Diagnostic utility of Freyss motor examination, House–Brackmann grading, topognostic tests, and electrophysiological assessments for unilateral peripheral facial nerve disorder

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Abstract. Unilateral peripheral facial nerve disorder is most often idiopathic, and the determination of the cause may be necessary to guide appropriate treatment. There is no standard diagnostic test for determining the cause or location of the lesion; therefore, clinicians may rely on a variety of diagnostic methods such as the Freyss system of motor examination, House–Brackmann grading, topognostic tests (Schirmer, stapedius reflex, gustatometry), and electrophysiological investigations (nerve conductivity, blink reflex, needle EMG). However, few studies have directly compared the suitability of these different diagnostic methods according to disease characteristics (severity, onset, etc.). This descriptive cross-sectional study compared results using these diagnostic modalities among 44 consecutive patients. There was a significant concordance (Kappa R = 0.5, p < 0.05) between the Freyss motor system examination results and House–Brackmann scoring for 32 patients with chronic onset and moderate-to-severe damage. Alternatively, there was poor agreement (Kappa R = 0.011, p = 0.935) between topognostic and electrophysiological investigation for determining lesion location. In 13 patients with chronic onset and moderate-to-severe damage, lesion location could not be determined based on electrophysiology, while topognostic examination was able to determine the location for both acute- and chronic onset cases. For lesions that cannot be assessed by electrophysiology, Freyss and House–Brackmann motor tests can be used for diagnoses and prognosis.

1. Introduction
Peripheral facial nerve disorder is the most common form of unilateral facial paralysis, and most cases exhibit acute onset of unknown cause. The incidence is 20–30 cases per 100,000 people per year with a peak incidence between 20 and 40 years of age. Only a small percentage requires surgical treatment. During the acute phase, surgical indications are not dependent on etiology, but are rather dependent on the potential for functional recovery [1-4]. Finsterer reported that 60%–80% of facial peripheral neurological disorders are idiopathic (Bell’s palsy) [3]. This disorder can also recur in about 4%–14% of reported cases. The incidence does not differ between sexes but the risk is 3.3 times higher in pregnant women (usually in a third trimester) compared to non-pregnant women.

The electrodiagnosis of facial nerve disorders is achieved by well-established electrophysiological examinations that aim to determine the severity of nerve dysfunction. In 1962, Campbell introduced a nerve excitability test (NET) that has proven useful for prognosis in cases of facial paresis with
various etiologies [5]. The current electrophysiological examinations used routinely for peripheral facial nerve examination include the NET, nerve conduction velocity (NCV), electromyography (EMG), maximal stimulation tests, needle electromyography (needle EMG), and blink reflex test [6-10].

Another type of routine examination is the topognostic test, which aims to determine the lesion location based on loss-of-function pattern. These tests include the Schirmer examination, stapedia reflex, and gustatometry. Each method has its own advantages and limitations, and, currently, there is no single examination that can determine the etiology and prognosis of all patients with facial nerve disorders [1,2,6-10].

Currently, there is no international consensus for the use of a specific examination system; therefore, some institutions and countries have developed their own grading systems for facial nerve motor function. The Freyss system was introduced in 1973 by Sjarifuddin at the Department of Otolaryngology Faculty of Medicine-Cipto Mangunkusumo Hospital, Indonesia, and is still used today by the Neurology Department of Faculty of Medicine-Cipto Mangunkusumo Hospital to assess peripheral facial nerve disorders of various etiologies [11].

In 2006, the Department of Otolaryngology Faculty of Medicine-Cipto Mangunkusumo Hospital conducted a comparative study on the scalability of facial nerve motor function scores between the Freyss System and House–Brackmann grading scale, another metric used routinely in the clinic. The results indicated the equivalence of both examination systems for light and moderate facial paresis (i.e., degree I–III with motor function of more than 66.8% according to the Freyss system and House–Brackmann degree I–III), but not for the more severe cases (degrees IV–VI). Therefore, we conducted a comprehensive comparison of several systems among the same patients with a wide range of severities and etiologies.

2. Methods
This study was conducted using cross-sectional descriptive methods to assess the concordance among the Freyss system, topognostic battery, House–Brackmann grading, and electrophysiological investigation (nerve conductivity, needle EMG, EMG, and blink reflex) according to Cohen’s kappa test of inter-rater agreement. The research was conducted by the neurotology clinic of the Department of Otolaryngology and polyclinic of EMG (peripheral nerve) Department of Neurology Faculty of Medicine-Cipto Mangunkusumo Hospital starting in August 2012 and continuing until sufficient numbers of patients were examined (within 10 months).

The inclusion criteria were the diagnosis of unilateral facial paresis/paralysis with fast or slow onset caused by various etiologies, 17–59 years of age, and willing to undergo the indicated series of examinations (with signed approval letter). The exclusion criteria were refusal to participate in the study, unconsciousness at presentation, central facial paralysis, and bilateral peripheral facial paralysis.

3. Results
The research cohort of 44 patients included a higher proportion of males than females (25 vs. 19). Patients ranged in age from 17 to 59 years with the largest proportion in the 40–49 year age range (18 patients, 40.9%). Paralysis was on the left side in 25 subjects (56.8%) and the right side in 19 subjects (43.2%). All the cases were slow onset (chronic) and the most common etiological classification was idiopathic (17 patients, 38.6%).

Table 1 presents the inter-rater agreement between two ENT specialists using the Freyss and House–Brackmann systems. Using both systems, the most common classification was degree V (House–Brackmann: 15 patients, 34.1%; Freyss: 23 patients, 52.3%) and there was good inter-rater agreement for Degrees I and VI (no cases) as well as Degrees II and IV, for which the proportions were nearly equal. However, there was poor agreement for Degrees III and V.
**Table 1.** Inter-rater agreement using house–brackmann and freyss systems.

| House–Brackmann | n (%) | Freyss | n (%) |
|-----------------|-------|--------|-------|
| Degree I        | 0 (0.0) | Degree I | 0 (0.0) |
| Degree II       | 12 (27.3) | Degree II | 13 (29.5) |
| Degree III      | 10 (22.7) | Degree III | 3 (6.8) |
| Degree IV       | 7 (15.9) | Degree IV | 5 (11.4) |
| Degree V        | 15 (34.1) | Degree V | 23 (52.3) |
| Degree VI       | 0 (0.0) | Degree VI | 0 (0.0) |

Table 2 presents an overview of the topognostic examination results for the same 44 patients. Of the idiopathic cases, the lesions were most often suprageniculate (7 of 17 patients, 41.2%). In infection-related cases, about half of the lesions were suprastapedial-suprageniculate (5 of 9, 55.6%). In trauma-related cases, most were infrastapedial-supracordial, while in malignancy-related cases most were suprageniculate (5 of 9, 55.6%).

**Table 2.** Overview of topognostic test results.

| Etiology     | Schirmer N | Abn | Stapedial Reflex N | Abn | Gustatometry N | Abn | Total N |
|--------------|------------|-----|--------------------|-----|----------------|-----|---------|
| Idiopathic   | 7 (41.2)   | 10 (58.8) | 6 (35.3) | 11 (64.7) | 3 (17.6) | 14 (82.4) | 17 (100) |
| Infection    | 5 (55.6)   | 4 (44.4)  | 3 (33.3)  | 6 (66.7)  | 3 (33.3)  | 6 (66.7)  | 9 (100)  |
| Trauma       | 5 (55.6)   | 4 (44.4)  | 7 (77.8)  | 2 (22.2)  | 5 (55.6)  | 4 (44.4)  | 9 (100)  |
| Malignancy   | 2 (22.2)   | 7 (77.8)  | 4 (44.4)  | 5 (55.6)  | 2 (22.2)  | 7 (77.8)  | 9 (100)  |

N: Normal, Abn: Abnormal

Table 3 presents the lesion locations as determined by electrophysiological examination. Of the 17 idiopathic cases, 12 could be classified as proximal or distal (6 patients in each group, 35.3%). Most of the lesions related to infection and trauma were localized (7 of 9), with more than half classified as distal (4 in each group, 44.4%), while only 5 of 9 lesions were localized in malignancy-related cases by electrophysiology, all of which were distal. Thirteen cases could not be localized by electrophysiological examination. The topognostic tests on these 13 subjects revealed 8 with suprageniculate lesions, one with a suprastapedial-infrageniculate lesion, one with an infrastapedial-supracordial lesion, and three with infracordial lesions.

**Table 3.** Overview of topognostic and electrophysiological test results.

| Etiology    | Electrophysiological | Topognostic test |
|-------------|----------------------|------------------|
|             | Proximal | Distal | Cannot be determined |          |
| Idiopathic  | 6 (35.3) | 6 (35.3) | 5 (29.4) | 1 (7.6) |
| Infection   | 3 (33.3) | 4 (44.4) | 2 (22.2) | 1 (7.6) |
| Trauma      | 3 (33.3) | 4 (44.4) | 2 (22.2) | 3 (23.1) |
| Malignancy  | 0 (0.0)  | 5 (55.6) | 4 (44.4) | 8 (61.5) |

Table 4 presents the electrophysiological measurements of NCV, blink reflex time, and Needle EMG. The median R1 value for the entire group was prolonged (12.81 [range, 4.67–55.50]), as was the median R2 (34.17 [23.80–64.00]), while R2C latency was actually shorter than normal (36.41
[0.80 to 56.0)]. Needle EMG revealed spontaneous activity on the paresis side in 14 subjects (31.8%). Normal recruitment features were found in 35 of 44 subjects (79.5%) and reduced recruitment was found in 9 subjects (20.5%).

Table 4. Results of electrophysiological screening.

| Side of Facial Paresis | Speed of Nerve Conductivity |
|-------------------------|-----------------------------|
|                         | Latency (ms) *               |
|                         | Median (Min–Max)             |
| Total Paresis side      | 3.28 (0−9.10)               |
| Right                   | 3.18 (0.0−1.76)              |
| Left                    | 3.38 (0−9.10)                |
| Amplitude (µV mV)       |
| Total Paresis side      | 1.17 (0−5.35)                |
| Right                   | 1.59 (0.0−5.35)              |
| Left                    | 1.88 (0.0−5.91)              |
| Blink Reflex (ms)       |
| *(Median, Min–Max)      |
| Paresis side R1, All patients | 12.81 (4.67–55.50) |
| R1 Right-side paresis   | 12.08 (4.67–55.50)           |
| R1 Left-side paresis    | 12.50 (7.50–24.33)           |
| Paresis side R2, All patients | 34.17 (23.8–64.0) |
| R2 Right-side paresis   | 34.17 (23.8–64.0)            |
| R2 Left-side paresis    | 35.33 (24.5–50.0)            |
| Paresis side R2C, All patients | 36.41 (0.80–56.0) |
| R2C Right-side paresis  | 40.08 (24.75–55.50)          |
| R2C Left-side paresis   | 36.00 (0.80–56.0)            |

**Needle EMG (n%)**

| Recruitment          |          |
|----------------------|----------|
| Normal               | 35 (79.5)|
| Abnormal             | 9 (20.5) |

| Spontaneous activity |          |
|----------------------|----------|
| Normal               | 30 (68.2)|
| Abnormal             | 14 (31.8)|

Cohen’s test for probability of matches (Kappa) indicated a highly significant concordance between the Freyss system motor examination and the Seddon class electrophysiological examination for diagnosis of moderate-to-severe facial nerve damage (Kappa R = 0.50 and p < 0.001). There was also a moderately significant concordance between Brackmann–House system motor examination and
the Seddon classification for moderate-to-severe facial nerve damage (Kappa R = 0.43 with p = 0.05).

In contrast, there was no significant relationship between electrophysiological examination and topognostic tests for lesion location (Kappa R = 0.011 and p = 0.935). Nerve conduction latency cannot be used when damage is permanent or the degree of damage is moderate-to-severe. In this cohort, lesion location could not be determined in 13 patients by electrophysiological examination. Alternatively, the topognostic test revealed the location in these patients as detailed above (Table 3).

4. Discussion

This study involved 44 patients with facial paresis/paralysis of various etiologies examined by multiple modalities to assess concordance and the most appropriate for specific conditions. The demographic distribution (25 men and 19 women, median age 40.9 years with most between 40–49 years) is consistent with Alberton’s report of high Bell’s palsy risk at around age 40 [15]. Volk et al [1] and Thai [16] estimated an incidence of 20 to 30 cases per 100,000 annually with highest incidence between 20 to 40 years of age, while Finsterer reported highest incidence rate in the age range 15–45 years, with no difference in affected side [3]. Thus, the cohort examined here appears representative of the patients studied in other regions. All the study subjects presented with slow onset, indicating permanent moderate-to-severe facial nerve damage as the possibility of recovery (regeneration) drops substantially by 9 weeks after onset [12–14]. Micro-anatomic studies have concluded that nerve damage occurring in patients with slow onset (> 3 months) has likely reached the axon due to intraneural pressure, damaging the epineurium and possibly the perineurium and endoneurium. Axonal damage will impede the healing process, which can be rated by Freyss and House-Brackmann systems [13].

The most common etiological classification in this cohort was idiopathic (38.6%), while malignancy (scarring cell carcinoma of the ear canal) was deemed the cause in 9 patients (20.5%), and infection in 9 patients (20.5%) including chronic suppurative otitis media in 6 (15.9%), sine herpete in 2 (4.5%) and otitis external (OE) malignancy in one (1.2%). Of the 9 cases attributed to trauma (20.5%) which 4 experienced temporal trauma (9.1%), 3 surgery trauma (6.8%), and 2 facial trauma (4.5%). Volk et al. [1] and Junior et al [10] reported that 60%−75% of unilateral peripheral facial nerve paresis cases are idiopathic (Bell’s palsy), while the common known causes were accidental trauma, herpes zoster, otitis media, surgical trauma, and Schwannoma. Ozgur et al. reported an even higher prevalence of idiopathic cases (85%) [2]. Thus, although there were substantially fewer idiopathic cases, the distribution of etiologies in this cohort resembles that of previous studies. Further study is required to assess whether this reflects a true population difference or selection bias.

Based on NCV and amplitude examinations, conduction block was common in this cohort. Poernomo reported normal values of 10 ms for R1, 30 ms for R2, and 41 ms for R2C latency [17]. In eye-blink examination, median ipsilateral R1 for the entire group was 12.81 ms [4.67–55.50], ipsilateral R2 when on the right side was 34.17 ms (23.80–64.00), and contralateral R2C for right-side paresis was 36.41 ms (0.80–56.0). Thus, this cohort demonstrated prolonged R1 and R2 latencies but normal R2C latency. The R1 and R2 latencies are usually abnormal in peripheral facial nerve paresis compared to the normal side. In this study, the healthy side showed an even greater ipsilateral slow response stimulus (R2) than contralateral side (R2C) did. When connected with the side of paresis will give opposite response results. The latencies of R1, R2, and R2C during ipsilateral and contralateral stimulation can reveal whether the lesion is in the supraorbital nerve, the facial nerve, the pons, or the medulla oblongata, or whether it is a diffuse demyelinating lesion. The results of this study indicate facial nerve lesions based on previous findings that R1 and R2 latencies on the paresis side are elongated while R2C values are normal in cases of facial nerve damage.

Basuki and Wijaya reported no electrical activity on EMG needle examination at rest in normal subjects, while nerve/muscle disorders may be accompanied by spontaneous positive sharp wave (PSW) activity or fibrillation [18]. Spontaneous activity in the form of PSW and/or fibrillation as measured from the muscle at rest may indicate damage to the muscle due to loss of innervation and/or primary damage to the muscle itself. In this study, spontaneous activity on the paresis side was found by Needle EMG in 14 subjects (31.8%). This indicated that 14 patients already had damage to motor neurons, axons, or muscles. Recognizing the presence of spontaneous activity may assist in diagnosis, localization of the lesion, and prognosis. Spontaneous localized activity in chronic lesions suggests
poorer prognosis because there is no possibility of reinnervation [19,20]. Normal recruitment was found in 35 subjects (79.5%) and decreased recruitment was found in 14 subjects (20.5%). Thus, in 14 subjects, the number of motor units was already reduced. No increased recruitment was found in this study.

Electrophysiological examination of the facial nerve using the Seddon classification divided the cohort into 3 groups: neuropraxia, axonotmesis, and neurotmesis. Twelve patients were diagnosed with neuropraxia, which is characterized by focal demyelination without axon disturbance. In neuropraxia case, there is no Wallerian degeneration and ENMG reveals conduction block only in the damaged segment of the nerve. The other 32 patients were diagnosed with axonotmesis or neurotmesis. In axonotmesis, there is already damage to myelin and axons, leading to Wallerian degeneration. In such cases, ENMG revealed conduction block in less than 10 days from onset, while compound muscle action potential (CMAP) amplitude decreased 30%–50% by day 9 on the contralateral side and the EMG needle potential was negative (positive sharp wave, fibrillation) with morphological changes of the motor unit action potential (MUAP) after week 2. In cases of neurotmesis already involving the myelin layer, axon, and buffer layer (endo-peri-epineurium), regeneration requires surgery and ENMG will reveal conduction block in less than 10 days and reduced CMAP amplitude at day 9, while EMG needle examination will show denervation syndrome accompanied by MUAP changes after week 2. Electrophysiological examination is not influenced by etiology so no examination characteristics are unique to a specific etiology. However, this modality describes the current extent of functional damage to the facial nerve. The results of this study suggest that patients with new/slow onset (under 3 months) and mild degree of neurological damage have better prognosis than chronic onset patients (over 3 months) with moderate-to-severe neuronal damage.

5. Conclusion

Based on the results of this study, it can be concluded that there is a strong relationship between Seddon electrophysiological examination and both the Freyss System and House–Brackmann grading for patients with chronic onset moderate-to-severe facial nerve damage. However, there was no acceptable concordance between electrophysiology and topognostic testing for determining the location of the lesion in chronic onset moderate-to-severe cases because of the limitations of latency and amplitude measurements. Comprehensive multi-modal examination may be necessary for accurate diagnosis.

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