ORIGINAL ARTICLES

Prandial presbyopia: the muffin man

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Abstract

Transient blurring of near vision can be due to a variety of causes. We report the case of a 35-year-old man with a 10-year history of blurring of near vision that begins 30 to 45 seconds after he starts to eat and that lasts until 10 to 15 minutes after he stops eating. Magnetic resonance imaging and computed tomography of the brain and orbits did not reveal any abnormality, and stimulation of individual cranial nerves did not result in a loss of near vision. Retinoscopic refraction revealed the loss of 1.5 dioptres of accommodative power in each eye one minute after he began to eat. To the best of our knowledge such blurring of vision at near, immediately after initiating a meal, has not been previously reported. The neuroanatomy of the accommodation and of the gustatory pathways are discussed, as they may relate to this patient's visual complaint.

Transient blurring of vision at near can be due to a variety of causes, including gaze evoked amaurosis, amaurosis fugax, transient obscurations of vision due to optic disc anomalies, dynamic accommodation insufficiency, migraine, and convergence insufficiency. Gaze evoked amaurosis is related usually to the presence of an orbital mass lesion. Amaurosis fugax is usually monocular, often associated with a vascular aetiology, and found in older patients. Migraine is typically associated with a visual phenomenon, such as a fortification scotoma. Acquired convergence insufficiency typically presents as diplopia at near and is often related to trauma or to a systemic illness such as diabetes. Dynamic accommodative insufficiency is found usually in younger patients, many of whom have no other abnormal physical findings.

We describe an adult male who presented with blurring vision at near that begins immediately after he begins eating. The neuroanatomy and physiology of the accommodative pathway is discussed as it may relate to this patient's ocular condition.

Case report

A 35-year-old man was referred to the Doheny Eye Institute with a 10-year history of blurring of vision when eating. The patient stated that his vision begins to blur 30 to 45 seconds after he starts eating and remains blurry for 10 to 15 minutes after he ceases eating. Many foods provoke this, but particularly muffins, fruits,

and sour foods. He has had no other ocular complaints, but he gave a history of head trauma in a fall from a catwalk in 1968. He reported a transient loss of consciousness at that time, but no examination was performed. He is on no medications, denies tobacco use, and reports drinking only a couple of alcoholic beverages at weekends. There is no history of toxic exposure, and the family history contains nothing relevant to his condition.

On physical examination the patient's visual acuity at distance was 20/20 in the right eye and 20/25-2 in the left eye. He had a near point of accommodation of 3 cm in the right eye and 4 cm in the left. He also had a near point of convergence of 4 cm. He could discern 14 of 14 American Optical test plates with each eye. The results of brightness sense testing, Amsler grid testing, tangent screen testing, and orbital examination were normal in each eye. The pupils were briskly reactive to light, and there was no afferent pupillary defect. The findings on slit-lamp biomicroscopy and dilated fundus examination were normal in each eye. Ocular motility examination revealed 10 dioptres of right esophoria at both distance and near. The patient also had minimal lid lag in the right eye. A general neurological examination, including the cranial nerves, gave results within normal limits.

Magnetic resonance imaging as well as computed tomography of the brain and orbits did not reveal any abnormalities. The patient's distance visual acuity was unchanged until he began to eat, at which time it decreased to 20/40 in each eye. However, he was refracted to 20/20 in each eye with the following correction: OD, +1.00+0.50 $\times$ 90, and OS, +0.75+1.00 $\times$ 90. More remarkable was his loss of vision at near, which went from Jaeger 1+ before eating to Jaeger 7 after eating. However, he could see Jaeger 1+ with the aid of a +3.00 lens over each eye. During a subsequent visit an attempt was made to elicit the loss of near vision by stimulation of individual cranial nerves. Following cranial nerve stimulation, baseline retinoscopy was performed, after which he was allowed to eat a large meal, during and after which his retinoscopic refraction at distance and near was monitored.

Results

Stimulation of individual cranial nerves did not result in a loss of near vision (Table 1). However, one minute after beginning to eat the patient could read at near, without correction, only

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Table 1 Effect of cranial nerve stimulation on near vision

Task*	Cranial nerve stimulated	Effect
Smell cloves	I	None†
Sustained upgaze	III	None
Sustained downgaze	III	None
Mastication	V	None
Taste salt on anterior 2/3 of tongue	VII	None
Taste grapefruit juice on posterior 1/3 of tongue	IX	None
Drink water	IX,X	None

*All tasks were performed for 2 minutes.

†None=no change from baseline visual acuity at near, as assessed by Rosenbaum card.

Jaeger 10 with the left eye and Jaeger 7 with the right eye. Fifteen minutes after finishing the meal he could read Jaeger 5 with the left eye and Jaeger 3 with the right. Thirty minutes after finishing the meal his visual acuity at near had returned to Jaeger 3 in the left eye and Jaeger 1+ in the right (baseline). These results were comparable to the changes in his accommodative power (Fig 1). Furthermore, the patient's visual acuity at distance was only slightly affected by eating, going from 20/20 OD and 20/25 OS before eating to 20/25 OD and 20/40 OS one minute after he began to eat. His retinoscopic refraction at distance was also only minimally affected by eating. No change in pupil size, reactivity to light, or reactivity to near was noted throughout the course of the examinations.

Discussion

Blurring of vision at near immediately after initiating a meal has never to our knowledge been reported. Normally with accommodative effort caused by either a blurred retinal image or conscious visual fixation on a near object of regard a 'near synkinesis' is evoked, including increased accommodation of the lens,¹ convergence of the visual axes of the eyes,² and pupillary constriction.³ A defect in any one of these elements could result in poor near visual acuity.

Figure 1 Graph of accommodative power (vertical axis), defined as distance of retinoscopic refraction minus near retinoscopic refraction (in dioptres of sphere), versus time (horizontal axis). Note the almost total loss of accommodative power one minute after beginning to eat. The dioptres of cylinder, as well as the cylinder axis, remained unchanged throughout the examination (not shown).

However, there was no evidence of convergence insufficiency or pupillary abnormalities in our patient, though retinoscopic refraction at near showed a loss of +1.5 dioptres of sphere in each eye one minute after he started to eat. This loss of accommodative power of the lens returned approximately 30 minutes after the meal was finished. Several explanations for a bilateral, transient loss of accommodative power of the lens related to eating could be considered.

The patient did give a history of head trauma, though patients who suffer posterior cortical injury typically have a permanent bilateral accommodative paresis.¹ Ocular trauma can also result in a loss of accommodative power, but this is ipsilateral to the injury, and therefore unlike that seen in our patient.

A synkinesis between the oculomotor nerve and another cranial nerve could be postulated as a cause of this patient's symptoms, but it would be extremely unlikely given the bilaterally symmetric nature of the accommodative loss. In addition, stimulation of individual cranial nerves did not evoke a loss of accommodative power in this patient. Moreover, one would expect stimulation of the ciliary fibres of the oculomotor nerve, via aberrant regeneration from another cranial nerve, to result in accommodative spasm, not paresis.

Review of the neuroanatomy of the accommodation pathway, as well as that for eating, swallowing, and digestion, may provide a clue to our patient's problem. The neural mechanisms of the accommodation pathway are not well understood, but it is likely that awareness of decreased distance of the object viewed evokes accommodative effort that originates in frontal centres; blurred retinal images are sensed in the prestriate cortex and are corrected via occipitotectal tracts.² A midbrain centre for accommodative vergence has not been shown anatomically but it is suggested on theoretical grounds.³ However, the Edinger-Westphal nucleus in the rostral midbrain has been stereotactically mapped and may be divided functionally into a rostral part concerned with accommodation, a caudal part concerned with pupillary constriction, and an intermediate area that, when stimulated, results in both accommodation and pupillary constriction.⁴ The parasympathetic fibres that innervate the ciliary body originate in the rostral part of the Edinger-Westphal nucleus and then course via the oculomotor nerve, the ciliary ganglion, and the short ciliary nerves. Stimulation of these fibres evokes constriction of the ciliary body, relaxation of the zonular fibres, and thickening of the lens, resulting in accommodation. This neuroanatomical pathway is far removed from the glossopharyngeal and vagal nuclei, which are located in the medulla and are involved in swallowing as well as in gastric secretion and motility.

It is intriguing, however, to speculate that the hypothalamus may provide the mediating pathway. The paraventricular hypothalamic nucleus (PVN) is believed to play an important part in the feeding mechanism. Feeding behaviour in satiated rats can be elicited by injecting a noradrenergic agonist into the PVN.⁵ Furthermore, paraventricular nucleus activity can be

modulated by electrical stimulation of the central stump of the vagus.⁶ It is likely that the PVN sends efferent fibres to the dorsal motor nucleus of the vagus, from which the vagus nerve originates.⁷ Afferents from the mouth, via cranial nerves IX and X, reach the paraventricular nucleus via the nucleus of the tractus solitarius.⁸

The PVN has been shown by autoradiographic tracing in cats to send efferent fibres to the Edinger-Westphal nucleus,⁹ and in another study electrical stimulation of the diencephalon of sympathectomised cats and monkeys caused pupillary dilatation and abolished the light reflex.¹⁰ This suggests the existence of a direct hypothalamic-mesencephalic pathway for inhibition of the Edinger-Westphal nucleus that may inhibit accommodation as well.

Eating (feeding) may thus result in stimulation of the PVN via vagal input to the nucleus of the tractus solitarius. Aberrant stimulation of the PVN may lead to firing of inhibitory fibres projecting to the rostral portion of the Edinger-Westphal nucleus. This could, at least theoretically, result in a loss of accommodation such as that seen in our patient. This aberrant pathway

may have been present since birth, and may have become symptomatic only as the patient aged and his accommodative reserve diminished.

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