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Is Electroneurography Beneficial in the Management of Bell's Palsy?

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BACKGROUND

The management of acute peripheral facial nerve palsy is complex, challenging, and controversial. This article focuses on the management of acute, unilateral, idiopathic facial nerve palsy, more commonly known as Bell's palsy. The annual incidence of Bell's palsy is 20 to 30 per 100,000 population, and facial weakness generally resolves in 6 months either with medical treatment or by observation alone.[1,2] However, a small subset (10%–29%) of affected individuals display persistent facial nerve dysfunction. These patients can suffer from corneal abrasions, dysarthria, facial contracture, synkinesis, and the social-psychological challenges of facial asymmetry.[1,2]

The cause of Bell's palsy is by definition uncertain, but evidence implicates facial nerve edema due to viral infection.[1,2] Swelling within the narrow bony confines of the fallopian canal leads to damage, with the meatal foramen and labyrinthine segments common sites of conduction block in cases taken to surgery.[1,3] The actual extent of nerve damage varies significantly, with some patients showing only mild weakness and others permanently disfigured. Ideal assessment of Bell's palsy patients would allow the clinician to definitively identify patients who will not regain full facial nerve function; unfortunately, such a test does not exist. A variety of useful assessments do exist however, including electrodiagnostic and function-based (House-Brackmann [HB] and Yanagihara grading systems) evaluations. This article discusses the prognostic implications of electroneurography (ENoG), a tool that provides reliable objective measurements of facial nerve function in acute facial nerve palsy.

LITERATURE REVIEW

Ugo Fisch and Erlo Esslen helped develop and popularized the use of ENoG. This technology measures the surface amplitude of facial muscle compound action potentials

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(CAP), evoked by a maximal transcutaneous stimulation of the common trunk of the nerve, providing objective indirect measurements of the conducting ability of the facial nerve (Fig. 1, published online as supporting information). Comparison of the CAP amplitudes between the two sides provides an estimate of the extent of axonal degeneration.[1,3,4] For example, if a recording from the affected side reveals amplitudes only 5% that of the unaffected site, the patient is considered to have 95% degeneration. Conversely, recordings taken in a normal patient would show nearly symmetric CAP amplitudes.

Early publications by Fisch address the use of ENoG in Bell's palsy patients. He readily admits the test's utility is not universal. Testing is restricted to the acute phase of Bell's palsy, as Fisch found the correlation between denervated facial muscles and degenerated motor nerve fibers most accurate within the first 2 to 3 weeks after onset. Training in test administration (e.g., optimized electrode placement, premeasurement evoked potentials) is also essential in decreasing intertest variability. Additionally, ENoG cannot differentiate between degrees of nerve damage (axonotmesis, endoneurotmesis, neurotmesis), and as a comparative modality is only useful in cases of unilateral paralysis.[3] Even with these limitations, Fisch's work showed ENoG has excellent prognostic qualities concerning Bell's palsy outcomes. In a review of his and other's observations, Fisch found patients with ≤90% degeneration at 3 weeks had an 80% to 100% chance of good recovery. Conversely, those with a maximal degeneration of ≥95% had a 60% to 70% chance of persistent facial dysfunction. Fisch further used ENoG scores to justify facial nerve decompressive surgery in patients with ≥90% degeneration within the first 3 weeks after onset.[3]

Sillman et al. published a key retrospective review of ENoG based on the experience at the University of Michigan.[4] This review of all facial paralysis patients who had undergone ENoG testing included 91 Bell's palsy patients (67% of all facial nerve palsies). They found that in facial paralysis patients not treated by surgery, those whose ENoG amplitudes did not degenerate beyond 90% regained normal (HB grade I or II) function regardless of etiology. Patients with ≥90% within the first 2 weeks after onset were offered surgical decompression, provided no voluntary motor potentials were recorded with subsequent standard monopolar electromyography (EMG) (Supporting Information Fig. 1). In these patients, surgical intervention was beneficial in restoring facial function (44% vs. 66% return to HB I or II).[4]

Gantz et al. provided further evidence of ENoG's utility as a prognostic tool in 1999, when they reported their experience in selecting candidates for surgical decompression of the facial nerve.[1] In this prospective, multi-institutional study of 169 Bell's palsy patients, those whose ENoG amplitudes did not exceed 90% degeneration within 14 days of onset all returned to normal or near-normal facial function (HB grade I or II). In patients with an ENoG response exceeding 90% degeneration, and showing no voluntary EMG motor unit potentials, surgical decompression of the meatal foramen, labyrinthine segment, and geniculate ganglion via the middle cranial fossa (MCF) approach was offered. Of those who underwent decompression within 14 days of onset in the Iowa patient subset, 91% had a good outcome (HB grade I or II) at 7 months ($P = .0003$). In those patients who chose medical therapy, treatment with high-dose steroids alone afforded a 42% chance of the same good outcome. Similar findings were reported by the Baylor and Michigan patient

sets as well. Statistically, this study provided a larger patient base to achieve a significantly improved statistical power (n = 169). In summary, Gantz's study confirmed Fisch's proposed 90% degeneration within 14 days as a cutoff for surgical decompression. The study also further confirmed that lesser degrees of nerve damage will likely resolve to satisfactory facial function without surgery.[1]

Recent work by Takemoto et al. compared ENoG against the Yanagihara grading system, the most commonly used clinical scale to evaluate facial movements in Japan (similar to the HB scale).[5] The study included 142 Bell's palsy patients and 26 patients with Ramsay Hunt syndrome (facial weakness due to varicella-zoster virus). All patients were treated with steroid and antiviral medications and followed for at least 6 months or until complete recovery. ENoG testing was performed 7 to 10 days after onset of facial paralysis, with repeat tests if indicated. Statistical analysis validated ENoG as more sensitive in predicting long-term facial nerve disability than clinical exam (Yaganihara grading score) alone (P = .005247). Interestingly their analysis also suggested a cutoff point of ≤85% degeneration for return of satisfactory facial nerve function without surgery. Takemoto's work confirms ENoG as a useful prognostic tool and also raises the question of expanding the selection criteria for offering surgery for Bell's palsy patients to those with >85% degeneration.[5]

BEST PRACTICE

The course of Bell's palsy varies, with a minority of patients suffering significant residual facial weakness. Several studies confirm the prognostic value of ENoG testing performed between 3 and 14 days after onset of complete facial paralysis (Fig. 2, published online as supporting information). In those patients who do not exceed 90% degeneration, use of ENoG is useful as a prognostic tool to reassure patients of the high likelihood of recovery to acceptable (HB I or II) facial function. In patients with an ENoG response exceeding 90% degeneration, and who show no voluntary EMG motor unit potentials, surgical decompression by a qualified surgeon likely offers improved functional outcomes. One recent study suggests 85% degeneration is predictive of unfavorable outcomes; however, this remains to be validated by further studies.

LEVEL OF EVIDENCE

The best studies in ENoG use are well-designed case-control and cohort studies without randomization, level 2b. Concerning the use of ENoG for evaluation of facial nerve decompression, the ethical controversies of conducting sham surgeries and the associated morbidity of the MCF approach precludes randomized-controlled trials.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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BIBLIOGRAPHY

1. Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgical management of Bell's palsy. Laryngoscope 1999;109:1177–1188. [PubMed: 10443817]
2. Peitersen E Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl 2002;(549): 4–30. [PubMed: 12482166]
3. Fisch U Prognostic value of electrical tests in acute facial paralysis. Am J Otol 1984;5:494–498. [PubMed: 6393772]
4. Sillman JS, Niparko JK, Lee SS, Kileny PR. Prognostic value of evoked and standard electromyography in acute facial paralysis. Otolaryngol Head Neck Surg 1992;107:377–381. [PubMed: 1408222]
5. Takemoto N, Horii A, Sakata Y, Inohara H. Prognostic factors of peripheral facial palsy: multivariate analysis followed by receiver operating characteristic and Kaplan-Meier analyses. Otol Neurotol 2011;32:1031–1036. [PubMed: 21725266]