

Objective Evaluation of Infraorbital Nerve Involvement in Maxillary Lesions by Means of the Blink Reflex

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Objective: To objectively evaluate the effects of maxillary lesions on the infraorbital nerve (ION).

Methods: We investigated the latencies (R1, R2) of the blink reflex, stimulating the infraorbital foramen electrically (18 mA, 0.2 millisecond). Twenty-two patients with unilateral maxillary lesions were enrolled.

Results: Ten patients showed delayed or absent R1 on the lesion side. Four of 20 patients showed delayed difference in R2 latency. Ten (77%) of 13 patients with lesions along the ION pathway showed an abnormal blink

reflex. Only 3 patients demonstrated a normal blink reflex despite a lesion along the pathway of the ION. All patients whose ION pathway was intact showed a normal blink reflex.

Conclusions: These results suggested that lesions along the ION pathway may impair the afferent pathway of the blink reflex. The blink reflex may be valuable to evaluate maxillary lesions objectively. Furthermore, R1 is more effective than R2 in detecting ION defects.

Arch Otolaryngol Head Neck Surg. 2002;128:952-955

PATIENTS WITH maxillary lesions complain of various symptoms. Some of the symptoms are derived from trigeminal nerve deficits (eg, pain, tactile deficits, or temperature sensation). Maxillary lesions (eg, maxillary tumors and maxillary cysts) sometimes impair the infraorbital nerve (ION), which supplies sensation to the middle parts of the face. In general, clinicians examine ION functions subjectively on the basis of tactile sensation or temperature sensation, as a standard nerve conduction study is not feasible in the ION for anatomic reasons.

The blink reflex (BR) reflects the function of the trigeminal and facial nerves. In an attempt to evaluate ION deficits objectively, we investigated the changes in BR under the influence of maxillary lesions. We used a method to stimulate the ION supplying sensation to the maxillary region. As far as we know, there are no reports of the use of the BR to objectively investigate ION deficits induced by maxillary lesions.

RESULTS

All 9 control subjects showed ipsilateral early waves (R1) and bilateral late waves

(R2).¹ The latency of R1 was 11.2 ± 0.7 milliseconds (mean \pm SD). The time difference of the R1 latency of the stimulated side was 0.6 ± 0.3 millisecond. The time difference of the R2 latency on the stimulated side was 1.4 ± 1.1 milliseconds.

For R1, the latencies and time differences of the latencies between the healthy side and the affected side were evaluated, while for R2, the latencies of the stimulated side were studied. On the basis of the results from control subjects, the normal range of R1 latency and the time difference of R1 latency on the stimulated side and that of R2 latency on the stimulated side were placed at 9.7 to 12.6 milliseconds (within mean \pm 2 SDs), 0 to 1.3 milliseconds (within mean \pm 2 SDs), and 0 to 3.6 milliseconds (within mean \pm 2 SDs), respectively.

Two of 22 patients who showed absent R1 bilaterally were excluded from the following results. Ten (50%) of 20 patients showed delay or absence of R1 on the affected side (**Figure 1** and **Table**). Of these 10, 4 patients showed delayed R1 and 6 patients showed absent R1. Four (20%) of 20 patients showed a delayed time difference in R2 (Figure 1). No patients showed absent R2 on the affected side. The R1 tended to show a more abnormal response than R2.

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SUBJECTS AND METHODS

Twenty-two patients (14 men and 8 women) with unilateral maxillary lesions, who gave informed consent, were enrolled. Their ages ranged from 26 to 78 years (median, 58 years). Their maxillary lesions were postoperative maxillary cysts in 13 patients, maxillary cancer in 3, maxillary papilloma in 1, unilateral chronic sinusitis in 1, mucocele in 1, chronic sinusitis with orbital cellulitis in 1, ameloblastoma in 1, and traumatic maxillary injury in 1. None had experienced facial nerve paralysis. In addition, we enrolled 9 volunteers (7 men and 2 women) without maxillary diseases as control subjects. Their ages ranged from 22 to 40 years (median, 25 years).

Active recording electrodes were placed on the bilateral upper eyelids while reference electrodes were placed on the bilateral temples. Electrodes (cathode and anode) for electrical stimulation were placed on the skin over the infraorbital foramen. A ground electrode was set on the forehead. We used 18-mA (0.2-millisecond) electrical stimulation. Electromyographic activities were amplified and bandpass filtered (20-2000 Hz). The stimulation was done 4 to 5 times at random and the analysis time was 100 milliseconds. When being measured, patients were instructed to lie on a bed with their eyes gently closed. We used evoked potential/electromyographic measuring systems (Neuropack 8; Nihon Kohden Corporation, Tokyo, Japan) to measure the responses. The latencies of the waves were measured. All patients had a computed tomographic scan of the sinus.

Seven (78%) of 9 patients with bony destruction of the orbital floor showed abnormal BR (Figure 1, **Figure 2** and Table). These 7 patients included 3 with bony destruction in the anterior half of the orbital floor, 2 in the posterior half, and 2 in both. The BR seemed to be irrelevant to the site of bony destruction between the anterior and posterior half of the orbital floor. Eight (73%) of 11 patients without bony destruction of the orbital floor showed normal BR, while the other 3 patients showed abnormal BR (Table). However, 2 of these 3 had bony destruction of the upper anterior wall of the maxillary sinus and the other one had tumor invasion into the pterygopalatine fossa. In other words, these 3 patients with abnormal BR had lesions along the ION pathway to the maxillary nerve.

Only 3 patients showed normal BR, despite lesions along the ION pathway to the maxillary nerve. Two of these patients had bony destruction of the orbital floor, and 1 had bony destruction of the upper anterior wall of the maxillary sinus. On the whole, all patients with intact ION pathways to the maxillary nerve showed normal BR, while the majority of patients (10/13 [77%]) with lesions along the ION pathway to the maxillary nerve showed abnormal BR.

Seven (70%) of 10 patients with sensory deficits (ie, tactile deficits, pain, and temperature sensation) in the maxillary region showed abnormal BR, while results in the other 3 patients were normal. On the other hand, 7 (70%) of 10 patients without sensory deficits had normal results, while the other 3 patients had abnormal results without clinical sensory deficits. In all, the clinical sensory deficits were likely to be compatible with the results of BR testing.

No apparent relationship was suggested between periods from the onset of sensory deficits and the BR in this study (**Figure 3**).

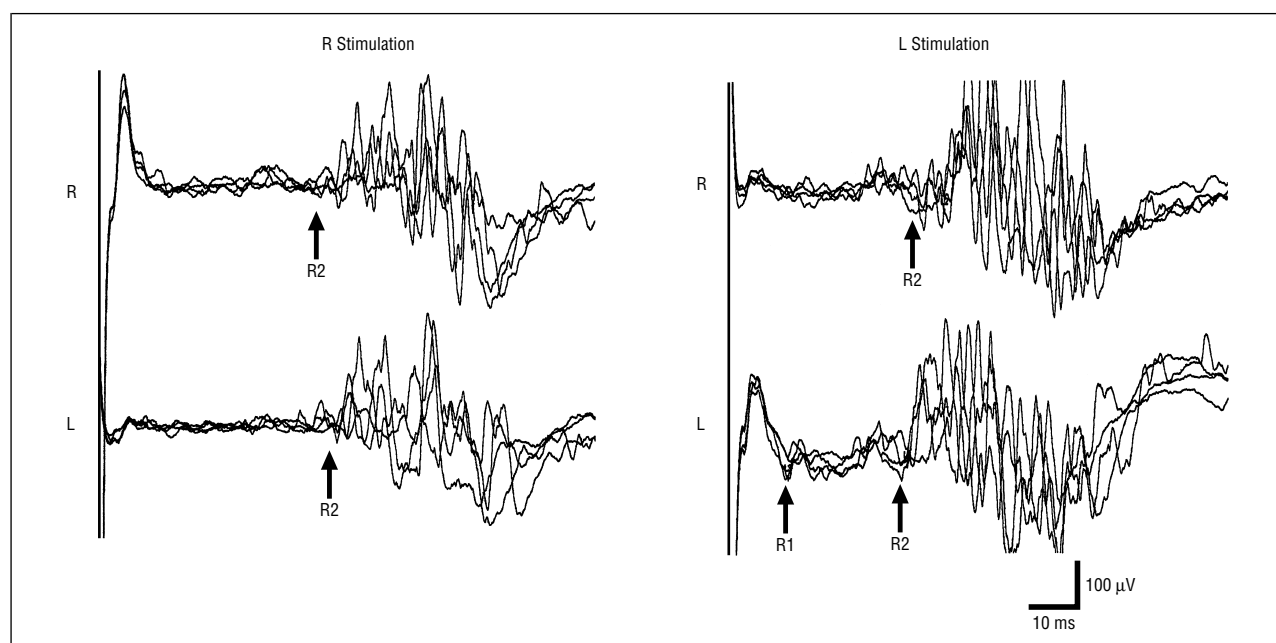


Figure 1. Recordings of the blink reflex in a 79-year-old woman with right maxillary mucocele. The right ipsilateral early wave (R1) was absent and bilateral late wave (R2) was delayed when the right side (R) was stimulated, whereas the blink reflex was normal when the left side (L) was stimulated.

Patient Characteristics			
Orbital Floor	Lesion Sites	Blink Reflex, No.	
		Abnormal	Normal
Bony destruction	Anterior half	7	2
	Posterior half	3	0
	Both	2	0
No bony destruction	Both	2	2
	Bony destruction of upper anterior wall	3	8
	Pterygopalatine fossa invasion	2	1
		1	0

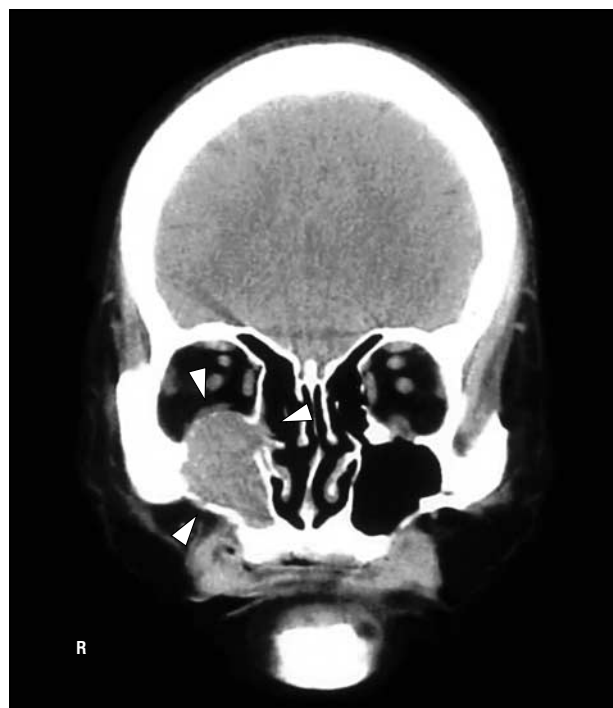


Figure 2. Computed tomographic scan of the patient in Figure 1. Coronal view shows the right maxillary mucocoele compressing the right orbital floor (arrowheads).

COMMENT

The BR reflects the integrity of the trigeminal and facial nerves. It is generally examined to evaluate the facial nerve, trigeminal nerve, pontine lesions, or medullary lesions.¹⁻³ The stimulation elicits 2 separate reflex responses (R1 and R2) of the orbicularis oculi muscle.¹⁻³ The BR can be evoked by not only the supraorbital nerve but also the ION or mental nerve.^{2,3} However, as the supraorbital nerve can most frequently evoke BR in healthy subjects,^{2,3} this nerve is, in general, stimulated electrically or mechanically in clinical examinations. Since the purpose of this study was to evaluate dysfunction of the ION, we applied a method to stimulate the ION. We frequently detected R1 in all control subjects and in the healthy side of 20 (91%) of 22 patients, when attempting to record on upper lids and stimulating only over the nerve. The R2 was detected in all control subjects and in the healthy

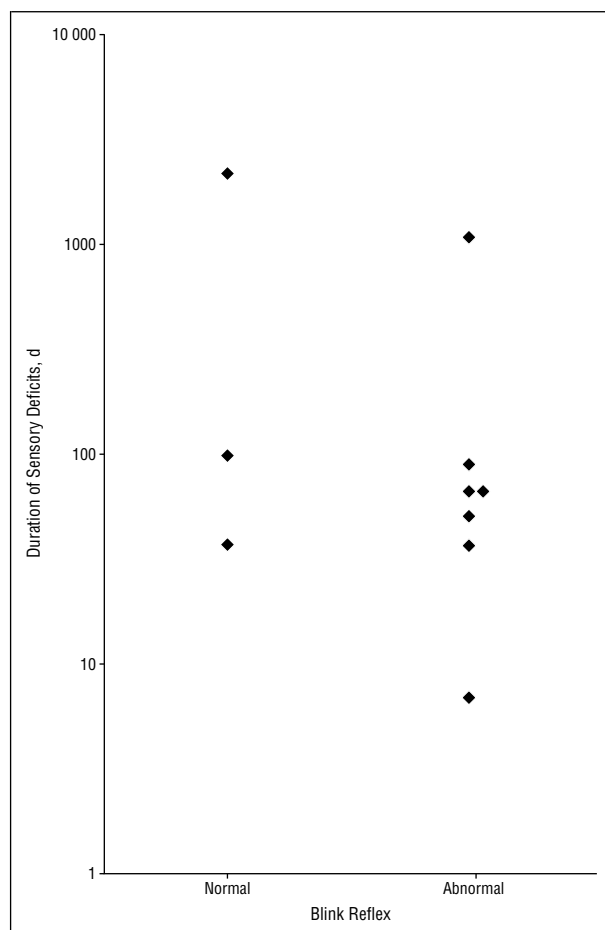


Figure 3. Interval from the onset of sensory deficits with blink reflex. No apparent relationship was seen.

side of all patients. The tendency for R1 to be evoked less often than R2 is compatible with results of a trial by Kimura.^{2,3}

The ION passes through the infraorbital foramen and inferior orbital fissure. This pathway lies in the orbital floor between the maxillary sinus and orbit. After that, it becomes the maxillary nerve in the pterygopalatine fossa. In this study, all 10 patients who showed abnormal BR had lesions along this pathway. It was suggested that lesions along this pathway impaired these afferent pathways of the BR. Moreover, abnormal BR indicated the presence of the lesion along this pathway. However, 3 patients showed normal BR despite lesions along the pathway. This finding may depend on the degree of nervous impairment.

In this study, all patients with abnormal BR showed abnormal R1, while R2 was shown to be abnormal in only 40%. This means that R1 tended to become abnormal more often than R2, and the 2 latencies were not always abnormal together. This may be why R1 and R2 pass through different characteristic nerve fibers, although they both share the same root of the ION. The R1 fibers are concerned with tactile sensation, whereas those of R2 are involved with pain and temperature sensation.² The different kinds of nerve fibers are thought to have a different susceptibility to lesions.⁴

It is still controversial what kind of nerve fibers are responsible for R1 and R2. Recently, it was suggested that R1 is mediated by A β fibers, while R2 is mediated by A β and A δ fibers.^{5,6} In general, tactile sensation activates the larger fibers in the cutaneous nerve in the A β range, while pain and temperature sensation activates the smaller fibers of the A δ -to-C range.⁴ The fibers in descending order from thickest to thinnest are A α , A β , A δ , and C. The thicker the fibers are, the more susceptible they are to compression or inflammation⁷; the fibers related to R1 may be more fragile. These facts suggest one reason why R1 becomes abnormal more frequently.

In practice, patients with sensory deficits were inclined to show a BR abnormality to some degree in this study. As minor sensory deficits may not be detected clinically, some patients showed abnormal BR without apparent sensory deficits. Duration of sensory deficits, in this study, seemed not to be related to an abnormality of BR. As chronic compression or inflammation may induce demyelination or axonal degeneration of neurons subclinically, the appearance of sensory deficits and the grade of a nervous impairment may not necessarily agree.

These results suggested that the lesions along the ION pathway to the maxillary nerve impaired the afferent pathway of BR. The BR may be a useful tool to evaluate maxillary lesions objectively. Furthermore, R1 is more

effective than R2 in detecting ION defects. To our knowledge, this is the first report to use BR to evaluate the effects of maxillary lesions on the ION. Further investigation is still needed.

Accepted for publication December 17, 2001.

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