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Brief Communication

Segmental Reflex, Long Latency Reflex, and Mixed Nerve Silent Period in Dystonia

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ABSTRACT: We hypothesized that "long latency reflexes" (LLRs), associated segmental reflex (SR), and mixed nerve silent periods (MnSPs) recorded on the distal upper extremity muscles would behave differently in patients with cervical dystonia and focal hand dystonia. We enrolled patients with cervical dystonia, generalized dystonia, focal hand dystonia, and healthy individuals. We recorded SR, LLRs, and MnSPs. The mean amplitude of SR on the affected side of focal hand dystonia was significantly lower ($p = 0.010$). The parameters related to LLRs and MnSPs were not different between groups. We suggest, using SR, LLRs, and MnSPs, we could not show an electrophysiological signature specific to dystonia.

RÉSUMÉ : La dystonie ET les réflexes segmentaires, les réflexes de longue latence et les périodes de silence. L'hypothèse de départ était que les réflexes de longue latence (RLL), les réflexes segmentaires (RS) associés et les périodes de silence des nerfs mixtes (PSNM), enregistrés sur les muscles distaux des membres supérieurs réagissaient de manière différente dans la dystonie cervicale et dans la dystonie focale de la main. Des patients souffrant de dystonie cervicale, de dystonie généralisée ou de dystonie focale de la main ainsi que des témoins en bonne santé ont participé à l'étude, puis il y a eu enregistrement des RS, des RLL et des PSNM. L'amplitude moyenne des RS du côté affecté chez les sujets atteints de dystonie focale de la main était significativement plus faible ($p = 0{,}010$) que chez les autres sujets. Par contre, les paramètres relatifs aux RLL et aux PSNM ne différaient pas d'un groupe ou à l'autre. Aussi sommes-nous d'avis qu'il n'était pas possible démontrer l'existence d'une signature électrophysiologique spécifique de la dystonie, fondée sur les ST, les RLL et les PSNM.

Keywords: Dystonia; focal hand dystonia; segmental reflex; long latency reflex; mixed nerve silent period

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Dystonia is an involuntary movement disorder characterized by abnormal sustained or intermittent muscle contractions.[1,2] It is characterized by prolonged muscle contraction, co-contraction, and overflow. For a long time, dystonia was considered a psychiatric disorder. However, the evidence in the last thirty years indicates that dystonia most likely results from the dysfunction of a network, including basal ganglia, cerebellum, thalamus, and sensorimotor cortex.[3,4] In the pathophysiology of dystonia, a defect in the striato-pallido-thalamic pathway causing disinhibition of excitatory thalamocortical neurons[5] and sensory dysfunction, decreased inhibition, and abnormal plasticity[6] have been suggested. While these findings are shared by focal dystonia and generalized dystonia, what is the reason for the variations in clinical phenomenology? Latorre *et al.*[7] indicated that the various forms of dystonia (e.g., focal, generalized, or task-specific) probably reflect derangements at different levels of the network and may have an electrophysiological signature. For example, Sabbahi *et al.*, using soleus H-reflex measures, identified neurophysiologic differences between generalized dystonia, cervical dystonia, and normal subjects.[8] Or, the recovery cycle of the blink reflex assessing brainstem excitability was abnormal due to reduced inhibition of the R2 component, mainly in patients with blepharospasm[9,10] and also in patients with segmental/generalized dystonia or torticollis, but not in those with focal arm dystonia, suggesting proximity is critical in dysfunction and one plausible explanation may be that each dystonia subtype might have an electrophysiological signature.

In this study, we hypothesized that "long latency reflexes" (LLRs), associated segmental reflex (SR), and mixed nerve silent periods (MnSPs) recorded on the distal upper extremity muscles may represent one of the electrophysiological signatures of the focal hand dystonia. Stimulation of a mixed nerve while recording on a distal hand muscle during slight contraction creates an M-response due to direct excitation of the motor axons, an SR (probably an H-reflex) mediated through Ia afferents, and up to three subsequent LLRs, I, II, and III.[11] One of the most critical applications of testing LLRs is diagnosing and classifying myoclonus.[12] In dystonia, one study reported high-amplitude LLR I and reduced or absent LLR II.[13] The MnSPs are electrophysiological inhibition periods mediated by descending volleys and activated by high-threshold cutaneous fibers of the mixed nerve.[14,15]

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Table 1: Demographic findings of patients and healthy subjects

	CD ($n = 10$)	GD ($n = 12$)	FHD ($n = 4$)	Control group ($n = 38$)	p
Gender, F/M	3/7	8/4	1/3	17/21	0.281
Age, years, (mean ± SD)	46.7 ± 8.8	36.7 ± 12.6	24.5 ± 10.4	30.1 ± 8.8	<0.001

LLR = long latency reflex; MnSP = mixed nerve silent period; SR = segmental reflex.

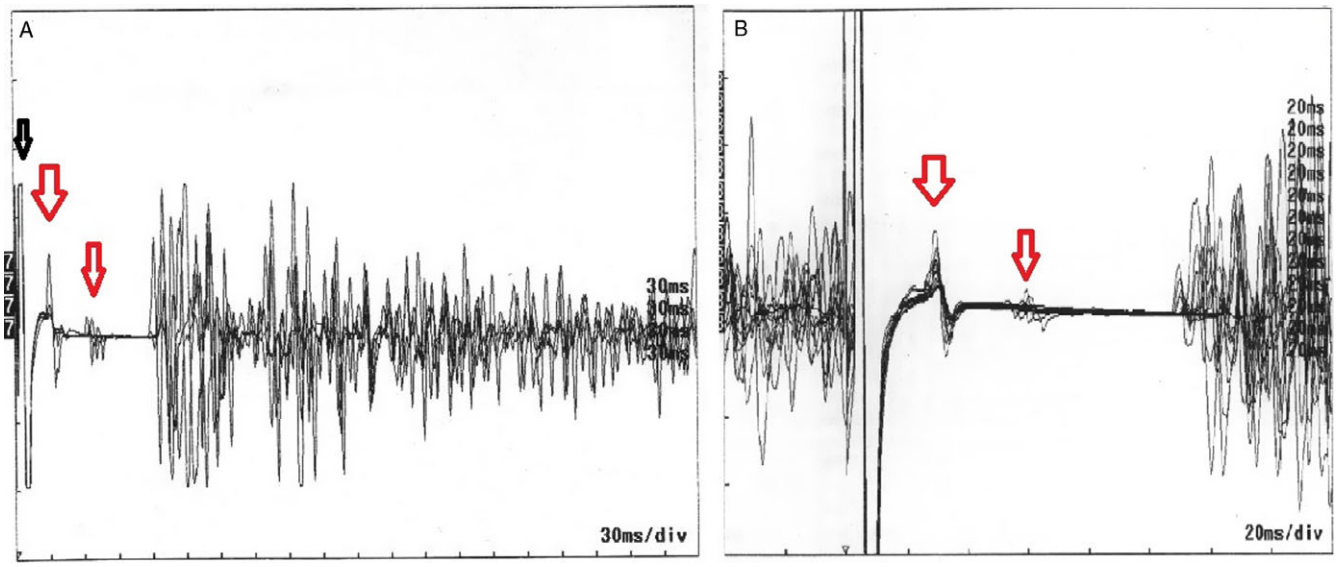

Figure 1: ***a***. M-response (black arrow), segmental reflex (broad red arrow), and long latency reflex (thin red arrow) in a 26-year-old male patient with cervical dystonia. 1***b***. Segmental reflex (broad red arrow) and long latency reflex (thin red arrow) in a 34-year-old female patient with generalized dystonia (gain 0.5 mV/div).

This was a cross-sectional study. We included ten patients with cervical dystonia, 12 with generalized dystonia, and four with focal hand dystonia who were admitted to our movement disorder outpatient clinic between January 2018 and January 2020. A control group of 38 healthy volunteers was also recruited. The gender and age of all participants were noted (Table 1). There was a significant age difference between groups due to the natural history of patients with focal hand dystonia and cervical dystonia. Patients with cervical dystonia were evaluated using the Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS), and those with generalized dystonia or focal dystonia were assessed using the Burke-Fahn-Marsden dystonia rating scale. The institutional review board approved the study. We received informed consent to perform the analysis.

We recorded LLRs, associated SR, and MnSPs in all participants. All studies were performed with a Neuropack Sigma MEB-5504k (Nihon Kohden Medical, Tokyo, Japan). All patients in the study had botulinum toxin treatment previously. However, we performed the electrophysiological investigations at least six months after the last botulinum toxin injections. The recordings were done according to the previously published methods.[11,16] All measurements were performed for the symptomatic extremity in the focal hand dystonia group. The recordings were done on the right side for other patient groups and healthy subjects. We used a square wave electrical stimulus at 0.2 ms duration at 2 Hz. We have collected 20 trials for each analysis.

1. ***Long latency reflexes and segmental reflexes:*** We placed surface silver-silver chloride recording electrodes on the belly of the abductor pollicis brevis (APB) muscle. We stimulated the median nerve at the wrist. The SR and LLRs (LLRI, LLRII, LLRIII) were recorded at rest while the subjects performed a mild contraction of the APB muscle (approximately 25% of the maximum). Both auditory and visual feedback maintained the level of contraction. Figure 1 shows representative examples of SR and LLRs.
2. ***Mixed nerve silent period:*** The MnSPs were measured while the recording electrodes were still on the APB muscle. The median nerve was stimulated with an electrical stimulus 25% above the supramaximal stimulus intensity threshold, producing a motor response at the wrist level while the subjects were performing a mild contraction of the APB muscle.

The onset latency of LLRs (distance from electrical stimulus artifact until the first negative deflection after segmental reflex) and the amplitude LLRs (peak-to-peak) were measured. The LLRs were classified into three groups based on Deuschl 1999:[12] 35-46 ms for LLR I, 45-58 ms for LLR II, and > 68 ms for LLR III.

The minimum latency and the maximum amplitude of the segmental reflex were also measured. We calculated the percentage of presence of each wave as follows: the number of participants with LLRx100/ the total number of participants in the specific group. For MnSPs, the mean end latency was measured. Data were presented as mean ± standard deviation (SD) or percentages (n). We used multivariate analysis. Fixed factors were phenotypes (cervical dystonia, generalized dystonia, focal hand dystonia, healthy subjects) and age (<20 years, 20–40 years, >40 years) because there was a significant age difference between groups. Post

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Table 2: Electrophysiological findings of patients and healthy subjects

	Cervical dystonia ($n = 10$)	Generalized dystonia ($n = 12$)	Focal hand dystonia ($n = 4$)	Control group ($n = 38$)	p
SR amplitude, μV (mean ± SD)	861.5±594.5	718.1±410.5	206.6±11.5	1133.5±812.0	0.045
LLR I n, (%)	2(80)	2(16)	0(0)	4(10.5)	0.703
LLR II n, (%)	8(80)	8(66)	2(50)	31(81)	0.419
LLR III n, (%)	3(30)	2(16)	1(25)	10(26)	0.893
LLR II amplitude, μV (mean ± SD)	1178.5±652.1	960.0±710.6	633.3±562.0	1026.0±615.1	0.484
MnSP end latency, ms(mean ± SD)	115.6±15.9	112.3±22.1	109.3±8.5	108.4±14.3	0.961

LLR = long latency reflex; MnSP = mixed nerve silent period; SR = segmental reflex.

hoc analysis was done using the Bonferroni test. The chi-square test has been used for the comparison of qualitative data. The data analysis was done using the SPSS 20.0 statistical package, and a p-value ≤ 0.05 was considered significant.

The mean TWSTRS scores of cervical dystonia patients were 21.0 ± 5.5 points. The neck and upper extremities were involved in seven patients with generalized dystonia. In other patients, axial muscles and lower extremities were also involved. The mean Burke-Fahn-Marsden score was 46.7 ± 9.1 points. The patients with focal hand dystonia did not have only task-specific symptoms and had symptoms provoked by many actions. The signs were mild in patients with focal hand dystonia.

The latencies of the SRs were similar between the diagnostic groups ($p = 0.099$). There was a significant difference in amplitudes of the SRs according to phenotypes ($F = 2.248$, $p = 0.045$, Table 2); however, not according to the age groups ($F = 1.181$, $p = 0.324$). Post hoc analysis showed that the mean amplitude of segmental reflexes was significantly lower on the symptomatic side of the focal dystonia group than the healthy subjects ($p = 0.010$). In the other groups, it was similar.

None of the participants in any of the groups had C reflex, i.e., LLR response at rest. Regarding the responses obtained during contraction, LLR II (69.2%) was commonly seen, followed by LLR III (23.1%) and LLR I (15.4%) among all patients with dystonia. The figures were very similar to those among healthy subjects. Comparing the whole group of dystonia patients with healthy subjects, the LLR amplitudes were not statistically different ($p = 0.831$).

The LLR I (during muscle activity) was obtained in two (20%) patients with cervical dystonia and two (16%) patients with generalized dystonia. None of the patients from the focal extremity dystonia group had an LLR I response, whereas there was an LLR I response in four (10.5%) of 38 healthy individuals ($p = 0.703$). The LLR II was obtained in eight (80%) patients with cervical dystonia, eight patients with generalized dystonia (66%), and two (50%) patients with focal hand dystonia, and there was LLR II response in 31(81%) healthy individuals ($p = 0.419$). The LLR III response was obtained from three out of 10 (30%) patients with cervical dystonia, two (16%) patients with generalized dystonia, one (25%) patient with focal extremity dystonia, and 10 (26%) healthy individuals ($p = 0.893$). The amplitude of LLRs was not different among patients with different phenotypes ($F = 0.352$, $p = 0.788$) or according to other age groups ($F = 0.833$, $p = 0.484$). Table 2 shows all the electrophysiological findings. The mean end latencies of MnSPs were not different among groups ($F = 0.097$, $p = 0.961$).

The significant finding in this study was the smaller SR amplitude in the group with focal hand dystonia and no change in LLR or SR in generalized dystonia.

Several physiological conditions modulate the SR. The H-reflex changes under certain pathological conditions. For example, the soleus H-reflex was suppressed throughout all phases of the contralateral rhythmic ankle movement.[17] Again, the amplitude of the H-reflex of the flexor carpi radialis muscle increases in association with teeth clenching even before the onset of the EMG activity of the masseter muscle.[18] Soleus H-reflex is inhibited during gait in Parkinson's disease,[19] whereas the amplitudes of H-reflexes are increased in spasticity.[20] The excitability of H-reflex depends on the excitability of the lower and upper motor neuron pools, and basal ganglia, cerebellum, or sleep states modulate it.[21,22] In our study, the amplitude of the SRs was low only in the group with focal hand dystonia, suggesting reduced excitability in this motoneuron pool. Regarding H-reflex, there was no change in its amplitude or H/M ratio in a group of patients with cervical or generalized dystonia in a previous study.[23] If the SRs are representative of H-reflex, to see no change in patients with cervical dystonia or generalized dystonia is compatible with the previous reports. Although the studies on this subject are limited if the cause of dystonia is related to pathophysiology, we would anticipate an increase in the excitability of the motoneuron pool compared to what we found in our study. Furthermore, SR is facilitated by muscle contraction, and we have recorded the reflex at the same limb that had dystonia, in other words, contractions. However, recordings did not coincide with the contractions, and reduced amplitudes may be related to the reduction of excitability after the powerful contractions. In a previous study, the shortest F-wave latency, the mean F-wave latency, and F-wave persistence of untreated muscles were measured before, one week, and five weeks after the treatment with botulinum toxin in patients with spasmodic torticollis and writer's cramp.[24] The latencies were slightly prolonged one week after the treatment and returned to baseline five weeks later. The F-wave persistence was reduced one week after the treatment. The authors concluded that although there was a decreased excitability of alpha-motoneurons supplying non-treated muscles, it was transient. We also performed recordings in a relatively remote, toxin-naive muscle six months after the botulinum toxin injections. However, patients in this study received the treatment more than once. In focal hand dystonia, muscles located in the forearm are injected. Usually, it should not affect remote sites. Distal hand muscles may be affected by diffusion. Therefore, botulinum toxin could have led to, more or less, permanent excitability changes. Thus, hypoexcitability may result from phasic relation to powerful contractions or botulinum toxin injections. It is still interesting that there were no changes in other groups. Classically, other groups received higher doses of botulinum toxin, and some cases of generalized dystonia were also injected toxin in the upper extremities. At this point, we should acknowledge the

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limitation that there was a small number of patients in the focal dystonia group.

The LLRs were used to understand the underlying mechanism of movement disorders such as Parkinson's disease, essential tremor, myoclonus, or dystonia.[25] Naumann and Reiners showed alterations in LLRI response in patients with cervical dystonia and upper limb dystonia.[13] Although increased amplitudes in LLRI responses were on the affected side, they showed that bilateral abnormality of LLRI response could be present. LLR II, occurring at approximately 50 ms, was obtained bilaterally in all controls but was reduced or absent in some patients, mainly on the clinically affected side. Therefore, there was a differential involvement in LLR I and II in this study. Another important finding in this study was more minor LLR II amplitudes after botulinum toxin injections. However, we were unable to replicate their results. There were two differences between this study and our study. First, these authors used a different statistical analysis than ours. They classified the LLR responses as normal or abnormal. For LLR I, they considered it abnormal when the response did not occur or when there was an increase in the amplitude. Second, they analyzed idiopathic dystonia patients without subgrouping. We also added an analysis comparing the entire group with healthy subjects and found no difference. LLRs were also recorded in patients with DYT11 myoclonus-dystonia syndrome and were normal.[26]

Electrical stimulation of peripheral nerves may elicit different kinds of reflex responses. The cutaneous silent period (CSP) is a robust and reproducible nociceptive electromyographic suppression mediated at the spinal level by small-diameter A-delta afferents.[27] A recent review nicely presented all studies regarding CSP in focal dystonia, functional dystonia, and change of CSP after treatment with botulinum toxin or pallidal stimulation.[28] The CSP in abductor pollicis brevis following D2 stimulation on the affected and the contralateral side showed delayed end latencies in patients with various forms of focal dystonia CSP.[29,30] Notably, there were similar abnormalities in both organic and psychogenic dystonia.[30] Interestingly, the CSPs remained constant over one year in two dystonia patients, before and during relief from a sensory trick in one patient, and before and after botulinum toxin treatment in the same patient.[29] In patients treated with bilateral pallidal stimulation, there was a trend towards shorter CSP duration due to later CSP onset, which did not change by switching the neurostimulator on.[23] The MnSP is made up of three different parts, including the collision of antidromic with orthodromic motor impulses, Renshaw cell inhibition activated by an antidromic motor volley, and activation of high-threshold cutaneous fibers within the mixed nerve.[17,18] The last half of the MnSP corresponds entirely to cutaneous afferent impulses, which, in isolation, produce a complete silent period between 70 and 120 ms after digital stimulation in contrast to the first half, where there are effects of voluntary and descending volleys as well as Renshaw cell inhibition.[18] However, the presence of Renshaw cell inhibition in the distal hand muscles is still controversial.[31] This is why we used the end latency, which represents the clear part of the silent period. The studies of MnSPs in dystonia are more limited compared to CSP. The MnSPs were abnormal in paroxysmal kinesigenic dyskinesia during the attack, in whom CSPs were normal.[32] We find MnSPs in dystonia comparable to healthy individuals.

Besides the abovementioned limitations, there were certain other limitations of the study. The patients were not toxin-naïve, and we could not appoint the muscles and doses injected. There were significant age differences observed within the four groups. Additionally, it should be noted that the number of subjects was not evenly distributed among these groups. We acknowledge that these are the main limitations of our study. However, the age difference and uneven distribution of numbers of subjects across groups originate from the natural history of these disorders. We conducted the statistical analysis according to different age groups. Naturally, isolated hand tremor is less frequent than cervical dystonia. Additionally, we excluded patients with secondary causes, spasticity or tremor, which is another reason for reducing numbers.

In conclusion, we analyzed different levels of the nervous system using electrophysiology in various dystonia phenotypes and determined only low-amplitude segmental reflex in focal hand dystonia. Considering the small number of patients in the focal hand dystonia group, we suggest that there was reduced spinal excitability in this group, which may be related to botulinum toxin treatment. We could not show significant changes in the LLRs or MnSPs in any dystonia. Therefore, using SRs, LLRs, and MnSPs, we could not offer an electrophysiological signature specific to dystonia.

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