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Prandial Presbyopia

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Abstract: Loss of accommodation amplitude during eating (prandial presbyopia) is a rare phenomenon that has been reported in only 1 patient who had had head trauma. We report 2 patients who had not had head trauma and whose accommodative amplitudes, measured by dynamic retinoscopy, became markedly diminished within 1 minute of starting a meal and did not recover for 55–60 minutes. Apart from this abnormality, there appeared to be no autonomic or other neurologic dysfunction. The cause of this isolated disturbance in these patients is a mystery.

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Prandial presbyopia is characterized by transient blurring of near vision immediately after initiating a meal. It has been described only once before in a single patient who had previously sustained head trauma (1). We describe 2 patients with prandial presbyopia unassociated with head trauma or other neurologic disturbance.

CASE REPORTS

Case 1

A healthy 42-year-old man presented with a 9-month history of blurred vision when eating. It occurred within 1 minute of his beginning a meal and generally persisted throughout it. He was unable to see his food clearly and could not read the bill at the end of a restaurant meal. Such blurred vision was related exclusively to eating and drinking, and there was no pertinent personal or family history.

Best-corrected visual acuity was 20/20 in both eyes with a distance correction of +2.50 sphere in the right eye and +1.50 sphere in the left eye. Results of a complete ophthalmologic and neurologic examination, including dilated ophthalmoscopy and cranial nerve and orthoptic evaluation, were normal. There were no abnormalities in his cardiovascular response to variations in posture, ambient temperature, and exercise level either before or just after a meal. Infrared pupillometry demonstrated no abnormalities in the amplitude or latency of the pupillary response to light or a near target before, during, or after a meal.

An investigation of the patient's accommodation was performed immediately before, during, and after a test meal. Heart rate and blood pressure were assessed, and amplitude of accommodation was measured by the "push-up" technique and a Royal Air Force (RAF) rule modified to measure up to 2 m. With the patient wearing his distance correction, each eye was tested using the N5 (20/30) test print, which was gradually brought as close to the patient as possible until the image was reported as blurred. Convergence was tested with a near stimulus at 15 cm. All measurements were taken just before and 1, 5, 10, 15, 25, 40, and 55 minutes after the patient began the meal. Blood glucose was measured before the meal and at 5, 15, and 55 minutes after initiation of the meal.

Accommodative amplitude fell rapidly within 1 minute of the beginning of the meal, reaching its nadir after 10 minutes and returning to the preprandial level at 55 minutes after the meal was begun (Fig. 1). During the test period, the symptoms experienced by the patient were typical of those during previous untested episodes. Apart from accommodative amplitude, all tests yielded normal results. F1

Case 2

A 34-year-old man had a 10-year history of blurred vision during eating. He stated that the symptoms occurred every time he ate, irrespective of food type. He complained that the blurring would begin within 1 minute of eating and continue for about 15 minutes afterwards. The blurred vision episodes had become more pronounced and longer in duration over the last few months. He did not complain of diplopia or any other ocular or systemic symptoms. There was no history of head trauma. There was no pertinent past medical history, and he was not taking any medications. He did not drink alcohol or smoke cigarettes.

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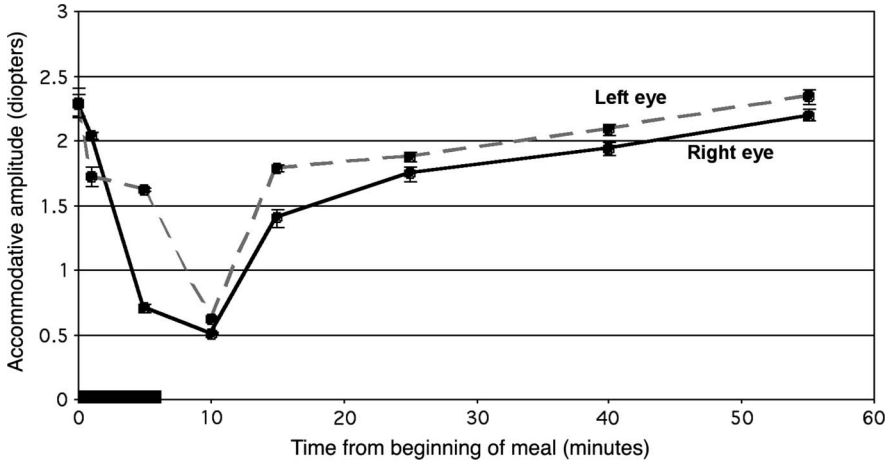

FIG. 1. Case 1. Accommodative amplitude related to time after beginning a meal. The horizontal black bar represents the time taken to complete the meal. Mean values are plotted with 95% confidence intervals represented by error bars.

Visual acuity was 20/20 in both eyes at 20 feet and 20/30 (Snellen equivalent) at 35 cm. He had a near point of convergence of 6 cm in both eyes and a near point of accommodation of 20 cm in both eyes. Visual fields were full to confrontation. Pupil size and reactivity were normal without a relative afferent pupillary defect. Color vision was intact in both eyes with Ishihara test plates. Ocular movements, ocular alignment, and results of ophthalmoscopy were normal.

Distance and near visual acuity, convergence, and dynamic retinoscopy were performed immediately before and after eating. Dynamic retinoscopy involved positioning a small letter chart close to the retinoscope peephole and holding the retinoscope-small letter chart complex at a distance of 35 cm from the patient. The patient was then asked to alternate fixation between the distance and the near fixation targets while retinoscopy was performed. The patient was then asked to maintain fixation on the near target to assess the sustainability of accommodation.

Immediately before eating, dynamic retinoscopy revealed +1.00 sphere in the right eye and +1.50 sphere in the left eye with the patient fixating at distance and 2.50 sphere in both eyes with the patient fixating at 35 cm. This accommodative effect was rapid, complete, and steady. At 1 minute after a meal was initiated, visual acuity was 20/20 and reading vision had been reduced to 20/400 unaided in each eye at 35 cm. Dynamic retinoscopy with the patient fixating at distance revealed a +1.50 diopter sphere in both eyes and remained constant when the patient fixated at 35 cm. Near vision improved to 20/30 in both eyes with a +1.50 diopter sphere in each eye.

The results of preprandial dynamic retinoscopy for distance and near noted immediately before eating were restored 60 minutes after the meal was initiated.

The patient was given a diagnosis of prandial presbyopia and was prescribed +1.50 diopter sphere reading glasses for use when eating.

DISCUSSION

In both patients there was a reduction in the ability to accommodate initially after eating a meal, demonstrated through the techniques of dynamic retinoscopy and measurement of accommodative amplitude.

The neural control of the accommodative component of the near triad is thought to lie in the rostral part of the Edinger-Westphal nucleus. Its caudal portion coordinates the association with pupillary constriction (2). AU1

The mechanism of accommodative failure during eating is a mystery. A change in lens volume induced by a change in osmolality of the aqueous would be too slow to cause the rapid onset of the deficit in this setting. Without a history of head trauma, a central failure of synkinesis seems unlikely too.

The single previously published report of prandial presbyopia (1) involved a 35-year old man with a 10-year history of blurring of near vision that began 30–45 seconds after he began to eat and lasted 10–15 minutes after he stopped eating. He reported that many foods exacerbated the phenomenon, particularly muffins, fruits, and sour foods. The patient did give a history of head trauma when he was 15 years old after a fall from a catwalk, but no examination or investigation were performed at that time. Retinoscopic refraction revealed a loss of 1.5 diopters of accommodative power in each eye 1 minute after he began to eat. In that report, brain and orbit MRI and CT did not reveal any abnormality. Stimulation of individual cranial nerves alone did not result in the loss of near vision. The main differences between this patient and the 2 patients

reported here are that our patients had no history of trauma, there was no food-specificity, and there was a longer course of accommodative paresis.

As an explanation for the prandial presbyopia, the authors of the single previous case report (1) suggested an abnormal pattern of activity in a pathway between the paraventricular nucleus of the hypothalamus associated with eating and the part of the Edinger-Westphal nucleus that controls accommodation. They hypothesized that their patient was at an age when his accommodative reserve had diminished, allowing a pathway that was probably present since birth to become symptomatic. The lack of any obvious cause for an acquired abnormality in our patients and their hypermetropia would be consistent with this theory.

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