Electrodiagnosis of the Cranial Nerves

Jun Kimura

Abstract- Isolated facial weakness suggests either a contralateral hemispheric lesion or a disease of the facial nerve per se. The presence of sensory symptoms usually indicates a central facial weakness, which characteristically involves the lower part of the face. In contrast, the absence of sensory disturbances suggests a peripheral nerve lesion, some system diseases such as amyotrophic lateral sclerosis, or a stroke sparing the sensory cortex. Sporadic cases of Bell's palsy rank the first in incidence. Although its exact etiology remains unknown, accumulating evidence suggests reactivation of herpes simplex virus type I. A facial palsy that develops in patients with diabetes mellitus tends to show a more severe involvement with substantial denervation. Acoustic neuroma, strategically located at the cerebellopontine angle, may compress the facial nerve. Peripheral facial palsy may herald other symptoms of multiple sclerosis in young adults. Serial electrodiagnostic studies help delineate the course of the illness. The amplitude of the direct response elicited by stimulation of the facial nerve after the fourth to fifth day of onset serves as the best means predicting the eventual outcome of recovery. Blink reflex studies usually show an absent or delayed R1, implicating the central reflex arc, which includes the intrapontine portion of the facial nerve.

Key Words: Blink reflex, Facial nerve stimulation, Facial weakness, Bell's palsy

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INTRODUCTION

Despite the unpredictable nature of traumatic injuries, certain individual nerves are predisposed to isolated damage[1]. These include not only the limb nerves, but also the facial and spinal accessory nerves. Injuries resulting from acute or chronic repetitive external pressure produce compressive neuropathy, whereas chronic distortion or angulation of the nerve from an internal source causes entrapment neuropathy[2]. Entrapment syndromes develop at the common sites of chronic or recurrent constriction of the facial nerve[3] as might be seen for the radial, median, ulnar, common peroneal and tibial nerves[4]. A number of different nerve lesions also result from stretch, ischemia, compression or laceration during a surgical procedure.

The diagnosis of a focal nerve lesion depends on elucidation of weakness and atrophy of all muscles supplied by the nerve distal to the lesion. Sensory findings, that usually provide localizing signs for limb nerves, do

From the Department of Neurology, Kyoto University Hospital, Kyoto, Japan.
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Reprint requests and correspondence to: Jun Kimura, MD. Department of Neurology, Kyoto University Hospital, Kyoto, Japan.
E-mail: kimurakyoto@aol.com

not help in assessing the facial or spinal accessory nerves. Nerve conduction studies help localize and characterize a focal lesion if conducted as an extension of a physical examination in a proper clinical context[5]. Electromyographic examination delineates the exact distribution of denervated muscles in localizing a focal nerve lesion. In a demyelinative condition, a reduced recruitment of motor units' signals a conduction block rather than a loss of axons. The pattern of distribution here also helps elucidate the zone of involvement.

Nerve conduction studies may provide the evidence of conduction abnormalities, which usually precede the axonal degeneration in a compression neuropathy. Excitability changes associated with such focal conduction slowing may not necessarily show characteristics typically attributable to demyelination[6,7]. Thus, other factors such as ischemia may contribute to the reversible conduction block seen in some of these cases. Stimulation above and below the suspected site of lesion will document not only the slowing of conduction velocity, but also changes in amplitude and area of the muscle or nerve action potential as indices of functional block. Such a pattern of abnormalities often helps differentiate a focal syndrome from a diffuse neuropathy. This distinction, however, may blur in certain types of polyneuropathy that, in early stages, mimic a localized pathology at the common sites of compression.

CRANIAL NERVES

Isolated cranial nerve palsies may result from lesions of the respective nerves along their extra-axial courses or as the sole manifestation of brainstem lesions[8]. Cranial nerves most commonly assessed in an electromyographic laboratory include the facial and spinal accessory nerves. They both travel superficially, which allows easy access to electrical stimulation from the surface. They also innervate the muscles readily approachable by needle or disc electrodes for recording.

Facial nerve

Bell's palsy affects the facial nerve sporadically in an isolated incidence. Although the exact etiology remains unknown accumulating evidence suggests that herpes simplex virus type I (HSV-1) reactivation causes facial nerve lesion in some patients[9-11]. This gives a rational for antiviral therapy with acyclovir[12]. Swelling and hyperemia in the intraosseous portion of the facial nerve suggests a focal pathology during the acute stage. Paralysis of the upper and lower portions of the face develops suddenly often associated with pain behind the ear. Additional features may include loss of taste in the anterior two thirds of the tongue and hyperacusis on the affected side. At least 80 percent of patients improve quickly without specific therapy[13]. Complete recovery follows the demyelinative form, whereas functions return slowly and poorly after degeneration of the facial nerve. Synkinesis nearly always develops with regeneration[14]. Patients may complain of sensory signs in the trigeminal distribution in an otherwise typical case of Bell's palsy. Patients with a rare familial type may suffer from recurrent episodes, which tend to leave increasing residual weakness after each attack[15]. Hyperostosis cranialis interna, a rare genetic bone disorder, also causes a recurrent facial palsy associated with impairment of the senses of smell, taste and vision[16].

The same principles apply to the electromyographic examination of facial and limb muscles. In the face, however, physiologically small motor unit potentials may mimic fibrillation potentials, and signs of denervation appear early in less than 3 weeks following injury, presumably because of the shorter nerve length. Serial electrodiagnostic studies, including the blink reflex (Fig. 1), help delineate the course of the illness (Tables 1 and 2). The amplitude of the direct response elicited by stimulation of the facial nerve provides the best means for prognosis after the fourth to fifth day of onset (Fig. 2). An amplitude greater than one half of the control value on the normal side indicates a good prognosis, although late degeneration can still occur. Preservation or return of Rl or R2 of the blink reflex also serves as a reliable measure in predicting a satisfactory recovery (Fig. 3), providing reasonable assurance that the remaining axons will survive. The reflex, however, rarely returns during the first few days after onset. In a series of 56 patients who recovered without substantial distal degeneration,

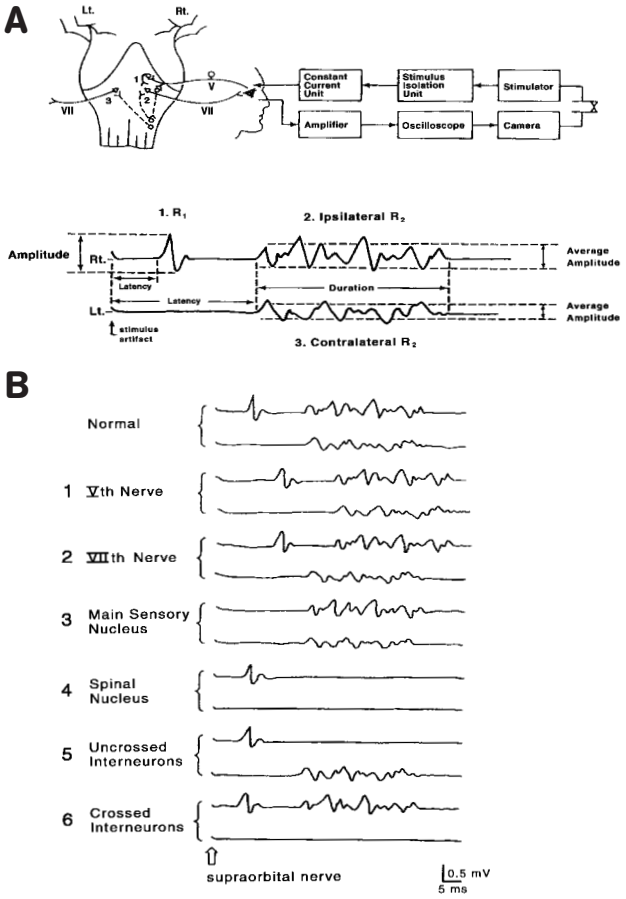

Figure 1. (A) Top: Stimulation and recording arrangement for the blink reflex, with the presumed pathway of R1 through the pons (1) and ipsilateral and contralateral R2 through the pons and lateral medulla (2 and 3). The schematic illustration shows the primary afferents of R1 and R2 shown as one fiber, as details of polysynaptic central connections of these reflexes are unknown. Bottom: A typical oscilloscope recording of the blink reflex after right-sided stimulation. Note an ipsilateral R1 response and bilateral simultaneous R2 responses. (Modified from Kimura, 1975, with permission). (B) Five basic types of blink reflex abnormalities. From top to bottom, the finding suggests the conduction abnormality of (1) afferent pathway along the trigeminal nerve; (2) efferent pathway along the facial nerve; (3) main sensory nucleus or pontine interneurons relaying to the ipsilateral facial nucleus; (4) spinal tract and nucleus or medullary interneuronal pathways to the facial nuclei on both sides; (5) uncrossed medullary interneurons to the ipsilateral facial nucleus; and (6) crossed medullary interneurons to the contralateral facial nucleus. Increased latencies of R1 usually indicates the involvement of the reflex arc itself, whereas the loss or diminution of R1 or R2 may result not only from lesions directly affecting the reflex pathway but also those indirectly influencing the excitability of the interneurons or motor neurons.

Table 1. *Blink reflex elicited by electrical stimulation of supraorbital nerve in normal subjects and patients with bilateral neurologic diseases (Mean ±SD)*

Category	Number of patients	Direct response right and left combined			R1 Right and left combined			Direct response (ms)	R1 (ms)	R/D ratio	Ipsilateral R2 (ms)	Contralateral R2 (ms)
		Abs	Delay	N1	Abs	Delay	N1					
Normal	83 (glabellar tap 21)*	0	0	166	0	0	166	2.9 ± 0.4	10.5 ± 0.8 (12.5 ± 1.4)*	3.6 ± 0.5	30.5 ± 3.4	30.5 ± 4.4
Guillain-Barré syndrome	90	12	63	105	20	78	82	4.2 ± 2.1	15.1 ± 5.9	3.9 ± 1.3	37.4 ± 8.9	37.7 ± 8.4
Chronic inflammatory polyneuropathy	14	4	13	11	7	13	8	5.8 ± 2.6	16.4 ± 6.4	3.1 ± 0.5	39.5 ± 9.4	42.0 ± 10.3
Flsher syndrome	4	0	0	8	0	1	7	2.7 ± 0.2	10.7 ± 0.8	3.9 ± 0.4	31.8 ± 1.3	31.4 ± 1.9
Hereditary motor sensory neuropathy type I	62	9	88	27	0	105	19	6.7 ± 2.7	17.0 ± 3.7	2.8 ± 0.9	39.5 ± 5.7	39.3 ± 6.4
Hereditary motor sensory neuropathy type II	17	0	0	34	1	0	33	2.9 ± 0.4	10.1 ± 0.6	3.6 ± 0.6	30.1 ± 3.8	30.1 ± 3.7
Diabetic polyneuropathy	86	2	20	150	1	17	154	3.4 ± 0.6	11.4 ± 1.2	3.4 ± 0.5	33.7 ± 4.6	34.8 ± 5.3
Multiple sclerosis	62	0	0	124	1	44	79	2.9 ± 0.5	12.3 ± 2.7	4.3 ± 0.9	35.8 ± 8.4	37.7 ± 8.0

Abs: absent response; N1: normal. *R1 elicited bilaterally by a midine glabellar tap in another group of 21 healthy subjects.
From Kimura[3].

the R1 reappeared by the latter half of the first week in 57 percent, by the second week in 67 percent, and by the third week in 89 percent[14]. Other signs for good outcomes include incomplete clinical paresis and the presence of voluntary motor unit potentials in electromyographic studies[17].

In the absence of substantial nerve degeneration, the direct response remains unaltered in latency and amplitude throughout the course on the affected side. In these patients R1 of the blink reflex, if present, shows a relatively normal latency during the first few days, a delay during the latter half of the first week to the fourth week, a notable recovery during the second month and a return to the normal range during the third to fourth months

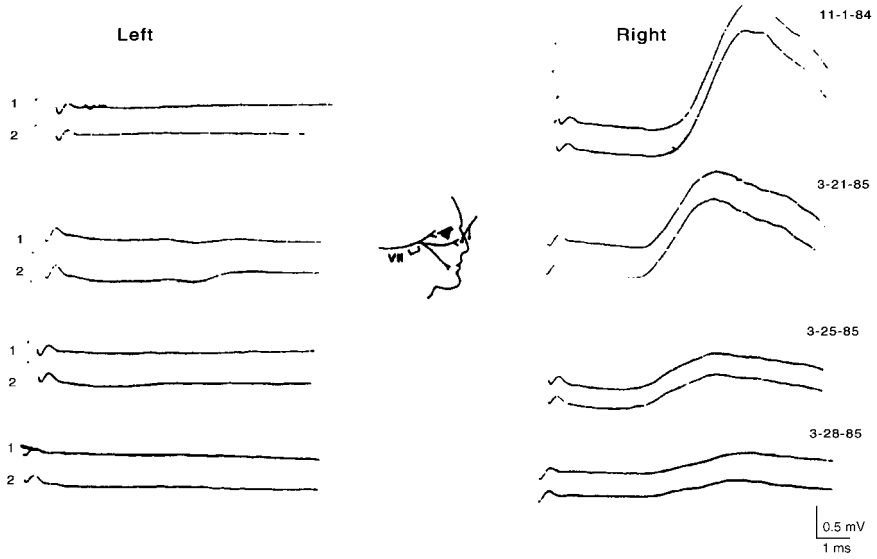

Figure 2. A 63-year-old man with acute facial palsy on the left in November, 1984, and on the right in March 1985. Stimulation of the left facial nerve elicited no response in the nasalis at the initial evaluation with no recovery thereafter. Stimulation on the right evoked a normal response in November but progressive reduction in amplitude of the compound muscle action potential in March. This finding indicates axonal degeneration during the first few days after the onset of illness. From Kimura[62].

Table 2. *Blink reflex elicited by electrical stimulation of supraorbital nerve on the affected and normal sides in patients with unilateral neurologic diseases (Mean ±SD)*

Category and side of stimulation	Number of patients	Direct response (ms)	R1 (ms)	R/D ratio	Ipsilateral R2 (ms)	Contralateral R2 (ms)
Trigeminal neuralgia						
Affected side	89	2.9 ±0.4	10.6 ±1.0	3.7 ±0.6	30.4 ± 4.4	31.6 ±4.5
Normal side	89	2.9 ±0.5	10.5 ±0.9	3.7 ±0.6	30.5 ± 4.2	31.1 ±4.7
Compressive lesion of the trigeminal nerve						
Affected side	17	3.1 ±0.5	11.9 ±1.8	3.9 ±1.0	36.0 ± 5.5	37.2 ±5.7
Normal side	17	3.2 ±0.6	10.3 ±1.1	3.4 ±0.6	33.7 ± 3.5	34.8 ±4.1
Bell's palsy						
Affected side	100	2.9 ±0.6	12.8 ±1.6	4.4 ±0.9	33.9 ± 4.9	30.5 ±4.9
Normal side	100	2.8 ±0.4	10.2 ±1.0	3.7 ±0.6	30.5 ± 4.3	34.0 ±5.4
Acoustic neuroma						
Affected side	26	3.2 ±0.7	14.0 ±2.7	4.6 ±1.7	38.2 ± 8.2	36.6 ±8.2
Normal side	26	2.9 ±0.4	10.9 ±0.9	3.8 ±0.5	33.1 ± 3.5	35.3 ±4.5
Wallenberg syndrome						
Affected side	23	3.2 ±0.6	10.9 ±0.7	3.6 ±0.6	40.7 ± 4.6	38.4 ±7.1
Normal side	23	3.2 ±0.4	10.7 ±0.5	3.4 ±0.4	34.0 ± 5.7	35.1 ±5.8

From Kimura[3].

(Fig. 4). These findings suggest that most patients with Bell's palsy, who develop little axonal degeneration, suffer from a focal demyelination. If the facial nerve undergoes substantial degeneration, the ultimate recovery depends on the completeness of regeneration. This process generally takes a few months to a few years, resulting almost always in an aberrant reinnervation[18] associated with synkinesis (Fig. 5) and hyperexcitability[19].

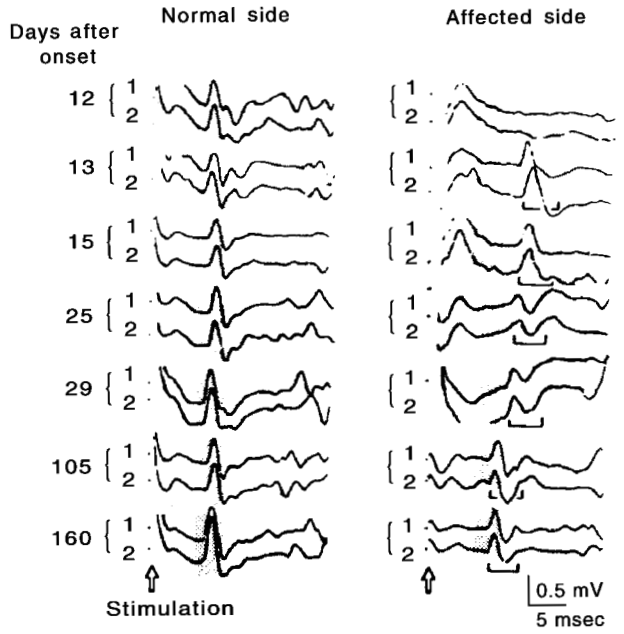

Figure 3. Serial changes of R1 in a 16-year-old girl with Bell's palsy on the right. Two consecutive tracings recorded on each side show consistency of R1 on a given day. On the affected side, delayed R1 first appeared on the 13th day of onset, recovering progressively thereafter. Shaded areas indicate normal range (mean 3SD in 83 subjects). From Kimura.[63]

Peripheral facial paresis secondary to herpes zoster infection carries a less favorable prognosis although early administration of acyclovir and prednisone may reduce the risk of nerve degeneration[20]. Patients with Bannwarth's syndrome may develop unilateral or bilateral facial palsy as part of multiple mononeuritis associated with erythema, pain, elevated cerebrospinal fluid protein and pleocytosis[21]. Peripheral facial palsies may also accompany systemic infection such as Lyme borreliosis[22,23] and human immunodeficiency syndrome or complicate an inferior dental and, less commonly, upper dental anesthetic block[24].

Diabetic patients who develop a facial palsy also tend to have a more severe paresis and evidence of substantial denervation[25]. Patients with Guillain-Barre syndrome usually develop prominent facial paresis as the consequence of acute demyelinative conduction block (Figs. 6 and 7)[26]. In contrast the chronic insidious progression in hereditary in Charcot-Marie-Tooth disease Type 1 allows compensation for motor function and thus minimal weakness despite marked delay in conduction.

An acoustic neuroma strategically located at the cerebellopontine angle may compress not only the facial nerve but also the trigeminal nerve and the pons, i.e., the efferent, afferent and central arcs, of the blink reflex[27-30].

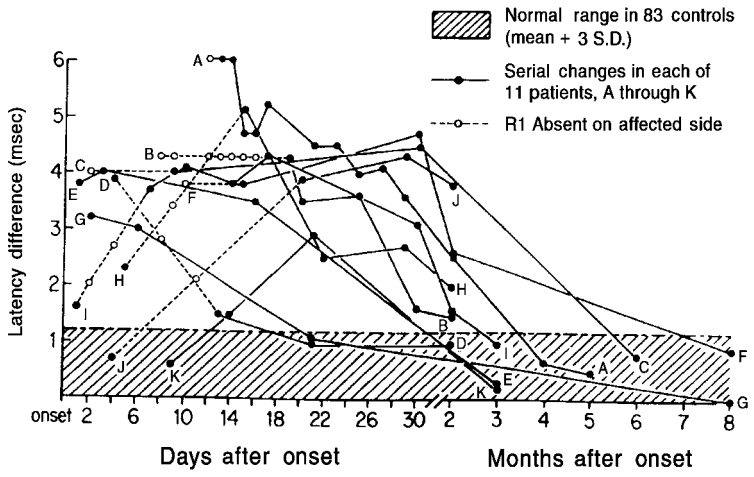

Figure 4. Serial changes in latency difference of R1 between normal and paretic sides in 11 patients recovering without nerve degeneration (A through K). Shaded area indicates the normal range (mean 3 SD in 83 subjects). The response, if present at onset, showed relatively normal latencies but rapidly deteriorated during the first few days. Delayed R1 usually returned during the second week, plateaued for 2 to 4 weeks and progressively recovered in latency during the next few months. From Kimura, Giron, Young.[14]

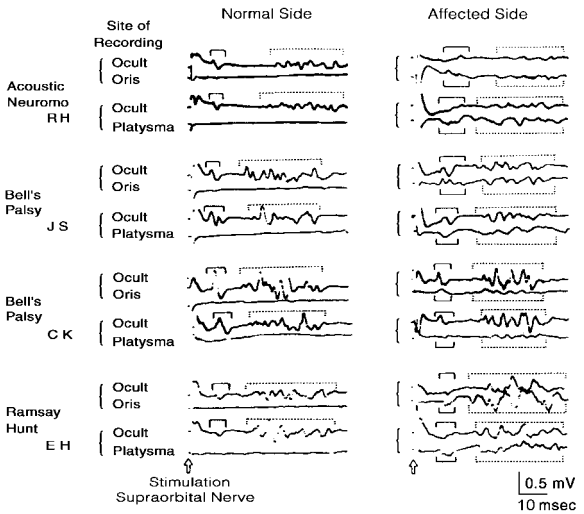

Figure 5. The blink reflex in the orbicularis oris and platysma in four patients following various diseases of the facial nerve. Stimulation on the affected side of the face elicited both R1 (small bracket) and R2 (dotted bracket) not only in the orbicularis oris but also in the platysma, indicating widespread synkinesis. The blink reflex elicited only in the orbicularis oculi on the normal side of the face served as a control in each patient. From Kimura, Rodnitzky, Okwara.[18]

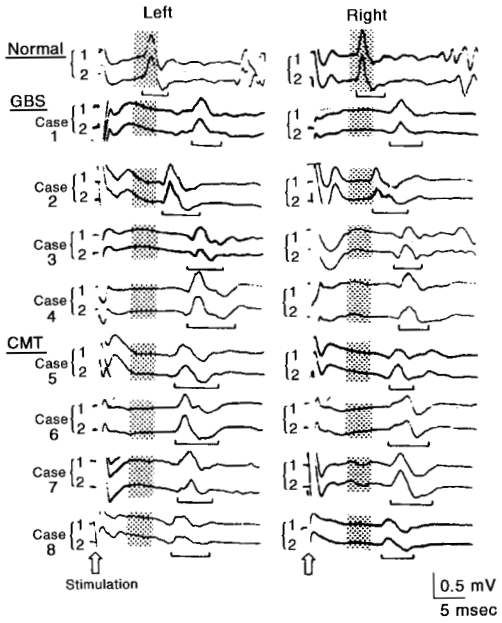

Figure 6. Bilateral delay of R1 in four patients with Guillain-Barré syndrome (GBS) and four patients with hereditary motor sensory neuropathy type 1 (CMT). Two tracings recorded on each side in each subject shows consistency. The top tracings from a healthy subject serve as a control, with shaded areas indicating the normal range. From Kimura.[63]

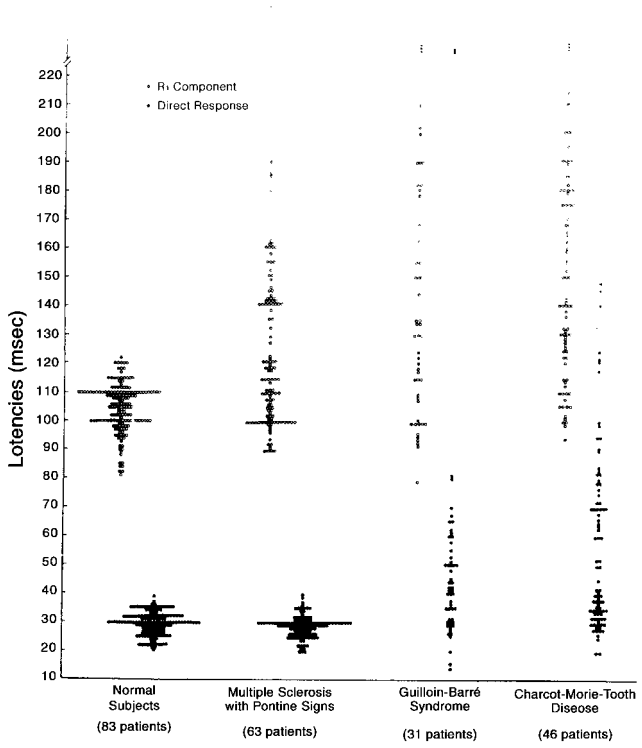

Figure 7. Latency distribution of the direct response and R1 of the blink reflex in normals and in patients with central or peripheral demyelination of the reflex pathways. The histogram shows delayed direct response in Charcot-Marie-Tooth disease, and to a slightly lesser extent in Guillain-Barré syndrome, and normal response in multiple sclerosis. The R1 response is delayed equally in the two polyneuropathies but to a lesser degree in multiple sclerosis. From Kimura.[63]

Table 3. *Direct response and R1 and R2 of the blink reflex*

Disorders	Direct response	R1	R2
Trigeminal neuralgia	Normal	Normal (95%)	Normal
Compressive lesion of the trigeminal nerve	Normal	Abnormal on the affected side (59%)	Abnormal on both sides when afffected side stimulated (afferent type)
Bell's palsy	Normal unless distal segment degenerated	Abnormal on the affected affected side (99%)	Abnormal on the affected side regardless of the side of stimulus (efferent type)
Acoustic neuroma	Normal unless distal segment degenerated	Abnormal on the affected affected side (85%)	Afferent and/or efferent type
Guillain-Barré syndrome	Abnormal (42%)	Abnormal (54%)	Afferent and/or efferent type
Hereditary motor sensory	Abnormal (78%)	Abnormal (85%)	Afferent and/or efferent type
Diabetic polyneuropathy	Abnormal (13%)	Abnormal (10%)	Afferent and/or efferent type
Multiple sclerosis	Normal	Abnormal with pontine lesion, variable incidence determined by patient's selection	Afferent and/or efferent type
Wallenberg syndrome	Normal	Normal or borderline	Afferent type
Facial hypesthesia	Normal	Abnormal with lesions of the trigeminal nerve or pons	Afferent type
Comatose state, akinetic mutism, locked-in syndrome	Normal	Abnormal with pontine lesion; reduced excitability in acute supratentorial lesion	Absent on both sides regardless of side of stimulus

From Kimura[3].

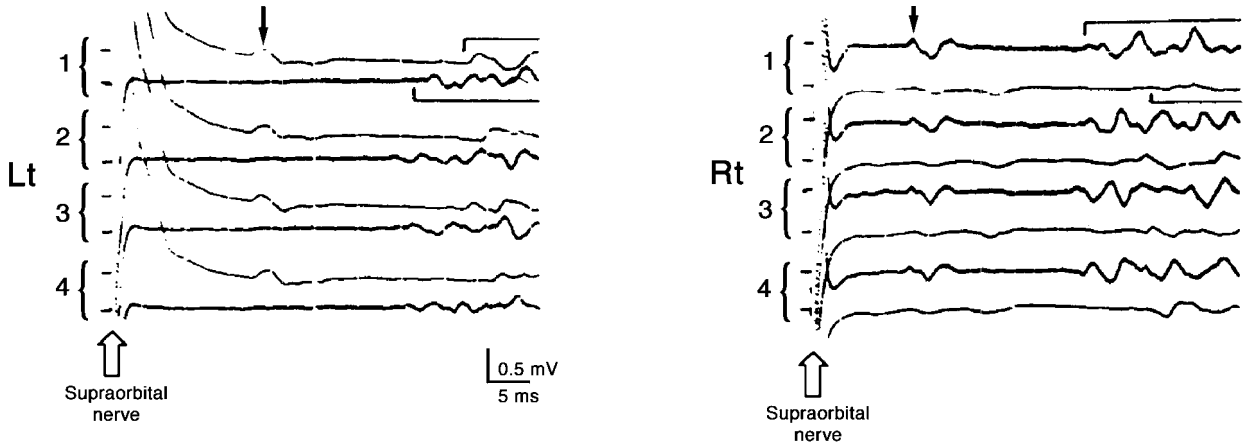

Figure 8. R1 and R2 in a 35-year-old woman with multiple sclerosis and mild facial and abducens paresis on the left. Stimulation on the right elicited normal R1 and delayed R2 contralaterally, whereas stimulation on the left evoked delayed R1 and delayed R2 ipsilaterally. This finding suggests a lesion involving the efferent arc of the reflex on the left, that is, the intrapontine portion of the facial nerve. From Kimura.[63]

Thus, the electrically elicited blink reflex reveals various degrees of abnormality in a most patients (Tables 2 and 3) showing a high correlation with the tumor size[31]. Hypoglossal facial nerve anastomosis may partially restore function after sacrifice of the facial nerve for removal of cerebellopontine angle tumors[32]. Sarcoidosis may also involve the facial nerve probably at the cerebellopontine angle[33].

Peripheral facial palsy may herald other symptoms of multiple sclerosis in young adults (Fig. 8). In these cases, blink reflex studies usually show an absent or delayed R1, indicating demyelination of the central reflex arc, which includes the intrapontine portion of the facial nerve[34]. Myokymic discharges, although characteristic of this disorder, may also appear in other conditions such as pontine glioma[35], and subarachnoid hemorrhage[36]. Progressive hemifacial atrophy may develop in scleroderma with or without associated hemiatrophy of the body[37-39].

Weakness of the orbicularis oculi and frontalis usually suggests a peripheral as opposed to a central type of facial palsy. In equivocal cases, an increase in minimal R1 latency will confirm a peripheral abnormality. Reduced excitability may cause an apparent delay in R1 latency during an acute stage of contralateral hemispheric lesions especially if elicited by the glabellar tap[40]. In doubtful cases, paired stimuli counter the effect of supranuclear hypoexcitability giving rise to the shortest R1 latency as the accurate measure of the conduction time along the reflex arc. The excitability of polysynaptic R2 may change substantially with a hemispheric lesion, showing either an afferent or efferent pattern (Fig. 9).

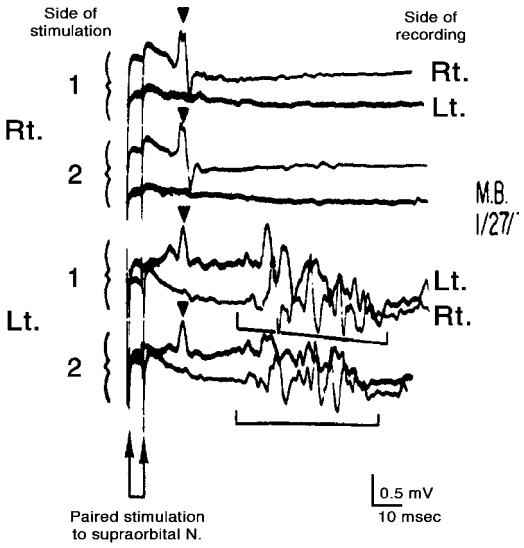

Figure 9. Left cerebral stroke. Paired stimuli delivered to the right supraorbital nerve elicited normal R1 but no R2 on either side. Stimulation on the left, however, evoked an ipsilateral R1 and bilateral R2. From Kimura, 1974.[64]

Trigeminal nerve

Trigeminal sensory neuropathy characteristically evolves with unilateral or bilateral facial numbness sometimes accompanied by pain, paresthesia and disturbed taste. This type of neuropathy may accompany systemic sclerosis or mixed connective tissue disease[41]. Patients with trigeminal neuralgia have altered cutaneous sensation in both the affected and unaffected adjacent divisions, suggesting combined peripheral and central pathology[42]. A mandibular fracture may result in an isolated lesion of the mandibular nerve[43]. Demyelinating lesions affecting pontine trigeminal pathways may cause trigeminal neuralgia in patients with multiple sclerosis[44,34]. Exposure to trichloroethylene causes a cranial neuropathy with peculiar predilection for trigeminal root damage[45]. Facial numbness may herald other symptoms of an expanding tumor involving the trigeminal nerve[46]. Other causes of trigeminal nerve lesion include perineural spread of carcinoma[47]. The blink reflex helps establish abnormalities of the trigeminal nerve. Other techniques of interest include conduction studies of the trigeminal motor nerve[48] and of the mandibular nerve[49].

Accessory nerve

Pressure from a tumor or surgical procedures of the posterior triangle can damage the spinal accessory nerve[50]. Other causes include stretch induced injury[51], cargo loading[52], coronary artery bypass[53], carotid endarterectomy[54,55], and ligature injury during surgical exploration[56]. In trapezius palsies following injury of the

accessory nerve, the upper vertebral border of the scapula moves away from the spinal vertebrae. With the lower angle of the scapula relatively fixed by muscles supplied by the C3 and C4 roots through the cervical plexus, the whole scapula slips downward and the inferior angle rotates internally, or clockwise for the right and counterclockwise for the left scapula as viewed from the back. This type of winging tends to worsen with abduction of the arm to the horizontal plane, which displaces the superior angle further laterally. The paralysis of the sternocleidomastoid causes weakness when the face is rotated toward the opposite shoulder in proportion to the degree of muscle atrophy. Bilateral involvement of the muscles makes flexion of the neck difficult. In a sequential study of patients with trapezius palsy, nerve conduction changes revealed evidence of spontaneous regeneration after complete axonal degeneration[57].

Other cranial nerves

Hypoglossal nerve palsy may result from compression of the vertebral artery[58,59] or aneurysm[60], or kinking of the vertebral artery[58,59] or as a complication in approximately 5 percent of endarterectomies[61].

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