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Contralateral Reinnervation in Patients With Facial Nerve Palsy

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Objective: To demonstrate a phenomenon of contralateral reinnervation in facial nerve palsy patients.
Methods: Retrospective case review of 9 patients whose facial muscles were electrophysiologically proven to be reinnervated contralaterally.
Results: The duration from symptom onset spanned from 3 to 114 months. All subjects had moderate-to-severe facial palsy initially. Contralateral reinnervation was observed in both traumatic and idiopathic causes, also in both complete and incomplete palsies. Cross-innervation is more frequently reported in muscles near the midline; however, this is the first report demonstrating evidence of cross-innervation in muscles far from the midline—the frontalis, the orbicularis oculi, and the zygomaticus.
Conclusion: Although contralateral reinnervation after facial nerve palsy is a common observation, it has not gained appropriate attention. Without recognition of this phenomenon, misinterpretation of the electromyography may mislead the proper timing of nerve repair or reanimation procedure. Therefore, routine examination of motor action potential by contralateral stimulation during electromyography, especially in patients with moderate-to-severe palsy, would provide accurate assessment of the injured nerves and would help in appropriate decision making for further treatment. **Key Words:** Electromyography—Facial nerve—Nerve regeneration.

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Facial nerve palsy is one of the most frequent nerve injuries, which can cause patients to withdraw socially. The interval between the injury and nerve repair or reanimation surgery is a critical factor for favorable outcome (1–3). Electromyography (EMG) gives objective and quantitative information regarding the severity of the injury and recovery status of the nerve, thereby guiding whether or when surgery should be performed (4).

In the usual electrodiagnostic techniques, compound muscle action potential (CMAP) responses are recorded after ipsilateral facial nerve stimulation (5). However, there have been several reports that have demonstrated the reinnervation of facial muscles by the contralateral nerve (6–9). This can result in discrepancy between the voluntary recruitment of motor units and the ipsilateral CMAP response, consequently making the interpretation difficult. Most clinicians fail to recognize this possibility of contralateral reinnervation after facial nerve palsy, and misinterpretations may delay proper timing of surgical intervention.

In this study, we describe a series of patients with facial palsies from various etiologies, in which the facial muscles were electrophysiologically proven to be reinnervated by the contralateral facial nerve, and review the relevant literature.

MATERIALS AND METHODS

Subject Selection

Medical records of all patients who visited an EMG unit for evaluation of facial palsy from March 2010 to March 2013 were initially screened (n = 39). Nine patients whose contralateral reinnervation in facial muscles was electrophysiologically proven were included in this study. Three patients had Bell's palsy; 3 had a history of previous surgeries for a vestibular schwannoma (n = 2) or cholesteatoma (n = 1) in the cerebellopontine angle; 2 had undergone a parotidectomy for parotid gland cancer; 1 had multiple traumatic injuries from a motor vehicle accident, which resulted in left cerebellar, temporal, and periventricular hemorrhage.

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The institutional review board of the Seoul National University Hospital approved the use of patient data.

Facial Nerve Evaluation

Before the electrodiagnostic evaluation, a detailed history was taken, and House-Brackmann (HB) score was determined by asking to relax and activate their facial muscles.

All electrodiagnostic examinations were performed by one physician with a commercially available Synergy system (Oxford Medelec, Wiesbaden, Germany). Subjects were positioned in a comfortable supine position during the measurements. The electrodiagnostic studies of both facial nerves were performed according to the standard techniques (5). In particular, facial CMAP responses were obtained from both sides with ipsilateral preauricular stimulation. A round self-adhesive surface recording electrode was placed symmetrically on either side of the facial muscles, including the frontalis, the orbicularis oculi, the nasalis, the orbicularis oris, and/or the zygomaticus. At initial evaluation, blink reflex studies were also conducted on both sides with supraorbital nerve stimulation. A needle EMG was carried out for each facial muscle with monopolar needle electrodes (CareFusion, Steedman, MO, USA); abnormal spontaneous activities were inspected at rest, and interference patterns were assessed at volition.

After routine electrodiagnostic studies, additional conduction studies were performed in order to evaluate the contralateral innervation of the facial muscles. A needle electrode was inserted into the facial muscles as a recording electrode, and then, electrical stimulation was applied at both ipsilateral and contralateral tragus, one after the other. Starting at 50 mA and 0.1 ms, respectively, stimulation intensity and duration were gradually increased until motor units were observed, and there was no additional recruitment of motor unit action potentials (MUAPs). The maximal stimulation intensity and duration were set at 100 mA and 0.5 ms, respectively.

Statistical Analysis

Linear regression was performed for the correlation between the latency and duration from symptom onset. The significance level was determined at 5%. Analyses were performed using the SPSS software (SPSS Inc, Chicago, IL, USA).

RESULTS

Median age was 35 years (range, 12–76 yr) and male-to-female ratio was 7:2. Regarding the etiology, 1 patient had multiple traumas, 5 had postoperative facial palsy, and 3 had idiopathic palsy. The lesion locations were intracranial in 4 patients and extracranial in 5. In cases with parotidectomy, all operation records documented that the facial nerve was completely sacrificed. Regarding the severity, 5 had HB Grade V, 3 had HB Grade IV, and 1 had HB Grade III. Duration of facial palsy at the time of evaluation varied from 3 to 114 months (median, 13 mo). Two (Patients 3 and 9) patients underwent additional follow-up electromyography (Table 1).

Contralateral reinnervation was confirmed by CMAP response elicited by stimulation of the contralateral facial nerve. Based on the configuration of CMAP recorded by a needle electrode, we verified that the response was not from volume conduction (Fig. 1). In 4 patients (44.4%), the facial muscles were innervated exclusively by the contralateral facial nerve, whereas part of the facial muscles were bilaterally innervated in the rest (Table 2). Muscles near the midline, such as the nasalis and the orbicularis oris, were more frequently innervated by the contralateral nerve. The nasalis muscle had contralateral reinnervation in all 9 patients, the orbicularis oris muscle in 8 patients, the frontalis in 6 patients, and the orbicularis oculi or the zygomaticus only in 2 patients. Of note, the contralateral reinnervation in the frontalis, orbicularis oculi, and zygomaticus has not been reported in previous studies.

The onset latencies of MUAPs ranged from 3.20 to 45.73 ms, and their amplitudes ranged from 0.1 to 2.4 mV with stimulation of the contralateral undamaged nerve. With stimulation of the ipsilateral damaged nerve, onset latencies ranged from 3.20 to 43.65 ms with MUAP amplitudes of 0.4 to 5.7 mV. Univariate linear regression analysis revealed significant tendency of the onset latency in the nasalis to decrease when the duration from symptom onset increased ($p = 0.045$, $R^2 = 0.412$) (Fig. 2). The interference patterns of the reinnervated muscles varied from discrete to complete. The amount of recruited motor units differed across the muscles in each patient; there was a tendency for the muscles more proximal to the midline, including the nasalis and the orbicularis oris, to have better interference patterns than the muscles more laterally located, including the frontalis and the orbicularis oculi (Table 2).

DISCUSSION

In this study, we electrophysiologically confirmed contralateral reinnervation of the facial muscles in 9 of 39 patients who underwent facial EMG. Etiology, symptom duration intervals, and completeness of injury varied widely among the participants. Contralateral reinnervation was more frequently observed in the midline muscles (orbicularis oris and nasalis) than in the lateral muscles (orbicularis oculi, frontalis, and zygomaticus), which is in accordance with previous reports (6–9). Also, there was a

TABLE 1. *Clinical characteristics of 9 patients*

Patient	Age (yr)	Sex	Etiology	Symptom duration (mo)[a]
1	12	F	Multiple trauma	114
2	76	M	Bell's palsy	13
3	31	M	Postoperative, vestibular schwannoma	34
4	35	M	Postoperative, cholesteatoma	5
5	59	F	Postoperative, parotid cancer	5
6	35	M	Postoperative, parotid cancer	12
7	29	M	Bell's palsy	36
8	69	M	Postoperative, vestibular schwannoma	31
9	52	M	Bell's palsy	3

[a]At the time of the initial evaluation.

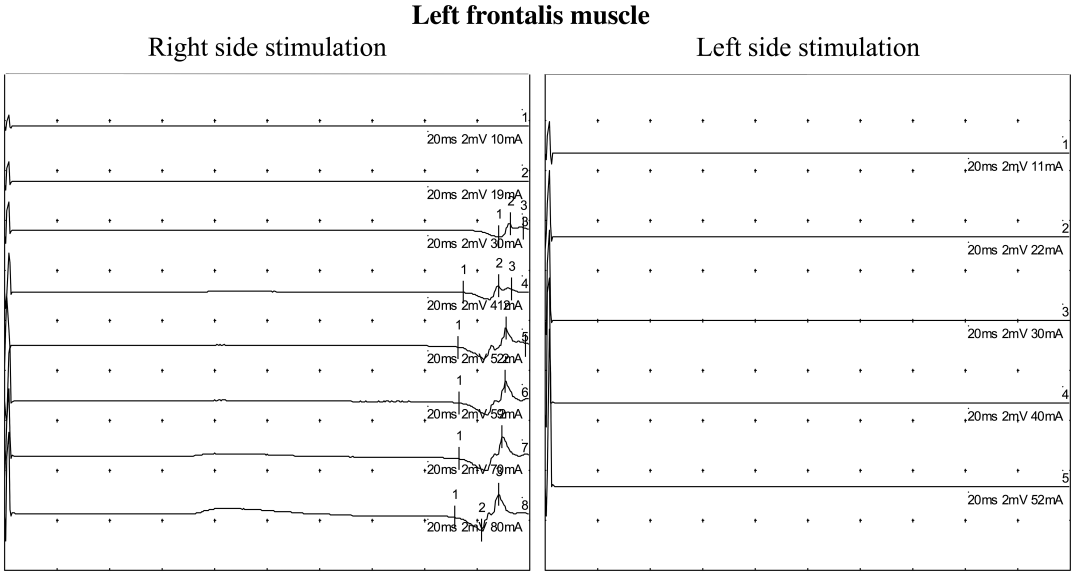

FIG. 1. Typical CMAP responses, which are recorded with a needle electrode placed in the paralyzed left frontalis (Patient 1). The needle electrode was located halfway between the hairline and eyebrow on a line passing vertically through the patient's pupil.

tendency for more motor units being recruited in the midline muscles than in the lateral muscles. Onset latencies with contralateral stimulation varied across patients and showed significant negative correlation with symptom duration. Functional outcome of the patients was poor (HB score ≥ IV in 8 of the 9 patients), despite remarkable recovery of the interference patterns, up to ''reduced'' or ''complete'' interference patterns, in the contralaterally reinnervated muscles.

The uptake of CMAP with contralateral stimulation in facial palsy is primarily explained by ''axonal regrowth'' from the contralateral facial nerves (7,8). It is supported by a distinct anatomy of the facial muscles which are not separated by a fascia, not like other skeletal muscles. The facial muscles share the subcutaneous musculoaponeurotic system (10), in place of the fascia, which enables the axon to sprout across the muscle. However, some researchers suggested ''myofiber conduction'' or ''motonucleus hyperexcitability'' to explain the phenomenon (9,11,12). In this study, we could observe contralateral reinnervation in far lateral muscles such as the zygomaticus and orbicularis oculi, which cannot be explained by ''myofiber conduction.'' In addition, we recorded variable onset latencies in this study. If the CMAP responses were from overactive motonucleus in the brainstem, the onset latency should be invariably delayed because it should involve brainstem reflex. Above all, decreasing tendency of onset latency with time strongly evidenced ''axonal regrowth'' (6,9). In particular, in a patient with serial follow-up (Patient 3), a latency of the nasalis changed from 10.15 to 4.27 ms and that of the orbicularis oris from 5.70 to 3.96 ms.

TABLE 2. *Needle electromyography and nerve conduction study results*

Patient	Study no.	HB	Nasalis			Orbicularis oris			Frontalis			Orbicularis oculi			Zygomaticus		
			IP[a]	Contr[b]	Ipsil[c]	IP[a]	Contr[b]	Ipsil[c]	IP[a]	Contr[b]	Ipsil[c]	IP[a]	Contr[b]	Ipsil[c]	IP[a]	Contr[b]	Ipsil[c]
1	#1	IV	R/C	0.3, 4.85	No	R/C	0.3, 3.7	No	D	0.9, 17.15	No	No	No	No	D	0.2, 17.25	No
2	#1	IV	R	0.6, 15.1	No	D	1.3, 4.5	No	D	0.2, 36.35	0.6, 6.7	D	NA	NA	NA	NA	NA
3	#1	V	R/C	0.9, 10.15	No	S	0.5, 5.7	No	R	0.2, 3.2	No	No	NA	NA	NA	NA	NA
	#2	V	R/C	0.4, 10.75	No	R	NA	NA	NA	NA	NA	D	NA	NA	NA	NA	NA
	#3	V	R/C	0.3, 4.27	No	R/C	0.7, 3.96	No	NA	NA	NA	R	0.2, 4.53	No	NA	NA	NA
4	#1	III	R	0.4, 15.6	No	C	0.6, 18.95	0.4, 33.8	R	No	0.9, 43.65	R	NA	NA	NA	NA	NA
5	#1	V	D	UC	No	No	NA	NA	No	NA	NA	NA	NA	NA	No	NA	NA
6	#1	V	R/C	0.6, 10	No	D	0.5, 9.4	No	R	0.6, 40.4	No	S	0.1, 34.35	No	D	No	No
7	#1	IV	C	0.4, 3.55	2.7, 4.45	R/C	2.4, 29.55	5.7, 3.2	R	1.9, 36.5	2.7, 5.9	R	0.4, 37.75	0.6, 3.75	NA	NA	NA
8	#1	V	C	0.6, 11.7	1.2, 5.75	C	NA	NA	R	No	0.5, 10.8	C	NA	NA	NA	NA	NA
9	#1	V	R	0.5, 10.15	0.8, 5	D	No	0.5, 9.9	D	NA	NA	R/C	NA	NA	NA	NA	NA
	#2	III	R/C	No	1.4, 4.04	C	0.6, 45.73	0.4, 4.38	C	No	7.4, 4.9	C	No	0.5, 3.33	NA	NA	NA

[a]Interference pattern by electromyography during maximal volition, represented by the following: C, complete; R/C, reduced-to-complete; R, reduced; D, discrete; S, single; No, no activity.

[b]Compound motor action potential by contralateral stimulation, represented by amplitude in mV and onset latency in ms.

[c]Compound motor action potential by ipsilateral stimulation, represented by amplitude in mV and onset latency in ms.

HB indicates House-Brackmann score; NA, not acquired.

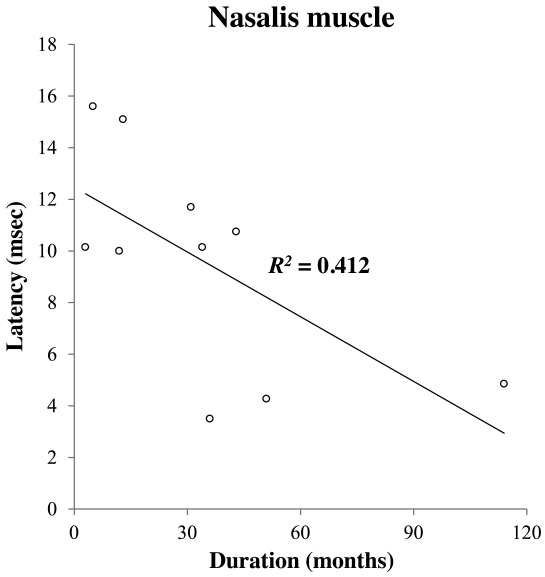

FIG. 2. A scatter diagram of the onset latency versus duration from symptom onset in the nasalis muscle. There is a significant tendency of the onset latency in the nasalis to decrease when the duration from symptom onset increased ($p = 0.045$, $R^2 = 0.412$).

Contralateral reinnervation after facial palsy is of clinical significance. First, it might be a more common phenomenon than generally expected (6–9,13). We observed it in 9 of 39 patients who underwent EMG for facial palsy. In particular, among 20 patients with severe palsies (HB score, >III), 8 patients (40%) exhibited contralateral reinnervation. If the clinician fails to recognize it, he or she might conclude that the injured nerves are actively regenerating based on the increased recruitment of motor units even in cases in which they are not. Second, and more importantly, functional outcome did not correlate with the improved interference patterns of the contralaterally reinnervated muscles. The discrepancy can be explained because contralateral reinnervation mainly occurs in the midline muscles rather than in the lateral muscles by which facial expression is largely produced. Consequently, if a clinician fails to recognize contralateral reinnervation during the EMG, one might misinterpret the result and, in turn, miss proper timing of nerve repair or reanimation. As a result, this might lead to unfavorable outcome, given that functional outcome solely from contralateral would not be favorable. Thus, we suggest that clinicians should be aware of the possibility of contralateral reinnervation after facial palsy, and electromyographers should perform bilateral facial nerve stimulation to examine it, especially in patients with moderate-to-severe facial palsy.

In conclusion, we present a series of patients with contralateral reinnervation after facial palsy in various clinical conditions. It was observed more frequently and actively in the midline muscles. Although the recovery of motor unit recruitment was variable, it did not correlate with functional status. Without index of suspicion, one might misunderstand the status of injured facial nerve as being regenerated and misjudge the treatment plan, consequently leading to poor prognosis. Therefore, we suggest that motor action potential by contralateral stimulation should be routinely surveyed during electromyography, especially in patients with severe palsy.

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