Clinical Neurophysiological Testing

All patients underwent unilateral neuronography and electromyography (EMG) facial tests, and bilateral blink reflex (BR) following international standard procedures.[24]

Facial nerve neuroconduction

Patients were positioned in a relaxed, supine posture on the examination table. Manual stimulation of the facial nerve was applied below the ear, anterior to the mastoid, or directly anterior to the tragus. The cathode was placed anterior and superior to the anode, although in some cases, the anode had to be rotated to minimize stimulation artifacts or avoid direct stimulation of the masseter muscle. For recording, the active electrode was placed over the nasalis muscle, with the reference electrode positioned over the same nasal area on the contralateral side. A similar setup was used for the orbicularis oculi, with the active electrode placed on the skin just inferior to the pupillary space in the mid-position and the reference electrode on the skin over the lateral canthus. The setup for the orbicularis oris facial muscles was similar to that of the nasalis muscle. This configuration ensured precise monitoring of facial nerve conduction without interference from adjacent structures. The stimulation parameters, such as intensity and pulse duration, were carefully adjusted to optimize nerve activation while avoiding unnecessary discomfort for the patient.

Facial nerve EMG: Facial nerve EMG was performed on the ipsilateral facial muscles, including the frontalis, orbicularis oculi, nasalis, and orbicularis oris, following previously described protocols.[3] Motor unit action potentials (MUAPs) were recorded using small concentric needle electrodes with a sensitivity of 100–500 μV/Div and a time base of 10 ms/Div (filters 30-1500 KHz). We specifically analyzed MUAP morphology at rest, as well as during partial and maximal effort, to identify signs of chronic denervation and reinnervation, such as increased duration, polyphasia, or reduced recruitment. Additionally, synkinesis was assessed by monitoring for involuntary MUAP firing in the mentalis muscle during voluntary eye closure.

Blink Reflex (BR): To complement the distal nerve evaluation, BR was utilized to assess the integrity of the reflex arc between the trigeminal and facial nerves, including their proximal segments. Following the criteria established by Aramideh et al.[18]. and our prior methodology.[3]. We analyzed the R1 and R2 components. A facial nerve lesion was identified by abnormalities in the ipsilateral R1 and R2 responses upon stimulation of the affected side, while ensuring the contralateral

R2 remained normal to confirm the afferent pathway's integrity. This combined approach of direct stimulation and reflex testing allowed for a comprehensive mapping of cranial nerve dysfunction

Severity of PRS: Disease severity was classified using the scale proposed by Guerrerosantos et al., which categorizes atrophy in PRS into four types: 1) shows mild thinning of facial soft tissues, noticeable only to the patient and close acquaintances; 2) involves more evident soft tissue thinning without bone or cartilage involvement, noticeable to others; 3) both soft and bony/cartilaginous tissues are thinner, creating a pronounced deformity; 4) the most severe, features extreme atrophy, with skin appearing against bones, along with significant cartilage and bone thinning, causing both aesthetic and functional issues, particularly with the lips and nose.[21]

Statistical analysis

The data were analyzed descriptively using IBM SPSS Statistics version 30.0 (IBM Corp., Armonk, NY). We calculated measures of central tendency (means, frequencies, and percentages) and dispersion (ranges, standard deviations).

Declarations

Competing interests

The authors declare no competing interests.

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Author Contribution

F.C.F., G.Q.P., J.B.C., and D.A.M.P. designed the study and performed the neurophysiological evaluations. C.G.G., A.N.L.O., K.A.F.I., and A.C.M. contributed to patient recruitment, clinical diagnosis, and plastic surgery assessment. A.V.H., J.U.S.P., M.B.C.S., E.Z.C.C., and D.S.J. performed data analysis, English translation, and drafted the manuscript. D.S.J. and A.C.M. served as principal supervisors and critically revised the manuscript. All authors reviewed, edited, and approved the final version of the manuscript.

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None

Data Availability

The datasets generated and/or analysed during the current study are not publicly available due to institutional ethics committee restrictions and patient privacy regulations, but are available from the corresponding author on reasonable request.

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Figures

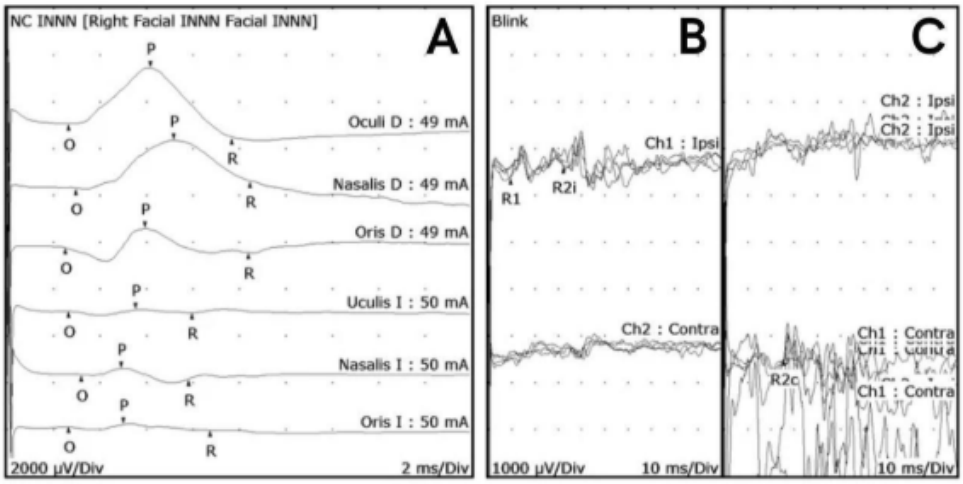


Figure 1

Illustration of facial neurophysiological studies performed on a 23-year-old female with an affected left side. A) Facial nerve neurography shows a decrease in amplitudes of 77, 94 and 63% respectively for the orbicularis oculi, nasalis, and orbicularis oris muscles. B-C) Left and right blink reflex with normal values. This neurophysiological finding indicates severe neuropathic involvement characterized by axonal degeneration of 63 to 94% of the functional fibers for the right facial nerve and trigeminal facial reflex preserved on both sides. L; Left R: Right. Ipsi: Ipsilateral; Contra: Contralateral

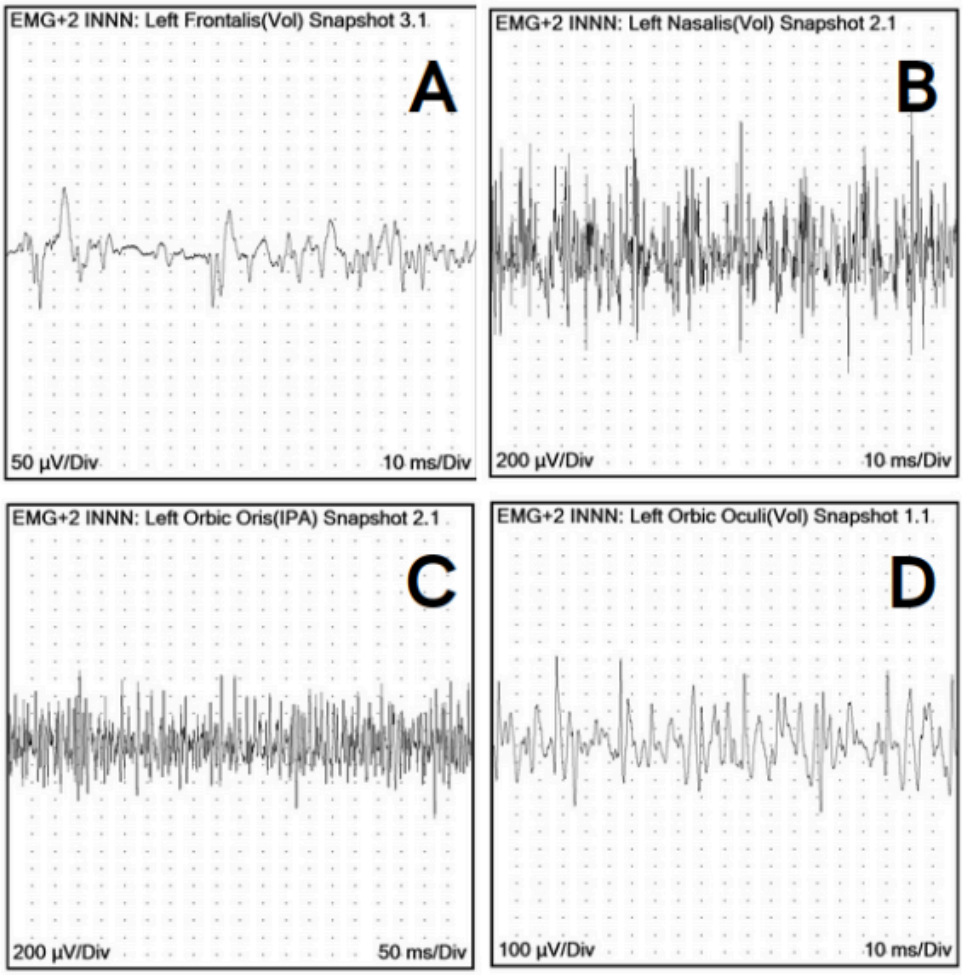


Figure 2

Illustration of EMG neuropathic findings performed on the same patient as Table 4 (Patient #3, 59-year-old female), with the left side affected.

A) Left frontalis muscle in resting state, B) Left nasalis muscle in partial contraction, and C) Left orbicularis oris muscle at full contraction, D) Left orbicular oculi muscle in partial contraction.