Chapter 6.2

The orbicularis oculi reflexes

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Introduction

The orbicularis oculi reflex can be evoked by stimuli of various modalities. In clinical practice, a supraorbital nerve stimulus is often used to evaluate the trigemino-facial blink reflex.

The blink reflex involves an early response (R1) ipsilateral to the stimulated supraorbital nerve and a late bilateral response (R2). The common afferent limb of the reflex loop is made up of the sensory trigeminal root and the ophthalmic division, whereas the common efferent limb consists of the facial nerve. R1 is relayed centrally through an oligosynaptic arc including one to three interneurons in the mid pons, probably located in the vicinity of the main sensory nucleus of the trigeminal nerve. R1 is not visible clinically but may have a preparatory function, by shortening the late blink reflex latency.

The R2 blink reflex correlates with closure of the eyelid. Nerve impulses responsible for R2 are conducted by the descending spinal tract through the dorsolateral region of the pons and medulla oblongata to the lower spinal trigeminal nucleus. From there impulses are relayed through polysynaptic medullary pathways ascending both ipsilaterally and contralaterally to the stimulus side, before making connections with the facial nuclei (Ongerboer de Visser and Kuypers 1978; Kimura 1989). The impulses cross in the lower medullary region. Trigemino-facial connections are thought to pass through the reticular formation and lie medial to the spinal trigeminal nucleus (Ongerboer de Visser and Kuypers 1978; Aramidéh et al. 1997). The blink reflex pathways are represented schematically in Fig. 1.

The orbicularis oculi reflex can also be evoked by stimulation of the cornea. The corneal reflex consists only of a late bilateral response, which differs in several ways from the R2 component of the blink reflex both in normal subjects and in patients (Accornero et al. 1980). The cornea is exclusively innervated by unmyelinated (C) and small myelinated (Aδ) fibers. The Aδ fibers, which constitute the afferent units of the corneal reflex, pass through the long ciliary nerves and the ophthalmic division of the trigeminal sensory

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root to reach the pons. Although the central pathway is similar to that of the R2 component of the blink reflex the anatomical and functional properties of the two responses differ considerably. The corneal reflex is a purely nociceptive response, mediated by fewer interneurones, less susceptible to suprasegmental modulation (including cognitive factor), and more sensitive to analgesic than sedative drugs (Berardelli et al. 1985a; Crucu et al. 1991).

**Stimulation and recording**

While recording the blink reflex and the corneal reflex the subject keeps the eyes open. The supraorbital nerve is stimulated transcutaneously with the cathode placed over the supraorbital foramen and the anode about 2 cm higher and rotated laterally at an oblique angle to avoid spread of current to the contralateral supraorbital nerve. If the second trigeminal division is to be examined, the infraorbital nerve is stimulated by placing the cathode over the nerve as it exits through the infraorbital foramen at the inferior rim of the orbit and the anode is placed about 2 cm below the cathode.

To avoid habituation, shocks should be delivered at intervals of 7 s or more while the subject is kept in an alert state. Shocks of optimal intensity should elicit maximum and nearly stable responses with repeated trials. To facilitate responses, the patient is asked to close the eyes, or stimuli are given in pairs at a short interval (5 ms). Responses from the mid-lower half of both orbicularis oculi muscles are recorded by surface electrodes simultaneously. The reference electrode is placed 2 cm lateral to the active electrode and a ground electrode under the chin or around the upper arm. The passband extends from 20 to 2000.

For recording the corneal reflex the cornea can be stimulated either with mechanical or with electrical stimuli. When the corneal reflex is evoked by electrical stimulation, the cornea is touched with a thin, saline-soaked cotton thread connected to the cathode of a constant-current stimulator. The anode is placed on the earlobe or forearm.

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**Fig. 1.** The left part of the figure shows R1 and R2 in a normal subject. The upper two traces illustrate the responses from the right and left orbicularis oculi to right and the lower traces to left supraorbital nerve stimulation. On the right of the figure the diagram shows the circuit of the two components of the blink reflex: (1) interneurones subserving the ipsilateral early component; (2) interneurones subserving the bilateral late component. Vm, trigeminal motor nucleus; SpV Co, spinal trigeminal complex; SpV Tr, spinal trigeminal tract; VI, abducens nucleus; VII, facial nucleus; VII N, facial nerve; V N, ophthalmic trigeminal sensory root; XII, hypoglossal nucleus; LAT RET, lateral reticular formation; MED RET, medial reticular formation. From Ongerboer de Visser and Crucu 1993.
Responses are recorded from the orbicularis oculi muscles.

Measurement and normal values

Latencies are measured from the stimulus artefact to the initial deflection of the EMG responses. Using mean latency plus 3 SD, R1 is delayed if it exceeds 13 ms, R2 if it exceeds 41 ms. A difference between the two sides greater than 1.2 ms for R1 and 8.0 ms for R2 is also considered abnormal. In addition, the difference between the ipsilateral and contralateral R2 should not exceed 5 ms or 8 ms. Amplitudes vary considerably from one subject to the next. The normal values (mean ± S.D.) are 0.38 ± 0.23 mV for R1, 0.53 ± 0.24 mV for ipsilateral R2 and 0.49 ± 0.24 mV for the contralateral R2.

Stimulation of the infraorbital nerve always evokes an R2 response but not necessarily an R1. When R1 is absent, R2 is difficult to evaluate because of the wide range of latencies. An absent R2, however, is certainly abnormal.

The corneal reflex is considered abnormal when its latency is greater than 55 ms or the latency difference between sides is greater than 8 ms; the electrical stimulation also allows measurement of the threshold, which should not exceed 0.5 mA (Cruccu et al. 1987).

The general conditions of stimulation and laboratory environment should be maintained identical because the amplitude and the threshold of the EMG responses are sensitive to extraneous stimuli and stimulus anticipation.

Some special techniques, namely the study of the habituation, the recovery cycle and the prepulse modulation, use the R2 blink reflex as a test for assessing the excitability of the brain-stem reticular formation and the cortico-reticular drive.

Habituation to rhythmic stimulation is usually studied by delivering 4–5 series of 8 stimuli at a rate of 0.2, 0.5 and 1 Hz. The size of the response to the eighth stimulus, expressed as a percentage of the size of the control response, can be taken as an index of habituation. The recovery cycle is studied by applying two shocks of equal intensity (conditioning and test stimuli) to the supraorbital nerve, at intervals ranging from 100 ms to 2 s. The size of the response to the test stimulus, measured as the rectified and integrated EMG activity, is expressed as a percentage of the size of the response to the conditioning stimulus (Kimura 1973; Berardelli et al. 1985a).

The excitability of the blink reflex is modulated by afferent inputs from peripheral nerves or even with stimuli of different modalities. When the first (conditioning) stimulus is of low intensity, incapable of eliciting any response by itself, it is called ‘prepulse’. Prepulse stimuli can be of any sensory modality, including visual, auditory and somesthetic. Their effects on cranial reflexes occur at intervals that vary according to the sensory modality (Valls-Solé et al. 1994). For somatosensory stimuli (a tap to the forearm or a weak electrical shock on the 3rd finger), the peak of the maximum inhibition of the R2 is at about 100 ms. Studies in animals indicate that prepulse effects are a feature of the brain-stem. When the prepulse and the response-eliciting stimuli are repeated several times, another phenomenon occurs: the leading stimulus, previously unable to induce a reflex response, becomes generator of a small (‘postpulse’) response (Valls-Solé et al. 1996).

Clinical use

Orbicularis oculi reflex recordings provide quantitative analysis for functions that involve the fifth and seventh cranial nerves and the described portion of the trigeminal system situated in the dorsolateral areas of the lower half of the pons and the medulla oblongata to its caudal region. The indications for a study of the orbicularis oculi reflex include two categories: diseases that directly affect the reflex arc, and diseases that alter its excitability but lie outside the reflex arc itself, such as hemispheric or basal ganglia lesions.

Numerous clinical conditions affect the reflex arc of the orbicularis oculi reflex (Kimura 1989). Trigeminal nerve lesions are characterized by a delay or block of R1 ipsilaterally and R2 or CR bilaterally when the affected side is stimulated (afferent type of abnormality). Facial nerve lesions cause abnormalities in the R1 and R2 components
of the blink reflex in the ipsilateral orbicularis oculi muscles, regardless of the side of stimulation (efferent type of abnormality).

In hemifacial spasm and postparalytic facial syndrome, stimulation of the supraorbital nerve evokes anomalous responses, resembling the blink reflex in lower facial muscles, including the orbicularis oris or mentalis.

Blink reflex studies provide clinically useful information in diabetic neuropathy, Guillain-Barré syndrome and other types of polyneuropathies.

Lesions affecting the lower pons and the dorsolateral medulla oblongata cause several types of blink reflex abnormalities (Fig. 2). Lesions of the sensorimotor cortex depress the orbicularis oculi reflex because of loss of facilitatory inputs to the brain-stem descending from the cortex via the pyramidal tract (Berardelli et al. 1983). Other conditions causing an altered excitability of interneurons include Parkinson’s disease, dementia, Huntington’s chorea and facial dystonia. In these disorders, the paired shock technique often reveals abnormal recovery curves. In Parkinson’s disease, the R2 component of the blink reflex is less inhibited by preceding impulses, whether delivered in pairs (Kimura 1973) or repetitively. In patients with on-off fluctuations, the facilitation of R2 seen in the off period is reduced during the on period. This indicates that in Parkinson’s disease, changes in excitability are strictly related to variations in central dopamine activity (Agostino et al. 1987). In patients with blepharospasm and oromandibular dystonia the recovery cycle of R2 (Berardelli et al. 1985b) and of the R1 (Aramideh et al. 1995) is also enhanced. Similar changes are also present in patients with dystonia affecting sites other than the facial muscles. In Parkinson’s disease and dystonia, the recovery cycle of R1 is usually normal, indicating that the changes in the excitability of the R2 occur at the interneuronal rather than at the motoneuronal stage. In Huntington’s disease, habituation of the R2 component is reduced in some patients, particularly those with involuntary movements in the face (Agostino et al. 1988). In Parkinson’s disease and dystonia the net result is an enhanced excitability of the segmental interneurons, while in Huntington’s disease excitability is reduced.

The corneal reflex is clinically useful to assess small-fibre function; it is less sensitive than R2 in patients with motor disturbances or with supraorbital lesions in general, probably because the corneal reflex is relayed through fewer synapses than R2 (Crucchi et al. 1987, 1991). Prepulse modulation of the blink reflex is also useful for clinical neurophysiological studies and has disclosed abnormalities in patients with Parkinson’s disease, Huntington’s disease and dystonia. However, further work is needed to increase our knowledge on the circuits of the prepulse effects, as well as on the relationship between prepulse, startle, and blink reflex.

![Diagram of blink reflex responses in normal subjects and in patients with different patterns of abnormalities.](image)

**Fig. 2.** Diagram of blink reflex responses in normal subjects and in patients with different patterns of abnormalities. From Ongerboer de Visser and Crucchi 1993.

**References**

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