5

Blink Reflex Alterations in Various Polyneuropathies

Figen Guney

Department of Neurology, Selçuk University, Meram Medical Faculty Konya,
Turkey

1. Introduction

An eyelid closure in response to some stimulus is a blink reflex (BR), which is normally isolated. In humans and primates, the closing is bilateral while in other animals, mostly those with eyes set laterally, the closure is frequently unilateral. In clinical practice, a BR is characteristically provoked by light corneal or eyelash touching or glabellar tapping. The British physician Overrend first elicited the blink reflex by tapping one side of the forehead. Kugelberg analysed the blink reflex electromyographically by electrically stimulating the supraorbital nerve. Since the original description of the blink reflex 100 years ago the study of this reflex has given understanding the central and peripheral mechanisms of the trigeminofacial pathways in normal and different disorder. Stimulation of the supraorbital nerve, a distal branch of the ophtalmic division of the trigeminal nerve , is the common technique in clinical neurophysiology to obtain a BR (Figure 1). Characteristically, an electrical stimulus on the supraorbital nerve induces two recordable responses in the orbicularis oculi muscles: an early one R1, ipsilateral to the stimulated side, and a later one, R2, which is bilaterally expressed. R2 response ipsilateral to the stimulus is frequently cited as R2i, and the R2c is the one obtained on the contralateral side. R1 component of BR has a rather stable latency. R2 typically shows relative variable latencies and larger magnitudes than R1 and its threshold is lower. R2 component is responsible for the eyelid closure of the blink. It has been observed that common motor unit potentials contribute to the buildup of both responses, that is, the same orbicularis oculi motor unit is depolarized after the stimulation at times corresponding to the latencies of R1 and R2 responses, the latter presumably in a repetitive reverberating pattern. Both response are cutaneous and nociceptive in origin. The classical findings, indicative of such a lesion are an afferent defect with prolonged latencies of R1, ipsilateral R2 and contralateral R2. The efferent type pattern occurs also with intraaxial lateral pontine lesions involving the pons at the trigeminal entrance zone. In facial nerve lesions there is a delay in the reflex latency only on the affected side, regardless of the side of stimulation. Such abnormalities were found in Bell's palsy or other lesions of the facial nerve.

2. Discussion

Generalized polyneuropathy may induce bilateral abnormalities of the trigeminal reflex. Blink reflex alterations were first described in large series of patients with polyneuropathy

in 1982 by Kimura. He analyzed the blink reflex obtained from the patients with Guillain-Barre syndrome (GBS), chronic inflammatory polyneuropathy (CIPN), Fisher syndrome (FS), hereditary motor and sensory neuropathy (HMSN) Types I and II, diabetic polyneuropathy. The patients with diabetic polyneuropathy were selected as having maturity-onset diabetes with diffuse symmetrical neuropathy. None had renal complications, mononeuropathy, distinctly asymmetrical features, dysautonomia, or predominantly proximal weakness. In GBS, CIPN, and HMSN Type I, R1 responses were either absent or delayed in a majority of patients. The incidence of abnormality was considerably less in DPN. In FS, the direct and R1 responses were normal in all except for 1 patient who had peripheral palsy associated with delayed R1 on the affected side. In HMSN Type II, the studies were normal in all except for 1 patient who had an absent R1 on one side. The average latencies of the much lesser degree in DPN, and were normal in FS and HMSN Type II. The latency ratio of R1 to the direct response showed a mild increase in GBS, a moderate decrease in CIPN and HMSN Type I and a mild decrease in DPN. In GBS, the latency ratio of R1 to direct response is increased slightly, indicating more proximal involvement of the facial nerve, provided, trigeminal nerve is relatively intact. This ratio is decreased significantly in CIPN and DPN, as migh be expected from a distally prominent involvement in a chronic neuropathic process. Although latencies of R2 were commonly within the normal range when analysed individually, the average value was greater significantly in the neuropathies than in the controls. In normal subjects, R2 begins clearly after R1. This distinction became unclear in some patients with demyelinating neuropathy, in which R1 was temporally dispersed. However, the ipsilateral and contralateral R2 were of nearly identical latency in neuropathy. Thus, a simultaneously recorded contralateral R2 helped to identify that the apparent initial portion of the ipsilateral response contained a delayed R1 continuous with R2. Abnormalities of the blink reflex and direct response generally were well correlated with slowing of motor nerve conduction velocities. A marked delay in latency of R1 and the direct response offers conclusive evidence that the facial nerve is severely involved in some polyneuropathies. This is expected in GBS, which is associated characteristically with facial weakness, but not in HMSN Type I, in which weakness of the facial muscle, if any, is clinically very subtle. The blink reflex abnormalities in CIPN are similar to those in GBS, but, interestingly, studies are normal in FS unless associated with facial nerve palsy. Analogous to a bimodal distribution of motor nerve conduction velocities in HMSN, the latency of direct and R1 responses usually are prolonged in Type I and normal in Type II.

Blink reflex provides clinically useful information in the assessment of the cranial nerves in polyneuropathies. The blink reflex reflects the integrity of the afferent and efferent pathways, including the proximal segment of the facial nerve. Thus, the latency of R1 represents the conduction time along the trigeminal and facial nerves and pontine relay. R2 is less reliable for this purpose because of inherent latency variability from one trial to the next. Furthermore, the latency of R2 reflect excitability of interneurons and synaptic transmission in addition to axonal conduction.

Polo et al. found that in patients with polyneuritis cranialis which is Guillain Barre syndrome's rare varient both R1 and R2 components of blink reflex were absent. Five months later blink reflex could be elicited, the R1 latency was significantly increased.

Neau et al. have studied blink reflex in 50 patients with Guillain-Barre polyradiculitis. They detected bilateral lenghtening of the early and late reflex responses of the blink reflex with unilateral or bilateral increase of motor facial latency. The blink reflex showed various

abnormalities in slightly more than one third of the cases of polyradiculoneuritis presenting no clinical signs of facial involvement, thus constituting subclinical lesional evidence of the reflex arc.

Abnormalities of blink reflexes have been reported in acute inflammatory demyelinating polyneuropathy (AIDP). Ropper et al. examined blink reflex in AIDP within 21 days of symptom onset. In that study, it was documented that 46% of patients with AIDP exihibited abnormality of blink reflexes, 96% of these patients exhibited facial weakness. In addition, R1 onset latency correlated positively with prolonged median nerve distal motor latency. One study has examined the blink reflex abnormalities in AIDP within 10 days of symptom onset. Abnormalities of blink reflexes, absent or prolonged ipsilateral R1 and R2 responses and contralateral R2 responses, were noted in 16 of 31 patients (52%) tested. Abnormalities of R1 component (51%) were slightly more frequent than abnormalities of the R2 component (48%). In those patients with abnormalities of blink reflexes, facial weakness was evident in 11 (69%) patients. This would suggest that blink reflexes, might be abnormal in some AIDP patients with apparently normal facial strength, and hence should be performed systematically if AIDP was suspected. Furthermore, abnormalities of blink reflexes also correlated positively with prolonged mean summated distal motor latency in this study, suggesting that in early AIDP, distal demyelination occurs in parallel in many nerves. These abnormalities of blink reflexes most likely represent demyelination in either the facial and/or the trigeminal nerves, reflecting the multifocal nature of demyelination in AIDP.

Cruccu et al. made blink reflex study in 14 patients with chronic inflammatory demyelinating polyneuropathy (CIDP), in 23 patients with severe distal sensory or sensorymotor form of diabetic polyneuropathy, and in 12 patients with mild diabetic polyneuropathy. Recordings of the blink reflexes showed abnormal R1 blink reflex, and on 7/29 an abnormal R2. Recording of the blink reflexes showed on 11/29 sides an abnormal R1 blink reflex, and on 7/29 an abnormal R2 in patients with chronic inflammatory demyelinating polyneuropathy. The mean R1 and R2 latencies were slightly longer than control values. R1 blink reflex was abnormal on 8/43 sides and R2 on 3/43 in patients with severe diabetic polyneuropathy. On one side of 1 patient the R1 and R2 delays originated from the facial-nerve reflex efferents. They examined 20 sides in 12 patients with mild diabetic polyneuropathy. They detected only the latency of R1, which might reflect the sum of mild trigeminal and facial abnormalities, was slightly longer than control values. In patients with CIDP, the motor and sensory nerves were affected symmetrically. The long delays in the latency of R1 were probably due to demyelination in the facial, supraorbital nerves. Even the absent responses did not necessarily imply axonal loss. In diabetic patients, the abnormalities had a far more irregular distribution. The supraorbital R1 was affected unilaterally in several patients. The mean R1 latency was only slightly longer in the severe than in the mild neuropathy group. Individual patients had relatively small delays, compatible with slightly longer synaptic times due to the defect of spatial summation entailed by axonal loss.

Kokubun et al. examined 20 patients with CIDP using blink reflex. All patients had symptomatic motor and sensory neuropathies for more than 2 months, and demyelinating polyneuropathy was diagnosed by conventional nerve conduction studies. The latency of the R1 response was prolonged on the left side in 16 of 20 patients and on the right in 16 patients. Only 3 patients had a normal R1 response bilaterally. The ipsilateral R2 response was abnormal on the left in 8 patients and on the right in 7, but was normal bilaterally in 10

patients. The contralateral R2 response was prolonged on the left in 8 patients and on the right in 3, could not be evoked in 2 patients, and was normal bilaterally in 11. The latencies of the R1, and R2 responses in CIDP patients were significantly prolonged compared with those of normal subjects. The prevalence of subclinical facial neuropathy in their CIDP patients was high; an abnormal direct response was observed bilaterally in 10(50%) and unilaterally in 2 patients (10%). However, none of the patients showed unilateral trigeminal nerve involvement, based on the result of the blink reflex. The R1 and R2 latencies were prolonged and the responses were not evoked bilaterally in 4 patients, perhaps because of trigeminal nerve or brainstem involvement in addition to bilateral facial nerve involvement. Bilateral or unilateral prolongation of R1 latency alone was observed in 10 patients. This result may be due to some defect in the pons (5). Only one patient's R1 latency was within the normal range bilaterally; however R2 latency was markedly increased. This patient possibly had medullary involvement in addition to trigeminal or facial nerve involvement. Kokobun's study results suggested that involvement of trigeminal and facial nerves probably occurred early during the onset of CIDP because there was no correlation between clinical disability or disease duration, and direct or R1 response. The study of the blink reflex was used widely and it was an established test. Although their study had certain limitations, the direct response and the blink reflex are useful for functional evaluation of trigeminal and facial nerves in patients with CIDP, especially since the data has indicated a lack of correlation between clinical features and electrophysiological detections in CIDP patients.

Varela et al. reported an 83 year-old-woman with a remote history of localized melanoma and breast carcinoma (treated with resection without chemothreapy or radiation in the early 1980s), but no history of systemic medical disease such as diabetes, who presented with a 2-month history of progressive tongue and perioral numbness and difficulty sipping liquids, in 2009. She described a decrease in sensation and occasional "tingling" mostly involving the perioral region but occasionally throughout the face. She denied diplopia, ptosis, or other cranial nerve symptoms and had no limb weakness or sensory loss, apart from intermittent numbness in her hands that would awaken her intermittently at night. Neurologic examination demonstrated mild weakness (Medical research Council grade 4) of her orbicularis oculi, orbicularis oris, and frontalis and a mild decreased pinprick sensation in her feet bilaterally. Reflexes were present but mildly reduced diffusely. On routine nerve conduction studies, only median motor distal latencies were prolonged,& conduction velocities slowed in the forearm, but there was no abnormal temporal dispersion or conduction block. Median sensory responses were absent bilaterally. Blink reflex study demonstrated prolonged R1 and R2 latencies bilaterally. Cerebrospinal fluid demonstrated an elevated protein of 87 mg/dl. Patient was diagnosed as chronic inflammatory demyelination neuropathy, prednisone (60 mg daily) was initiated after intravenous immunoglobulin teratment. One month after initation of prednisone, her symptoms and examination improved, prolonged latencies were detected on blink reflex study leading to the diagnosis of demyelinating neuropathy. Therefore, blink reflex study is useful in helping to confirm selected cases of suspected CIDP.

Ogawara et al. reported a case of anti-GQ1b antibody syndrome. The patient had symptoms and signs suggesting both peripheral lesions i.e. asymmetric ophthalmoplegia, facial diplegia, upper limb weakness, areflexia and central lesions i.e. drowsiness, and hemisensory loss. The anti-GQ1b antibody titres were very high. Extensive

electrophysiologic studies supported the involvement of both peripheral and central nervous systems. The coexistence of central and peripheral components refutes the idea of simple relationship between Bickerstaff encephalitis and purely central involvement, or between Fisher syndrome and a simple peripheral neuropathy. Published reports have described cases of Fisher syndrome associated with evidence of brainstem or cerebellar lesions on MRI and indicated that central components were occasionally associated with Fisher syndrome. Magnetic resonance imaging of the brain T1- and T2- weighted images showed no abnormalities. R1 responses were small with normal latency (9.1 ms), in this patient's blink reflex study. The R2 responses were not elicited on either side. Absence of R2 despite detectable R1 after repeated trials raised the possibility of central abnormality. Therefore, blink reflex study is useful in differentiating the diagnosis from Bickerstaff encephalitis, too.

Urban et al. examined both electromyographical investigation and blink reflex study in 40 patients with diaetes mellitus. Compared with age related normal values, the patient group showed abnormal nerve conductions to the sural nerve (absent sensory nerve action potential in 33%, reduced amplitude in 17.9%, reduced sensory conduction velocity in 33.3%), the peroneal nerve (reduced motor conduction velocity in 28.5%, reduced amplitude in 26%), the median motor fibers (reduced motor conduction velocity in 7.7%) and the median sensory fibers (abolished the sensory nerve action potential in 5%, reduced sensory conduction velocity in 18%, reduced amplitude in 51%). Nerve conduction studies confirmed sensory or sensorimotor polyneuropathy in all 24 of the clinically affected patients. Urban et al. detected significantly prolonged R1 latency concluding that subclinical facial and trigeminal nerve involvement is not unusual in diabetes mellitus although it is significantly less frequent than the involvement of limb nerves. Nazliel et al. showed abnormal blink reflex responses in 55% of 20 diabetic patients with polyneuropathy. They detected prolonged R2i and R2c latencies but R1 values in diabetic patients with polyneuropathy did not differ sigificantly from those of normal controls. Guney et al. studied 95 diabetic patients and found bilateral significantly increased R1 and R2i and R2c latencies in diabetic patients with polyneuropathy (Figure 2). And as an interesting result, R1 values in diabetic patients without polyneuropathy did not differ significantly from normal controls. These results suggest R1 is mainly conducted by exteroceptive, medium thick myelinated A-beta fibres, whereas R2 is predominantly conveyed by the nociceptive, thin myelinated A-delta fibers. As a result of these studies, there is no surprise sparing of R1 in diabetic patients without clinical or subclinical neuropathy. The reflex arc of R1 has its central representation in main trigeminal nucleus of the pons, its circuit bases in an olygosynaptic organisation with three neurons, respectively on the Gasser's ganglion, the trigeminal principal nucleus and the facial nucleus, and at least one interneuron between two latter structures, as reported by Trontelj and Tamai et al. It is not completely known if these interneuron's location is on the multisynaptic way of R2 in man. For R2 response, the central way is multisynaptic. Holstege et al. postulated that blink premotor area located at the pontine and medullary tegmental fields projecting into the blink motoneuronal pool would probably be involved in R2 blink reflex component. Prolongation to Gasser's ganglion, throughout the tractus spinalis trigeminalis reached the second-order neurons located on the nucleus spinalis trigeminalis. From here, a long interneuronal ascending system connected to the ipsilateral and contralateral facial nuclei. This multisynaptic pathway included the lateral

propriobulbar system of the reticular formation, lying medial to the trigeminal spinal nucleus. A bilateral delayed R2i and R2c response in all diabetic patients with or without polyneuropathy indicates interneuron subclinical dysfunction in the low brainstem reticular formation.

Four published works on blink reflex in chronic renal failure were found. In one 47 patients undergoing hemodialysis, 24 showed clinical or electromyographical evidence of peripheral neuropathy, and in 13 patients abnormal early R1 blink response was noted. In another, there was a 64% coincidence of abnormal peroneal nerve conduction and R1 blink reflex abnormalities (9/15 patients); delayed blink responses with simultaneous reduced conduction velocity in the median nerve were only found in four cases (29%). In the third study there were no reports of conduction studies of the peripheral nerves. Resende et al. studied blink reflex in 20 non-diabetic adult males who had chronic renal failure. They detected abnormal blink reflex in 10 (50%) patients and axonal sensorymotor peripheral neuropathy in 8 (80%) of these 10. They detected a bilateral delayed early R1 response, and this indicated bilateral dysfunction of the trigeminal and/or facial nerve, elongation at limit in R2i, and R2c latencies, and this was indicative of interneuron subclinical dysfunction in low brainstem reticular formation. Late response abnormalities in the blink reflex suggest subclinical brainstem dysfuncion in chronic renal failure with this study.

Ishpekova et al. studied blink reflex in 27 patients with hereditary motor and sensory neuropathy. In these patients , they detected three components (R1, R2 and R3) of blink reflex instead of usual two which are recordable in normal subjects on the side ipsilateral to the stimulation. On the contralateral side the latter two components (R2 an R3) were present. The responses were distinct in all patients and their mean latencies were prolonged in comparison with those of healthy subjects. An unusual three component blink reflex was observed in the HMSN patients. R3 component appeared to be distinct temporally from R2. It could only be evoked by strong electrical pulses and had a latency about 50 ms longer than R2 in healthy subjects. R1 latency was increased 2.5 times over normal values. Latencies of R2 and R3 components were prolonged in these patients. The medium thick myelinated A-beta fibres were mainly responsible for R1 component, whereas R2 was mediated by the nociceptive thin myelinated A-delta fibres. The cutaneous A-beta and nociceptive A-delta fibres contributed to the generation of the very late component R3. Identical changes of R2 and R3 components in HMSN patients supported the conclusion that the reflex arc for R2 and R3 uses the same brain stem pathways.

Charcot-Marie-Tooth (CMT) is the most frequent inherited neuropathy. X-linked CMT (CMTX) is the second most frequent form after CMT1A, with a frequency about 20%. Signs of CNS involvement have been reported in a few CMTX patients. Moreover, the use of modern imaging and electrophysiological methods has provided evidence of occasional subclinical CNS involvement in some patients with CMT. Zambelis et al. studied blink reflex in all members of X-linked Charcot-Marie-Tooth family (seven probands; 4 male and 3 female) . Blink reflex showed bilaterally prolonged R1 and R2 responses in four symtomatic patients with normal distal latency of the facial nerve. This is supporting electrophysiological evidence of subclinical involvement of central nervous system in CMTX neuropathy.

Table 1 summarizes blink reflex alterations in various polyneuropathies.

	R1 (latency)	R2i (latency)	R2c (latency)
Diabetic polyneuropathy	**Delayed**	**Delayed**	**Delayed**
Uremic neuropathy	**Delayed**	**Delayed**	**Delayed**
GBS	**Delayed**	**Delayed**	**Delayed**
CIDP	**Delayed**	**Delayed**	**Delayed**
Fisher syndrome	**Normal**	**Normal**	**Normal**
Polyneuritis cranialis	**Absent**	**Absent**	**Absent**
AntiGQ1b antibody syndrome	**Normal**	**Absent**	**Absent**
HMSN	**Delayed**	**Delayed**	**Delayed**

Table 1. Blink reflex alterations in various polyneuropathies.
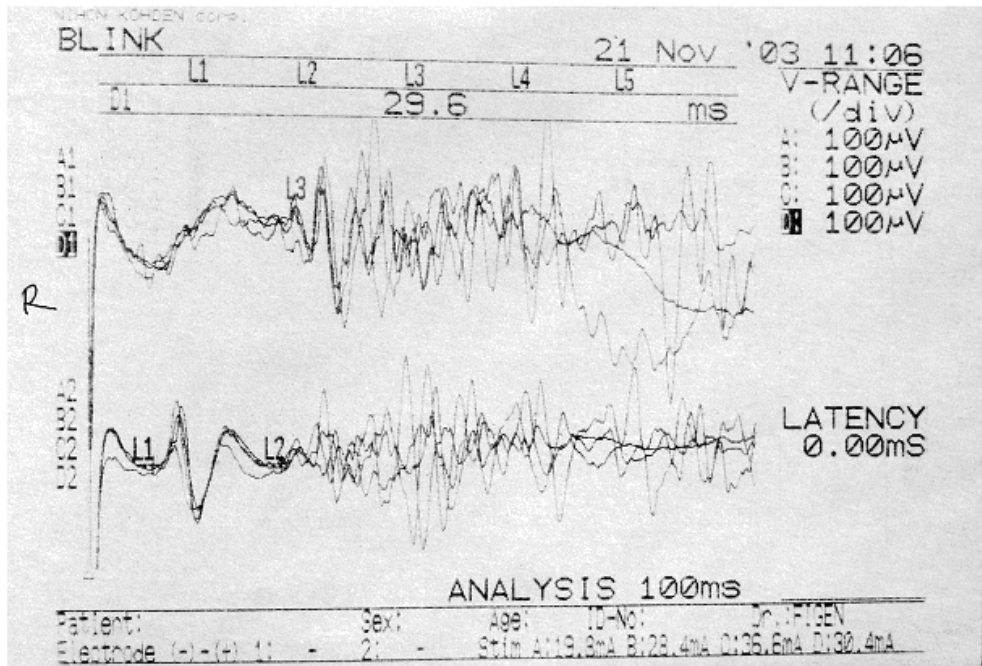
Fig. 1. Blink reflex in normal subject.
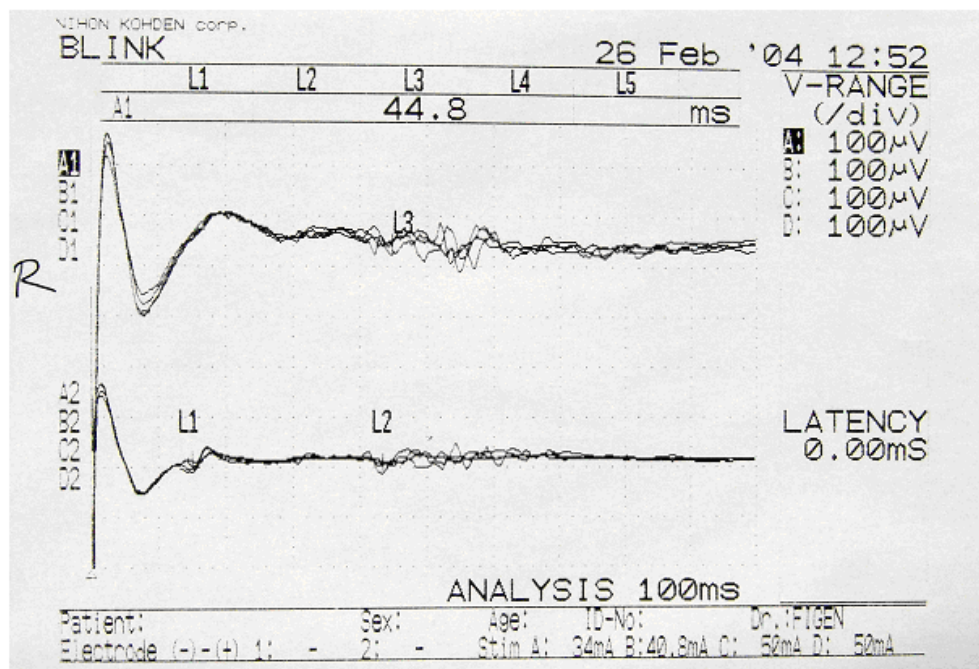
Fig. 2. Increased R1, R2i and R2c latencies in diabetic patient with polyneuropathy.

3. Conclusion

Finally blink reflex can be useful tool for detection of clinically silent intraaxial brainstem functional abnormalities or extraaxial lesions patients with polyneuropathy.

4. References

[1] Overend W. Preliminary note on a new cranial reflex. Lancet 1896; 1:619.
[2] Kugelberg E. Facial reflexes. Brain 1952; 75: 385-396.
[3] Kimura J. Conduction abnormalities of the facial and rigeminal nerves in polyneuropathy. Muscle Nerve 1982; 5: 149-154.
[4] Sanes JN, Ison JR. Conditions that affect the thresholds of the eyeblink reflex in human. J Neurol Neurosurg Psychiatry 1982; 45:543-9.
[5] Shahani BT, Young RR. Human orbicularis oculi reflexes. Neurology 1972; 22:149-54.
[6] Snow BJ, Frith RW. The relationship of eyelid movement to the ik reflex. J Neurol Sci 1989; 91: 179-89.
[7] Dengler R, Rechl F, Struppler A. Recruitment of single motor units in the human blink reflex. Neuroscience Lett 1982; 34: 301-5.
[8] Hiraoka M, Shimamura M. Neural mechanisms of the corneal blinking reflex in cat. Brain Res 1977; 125: 265-75.
[9] Esteban A. A neurophysiological approach to brainstem reflexes. Blink reflex. Neurophysiol Clin 1999; 29: 7-38.

[10] Polo A, Manganotti P, Zanette G, Grandis D. Polyneuritis cranialis: clinical and electrophysiological findings. J Neurol Neurosurg Psych 1992; 55: 398-400.

[11] Neau JP, Gil R, Boissonnot L, Lefevre JP. The blink reflex and stimulus detection by the facial nerve in 50 cases of Guillain Barre polyradiculitis. Acta Neurol Belg 1987; 87(1):12

[12] Ropper AH, Wijdicks EF, Shahani BT. Electrodiagnostic abnormalities in 113 consecutive patients with Guillain-Barre syndrome. Arch Neurol 1990; 47:881-7.

[13] Vucic S, Cairns KD, Black KR, Chong PST, Cros D. Neurophysiologic findings in early acute inflammatory demyelinating polyradiculoneuropathy. Clin Neurophysiol 2004; 115: 2329-2335.

[14] Cruccu G, Agostino R, Inghiller M, Innocenti P, Romaniello A, Manfredi M. Mandibular nerve involvement in diabetic polyneuropathy and chronic inflammatory demyelinating polyneuropathy. Muscle Nerve 1998; 21: 1673-1679.

[15] Kokubun N, Hirata K. Neurophysiological evaluation of trigeminal and facial nerves in patients with chronic inflammatory demyelinating polyneuropathy. Muscle Nerve 2007; 35: 203-207.

[16] Varela H, Rubin DI. Facial and trigeminal neuropathies as the initial manifestation of chronic inflammatory demyelinating polyradiculopathy. J Clin Neuromuscul Dis 2009; 10: 194-98.

[17] Ogawara K, Kuwabara S, Yuki N. Fisher syndrome or Bickerstaff brainstem encephalitis? Anti-GQ1b IgG antibody syndrome involving both the peripheral and central nervous systems. Muscle Nerve 2002; 26: 845-49.

[18] Urban PP, Forst T, Lenfers M, Koehler J, Connemann BJ, Beyer J. Incidence of subclinical trigeminal and facial nerve involvement in diabetes mellitus. Electromyogr Clin Neurophysiol 1999; 39: 267-72.

[19] Nazliel B, Yetkin I, Irkec C, Kocer B. Blink reflex abnormalities in diabetes mellitus. Diabetes Metab Res Rev 2001; 17: 396-400.

[20] Guney F, Demir O, Gonen MS. Blink reflex alterations in diabetic patients with or without polyneuropathy. Int J Neurosci 2008; 118(9): 1287-98.

[21] Trontelj MA, Trontelj JV. Reflex arc of the first component of the human blink reflex: a simgle motoneuron study. J Neurol Neurosurg Psychiatry 1978; 41: 538-47.

[22] Tamai Y, Iwamoto M, Tsujimoto T. Patway of the blink reflex in the brainstem of the cat: interneurons between the trigeminal nuclei and the facial nucleus. Brain Res 1986; 380: 19-25. Holstege G, Van Ham JJ, Tan J. Afferent projections to the orbicularis oculi motoneuronal cell group: An autoradiographic tracing study in the cat. Brain Res 1986; 374: 306-320.

[23] Schneider C. Le reflexe de clignement dans quelques polyneuropathies. Electrodiag Ther 1979; 16(1): 41-6.

[24] Strenge H. The blink reflex in chronic renal failure. J Neurol 1980; 222: 205-14.

[25] Stamboulis E, Scarpalezos S, Malliara-Loulakaki S, Voudiklari S, Koutra E. Blink reflex in patients submitted to chronic periodical hemodialysis. Electromyo Clin Neurophysiol 1987; 27: 19-23.

[26] Resende LAL, Caramori JCT, Kimaid PAT, Barretti P. Blink reflex in end-stage-renal disease patients undergoing hemodialysis. J Electromyogr Kinesiol 2002; 12: 159-63.

[27] Ishpekova BA, Christova LG, Alexandrov AS, Thomas PK. The electrophysiological profile of hereditary motor and sensory neuropathy-Lom. J Neurosurg Psychiatry 2005; 76: 875-78.

[28] Ellrich J, Hopf HC. The R3 component of the blink reflex: normative data and application in spinal lesions. Electroencephalogr Clin Neurophysiol 1996; 101: 349-54.

[29] Rossi B, Risaliti R, Rossi A. The R3 component of the blink reflex in man: a reflex response induced by activation of high threshold cutaneous afferents. Electroencephalogr Clin Neurophysiol 1989; 73: 334-40.

[30] Ellrich J, Bromm B, Hopf HC. Pain-evoked blink reflex. Muscle Nerve 1997; 20: 265-70.

[31] Ellrich J, Katsarava Z, Przywara S, Kaube H. Is the R3 component of the human blink reflex nociceptive in origin? Pain 2001; 91: 389-95.

[32] Zambelis T, Panas M, Kokotis P, Karadima G, Kararizou E, Karandreas N. Central motor and sensory pathway involvement in an X-linked Charcot-Marie-Tooth family. Acta neurol Belg 2008; 108: 44-47.

[33] Aramideh M, Ongerboer De Visser BW. Brainstem reflexes: Electrodiagnostic techniques, physiology, normative data, and clinical applications. Muscle Nerve 2002; 26:14-30.
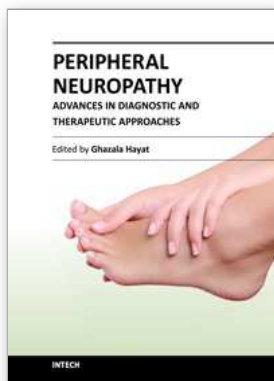
Peripheral Neuropathy - Advances in Diagnostic and Therapeutic Approaches

Edited by Dr. Ghazala Hayat

Over the last two decades we have seen extensive progress within the practice of neurology. We have refined our understanding of the etiology and pathogenesis for both peripheral and central nervous system diseases, and developed new therapeutic approaches towards these diseases. Peripheral neuropathy is a common disorder seen by many specialists and can pose a diagnostic dilemma. Many etiologies, including drugs that are used to treat other diseases, can cause peripheral neuropathy. However, the most common cause is Diabetes Mellitus, a disease all physicians encounter. Disability due to peripheral neuropathy can be severe, as the patients suffer from symptoms daily. This book addresses the advances in the diagnosis and therapies of peripheral neuropathy over the last decade. The basics of different peripheral neuropathies is briefly discussed, however, the book focuses on topics that address new approaches to peripheral neuropathies.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Figen Guney (2012). Blink Reflex Alterations in Various Polyneuropathies, Peripheral Neuropathy - Advances in Diagnostic and Therapeutic Approaches, Dr. Ghazala Hayat (Ed.), ISBN: 978-953-51-0066-9, InTech, Available from: http://www.intechopen.com/books/peripheral-neuropathy-advances-in-diagnostic-and-therapeutic-approaches/blink-reflex-alterations-in-various-polyneuropathies

INTECH
open science | open minds