

Observations on the Blink Reflex

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Key Words

Blink reflex · Glabella tap reflex · Trigemino-facial reflex · Orbicularis oculi

Abstract

The blink reflex and its equivalent glabella tap reflex are behavioural motor responses normally found in neonates and in the physiological startle reflex. Historically, it was described by Overend in 1896 and soon afterwards by McCarthy and by Bekhterev who disputed its origins. Kugelberg in 1952 recorded the early-latency R1 and the late-latency R2 electromyographic responses from orbicularis oculi and considered the blink reflex to be 'a true skin reflex'. The mechanisms and clinical diagnostic value are discussed.

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Many years ago, Blount [1] and Hall [2] showed that each mammalian species has a characteristic blink rate that tends to be constant under unchanging conditions. In man, the eye blink response is the first and most reliable component of the startle reflex. From an evolutionary point of view, the startle reflex is an integral part of avoidance responses induced by a threatening stimulus, and is increased by emotional stimuli and suppressed by lesions of the amygdala.

In 1888 Mercier [3] described

'a nerve-centre which actuates a single blink of the lids, and then the action ceases ...'

Walker Overend (1858–1926) in the *Lancet* in 1896 [4] first described the blink reflex. It is one of many behavioural motor responses which are found in neonates with normal early development. It is subsequently inhibited, but may be released from inhibition by cerebral lesions. This reflex has been referred to as the glabella (feminine of Latin *glabellus*, hairless) tap sign and nasopalpebral reflex [5]. But these are the same phenomenon; the different terms indicate artefacts arising from spurious transmission of the stimulus across bony structures in the face [6]. When the supra-orbital area, conjunctiva, or cornea are stimulated, the eyelids blink. Afferent impulses travel in the ophthalmic branch of the trigeminal nerve to its sensory nucleus. This projects to the motor nucleus of the facial nerve through three kinds of synaptic connections. First, a direct and monosynaptic connection; second, an indirect polysynaptic connection with the contralateral facial nucleus, and third, a polysynaptic connection to the ipsilateral facial nerve nucleus. The facial nerve innervates the orbicularis oculi muscle, causing the blink.

History

Overend [4] in 1896 wrote:

'When the skin of the forehead is gently tapped with the edge of an ordinary wooden stethoscope, a twitch in the lower eyelid of the same side may be observed ... severe percussion elicits a simultaneous movement of the opposite lids ... It is a true skin

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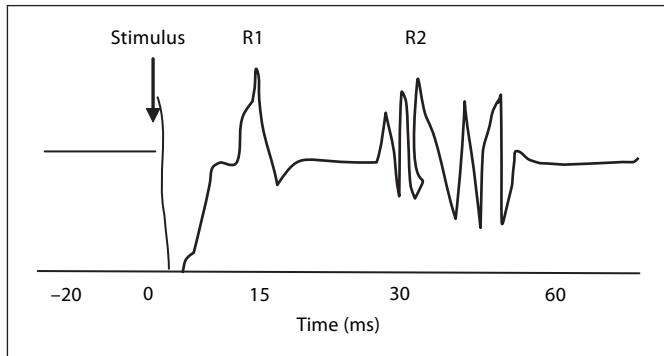


Fig. 1. Schema of the blink/glabella reflex.

reflex and the motor pathway is identical to the conjunctival reflex; the sensory channels lie in the ... supraorbital division of the frontal nerve and the centre is probably located in the midbrain.'

He noted that the reflex was evoked from a wider area of stimulus in choreics.

In 1901 and 1902, Daniel J. McCarthy (1874–1958) [7, 8], Professor of Medical Jurisprudence at Philadelphia, reported that tapping the skin over the supra-orbital ridge elicited bilateral contraction of the orbicularis oculi and deduced that this was a 'pure nerve reflex' identical to tendon reflexes. He wrongly claimed priority. He commented that division of the supra-orbital nerve abolished the blink reflex, and, therefore, the afferent limb was through the trigeminal nerve, confirmed by sectioning the sensory root of the Gasserian ganglion which also abolished the reflex.

The paper by Fine and Vicente [9] notes that in 1902 Vladimir Mikhailovich Bekhterev (1857–1927), Russian neurologist and psychiatrist (after whom the superior vestibular nucleus was named), described orbicularis oculi contraction stimulated by a stroke on forehead, temple, or cheek [10]. Bekhterev overlooked Overend's report [4] and disputed McCarthy's claim [7] for priority.

Mechanisms

Erik Kugelberg [6], some 50 years later, electrically stimulated the supra-orbital nerve and recorded the early-latency R1 and the late-latency R2 electromyographic responses from orbicularis oculi (fig. 1). The blink reflex, he said, was:

'“A true skin reflex”: pain and light touch were adequate stimuli for the R2, but inadequate for the R1 for which “distortion of skin or underlying structures are apparently required”.'

The R1 component was thought to be myotatic. Both R1 and R2 were delayed or abolished by lesions of the trigeminal nerve, in keeping with Overend's original observations [4]. But the central nexus was in the pontomedullary region, not the midbrain, as Overend had suggested.

Shahani and Young [11] showed that the latency of the first component is variable, briefer in duration, and more consistent in shape and size than the R2:

'The first component, as well as the second, is cutaneous rather than “proprioceptive” in nature, as suggested previously. The second component of this reflex has been shown to correlate with closure of the eyelids. The significance of the first component remains to be elucidated, but it appears not to be related to afferent fibers from facial muscles.'

Electrical stimulation of the supra-orbital nerve evokes the trigeminofacial reflex, consisting of an early R1 (oligosynaptic) component on the ipsilateral side with an onset latency of 9–12 ms; R2, a bilateral (polysynaptic) polyphasic burst, latency 25–35 ms, and R3, a bilateral polyphasic component with a latency of 70–90 ms. Different methods of stimulus and response measurements are important in research [12].

R1 is a pontine component, and R2 is a medullary-C1 component [13]. R1 and R2 can be evoked by innocuous stimuli via A β -afferents, but the R2 response is also triggered by pain or heat, suggesting that the R2 is mediated by wide dynamic range neurons of the spinal trigeminal nucleus [14].

Interestingly, the human blink, elicited by a glabella tap, can be inhibited, if a relatively weak acoustic signal precedes the tap by about 100 ms [15]. Nicotine decreases the magnitude of the orbicularis oculi electromyogram and increases selectively the latency of the long-latency (R2) component, probably by causing dopamine release in the striatum [16].

Autopsy of 2 patients, one with a lesion at the dorsal lateral tegmental field and the second with a lesion of the dorsal lower medulla oblongata, indicated that crossed and uncrossed ascending trigeminofacial connections are mediated through the lateral tegmental field. The uncrossed trigeminofacial connection originates at the level of the lower medulla oblongata, and the contralateral R2 response is established via an ascending pathway which crosses the midline at the level of at least the lower third of the medulla oblongata [17].

Clinical Features

The blink reflex [18] is one component of the startle reflex in normal subjects. When the glabella is tapped lightly with a fingertip or reflex hammer, a brisk blink reaction is seen bilaterally which repeats and is fatigable in normal subjects (adults show 2–5 blinks). The blink reflex is equivalent [6] to this glabella tap reflex.

In unilateral trigeminal nerve lesions, all three electrical responses are affected. In unilateral facial palsy, stimulation on the same side of the lesion will result in delayed or absent direct and indirect responses ipsilaterally, but in a normal indirect response contralaterally. When the nerve is stimulated on the healthy side, both the direct and indirect responses are spared, while the contralateral indirect response is affected. The absent corneal reflex is another useful sign of a trigeminal V1 afferent lesion, e.g., acoustic tumour or efferent facial nerve palsies.

The related glabella tap sign is non-specific, being abnormal in diffuse cerebral hemisphere degenerative and

vascular diseases and in parkinsonism and senescence. There is evidence that these suprasegmental influences upon the reflex derive from cortex and basal ganglia, sites at which damage will modify the responses. Pearce et al. [19] reported that 19 of 20 patients with parkinsonism failed to show fatigue of the blink response to glabella tap. They also reported persistence of the reflex after continued tapping in 13 of 56 patients with parenchymal brain disease (senile and presenile dementia, cerebral anoxia). In contrast, all of their 23 normal control subjects showed fatigue of the reflex after 2–5 glabella taps. But the test is not diagnostic.

A possible explanation is the disordered output from the basal ganglia to the pontine tegmentum nuclei, so that dopamine depletion in the basal ganglia increases reflex blink excitability. Single subthalamic nucleus deep brain stimuli inhibit the R2 phase of the blink reflex in Parkinson's disease [20]. This finds support in the increased blink rate or in the absent glabella reflex in nearly one third of untreated schizophrenics [21, 22].

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