Available online at www.sciencedirect.com

ScienceDirect

journal homepage: http://www.elsevier.com/locate/pjnns

Diagnostic value of blink reflex in multisystem atrophy, progressive supranuclear palsy and Parkinson disease

Elzbieta Szmidt-Salkowska [a], *Malgorzata Gawel* [a,*], *Zygmunt Jamrozik* [a], *Joanna Salkowska-Wanat* [b], *Damian Gawel* [c], *Anna Kaminska* [a]

[a] *Department of Neurology, Medical University of Warsaw, Warsaw, Poland*
[b] *Department of Dermatology and Venereology, Medical University of Warsaw, Warsaw, Poland*
[c] *Centre of Postgraduate Medical Education, Department of Biochemistry and Molecular Biology, Warsaw, Poland*

ARTICLE INFO

Article history:
Received 15 March 2016
Received in revised form
18 May 2016
Accepted 14 June 2016
Available online 27 June 2016

Keywords:
Blink reflex
Multisystem atrophy
Progressive supranuclear palsy
Parkinson disease

ABSTRACT

Abnormal blink reflex (BR) is a result of reticular brainstem pathways dysfunction and seems to be one of the features of brain degenerative disorders.

The aim of the study was to estimate the diagnostic value of blink reflex in neurodegenerative diseases such as: multisystem atrophy (MSA), progressive supranuclear palsy (PSP) and Parkinson disease (PD).

Material consisted of 99 patients with clinically probable MSA (51), PSP (28) and PD (20). MSA patients were divided into two subgroups, with dominant cerebellar (MSA-C) and parkinsonian signs (MSA-P). The mean age of patients was 64.9 years (47–79 years); males – 55.3%.

Blink reflex was obtained in a typical way.

Results: The significant differences in mean values of blink reflex latencies between PD and other subgroups (MSA-P, MSA-C, PSP) were found, but all of them were in normal range. In individual patients with PD and PSP (50% and 18%, respectively) delayed R2 latencies were recorded.

Conclusions: The most frequently abnormal blink reflexes, comparing the MSA, PSP and PD groups, were present in PD patients. We postulate that this may be explained by pathological influence of nigrostriatal pathway on the circuit linking the basal ganglia, cerebellum and brainstem.

1. Introduction

Multisystem atrophy (MSA) and Parkinson's disease (PD) belong to a group of α-synucleinopathies with different localizations and pattern of synuclein pathology [1]. Progressive supranuclear palsy (PSP) is a rare taupathy which shares many clinical symptoms with MSA and PD. All of them are characterized by dopamine deficiency in subcortical nuclei, mainly in the substantia nigra and in the brainstem.

* *Corresponding author at*: Department of Neurology, Medical University of Warsaw, Banacha 1A, 02-097 Warsaw, Poland. Tel.: +48 225992857; fax: +48 225991857.
E-mail addresses: ela.szmidt@wp.pl (E. Szmidt-Salkowska), mgawel@wum.edu.pl (M. Gawel), zygmunt.jamrozik@wum.edu.pl (Z. Jamrozik), Polandgawel@cmkp.edu.pl (D. Gawel).
http://dx.doi.org/10.1016/j.pjnns.2016.06.001

Multisystem atrophy is a neurodegenerative disease and is characterized by different clinical symptoms due to autonomic, parkinsonian, pyramidal and cerebellar signs. Based on predominant clinical symptoms (*e.g.* cerebellar or parkinsonian), MSA is subdivided into a parkinsonian (MSA-P) and a cerebellar (MSA-C) type [2–5]. Cellular pathology in MSA was revealed mainly in upper motor neurons, in basal ganglia and sporadically in lower motor neurons [6–8].

Progressive supranuclear palsy (PSP) is characterized by progressive degeneration of basal ganglia, brainstem, cortex, facial and trigeminal nuclei, which has been documented in neuropathological examinations, as well as in magnetic resonance imaging and positron emission tomography studies [9–15].

The level and intensity of cortex, cerebellar and/or brainstem dysfunctions in MSA, PSP and in PD could be estimated using neuroimaging methods and electrophysiological test as blink reflex (BR).

The main external control of blink reflex consists of a dopaminergic system and olivo-cerebellar circuit.

Unconditioned stimulus pathway of the eye-blink includes inferior olive and climbing fibers of the cerebellar nuclei and cortex, while conditioned stimulus consists of pontine sensory nuclei, cerebellar interpositius nucleus, superior cerebellar peduncle and magnocellular red nucleus [16–18].

Basal ganglia could modulate the brainstem reflex blink circuits *via* descending cortical projections or alternatively, with input to the superior colliculus *via* tecto reticular projections [19,20].

The spinal trigeminal nucleus consists of three subnuclei and ascending/descending pathways probably exist between these subnuclei. Han et al. showed that excitatory and inhibitory synaptic responses are mediated by AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid), NMDA (N-methyl-D-aspartate) and by GABA (gamma-aminobutyric acid) (receptors with a differential contribution to the synaptic responses between subnuclei) [21].

Neurotransmission disturbed in MSA, PSP and PD and neural pathways underlying brainstem and spinal reflexes may influence blink reflex in different way.

The reduced blinking in Parkinson's disease and excessive blinking after overstimulation by dopaminergic agents associated with dyskinesia suggests involvement of efferents of nigrostriatal system to superior colliculus and brainstem [22]. Other structures involved in delayed components of blink reflex may be brainstem, cerebellum and olivo-cerebellar circuit. As shown previously, the brainstem is involved in many extremely complicated (*e.g.* vocal control or auditory startle reaction) [23–25]. In PSP the voluntary blinking is bradykinetic with an increased duration of opening phase without abnormalities reversed by correction of dopaminergic deficit [26].

The lesions of supratentorial could reveal prolonged, contralateral R2 latencies as a result of inhibitory inputs of pyramidal tracts to the contralateral reticular formation (afferent branch abnormality) [27].

Blink reflex is a polysynaptic reflex between the trigeminal and the facial system [28–31].

The afferent arc of BR consists of trigeminal nerve sensory fibers. The efferent arc of BR conveys the impulses through the motor fibers of the facial nerves. Blink reflex involves an early R1 response which terminates in midpoints. The two late bilateral R2 responses mediated by the spinal nucleus and trigeminal nerve tracts reflect the connection of trigeminal and facial systems in the lower medullary region by polysynaptic bilateral medullary pathways. They are terminated in the facial nuclei [32–37] (Fig. 1).

Likewise in Bentivollo's study, no differences in BR values between age and sex were observed [38].

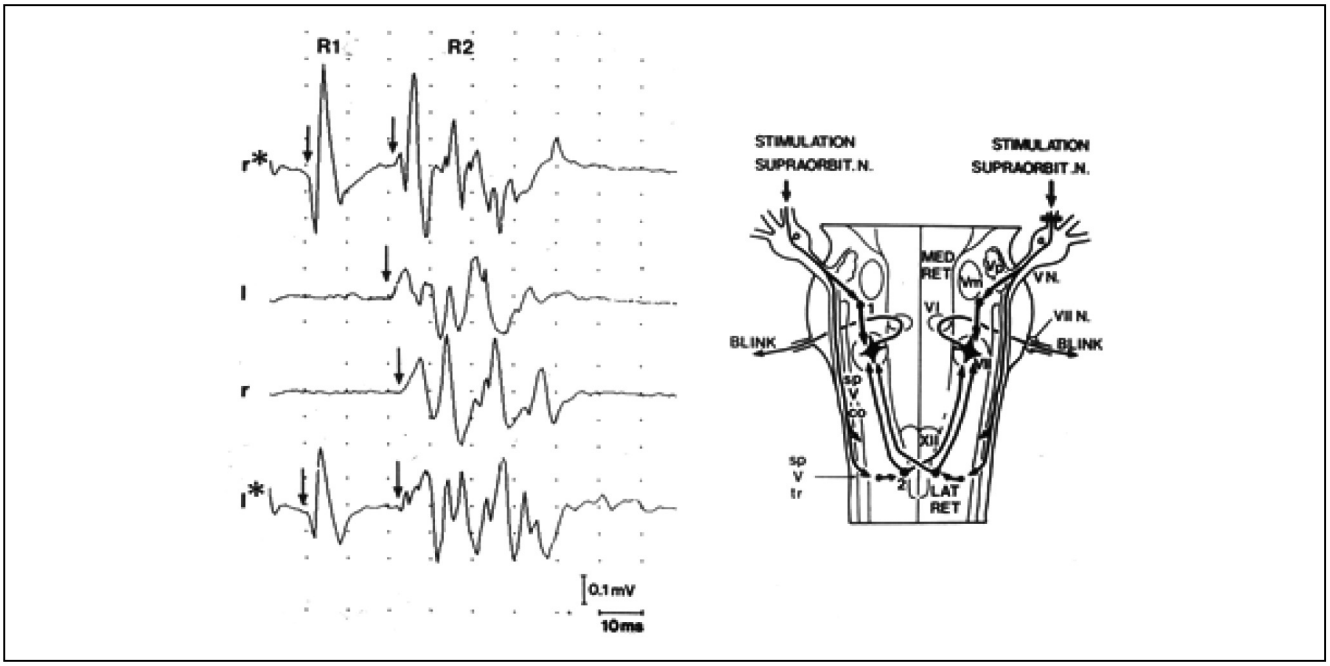

Fig. 1 – Pathways of blink reflex [39]. R1-R1 (early) response of blink reflex. R2-R2 (late) response of blink reflex.