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Electroneurographic Degeneration Index Before Facial Rehabilitation in Idiopathic Facial Palsy (Bell's Palsy): A Prospective Observational Cohort of 90 Patients

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Abstract: Background: Early oral corticosteroids, with or without antivirals, are the established first-line treatment for idiopathic facial palsy (IFP, Bell's palsy), and most patients recover. Clinical grading in the acute phase, however, cannot distinguish a conduction block from axonal degeneration, and rehabilitation is usually started without objective information about the state of the nerve. Electroneurography (ENoG) quantifies the proportion of degenerated motor axons as a degeneration index (DI). Objective: To describe how the pre-rehabilitation ENoG DI relates to clinical severity, and to describe recovery trajectories by DI stratum, in patients with IFP managed with medical therapy plus adjunctive, DI-informed rehabilitation. Materials and Methods: his prospective, single-centre, single-arm observational cohort study included 92 patients with acute IFP. Baseline ENoG was performed within 10 days of symptom onset (median: 6 days). The DI was calculated for five facial muscles representing the major peripheral branches of the facial nerve using the formula: DI (%) = 100 − [(CMAP amplitude on the affected side / CMAP amplitude on the unaffected side) × 100]. House–Brackmann (HB) grade and the Sunnybrook Facial Grading System (SBFGS) score were assessed by an independent physiotherapist at baseline and on Days 21 and 42 after baseline. All patients received oral corticosteroids; 40% initiated treatment within 72 hours of symptom onset and 60% thereafter. All patients received a structured facial rehabilitation programme, including facial exercises, massage and therapeutic ultrasound. Low-level laser therapy and extracorporeal shockwave therapy were allocated according to predefined DI strata: mild (<25%), moderate (25%–50%) and severe (>50%). Outcomes were compared across these strata. Results: The mean age was 35.4 ± 14.8 years, and 62 of 90 patients with recorded sex were men. Mean baseline DI was approximately threefold higher in the severe stratum than in the mild stratum (60.2% ± 10.3% vs. 19.4% ± 5.4%). The corresponding mean HB grades differed by less than one grade (3.69 vs. 2.78; $p = 0.001$). Across the cohort, DI, HB grade and SBFGS scores improved by Day 42 (all $p < 0.001$). Among the 90 patients included in the complete repeated-ENoG analysis, 81 (90.0%) achieved HB grade I by Day 42. The proportion of baseline DI resolved was similar across the mild, moderate and severe strata (80.5%, 83.3% and 83.9%, respectively; $p = 0.87$). However, Day-42 SBFGS scores remained lower in the severe stratum than in the mild stratum (93.2 vs. 97.3; $p = 0.003$). The proportion achieving HB grade I showed a non-significant downward trend across the strata (94.4%, 90.2% and 84.4%, respectively; $p = 0.52$). Higher baseline DI was associated with a greater number of rehabilitation sessions (mean: 22.0, 29.7 and 32.4 in the mild, moderate and severe strata, respectively; $p = 0.002$; Spearman's $\rho = 0.35$, $p < 0.001$). Conclusion: Pre-rehabilitation ENoG DI provided objective information about the extent of nerve injury that was not fully captured by clinical grading. Higher baseline DI

was associated with greater residual facial dysfunction at six weeks and a greater number of rehabilitation sessions. Recovery was frequent during follow-up under the combined medical and rehabilitation pathway. However, the absence of a control group and the potential contribution of spontaneous recovery preclude attributing these outcomes to rehabilitation or DI-based treatment allocation. Controlled studies are needed to determine whether DI-guided rehabilitation improves outcomes compared with standard rehabilitation.

Keywords: Bell's palsy, degeneration index, electroneurography, facial nerve, facial rehabilitation, House–Brackmann scale, prognosis, Sunnybrook Facial Grading System

INTRODUCTION

Bell's palsy, or idiopathic facial palsy (IFP), is the most common cause of acute unilateral peripheral facial paralysis. It is a diagnosis of exclusion, with isolated facial weakness that typically peaks within 72 hours.[1,2] The facial muscles insert into skin rather than bone, are supplied only by cranial nerve VII, and mediate both emotional expression and protective eye closure, so even partial paresis has clinical and psychosocial consequences.[3]

The natural history of Bell's palsy recovery is favourable. In Peitersen's series of untreated patients, about 71% recovered completely, and most showed the first signs of recovery within three weeks.[4] Early oral corticosteroids improve the chance of complete recovery and are recommended for eligible adults within 72 hours of onset. Antivirals may be added but give little benefit on their own.[2,5-9] Even with optimal medical treatment, however, a minority of patients are left with incomplete recovery or synkinesis, the involuntary co-contraction that follows misdirected axonal regeneration.[10,11]

The outcome is decided by the type of nerve injury, not by how weak the face looks in the first days. A conduction block without axonal loss (Seddon neurapraxia; Sunderland grade I) recovers quickly and completely. Axonal degeneration (axonotmesis; Sunderland grade II or higher) requires regeneration, recovers more slowly, and carries the risk of aberrant reinnervation.[12,13] In the acute phase these patterns can produce identical clinical grades. Electroneurography (ENoG) helps to separates them. In this test stimulation is given to the nerve distal to the lesion and then the compound muscle action potential (CMAP) amplitude is compared on the affected side with that on the healthy side. Once Wallerian degeneration has reached the distal segment (about 72 hours after injury), the amplitude reduction estimates the proportion of degenerated motor axons. It is expressed as a degeneration index (DI): DI (%) = [100 − (CMAP amplitude affected side / CMAP amplitude unaffected side) × 100].[14,15] Higher early degeneration predicts slower and less complete recovery.[14,16-22] Current guidelines reserve ENoG mainly for patients with complete paralysis who are being considered for surgical decompression, and do not recommend it routinely in incomplete palsy.[2,9]

Rehabilitation is often offered alongside medical treatment. The evidence for it is limited and mostly of low certainty, but some studies suggest that facial exercise and related approaches may improve function and shorten recovery time.[23-25] One reason the evidence is inconsistent may be that rehabilitation is usually prescribed uniformly, without knowing whether a given patient has a conduction block or axonal loss. That distinction matters for rehabilitation planning. It sets the expected recovery time for counselling. It

indicates when strong, mass-movement effort and unguided electrical stimulation should be avoided because of the risk of synkinesis.[26,27] It also provides an objective baseline against which recovery can be measured.

This study therefore made the pre-rehabilitation ENoG and DI its focus. The aims were (i) to describe how the baseline DI relates to clinical severity measured by the House–Brackmann Facial Nerve Grading System grade and Sunnybrook Facial Grading System; and (ii) to describe the recovery of the DI and of clinical grades over six weeks, overall and by baseline DI stratum, in patients receiving medical therapy plus adjunctive rehabilitation informed by the DI. We did not set out to test whether rehabilitation is effective, and the design cannot test this.

MATERIALS AND METHODS

Study Design, Setting and Approvals

This prospective, single-centre, single-arm observational cohort study was conducted in the Department of Physiotherapy and Electrophysiology, Neuro Vihar, Bihar Neuro Diagnostic Centre, Patna, from September 2024 to February 2025. The study was approved by the Institutional Ethics Committee (IEC; approval number JVR/IEC/2023-24/2101). Written informed consent was obtained in English or Hindi from all participants or, for minors, from a parent or legal guardian; assent was also obtained from minors. The IEC approved the study protocol, including the prespecified outcomes (DI measured at five facial muscles, House–Brackmann [HB] grade and Sunnybrook Facial Grading System [SBFGS] scores at baseline and Days 21 and 42), on 30 June 2024. The study was registered with the Clinical Trials Registry–India (CTRI/2024/07/071286) in July 2024. The first participant was enrolled in September 2024, after both ethics approval and trial registration; therefore, registration preceded participant enrolment. A pilot phase, initiated in January 2023 to develop the study protocol, preceded the main study. Data from pilot participants were not included in the present analysis. The rehabilitation protocol was registered with the Government of India Copyright Office (registration number L-147405/2024; July 2024). This copyright registration protects the protocol as intellectual property; it is distinct from clinical study registration and does not constitute evidence of clinical effectiveness.

Participants and Eligibility

Consecutive patients referred with acute idiopathic facial palsy (IFP) were screened for eligibility. Inclusion criteria were: (i) acute, unilateral lower motor neuron facial weakness involving the forehead, reaching maximum severity within 72 hours of symptom onset, without other neurological deficits, and diagnosed by a neurologist; (ii) presentation permitting baseline electroneurography (ENoG) within 10 days of symptom onset; (iii) age 10–59 years; and (iv) ENoG findings consistent with unilateral facial nerve involvement and a measurable compound muscle action potential (CMAP) on the unaffected side.

Exclusion criteria were recurrent or bilateral facial palsy; herpes zoster oticus (Ramsay Hunt syndrome); recent head or neck trauma; active or recent otitis media or ear discharge; pregnancy; previous facial nerve or parotid surgery; central (upper motor neuron)

facial weakness; known secondary causes of facial palsy, such as tumour, stroke or Guillain–Barré syndrome; and inability to cooperate with the assessments.

Medical Management

Rehabilitation was provided as an adjunct to medical treatment, not as a replacement. Medical therapy was prescribed by the referring neurologist in accordance with clinical practice guidelines.[2,9] The corticosteroid regimen comprised oral prednisolone 60 mg daily for 5 days, followed by a 5-day taper. Patients with severe facial palsy were additionally prescribed an antiviral agent: either acyclovir 400 mg five times daily for 10 days or valacyclovir 1 g three times daily for 7 days. All 90 eligible patients received oral corticosteroids. Treatment was initiated within 72 hours of symptom onset in approximately 40% of patients and after 72 hours in approximately 60%. The timing of corticosteroid initiation relative to symptom onset and the use of antiviral therapy were recorded for each patient (Table 1). Patients with incomplete eye closure received eye protection, including lubricating eye drops during the day and ophthalmic ointment with eyelid taping at night.

Neuroimaging

Neuroimaging was not performed routinely in patients with typical Bell's palsy, consistent with clinical practice guidelines.[2] Ten patients underwent non-contrast brain MRI with a constructive interference in steady state (CISS) sequence to evaluate the facial nerve because of atypical clinical features, including progressive symptoms, recurrence, multiple cranial nerve deficits, or failure to improve after several weeks. The investigations were funded by Neuro Vihar, Bihar Neuro Diagnostic Centre. MRI findings were normal in all 10 patients.

Timing of Baseline Assessment

Baseline was defined as the visit at which electroneurography (ENoG) and clinical grading were performed. Patients presented to the centre at least 3 days after symptom onset before undergoing baseline ENoG. ENoG was performed before initiation of the formal degeneration-index (DI)-guided rehabilitation programme, allowing patients to be allocated to predefined DI severity bands. Patients who presented earlier received supportive care, comprising facial massage and eyelid taping where indicated, before the baseline ENoG assessment. This early supportive care was distinguished from the formal DI-guided rehabilitation programme, which commenced only after the baseline DI had been determined. Formal rehabilitation began on the same day as ENoG or the following day. ENoG timing was selected to allow Wallerian degeneration to be reflected in the distal compound muscle action potential (CMAP), consistent with its established prognostic application.[14,16]

Electroneurography and Degeneration Index

Electroneurography (ENoG) was performed using an RMS Salus 4C system (Recorders & Medicare Systems, India) by a physiotherapist trained in clinical electrophysiology. The

facial nerve trunk was stimulated at the stylomastoid foramen, anterior to the mastoid process, using a bipolar surface electrode and supramaximal stimulation. Compound muscle action potentials (CMAPs) were recorded using surface electrodes over five facial muscles: frontalis (temporal branch), orbicularis oculi (zygomatic branch), nasalis (buccal and zygomatic branches), orbicularis oris (buccal branch), and mentalis (marginal mandibular branch). These muscles were selected to represent the major peripheral branches of the facial nerve and the functions assessed by the House–Brackmann (HB) and Sunnybrook Facial Grading System (SBFGS), including brow elevation, eye closure, nasal flaring, smile and lip movement, and lower-lip depression. Both sides were examined during the same session using consistent electrode placement, and peak-to-peak CMAP amplitudes were recorded. The degeneration index (DI) for each muscle was calculated as:

DI (%) = [100 − (amplitude affected side / amplitude unaffected side) × 100].

A composite DI was calculated as the arithmetic mean of the DI values for the five muscles.

Stimulation Settings:

Stimulation Pulse Duration (Pulse Width): 0.1 ms to 0.2 ms (100 µs to 200 µs).

Stimulation Frequency: 1 Hz (1 pulse per second)

Recording Filter Settings:

Low-Frequency Filter (LFF / High Pass): 2 Hz to 5 Hz

High-Frequency Filter (HFF / Low Pass): 10 kHz

Notch Filter: 50 Hz

Electrode Configuration

The protocol uses a standard standard bipolar surface electrode arrangement:

- **Active Recording Electrode (G_1 or Cathode):** Placed directly over the motor belly of the target facial muscle branch. Common targets include:
    - *Frontalis* (Temporal branch)
    - *Orbicularis oculi* (Zygomatic branch)
    - *Nasalis* (Buccal branch)
    - *Orbicularis oris* (Buccal/Mandibular branch)
    - *Mentalis*
- **Reference Recording Electrode (G_2 or Anode):** Placed over an electrically neutral or relatively inactive site, typically on the contralateral side of the nasal bridge, the chin, or the tendon/bone area opposite the G_1 placement.
- **Ground Electrode:** Placed centrally, typically on the forehead (glabella) or the chin, to suppress ambient artifacts.

Inter-Electrode Distances

- **Recording Electrodes (G_1 to G_2):** A separation distance of approximately **3 cm to 4 cm** is maintained. Keeping this distance standard ensures you do not inadvertently truncate or artificially distort the wave morphology.
- **Stimulating Probe Distance:** The electrical simulator (placed anterior to the mastoid process, below the ear lobule where the facial nerve trunk exits the stylomastoid foramen) should remain roughly **5 cm to 7 cm** away from the active recording electrode (G_1) depending on the patient's anatomy.

ENoG was selected because it provides a quantitative comparison of CMAP amplitudes between the affected and unaffected sides and is used to estimate the extent of facial nerve degeneration. The blink reflex evaluates the integrity of the broader facial reflex pathway and can assist with lesion localization, whereas needle electromyography can identify denervation activity and changes in voluntary motor-unit recruitment as these become detectable over time. Distal motor latency provides different information and is less directly suited to quantifying side-to-side CMAP amplitude loss.[14,16]

Interpretation of the DI

The degeneration index (DI) was interpreted as an ENoG-derived estimate of the extent of facial nerve degeneration, based on the reduction in compound muscle action potential (CMAP) amplitude on the affected side relative to the unaffected side. A low DI in the presence of clinical weakness may be consistent with predominantly conduction block (neurapraxia; Sunderland grade I), whereas a high DI indicates greater CMAP amplitude loss consistent with more substantial axonal degeneration (Sunderland grade II or higher).[12,13] However, DI alone cannot definitively distinguish conduction block from axonal injury or assign a specific Sunderland grade. ENoG cannot reliably differentiate Sunderland grades II–V, all of which may involve Wallerian degeneration, and it does not directly localize or characterize the lesion proximal to the stimulation site. Accordingly, DI was used to estimate the extent of electrophysiological degeneration rather than to establish the definitive anatomical grade of nerve injury.

Clinical Assessment

Facial function was assessed at each study visit using the House–Brackmann (HB) grading system (grades I–VI)[28] and the Sunnybrook Facial Grading System (SBFGS; 0–100).[29] Assessments were performed by an independent physiotherapist who was not involved in patient treatment or other study procedures, including ENoG, and who was blinded to the DI results. The SBFGS composite score was calculated according to the scoring method, incorporating voluntary movement, resting symmetry and synkinesis. Standardized videos were recorded at each visit to document facial function. The recording protocol included facial appearance at rest, brow elevation, gentle and tight eye closure, open-mouth smiling, snarling and lip puckering.

Assessment Schedule

Assessments were performed at baseline, Day 21 and Day 42, with follow-up days calculated from the baseline assessment rather than symptom onset. Day 21 was selected because spontaneous recovery commonly begins to become clinically apparent within the first three weeks.[4] Repeat ENoG at this visit was intended to document changes in CMAP amplitudes and the DI during recovery. However, these measurements cannot independently establish whether improvement reflects resolution of conduction block or changes associated with axonal degeneration. Day 42 marked the end of the six-week study assessment period. Synkinesis typically emerges 3–6 months after symptom onset and therefore could not be fully evaluated within the study follow-up period.[11]

Adjunctive Rehabilitation

All patients received a structured facial rehabilitation protocol (Table 2). Formal DI-guided rehabilitation was delivered at the centre six days per week, with a home exercise programme on the seventh day. Patients presenting before baseline ENoG received early supportive care, comprising facial massage and eyelid taping where indicated; this was distinguished from the formal rehabilitation programme initiated after the baseline DI had been determined.

The formal protocol included facial neuromuscular re-education with mirror feedback (10 minutes per session), emphasizing slow, small-amplitude, isolated movements, together with a home exercise programme performed three times daily; facial massage (5 minutes); facial taping to support resting symmetry, where indicated; and eye care. Surface electrical stimulation was not used because of concerns that unguided stimulation may contribute to synkinesis.[26,27,30] Therapeutic ultrasound, low-level laser therapy and extracorporeal shockwave therapy were also included, with treatment allocation and parameters specified in Table 2.

Treatment allocation was determined by predefined DI severity bands. Laser and shockwave therapy were not used in the mild DI band but were used in both the moderate and severe bands. Laser parameters were identical in the moderate and severe bands, whereas shockwave frequency was 1.3–1.5 Hz in the moderate band and 1.5–2 Hz in the severe band. Other protocol components were common across the DI bands. Evidence supporting these physical agents in Bell's palsy remains limited; they formed part of the clinical pathway and are reported to support transparency and reproducibility, rather than being evaluated as independent interventions.

Rehabilitation sessions continued until clinical recovery, defined as HB grade I. Clinical and electrophysiological outcomes were assessed at Day 42 for all patients. Patients who had not achieved HB grade I by Day 42 continued rehabilitation beyond the scheduled assessment period, and these additional sessions were included in the total session count. The maximum recorded number of sessions was 57, in a patient classified in the severe DI band.

Predefined Severity Bands

As no validated DI cut-offs are established for rehabilitation planning, three severity bands were defined a priori using the mean baseline composite DI: mild (DI <25%), moderate (DI

25%–50%) and severe (DI >50%). These bands were used both to allocate rehabilitation components according to the predefined protocol and to group patients for analysis. No patient had a composite DI ≥90%, a threshold cited in the literature in the context of considering surgical decompression.[14,16] Accordingly, the severe category in this study represents substantial but incomplete degeneration, rather than near-complete degeneration.

Outcomes

The primary outcome was the relationship between baseline DI severity band and baseline clinical facial function, assessed using the House–Brackmann (HB) grade and Sunnybrook Facial Grading System (SBFGS). Secondary outcomes included the number of rehabilitation sessions until clinical recovery, compared across DI severity bands; changes in muscle-specific and composite DI, HB grade and SBFGS from baseline to Day 21 and Day 42; the proportion of baseline DI resolved by Day 42; and the proportion of patients achieving HB grade I by Day 42, both overall and within each DI severity band.

Statistical Analysis

Data were analysed using jamovi (version 2.3), based on R (version 4.1).[31,32] Continuous variables are summarized as mean ± standard deviation (SD), and normality was assessed using the Shapiro–Wilk test. Because the degeneration index (DI), House–Brackmann (HB) grade and Sunnybrook Facial Grading System (SBFGS) scores were not normally distributed, changes across baseline, Day 21 and Day 42 were assessed using the Friedman test, followed by Durbin–Conover pairwise comparisons. Differences between predefined DI severity bands were assessed using the Kruskal–Wallis test. The proportion of patients achieving HB grade I by Day 42 was compared across severity bands using the chi-square test. Associations between baseline composite DI and clinical scores at baseline and Day 42, as well as the number of rehabilitation sessions, were examined using Spearman's rank correlation coefficient. A two-sided p-value <0.05 was considered statistically significant. Analyses were descriptive and were not adjusted for multiple comparisons or potential confounders; therefore, p-values should be interpreted cautiously. Analyses were conducted using the available data for each outcome.

RESULTS

Participants

Ninety-two patients were initially enrolled. Two patients, aged 6 and 62 years, were excluded because they fell outside the predefined eligibility range of 10–59 years. The final study cohort therefore comprised 90 eligible patients, all of whom completed assessments at baseline, Day 21 and Day 42. Sex was recorded for all 90 patients. All eligible patients received oral corticosteroids, with approximately 40% initiating treatment within 72 hours of symptom onset and 60% thereafter. Antiviral therapy was prescribed to patients with severe facial palsy. Brain MRI was performed in 10 patients (11.1%), and all scans were normal.

All 90 eligible patients completed the three scheduled assessment time points, including five-muscle electroneurography (ENoG), clinical grading using the House–Brackmann (HB) scale and the Sunnybrook Facial Grading System (SBFGS). Patients were classified into the predefined mild, moderate and severe degeneration-index (DI) bands using their baseline composite DI. The distribution of patients across these bands and the corresponding clinical and rehabilitation outcomes are presented in the Results and tables.

Baseline DI and Clinical Severity

The baseline composite degeneration index (DI) was 41.2% ± 17.1%. Muscle-specific DI values ranged from 35.0% in the orbicularis oculi to 43.5% in the mentalis (Table 3). Baseline DI correlated moderately with clinical severity, as assessed by the House–Brackmann (HB) grade (Spearman's $\rho = 0.39$, $p < 0.001$) and the Sunnybrook Facial Grading System (SBFGS; $\rho = -0.34$, $p < 0.001$), indicating that clinical grading captured only part of the variation in electrophysiological degeneration.

Across the predefined DI severity bands (Table 5), the mean baseline HB grade was 2.78 ± 0.81 in the mild, 3.17 ± 0.92 in the moderate and 3.69 ± 0.78 in the severe band ($p = 0.001$). Corresponding mean SBFGS scores were 59.4 ± 21.5, 49.4 ± 22.0 and 39.5 ± 18.0, respectively ($p = 0.004$). The mean composite DI increased approximately threefold from the mild to the severe band, from 19.4% to 60.2%, whereas the mean HB grade differed by less than one grade. These findings illustrate that clinical grading and ENoG provide complementary information, with DI demonstrating differences in electrophysiological degeneration that were less apparent from clinical grading alone.

Recovery Over Six Weeks

The degeneration index (DI) decreased across the three assessment time points at all five muscles (Table 3). The mean composite DI decreased from 41.2% ± 17.1% at baseline to 25.6% ± 14.8% on Day 21 and 6.9% ± 4.6% on Day 42 (Friedman $x^2 = 169$, $p < 0.001$; $p < 0.001$ for each muscle). The mean House–Brackmann (HB) grade improved from 3.27 ± 0.91 at baseline to 1.98 ± 0.58 on Day 21 and 1.11 ± 0.31 on Day 42 ($x^2 = 166$, $df = 2$, $p < 0.001$). The mean Sunnybrook Facial Grading System (SBFGS) score increased from 47.9 ± 21.6 at baseline to 79.4 ± 13.6 on Day 21 and 94.9 ± 4.1 on Day 42 ($x^2 = 179$, $df = 2$, $p < 0.001$) (Table 4). By Day 42, 81 of 90 patients (90.0%) had achieved HB grade I.

Recovery by Baseline DI Stratum

The proportion of baseline degeneration index (DI) resolved by Day 42 was similar across the three predefined severity bands: 80.5% ± 20.3% in the mild band, 83.3% ± 7.9% in the moderate band and 83.9% ± 7.0% in the severe band (Kruskal–Wallis $H = 0.27$, $p = 0.87$) (Table 5). However, clinical recovery trajectories differed across the bands. Mean Sunnybrook Facial Grading System (SBFGS) scores differed significantly across the mild, moderate and severe bands on Day 21 (85.4, 79.6 and 75.9, respectively; $p = 0.009$) and Day 42 (97.3 ± 3.5, 95.0 ± 3.8 and 93.2 ± 4.1, respectively; $p = 0.003$). Baseline DI was negatively correlated with the Day-42 SBFGS score (Spearman's $\rho = -0.33$, $p < 0.001$), indicating that

greater initial electrophysiological degeneration was associated with poorer facial function at six weeks.

The proportion of patients achieving House–Brackmann (HB) grade I decreased across the mild, moderate and severe bands (94.4%, 90.2% and 84.4%, respectively), although the difference was not statistically significant ($x^2 = 1.31$, $p = 0.52$). Mean HB grade at Day 42 also did not differ significantly across the bands ($p = 0.52$). This limited discrimination near normal function may reflect a ceiling effect of the HB scale, which has relatively few categories for distinguishing subtle residual deficits.

Baseline DI and Rehabilitation Needs

Patients with higher baseline degeneration index (DI) required more rehabilitation sessions to achieve clinical recovery. The mean number of sessions was 22.0 ± 7.8 (median, 23) in the mild DI band, 29.7 ± 9.1 (median, 29) in the moderate band and 32.4 ± 10.6 (median, 32) in the severe band (Kruskal–Wallis H = 12.50, $p = 0.002$). Across the cohort, baseline composite DI showed a positive correlation with the number of rehabilitation sessions (Spearman's $\rho = 0.35$, $p < 0.001$). On average, patients in the severe band required approximately 10 more sessions than those in the mild band, equivalent to roughly two additional weeks at six sessions per week. These findings indicate an association between greater baseline electrophysiological degeneration and longer rehabilitation requirements; however, session counts also reflect the clinical recovery and treatment-discontinuation criteria used in this study.

Table 1: Baseline characteristics and medical management (N = 92)

Characteristic	Value
Age, years, mean ± SD (range)	35.4 ± 14.8 (10–59)
Sex, n (%) [recorded n=90; missing n=2]	Male 62 (68.9); Female 28 (31.1)
Side of palsy, n (%) [recorded n=91; missing n=1]	Right 49 (54.4); Left 40 (44.4); Ambiguous 1 (1.1)
Comorbidity, n (%)	None 79 (87.8); Hypertension 3 (3.3); Diabetes mellitus 5 (5.6); Both 3 (3.3)
Days from onset to baseline ENoG, median (range)	6 (5–10)
Days from onset to first formal rehabilitation session, median (range)	6 (5–8)
Oral corticosteroid given, n (%)	90 (100)
Started ≤72 h of onset, n (%)	40%
Started >72 h after onset, n (%)	60%
Antiviral added (severe palsy only), n	10
HB grade at baseline, n (II / III / IV / V / VI)	24 / 19 / 44 / 3 / 0
Composite DI at baseline, %, mean ± SD	41.2 ± 17.1
Rehabilitation sessions attended, mean ± SD (range)	29.2 ± 10.0 (10–57)

Brain MRI with CISS sequence performed, n	10 ; all normal

DI = degeneration index; ENoG = electroneurography; HB = House–Brackmann; CISS = constructive interference in steady state.

Table 2: Rehabilitation protocol and allocation by predefined degeneration-index band

Component	Mild (DI <25%)	Moderate (DI 25%–50%)	Severe (DI >50%)
Facial neuromuscular re-education with mirror feedback (10 min per session; home programme three times daily)	Yes	Yes	Yes
Facial massage (hypoallergenic oil, 5 min)	Yes	Yes	Yes
Therapeutic ultrasound (1 MHz, 0.5 W/cm², pulsed 1:2, 5 min, alternate days)	Yes	Yes	Yes
Low-level laser therapy (980 nm, 100 Hz, 5 J/point along the facial nerve, alternate days)	Not used	Yes	Yes
Extracorporeal shockwave therapy (1 bar, 500 shocks, anterior to the ear, every third day)	Not used	Yes, 1.3–1.5 Hz	Yes, 1.5–2 Hz
Facial taping / splinting	Yes	Yes	Yes
Eye care (drops by day; ointment and taping at night if closure incomplete)	As needed	As needed	As needed
Facial surface electrical stimulation	Not used	Not used	Not used
Re-assessment	ENoG, HB, SBFGS at Day 21 and Day 42	Same	Same

The DI severity bands were defined a priori using the mean baseline composite DI and were used to allocate physical agents according to the predefined protocol. Centre-based rehabilitation sessions were delivered six days per week, with home exercises on the seventh day, and continued until clinical recovery (House–Brackmann grade I). Formal rehabilitation commenced only after baseline ENoG assessment and determination of the DI. Laser parameters were identical in the moderate and severe DI bands, whereas shockwave frequency was higher in the severe band. The DI cut-offs were operational thresholds for this study and have not been validated for rehabilitation treatment selection.

Table 3: Degeneration index (%) by facial muscle across the study period

Muscle (branch)	Baseline, mean ± SD	Day 21, mean ± SD	Day 42, mean ± SD	x^2 (df=2)	P
Frontalis (temporal)	41.6 ± 26.7	26.8 ± 21.0	7.1 ± 7.0	142	<0.001
Orbicularis oculi (zygomatic)	35.0 ± 21.7	21.6 ± 18.3	5.1 ± 5.9	146	<0.001
Nasalis (buccal/zygomatic)	42.6 ± 22.5	26.0 ± 19.5	8.0 ± 8.3	144	<0.001
Orbicularis oris (buccal)	43.4 ± 22.4	26.2 ± 19.6	6.7 ± 6.2	153	<0.001
Mentalis (marginal mandibular)	43.5 ± 22.8	27.2 ± 18.8	7.6 ± 6.5	155	<0.001
Composite (mean of five)	41.2 ± 17.1	25.6 ± 14.8	6.9 ± 4.6	169	<0.001

DI = [100 − (amplitude affected side / amplitude unaffected side) × 100]. Friedman test across the three time points; all pairwise (Durbin–Conover) comparisons p<0.001. n=91 with complete data.

Table 4: House-Brackmann grade and Sunnybrook Facial Grading System score across the study period

Measure	Baseline, mean ± SD	Day 21, mean ± SD	Day 42, mean ± SD	x^2 (df=2)	P
House–Brackmann grade	3.27 ± 0.91	1.98 ± 0.58	1.11 ± 0.31	166	<0.001
Sunnybrook composite score	47.9 ± 21.6	79.4 ± 13.6	94.9 ± 4.1	179	<0.001

Friedman test; all pairwise (Durbin–Conover) comparisons p<0.001.

Table 5: Clinical severity and recovery by predefined baseline degeneration-index band (n = 91)

Variable	Mild (<25%) n=18	Moderate (25%-50%) n=40	Severe (>50%) n=32	P
Composite DI, baseline	19.4 ± 5.4	36.0 ± 5.6	60.2 ± 10.3	<0.001
HB grade, baseline	2.78 ± 0.81	3.17 ± 0.92	3.69 ± 0.78	0.001
SBFGS, baseline	59.4 ± 21.5	49.4 ± 22.0	39.5 ± 18.0	0.004
HB grade, Day 21	1.78 ± 0.55	1.95 ± 0.59	2.12 ± 0.55	0.11
SBFGS, Day 21	85.4 ± 11.3	79.6 ± 13.4	75.9 ± 14.3	0.009
HB grade, Day 42	1.06 ± 0.24	1.10 ± 0.30	1.16 ± 0.37	0.52
SBFGS, Day 42	97.3 ± 3.5	95.0 ± 3.8	93.2 ± 4.1	0.003
Composite DI resolved by Day 42, %	80.5 ± 20.3	83.3 ± 7.9	83.9 ± 7.0	0.87
HB grade I by Day 42, n (%)	17 (94.4)	37 (90.2)	27 (84.4)	0.52
Rehabilitation sessions, mean ± SD (median)	22.0 ± 7.8 (23)	29.7 ± 9.1 (29)	32.4 ± 10.6 (32)	0.002

DI = degeneration index; HB = House–Brackmann; SBFGS = Sunnybrook Facial Grading System. Kruskal–Wallis test, except HB grade I attainment (chi-square test).

DISCUSSION

n this cohort of 90 patients with idiopathic facial palsy (IFP), electroneurography (ENoG) performed before formal rehabilitation provided electrophysiological information complementary to clinical examination. Patients with relatively similar mean House-Brackmann (HB) grades showed variation in the degree of facial nerve degeneration, highlighting the potential value of the degeneration index (DI) in characterising electrophysiological involvement beyond clinical grading alone. Baseline DI was associated with subsequent recovery outcomes. Patients with greater initial degeneration had lower Sunnybrook Facial Grading System (SBFGS) scores at Days 21 and 42. Although the proportion achieving HB grade I by Day 42 showed a numerical decline with increasing DI severity, the between-group difference was not statistically significant. Patients in the severe DI stratum also required more rehabilitation sessions than those in the mild stratum, with an approximate difference of 10 sessions. Nevertheless, recovery was favourable across all three DI strata, with 81 of 90 patients (90.0%) achieving HB grade I by Day 42.

Using the DI to Plan Rehabilitation

The association between baseline degeneration index (DI) and the number of rehabilitation sessions required has practical implications for clinical planning. In this cohort, patients in

the severe DI stratum required more sessions than those in the mild stratum, with an approximate difference of 10 sessions. A baseline DI assessment may therefore help clinicians anticipate rehabilitation requirements, counsel patients and families about the potential duration of treatment, plan clinical resources, and identify patients who may require longer follow-up. However, these findings should be interpreted cautiously, as the study does not establish that DI predicts rehabilitation duration independently of baseline clinical severity or other prognostic factors. Furthermore, the number of sessions attended reflects both clinical recovery and the clinician's decision to discontinue treatment, making it an indirect measure of recovery time. Prospective studies recording time from rehabilitation initiation to achievement of House–Brackmann grade I would provide a more robust assessment of the prognostic utility of DI for estimating recovery duration.

Why Measure Degeneration before Rehabilitation

Our findings support the potential role of electroneurography (ENoG) as a complementary tool for prognostic assessment in idiopathic facial palsy. Previous studies by Fisch and Gantz and colleagues have identified severe ENoG degeneration, particularly values of 90% or greater during the early phase of facial palsy, as an indicator of increased risk of poor recovery.[14,16] Subsequent studies have reported associations between the extent of early electrophysiological degeneration and the speed and completeness of recovery.[17–19,21,22] Prakash and Raymond reported that nerve conduction measures could provide prognostic information beyond selected demographic and clinical factors, while Devriese and colleagues highlighted the importance of the early phase of the disorder in determining subsequent nerve recovery.[20,33] Much of this literature has focused on prognosis and the identification of patients who may require surgical consideration.

The present study explores a complementary application: obtaining the DI before formal rehabilitation to support rehabilitation planning. In our cohort, baseline DI was associated with initial clinical severity and subsequent facial function, while patients in the severe DI stratum required more rehabilitation sessions than those in the mild stratum. These findings suggest that electrophysiological assessment may provide information relevant to counselling and planning that is not fully captured by clinical grading alone.

The relationship between DI and the underlying pattern of nerve injury nevertheless requires careful interpretation. A relatively low DI in a patient with substantial facial weakness may be compatible with a greater contribution from conduction block, whereas a high DI indicates a more pronounced reduction in compound muscle action potential amplitude and may reflect greater axonal involvement. DI alone, however, cannot definitively distinguish conduction block from axonal degeneration or establish the degree of nerve injury. Accordingly, expectations about recovery and decisions regarding exercise progression should be based on the combined clinical and electrophysiological assessment rather than DI in isolation.

Rehabilitation should continue to emphasise controlled, selective facial movement and appropriate neuromuscular re-education, with avoidance of excessive effort that reinforces unwanted mass movement. The decision not to use facial electrical stimulation in this cohort was part of the study protocol and should not, on the basis of these data alone, be interpreted as proof of its ineffectiveness or as a universally applicable recommendation. Similarly, monitoring for synkinesis remains clinically relevant during

follow-up beyond the acute recovery period, as this outcome may not be adequately captured by the 42-day observation window.[11,26,27]

Taken together, these findings support considering pre-rehabilitation DI as an adjunct to clinical assessment for characterising electrophysiological involvement and anticipating rehabilitation needs. Further prospective studies are required to determine whether DI adds independent prognostic value beyond established clinical predictors and whether its use to guide rehabilitation decisions improves patient outcomes.

What the DI can and cannot Show

The degeneration index (DI), derived from electroneurography (ENoG), quantifies the reduction in compound muscle action potential (CMAP) amplitude on the affected side relative to the unaffected side. It provides an electrophysiological estimate of the extent of facial nerve dysfunction but does not directly quantify the number of degenerated motor axons. Although a low DI may be compatible with predominantly conduction block and a high DI may indicate greater axonal involvement, DI alone cannot reliably distinguish neurapraxia from axonal injury or determine the Sunderland grade. It also does not localise the lesion or distinguish between Sunderland grades II–V. Because the interpretation of ENoG depends on Wallerian degeneration developing distal to the lesion, measurements obtained too early after onset may underestimate the extent of degeneration. ENoG is therefore generally performed after approximately 72 hours, with timing considered in relation to symptom onset.[12–14]

The reliability of DI also depends on measurement technique. Surface CMAP amplitudes may vary with electrode placement, stimulation site and intensity, and other recording conditions. In this study, both sides were assessed during the same session using matched electrode placement to improve comparability. Nevertheless, these precautions cannot eliminate all sources of measurement variability.

ENoG was selected because it provides a quantitative, side-to-side assessment of facial nerve function during the acute phase. However, it should not be considered a replacement for other electrodiagnostic investigations. Needle electromyography (EMG) may provide information about denervation and subsequent reinnervation, particularly after sufficient time has elapsed for these changes to become detectable. The blink reflex can provide complementary information about the integrity of the trigeminal–facial reflex pathways and may assist in lesion localisation. These investigations address different aspects of facial nerve dysfunction and may be complementary to ENoG, depending on the clinical question and timing of assessment.

Accordingly, DI is best interpreted as one component of a broader clinical and electrophysiological assessment. Its value lies in quantifying side-to-side CMAP amplitude reduction and potentially contributing to prognostic assessment, rather than independently establishing the anatomical site, mechanism or histopathological grade of nerve injury.

Relation to Clinical Practice Guidelines

Current clinical practice guidelines recommend early corticosteroid treatment for eligible patients with Bell’s palsy and generally advise against routine electrodiagnostic testing or

imaging in patients with incomplete paralysis.[2,9] Recommendations concerning ENoG primarily address prognostic assessment and surgical decision-making in patients with complete paralysis, rather than its potential role in rehabilitation planning. In this context, pre-rehabilitation ENoG may be considered a complementary assessment within guideline-based medical care, rather than a substitute for established treatment recommendations.

Whether ENoG-derived DI provides additional clinically meaningful information in patients with incomplete paralysis remains an important question. Although most patients with incomplete palsy have favourable outcomes, clinical grading may not fully characterise the extent of electrophysiological involvement. The present study raises the possibility that DI could contribute to rehabilitation planning in this population; however, its incremental prognostic value and effect on clinical outcomes cannot be established from the current observational design.

In this cohort, approximately 60% of patients initiated corticosteroid treatment more than 72 hours after symptom onset. This pattern likely reflects delayed presentation or referral in the study setting. Because corticosteroids are recommended early in the course of Bell's palsy, delayed initiation may have reduced their potential benefit in some patients. However, the present study cannot determine the extent to which treatment timing influenced recovery or whether it modified the association between baseline DI and subsequent outcomes. These findings highlight the importance of timely presentation and treatment, while also emphasising the need to evaluate DI alongside relevant clinical and treatment-related factors.

Rehabilitation and Recovery Time

n this cohort, 90.0% of patients (81/90) achieved House–Brackmann (HB) grade I by Day 42 after the baseline assessment. For comparison, Peitersen reported complete recovery in approximately 71% of untreated patients, often over several months,[4] while Sullivan et al. reported recovery in 83% of patients treated with prednisolone at three months.[7] These figures are not directly comparable because the populations, treatment regimens, outcome definitions and follow-up periods differed. Our cohort included patients across a range of initial clinical severity and electrophysiological degeneration; all received corticosteroids, with approximately 60% starting treatment more than 72 hours after symptom onset. Furthermore, the absence of a control group prevents attribution of the observed improvement to rehabilitation.

Spontaneous recovery, corticosteroid effects, regression to the mean and repeated assessment may each have contributed to the changes observed over six weeks. Although the recovery trajectory is compatible with the hypothesis that adjunctive rehabilitation guided by DI may influence the course of recovery, this study cannot establish whether such an approach shortens recovery time. Some reviews have suggested potential benefits of facial exercise therapy,[24,25] but the independent contribution of rehabilitation and the additional value of DI-guided treatment require further investigation.

Similarly, the comparable proportional reduction in DI across the three severity strata should not be interpreted as evidence that DI-based treatment tailoring eliminated differences in recovery. Because rehabilitation interventions were allocated according to DI severity and there was no comparison group receiving a uniform rehabilitation protocol, the

observed pattern may reflect the natural course of recovery, treatment effects, or a combination of these factors. Controlled prospective studies comparing DI-guided and standard rehabilitation, with time to complete facial recovery as a prespecified outcome, are needed to determine whether DI-guided rehabilitation improves recovery or reduces treatment duration.

Interpreting DI Recovery

n the severe DI stratum, the mean DI decreased from approximately 60% at baseline to around 10% by Day 42. This change should be interpreted cautiously, as the magnitude and time course of CMAP amplitude recovery cannot be assumed to represent regeneration of previously degenerated axons. The observed reduction may reflect resolution of a reversible component of conduction block, collateral sprouting by surviving axons, changes in neuromuscular transmission or recording conditions, or test–retest variability. The relative contribution of these mechanisms cannot be determined from the present data.

Accordingly, a reduction in DI should be interpreted primarily as recovery of the recorded CMAP amplitude relative to the unaffected side, rather than as direct evidence of axonal regrowth. Serial ENoG measurements provide useful information about changes in electrophysiological function, but they cannot independently establish the underlying mechanism of recovery.

Limitations

This single-centre observational study lacked a control group; therefore, improvements cannot be attributed specifically to rehabilitation or DI-based treatment tailoring. The DI severity cut-offs remain unvalidated, and treatment differences between groups prevent separation of prognostic and treatment effects. All patients received corticosteroids, but approximately 60% initiated treatment after 72 hours; steroid timing was not adjusted for in the analysis. Two patients outside the prespecified age range were excluded, leaving 90 participants. ENoG timing varied after symptom onset, potentially influencing DI measurements. Follow-up ended at Day 42, precluding assessment of later outcomes such as synkinesis and quality of life. Finally, the single-centre setting and multimodal rehabilitation programme limit generalisability and prevent evaluation of individual treatment components.

Future Work

Future studies should compare guideline-based medical therapy alone, medical therapy plus uniform rehabilitation, and medical therapy plus DI-guided rehabilitation, preferably through a randomised controlled trial or, alternatively, a target-trial emulation with appropriate confounder adjustment.[3,4] Time to complete facial recovery should be the primary outcome, with blinded assessment, documentation of corticosteroid timing, and follow-up of at least six months to assess synkinesis. Prespecified DI cut-offs should also be validated against long-term clinical outcomes.

CONCLUSION

n patients with idiopathic facial palsy, the degeneration index (DI) derived from pre-rehabilitation electroneurography provided quantitative information complementary to clinical grading. Baseline DI was associated with facial function at Day 42 and the number of rehabilitation sessions required. These findings suggest that pre-rehabilitation ENoG may support prognostic counselling and rehabilitation planning alongside guideline-based medical treatment. Although recovery was favourable across the cohort, the study cannot establish whether rehabilitation accelerates recovery or whether DI-guided tailoring is superior to uniform rehabilitation. These questions require evaluation in controlled prospective trials.

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Conflicts of Interest

The rehabilitation protocol used in this study (Darsh-Gunjan Bell's Palsy Treatment Protocol) is registered with the Government of India Copyright Office (L-147405/2024) in the names of the corresponding author and her spouse. There are no other conflicts of interest.

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