측두공 내 종양을 암시하는 안면마비 표정의 특성

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Characteristic facial expressions in chronic facial paralysis suggest an incidental finding of an intratemporal mass

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Background : The objective of this study was to analyze the characteristics of facial expressions by their pathogenesis in subjects with chronic facial paralysis and to describe the partial defect of facial function in elucidating the etiology of facial paralysis.

Materials and Methods : The etiology of chronic facial paralysis could be categorized into 3 groups: Group I, total resection without any reanimation surgery; Group II, acute facial nerve injury such as Bell's palsy, and Group III, facial nerve resection and anastomosis. There was a total number of 104 subjects included in this study. Each subject with chronic facial paralysis and were investigated based on the severity of synkinesis, regression of muscular volume, latency and amplitude in electromyography, and facial movement by House-Brackmann grade system.

Results : Subjects from the Group I had no synkinesis with profound regression of muscular volume, H-B grade 5.7 ± 0.34. Subjects from the Group II had moderate to severe synkinesis with moderate to severe regression of muscular volume, H-B grade 3.4 ± 0.96. In this group, thirteen subjects with atypical facial expression also had an incidental finding of a hidden mass in the intratemporal area. Lastly, subjects from the Group III had severe synkinesis without regression of muscular volume, H-B grade 3.7 ± 0.76.

Conclusions : With careful examination of facial expression, a crucial point for elucidating the pathogenesis of facial paralysis may be provided. Severe facial palsy without muscular regression may indicate an incidental finding of a hidden mass in the intratemporal area.

Key Words Facial paralysis, Facial expression, Synkinesis

INTRODUCTION

When Facial paralysis occurs after acute facial paralysis and facial nerve injury, the regenerative phase often produces an involuntary static and dynamic alteration of facial expression, regardless of its etiology. This is due to failure of regeneration occurring in neurotmesis or grade III by Seddon and Sunderland classification of nerve injury.[1,2]

The most common sequelae of facial paralysis is synkinesis. This is defined as the presence of unintentional motion in one area of the face produced during intentional movement in another area of the face.[3,4] This is due to the aberrant regeneration of fibers in the neural repair process. This becomes particularly visible when spontaneous facial movement occur, especially during emotional expressions such as smiling.[5,6]

The second most common sequelae of facial paralysis is facial asymmetry. Even after modern successful procedures such as botulinum toxin injection or microsurgical muscle transplantation have been performed facial asymmetry may continue to persist. The non-paralyzed side, which acts chronically against the weak antagonism of the contralateral muscles, usually presents as facial muscular hypertrophy, wrinkles, furrows and deviation of the mouth. Changes of facial expression associated with facial sequela can also be attributed to 'unbalanced' muscular activity, muscular hypertrophy and pattern of facial expression. The worsening appearance of facial expression can progress through time. For some subjects, a progressive facial asymmetry and a more deteriorated synkinesis can lead to low self-esteem and poor quality of life with age.[7,8]

Recently, Babak and Kimberly (BK) classified chronic facial paralysis into 5 subtypes according to degree of synkinesis. This is easily applicable in clinical practice.[9] Other classifications for the discrimination of chronic facial paralysis such as Sunnybrook grading systems,[10] and revised House-Brackmann (H-B) grading systems,[11] have been introduced and currently applied in clinical setting. Although the aforementioned classifications for facial paralysis have been utilized, we now know that the cause of nerve damage, the site of facial nerve injury, and the initial grade of facial paralysis have all been attributed to the peculiar patterns of facial asymmetry exhibited following complete recovery from facial nerve injury.

The main location of injury along the course of the facial nerve is largely dependent on the underlying disease. However, relationships between the topognostic features of facial expressions as well as the cause or location of injury remain unknown. Also, recovery of some part of the facial muscle may either be incomplete or non-existent.[12] The unusual behavior of several facial muscles has not been thoroughly explained.

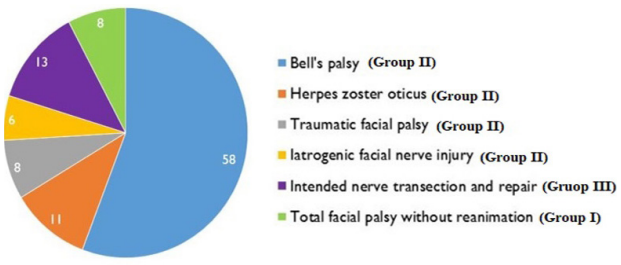
The purpose of this study was to analyze the characteristics of facial expressions by their pathogenesis in subjects with chronic facial paralysis and to describe the partial defect of facial function in elucidating the etiology of facial paralysis.

METHODS

1. Patients

One hundred and four subjects affected by facial synkinesis and facial asymmetry consequent to chronic facial paralysis seen from June 2013 to July 2015 at the Department of Otorhinolaryngology, Ilsan Paik Hospital were included in this study. Approval for this retrospective review study was obtained from the appropriate the

Fig. 1



Initial diagnosis of chronic facial paralysis (≥ 2 years).

Institutional Review Board of Ilsan Paik Hospital (IRB No. 5-20160076). All the subjects had facial sequela longer than 2 years duration from acute facial paralysis or facial nerve injury. The subjects were classified into 3 groups according to the etiology of facial paralysis.

Group I (n = 8): Total facial nerve palsy without any facial reanimation surgery. Group II (n = 83): Incomplete recovery after Bell's palsy, herpes zoster oticus, traumatic facial palsy or iatrogenic facial nerve injury with routine management and treatment. Group III (n = 13): Facial nerve resection and anastomosis (end to end anastomosis or inter-positional graft after surgery of parotid cancer, facial nerve schwannoma, or vestibular schwannoma) (Fig. 1).

Table 1. Classification of facial paralysis by House-Brackmann facial nerve grading system

Grade	Definition
I	• Normal symmetrical function in all areas
II	• Slight weakness noticeable only on close inspection • Complete eye closure with minimal effort • Slight asymmetry of smile with maximal effort • Synkinesis barely noticeable, contracture, or spasm absent
III	• Obvious weakness, but not disfiguring • May not be able to lift eyebrow • Complete eye closure and strong but asymmetrical mouth movement • With maximal effort • Obvious, but not disfiguring synkinesis, mass movement or spasm
IV	• Obvious disfiguring weakness • Inability to lift brow • Incomplete eye closure and asymmetry of mouth with maximal effort • Severe synkinesis, mass movement, spasm
V	• Motion barely perceptible • Incomplete eye closure, slight movement corner mouth • Synkinesis, contracture, and spasm usually absent
VI	• No movement, loss of tone, no synkinesis, contracture, or spasm

Table 2. Classification of facial paralysis by Babak and Kimberly classification

Type	Definition
A	Normal facial function
B	Partial paralysis with mild synkinesis
C	Partial paralysis with to severe synkinesis
D	Partial paralysis without synkinesis
E	Complete facial paralysis

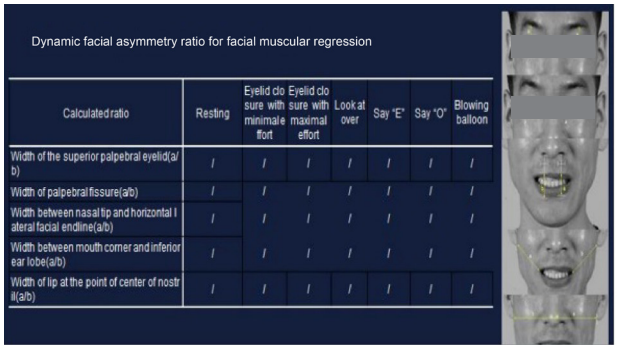
2. Measurement

To accurately measure facial synkinesis and asymmetry, both objective and subjective evaluations were utilized in this study.

Subjective evaluation of facial function was measured by H-B grading system since it is the most used system among otolaryngology clinicians. BK classification which classified chronic facial paralysis into 5 subtypes by degree of synkinesis was also utilized (Table 1, 2).

Objective evaluation for facial asymmetry such as "facial dynamic asymmetry ratio" which is an evaluation tool developed in our institution is utilized in this study.[13] Degree of facial asymmetry induced by regression of facial muscular volume on affected side after facial paralysis can be evaluated by the "facial dynamic asymmetry ratio". While facing forward, the subjects were tasked to express 3 specific eye movements (eyelid closure with minimal effort, eyelid closure with maximal effort and looking up with forceful wrinkling of the forehead), 3 mouth gestures (pronouncing "e", pronouncing "o", and acting out balloon blowing), and neutral facial expression at rest may be evaluated. The degree of facial asymmetry can be evaluated by measuring and comparing the superior

Fig. 2

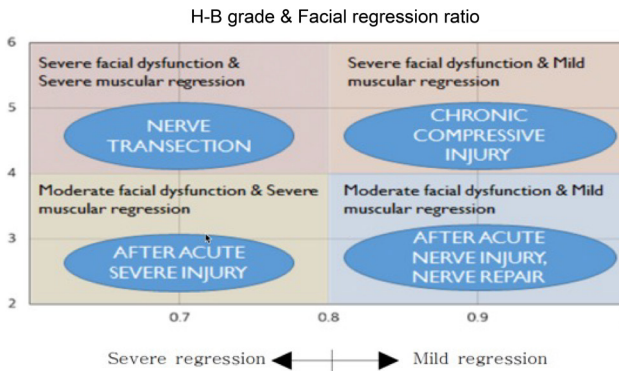


Measurement of dynamic facial asymmetry ratio.

palpebral eyelid width, the distance between nasal tip and horizontal lateral facial end line, the distance between the lip commissure and inferior ear lobe and the distance between the lip (Cupid's bow) and the point of center of nostril (Fig. 2). Mean ratio is sum of calculated ratio/35 (0 〈 calculated ratio 〈 1 [complete symmetry]).

The overall facial status of chronic facial paralysis could be expressed by the weakness of facial function (H–B grade) and facial muscular regression (Fig. 3).

Fig. 3

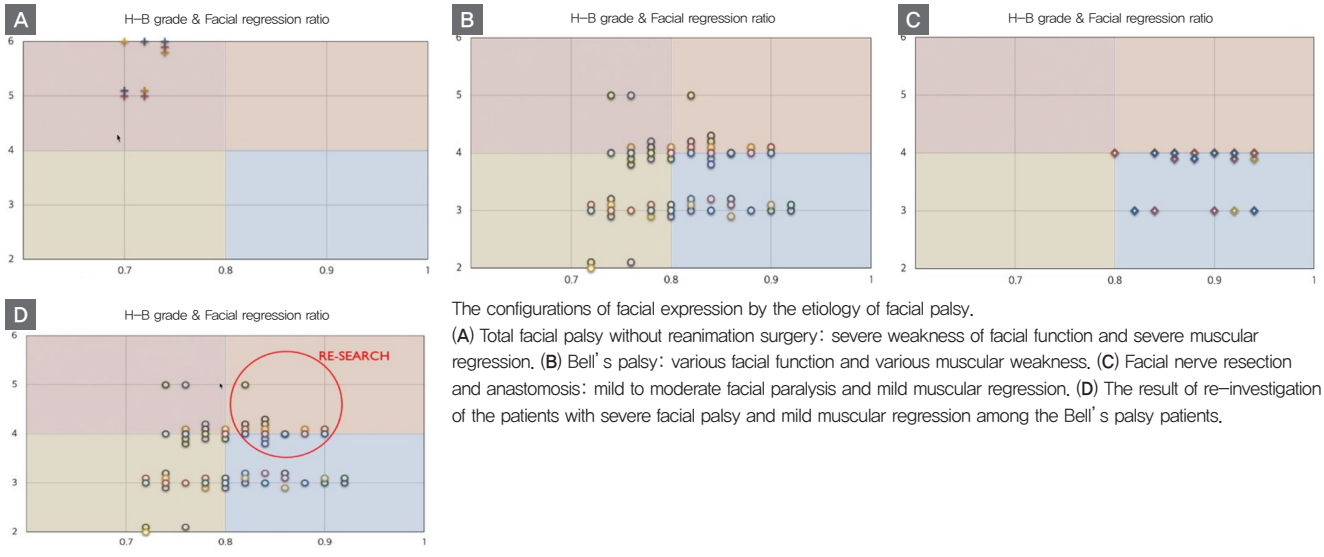


Four types of facial expression by House–Brackmann (H–B) grading system and facial asymmetry ratio.

RESULTS

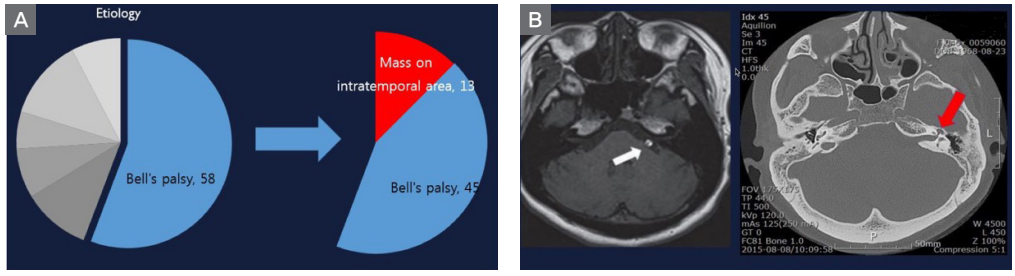
The subjects with total facial palsy without reanimation surgery were noted to have severe weakness of facial function and severe muscular regression (Fig. 4A). The subjects who had been diagnosed with Bell's palsy, herpes zoster oticus, traumatic facial palsy or iatrogenic facial nerve injury were shown to have various facial function and various muscular weakness (Fig. 4B). Lastly, the subjects with neural repair after facial nerve transection

Fig. 4



The configurations of facial expression by the etiology of facial palsy.
(**A**) Total facial palsy without reanimation surgery: severe weakness of facial function and severe muscular regression. (**B**) Bell's palsy: various facial function and various muscular weakness. (**C**) Facial nerve resection and anastomosis: mild to moderate facial paralysis and mild muscular regression. (**D**) The result of re–investigation of the patients with severe facial palsy and mild muscular regression among the Bell's palsy patients.

Fig. 5



(**A**) Out of the 83 patients with Bell's palsy, thirteen patients had an incidental finding of an intratemplral mass. (**B**) Radiologic evaluation of patients with Bell's palsy. Left: Temporal bone magnetic resonance imaging with lipoma in internal auditory canal left (arrow). Right: Temporal bone computed tomography imaging with facial nerve schwannoma left (arrow).

could be shown to have mild to moderate facial paralysis and mild muscular regression (Fig. 4C).

Among the subjects diagnosed with Bell's palsy, some patients were characterized to have severe facial dysfunction (high H-B grade) and mild muscular regression. This prompted for further diagnostic work ups. Additional radiologic evaluation was requested to further diagnose an intratemporal mass or lesion (Fig. 4D). Out of the 83 subjects diagnosed with Bell's palsy, herpes zoster oticus, traumatic facial palsy or iatrogenic facial nerve injury, thirteen subjects had an incidental finding of an intratemporal mass or lesion (Table 3, Fig. 5). The rest of the subjects had an unremarkable temporal imaging. We calculated sensitivity and specificity, positive predictive value (PPV), and negative predictive value (NPV) of severe facial dysfunction (high H-B grade) and mild muscular regression using 2 × 2 contingency tables (Table 4).

In Group I, subjects showed no synkinesis and a mean H-B grade was 5.7 ± 0.34. Severe regression of muscular volume was found by facial dynamic asymmetry ratio (0.72 ± 0.15). Among Group II patients who had been revealed to have hidden intratemporal mass, subjects showed mild synkinesis with a mean H-B grade of 3.7 ± 0.44 and mild regression of muscular volume was found by facial dynamic asymmetry ratio (0.82 ± 0.17). In Group II, the subjects showed mild to moderate synkinesis and mean H-B grade was computed at 3.4 ± 0.96. Mostly, moderate to severe regression of muscular volume was shown in facial dynamic asymmetry ratio (0.79 ± 0.08). In Group III, the subjects showed moderate to severe synkinesis and mean H-B grade was 3.7 ± 0.76. No regression of muscular volume was found by facial dynamic asymmetry ratio (0.88 ± 0.13) (Table 5).

Herein, we report a case of hidden mass found in Group II. A 56-year-old male referred to us with the complaint of chronic facial palsy left for 2 years. It was of sudden onset. He had no history of parotid surgery and trauma. The patient had only a history of hypertension. The patient was diagnosed with Bell's palsy at another institution without imaging such as temporal magnetic resonance

Table 3. Final diagnosis of Bell's palsy patients with severe facial dysfunction and mild muscular regression

Final diagnosis	No. of cases (n = 13)
Facial nerve schwannoma	7
Internal auditory canal lipoma	1
Cholesteatoma	3
Vestibular schwannoma	2

Table 4. Contingency table: Specificity and sensitivity of severe facial severe facial dysfunction (high House-Brackmann [H-B] grade) and mild muscular regression in chronic facial palsy

	Positive (mass)	Negative (mass)	Sum
H-B grade ≥ 4, FRR ≥ 0.8	13	2	15
H-B grade < 4, FRR < 0.8	0	68	68
Sum	13	70	83

Sensitivity = 100%, Specifity = 97.1%, PPV (positive predictive value) = 86.6%, NPV (negative predictive value) = 100%.
FRR: facial regression ratio.

Table 5. The characteristics of all of the patients with chronic facial palsy as their pathogenesis

	Group I (n = 8)	Hidden mass found in Group II (n = 13)	Group II (n = 70)	Group III (n = 13)
Age (yr)	49.5 ± 20.3		61.8 ± 14.6	38.1 ± 5.7
Onset (yr)	7.1 ± 4.5		2.8 ± 0.7	4.1 ± 1.5
House-Brackmann grade	5.7 ± 0.34	3.7 ± 0.44	3.4 ± 0.96	3.7 ± 0.76
Babak and Kimberly classification	Type E: 8	Type B: 12 Type C: 1	Type B: 42 Type C: 28	Type B: 3 Type C: 10
Facial dynamic asymmetry ratio	Severe: 0.72 ± 0.15	Mild: 0.82 ± 0.17	Moderate to severe: 0.79 ± 0.08	No regression: 0.88 ± 0.13

imaging (MRI) or computed tomography. Two years ago and had received steroid medical treatment at that time. However, has not shown any significant improvement of facial nerve function so far. On clinical examination, there was asymmetrical face with deviation to the right side on mouth opening, lack of movement of left forehead and eyebrows. The patient was unable to blow his mouth and close his left eye completely. H-B grade was evaluated IV and BK classification was Type B. And we evaluated facial dynamic asymmetry ratio and it was checked to 0.78. He was diagnosed with facial schwannoma on temporal bone MRI.

DISCUSSION

The important sequelae of facial nerve palsy include synkinesis, asymmetry and contracture; all of which have psychosocial effects on the subjects. Synkinesis due to mal regeneration may cause involuntary movements during a voluntary movement.[14] Chronic facial paralysis is accompanied by facial sequela including involuntary hyperkinetic movement and facial asymmetry in nearly all cases.[15] Common histological findings such as irregular course of regenerated fibers and multiple axon sprouting were seen in the facial nerve with synkinesis.[16] An expert clinician may still have difficulty in differentiating between acute and chronic facial paralysis because regardless of the cause of nerve injury, the site of injury, and the time of injury, subjects always present with just facial paralysis. In chronic facial paralysis, however, minute observation of facial expressions could show identical patterns of facial expressions based on its pathogenesis. Facial expressions elicited in chronic facial paralysis represent various appearances depending on the severity of facial nerve injury and the volume regression of facial musculature on affected side. This may signify an injured facial nerve etiology for the expressed facial paralysis. Apart from facial synkinesis being a common complication after facial nerve injury, facial asymmetry caused by an ongoing regression of facial musculature on affected side may continue to progress and eventually worsen.[14]

In our study, the subjects with chronic compressive injury (Group II) and the subjects with facial nerve resection and anastomosis (Group III) had neural patency with mild to moderate muscular regression including synkinesis. This phenomenon simply means that sustained neural patency could not involve facial muscular regression in cases of chronic facial paralysis. This especially holds true in Group II subjects, where most of the cases elicited showed mild synkinesis which could be attributed to the decreased probability of obtaining aberrant regeneration in cases of external compressive injury.

In Group II subjects, various aspects of facial expressions have been presented. Since facial complications after Bell's palsy, can be generated by only severe neural injury, volume regression by denervated muscles, together with synkinesis can be presented depending on its initial severity. In this study, some subjects with atypical facial expression in Bell's palsy have been encountered. As a result, these subjects were reevaluated further by diagnostic workups such as radiologic evaluation. Incidental findings of a hidden mass in the intratemporal area were then established and classified.

In this study, distinctive patterns of facial expression in patients with chronic paralysis were observed and further classified according to their respective pathogenesis. Furthermore, objective facial expression findings may also give a clue regarding the etiology of facial paralysis. Optimal management of chronic facial paralysis would ultimately depend on facial complications including the subject's compliance, and not solely on its etiology. If the clinician is aware that different pathogenesis could elicit different aspects of facial expression in chronic facial paralysis, he may be able to further diagnose underlying pathologies by doing complete history taking and proper physical examination of the subject.

In this study, identical patterns of facial expression were

noted based on the pathogenesis of facial paralysis. With careful and meticulous observation of facial movement, pathogenesis of facial paralysis may easily be provided. Also, severe facial palsy without muscular regression in subjective diagnosed with Bell's palsy could suggest an intratemporal mass.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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