

# The Spectrum of Electrophysiological Abnormalities in Bell's Palsy

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**ABSTRACT: Background:** As part of an investigation of a suspected "outbreak" of Bell's palsy in the Greater Toronto Area, a population-based sample of patients with Bell's palsy was investigated electrophysiologically to help understand the spectrum of abnormalities that can be seen in this setting. **Methods:** Two hundred and twenty-four patients were surveyed, of whom 91 underwent formal neurological assessment. Of the latter, 44 were studied electrophysiologically using standard techniques. Thirty-two of the 44 patients fulfilled clinical criteria for Bell's palsy. **Results:** A wide range of electrophysiological changes was observed. Blink responses were the most useful test showing diagnostic sensitivity of 81% and specificity of 94% compared to the contralateral control side. Needle electromyography was additionally helpful in only one patient of six with normal conduction studies. **Conclusions:** There is a wide spectrum of electrophysiological abnormalities in Bell's palsy. Blink reflex latencies may be under-utilized in the assessment of the facial nerve in Bell's palsy. Facial EMG is not generally useful in routine assessment.

**RÉSUMÉ: Anomalies électrophysiologiques dans la paralysie de Bell. Introduction:** Dans le cadre d'une investigation motivée par ce qui semblait être une éclosion de cas de paralysie de Bell dans la région de Toronto, un échantillon de patients atteints de la paralysie de Bell, tirés de la population de Toronto, a subi une investigation électrophysiologique afin d'aider à comprendre la gamme des anomalies qui peuvent être observées dans ce contexte. **Méthodes:** Deux cent vingt-quatre patients ont été dénombrés et 91 d'entre eux ont subi une évaluation neurologique. Parmi ceux-ci, 44 ont subi une évaluation électrophysiologique selon les techniques standards. Trente-deux des 44 patients satisfaisaient aux critères cliniques de la paralysie de Bell. **Résultats:** Une grande variété de changements électrophysiologiques a été observée. La réaction de clignement était le test le plus utile avec une sensibilité diagnostique de 81% et une spécificité de 94% en prenant le côté opposé comme contrôle. L'électromyographie à l'aiguille a été utile chez seulement un patient sur six dont les études de conduction étaient normales. **Conclusions:** Il existe une vaste gamme d'anomalies électrophysiologiques dans la paralysie de Bell. Les temps de latence du réflexe de clignement à la menace sont peut-être sous-utilisés dans l'évaluation du nerf facial dans la paralysie de Bell. En général, l'ÉMG facial n'est pas utile dans l'évaluation de routine de ces cas.

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Facial nerve electrophysiology in Bell's palsy has typically been used to try to predict facial nerve recovery. In the past, results were used to decide who might be a candidate for controversial and now out-of-vogue procedures such as intracranial decompression.<sup>1-5</sup> The natural history of Bell's palsy is that 50-60% of patients will recover in three weeks<sup>1</sup> and 80-90% have normal or near-normal recovery by six months.<sup>6</sup> If the maximal deficit is paresis rather than palsy, nearly all (>95%) patients will achieve a full recovery three to eight weeks after onset. A minority of patients (4-14%) will have residual facial nerve dysfunction.<sup>1,6-8</sup> Knowledge of the spectrum of electrophysiological findings in Bell's palsy is necessary to understand the possible use of these procedures for determining prognosis and in research trials. This subject was studied in detail in the pioneering era of electrophysiology, but there is no recent available data in which modern techniques are employed

and where the patient population is likely to present the full spectrum of pathology seen in Bell's palsy.

In the fall of 1997, a population-based investigation was conducted into an apparent "outbreak" of Bell's palsy in the Greater Toronto Area (GTA). The study was broad in scope including all patients with suspected Bell's palsy. As part of that

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study, a sub-group of patients underwent clinical examination and neurophysiological assessment by two neurologists. During the study, we were able to assess the spectrum of nerve conduction study abnormalities in clinically definite cases of Bell's palsy. The purpose of this report is to describe the spectrum of electrodiagnostic findings in a population-based sample of patients with Bell's palsy.

## METHODS

Since Bell's palsy is not a reportable disease, all physicians in the GTA were informed about our investigation by fax. Flyers were posted in Emergency rooms and distributed with laboratory test results from Gamma-North Peel, MDS, and Dynacare laboratories. Local public health officials published advisories regarding an unusual clustering of Bell's palsy. Advertisements were placed in all of the major English-language newspapers in the GTA. A toll-free 24-hour telephone hotline for health professionals and/or study subjects to call was established and the study received media coverage on radio and television.

The study was approved by the Human Subjects Review Committee of the University of Toronto and all patients provided informed consent. Individuals from the main survey were asked if they would voluntarily participate in detailed neurophysiological testing. Ninety-one patients consented and had a complete neurological history and physical examination performed by one of three neurologists (MDH, GM, or WCG). Bell's palsy was diagnosed if the history and physical examination and neuroimaging were compatible with idiopathic unilateral facial nerve palsy. Specific attention was placed on searching for other confounding diagnoses but patients were not excluded from a diagnosis of Bell's palsy based on electrophysiological results.

Electrophysiological assessment of the facial nerve consisted of bilateral facial nerve conduction studies using nickel electrodes stimulating at the stylomastoid foramen and recording over the ipsilateral nasalis, bilateral blink responses and monopolar needle electromyography (EMG) of the ipsilateral orbicularis oris, orbicularis oculi and frontalis. Where indicated, contralateral EMG was performed. Because of the nature of the study, patients underwent neurophysiological assessment at a variety of times after onset of their Bell's palsy.

Normal values for our laboratory are derived from the Mayo Clinic, Rochester and were as follows: facial nerve maximal distal latency (dlat) = 4.2 msec and minimum compound motor action potential (CMAP) amplitude = 1.0 mV. Blink response maximum ipsilateral R1 = 13 msec; maximal delta R1 = 1.2 msec; maximal ipsilateral R2 = 41 msec; maximal ipsilateral delta R2 = 5 msec; maximal contralateral R2 = 44 msec; and maximal contralateral delta R2 = 7 msec.

Comparisons of proportions were made using Fisher's exact test. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated using the contralateral side for comparison.

## RESULTS

The annualized incidence of Bell's palsy in the GTA in the study was 15.5 per 100 000. Two hundred and twenty-four patients were identified in the survey. Ninety-one underwent prospective neurological examination of whom 76% had

confirmed Bell's palsy. Of these 91 patients, a total of 44 patients volunteered for and underwent prospective electrophysiological examination; patients without a clinically confirmed diagnosis of Bell's palsy were included in the initial cohort of 44 studied electrophysiologically. Twenty-two were female and the median age was 45 years (range 18-84). These 44 patients were examined and studied electrically for a median of 47 days after symptom onset (range 11-126 days). Thirty-two of the 44 patients fulfilled clinical criteria for Bell's palsy; these 32 patients make up the cohort for analysis. The remaining 12 all had facial nerve lesions: four were diabetic, two had Ramsay-Hunt syndrome (herpes zoster), two had a brain stem stroke, two had cancer (testicular lymphoma, metastatic prostate carcinoma), one had suspected multiple sclerosis, and one had suspected chronic inflammatory demyelinating polyneuropathy. Patients were not followed long-term and therefore final diagnoses on the latter two patients were unavailable.

Eight patients had normal physical examinations such that our diagnosis was historical; patients had initially had Bell's palsy diagnosed by another physician. There were no significant demographic (age, gender) or symptomatic differences between the 44 patients studied electrophysiologically and the entire cohort of 224 patients ( $p > 0.05$ ). Similarly, there were no significant demographic (age, gender) differences among patients studied electrophysiologically who did or did not have a final diagnosis of Bell's palsy.

Among 32 patients with confirmed Bell's palsy, 27 (84%) patients had at least one neurophysiological abnormality. The five patients with normal studies underwent their neurophysiological testing between 42 and 64 days after symptom onset; this was not statistically different than the cohort with neurophysiological changes ( $p > 0.05$ ). Four of these five patients had normal clinical examinations. Among eight patients with normal physical examinations, four had neurophysiological abnormalities.

**Table 1:** Spectrum of electrophysiological change in Bell's palsy (n=32)

NCS (n=32)	N(%)	Median (range)
Prolonged facial dlat(msec)	9 (28.1)	6.9 (4.4-6.9)
Reduced ipsilateral facial CMAP(mV)	18 (56.3)	0.2 (0-0.9)
Prolonged ipsilateral R1(msec)	16 (50.0)	16.1 (13.2 – 49.6)
Prolonged ipsilateral R2(msec)	9 (28.1)	45.2 (41.2 – 82.6)
Prolonged contralateral R2(msec)	3 (9.4)	49.1 (45.6 – 84.6)
<b>Needle EMG (n=31)</b>		
EMG: normal	6 (19.4)	
EMG: fibrillation potentials in any ipsilateral muscle	20 (64.5)	
EMG: increased insertional activity only	1 (3.2)	
EMG: large MUPs in any ipsilateral muscle	11 (35.5)*	

\*7 patients had fibrillation potentials as well

CMAP = compound motor action potential; dlat = distal latency

**Table 2:** Sensitivity, specificity, positive predictive value and negative predictive value of nerve conduction studies in facial neuropathy due to Bell's palsy (n=32)<sup>†</sup>

	Ampl	dlat	Ipsi R1	ΔR1	Any ncs	Any blinks
SENS	0.56	0.28	0.50	0.59	0.56	0.81
SPEC	0.94	1.00	0.97	0.94	0.94	0.94
PPV	0.90	1.00	0.94	0.91	0.90	0.93
NPV	0.68	0.58	0.66	0.70	0.68	0.83

SENS = sensitivity; SPEC = specificity; PPV = positive predictive value; NPV = negative predictive value; dlat = distal latency; ipsi R1 = ipsilateral R1 latency; R1 = difference between ipsilateral and contralateral R1 latency; any ncs = nerve conduction study abnormality; any blinks = any blink reflex abnormality (R1, ipsilateral R1, contralateral R2, delta R1 or delta ipsilateral R2 or delta contralateral R2)  
<sup>†</sup>Note that the calculations are based on a comparison to the normal side so that the total number of compared studies is 64

**Table 3:** Blink Reflex Patterns in Bell's Palsy (n=32)

Test Results	n (%)
Prolonged ipsilateral R1 plus <sup>†</sup>	11 (34.4)
Normal	6 (18.8)
Prolonged ipsilateral R1, ipsilateral R2 and contralateral R2 (CLASSIC pattern)	5 (15.6)
Increased delta R1 only	6 (18.8)
Increased delta contralateral R2 only	3 (9.4)
Increased delta R1, delta ipsilateral R2 only	1 (3.1)

<sup>†</sup>"plus" implies other criteria for pathological responses were also met (eg. delta R1 > 1.2 msec)

One of 32 patients with confirmed Bell's palsy refused needle EMG examination. Fibrillation potentials were observed more commonly (in eight of eight patients) when the EMG was done within the first 21 days after symptom onset ( $p = 0.023$ ). The earliest a patient underwent needle examination was 11 days from symptom onset (median 43.5 days; range 11-126 days). Overall, 64.5% of patients showed fibrillation potentials. A third (35%) of patients had large motor unit potentials (MUPs) consistent with re-innervation. There was no statistical evidence of increased likelihood of observing large motor unit potentials between groups studied within 21 or after 21 days from symptom onset. Electrophysiological synkinesis was observed in two patients with clinically severe facial neuropathies; both were examined more than 100 days from symptom onset. Needle EMG showed abnormality in only one of six patients who had normal conduction studies (Table 1).

Two patients showed an electrophysiologically pure demyelinating type of lesion, defined as facial motor distal latency prolongation or blink reflex latency prolongation with

normal facial-to-nasalis CMAP and no fibrillation potentials or increased insertional activity on needle EMG. These two patients were examined 27 and 32 days from symptom onset; one had very mild weakness of the ipsilateral frontalis but not the lower face and the other had a normal clinical examination. An additional patient, with an atypical progression of facial palsy of several weeks associated with testicular lymphoma, showed an electrophysiologically pure demyelinating type of lesion. Despite intensive investigation meningeal lymphomatosis was not proven.

The patterns of nerve conduction study abnormalities are shown (Tables 1, 2). Among 32 patients, facial nerve conduction studies showed a high specificity and hence good positive predictive value but less useful negative predictive value. Blink responses were more useful showing both high specificity and sensitivity (Table 3).

## DISCUSSION

The unique feature of this study is the population-based nature of the sampling frame, which permitted us to observe the spectrum of electrophysiological findings among patients with Bell's palsy. Although there is potential for volunteer bias, there was no evidence that the group studied electrophysiologically was significantly different from the non-electrophysiologically studied group.

A majority of patients (84%) showed neurophysiological abnormalities. In the group with normal studies (16%), one would expect that these patients had very mild pathology such as focal demyelination and were therefore able to mount a rapid remyelinating response. Overall 56% (n=18) of patients showed a reduction in CMAP (median 0.2 mV) and 25% (n=8) showed a 90% or greater reduction in CMAP compared to the contralateral side. Similarly a wide range of delayed latencies was observed in facial nerve conduction studies and blink responses. Among blink responses, the classic pattern of a partial facial nerve lesion (prolonged R1, ipsilateral R2 with ipsilateral stimulation and prolonged contralateral R2 with contralateral stimulation) was seen only 15.6% of the time, while a prolonged ipsilateral R1 plus other changes was the most common. In contradistinction to previous literature, no patient had blink reflex evidence of trigeminal nerve dysfunction.<sup>9</sup> A significant proportion of abnormal tests (31%) were only classified as abnormal when compared to the unaffected side, emphasizing the importance of using latency difference in determining pathology.

Blink reflex studies were the most useful test, showing better sensitivity and specificity than other components of the neurophysiological assessment. This is similar to Hanner et al,<sup>9</sup> who reported that the efferent arc of the blink response was abnormal in 96% of patients with Bell's palsy tested within five weeks of disease onset. The most specific test was the facial nerve distal latency, but as reflected in the very low sensitivity, this was presumably because the study detects only the most severe lesions. In order to produce a prolonged motor distal latency the lesion (which is proximal to the stimulation site) would have to cause severe axonal loss and/or secondary demyelination.

The utility of blink reflexes in predicting outcome was studied by Kimura et al.<sup>10</sup> He studied 81 patients with Bell's

palsy, a majority of whom had their first study within 14 days of onset. They noted that there was a progressive loss of the ipsilateral R1 response in the first week after symptom onset; thereafter, the reflex returned in a majority of patients. The presence of the R1 after the first week was suggested to be a good prognostic sign.

Needle examination was additionally useful in only one patient. However, fibrillation potentials were observed in 65% of patients, which is comparable to previous reports.<sup>5</sup> Large motor unit potentials were not seen more often among patients studied late in the course of illness even among those in whom fibrillation potentials were initially seen (ie. with axonal injury). This is counter to accepted understanding of the time course of re-innervation. However, less than one third of patients were examined within 21 days of onset and none before 11 days. The relatively small sample size may have prevented the detection of a statistical difference.

This study has limitations insofar as there was an unavoidable selection bias (volunteer bias) and there was a wide range of times after symptom onset that patients were examined and studied. Our results may not apply to those patients studied within the first 10 days. However, the experience gained while participating in this population-based study of Bell's palsy has taught us that blink reflex latencies have been under-emphasized as a tool for detection of mild facial nerve abnormalities in patients with suspected Bell's palsy. The sensitivity of more standard techniques (motor conduction study, needle exam) is limited. Neurophysiologists who study such patients, either for clinical diagnosis and management, or as part of research protocols may benefit from our observations.

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#### REFERENCES

1. Olsen PZ. Prediction of recovery in Bell's palsy. *Acta Neurol Scand* 1975;52(suppl 61):1-120.
2. Danielides V, Skevas A, Van Cauwenberge P. A comparison of electroneurography with facial nerve latency testing for prognostic accuracy in patients with Bell's palsy. *Eur Arch Otorhinolaryngol* 1996;253:35-38.
3. Sinha PK, Keith RW, Pensak ML. Predictability of recovery from Bell's palsy using evoked electromyography. *Am J Otol* 1994;15:769-771.
4. Bouhanna S, Hagen R, Chabert R, Roumieux JL, Lallemand JG. Valeur pronostique de l'électroneurographie et du réflexe trigémino-facial dans la paralysie faciale a frigore. *Rev Laryngol Otol Rhinol* 1996;117:353-356.
5. Heath JP, Cull RE, Smith M, Murray JAM. The neurophysiological investigation of Bell's palsy and the predictive value of the blink reflex. *Clin Otolaryngol* 1988;13:85-92.
6. Peitersen E. The natural history of Bell's palsy. *Am J Otol* 1982;4:107-111.
7. May M, Klein SR, Taylor FH. Idiopathic (Bell's) facial palsy: natural history defies steroid or surgical treatment. *Laryngoscope* 1985;95:406-409.
8. Katusic SK, Beard M, Wiederholt WC, Bergstralh EJ, Kurland LT. Incidence, clinical features and prognosis in Bell's palsy, Rochester, Minnesota, 1968-1982. *Ann Neurol* 1986;6:22-627.
9. Hanner P, Badr G, Rosenhall U, Edstrom S. Trigeminal dysfunction in patients with Bell's palsy. *Acta Otolaryngol* 1986;101:224-230.
10. Kimura J, Giron LT, Young SM. Electrophysiological study of Bell's palsy. *Arch Otolaryngol* 1976;102:140-144.