Short report

Seizures triggered by blinking in a non-photosensitive epileptic

RD RAFAL,* KD LAXER,† JS JANOWSKY‡

From the Roger Williams General Hospital, Division of Neurology and Brown University, Providence, RI, Clinical Neurophysiology Laboratory, Good Samaritan Hospital, Portland, OR,† and University of California, San Diego, CA,‡ USA*

SUMMARY An epileptic girl with Lennox-Gastaut syndrome had seizures triggered specifically by blinking, but not by other eye movements or by photic stimulation. Electrographic and clinical seizures were most reliably precipitated by repetitive blinking produced voluntarily on command, by reflex blinking on corneal stimulation, or by psychogenic triggers of blinking such as social stress or cognitive effort.

The characteristic association of rhythmic eye blinking with the petit mal absence attack suggests that the neural substrate for blinking shares some common pathway with that involved in the generation of the corticoreticular epileptic discharge. Typically, the onset of the spike wave discharge precedes blinking,¹ but there have been several reports where blinking or eye closure have preceded or precipitated seizures.²⁻⁶ Many of these patients were photosensitive, and blinking may have activated epileptic discharges indirectly through a photoconvulsive response. In others the trigger appeared to have been oculomotor but was not specific to blinking, since sustained eye closure was also effective. We report a non-photosensitive epileptic patient in whom blinking specifically precipitated generalised electrographic and clinical seizures. A free-ranging EEG video telemetry technique was applied to the study of this phenomenon, and to its relation to stress.

Case report

This 15-year-old girl, with Lennox-Gastaut syndrome and intractable seizures from the age of five years, functioned intellectually as a four year old. She had numerous convulsive and non-convulsive seizures every day in spite of therapeutic doses of primidone, carbamazepine, phenytoin, phenobarbitone, and clonazepam. Her convulsive seizures

usually occurred during sleep or on awakening. Under stress of family strife or pressure in school it was common for her to have dozens of minor seizures each hour consisting of absences accompanied by blinking and myoclonic jerks of the face and extremities. Her EEG revealed slowing of background activity which was frequently interrupted by irregular, generalised, slow spike-wave paroxysms. There was no photoconvulsive response. During her convulsions, continuous, generalised polyspike and slow-wave discharges were present accompanied by rhythmic blinking.

Method

The general methods and applications of our free-ranging radio frequency EEG video-telemetry technique for the dynamic study of epileptic children has been described elsewhere.⁷ The patient's behaviour was monitored with a mobile television camera. Simultaneously, four EEG channels were displayed by video-reformer and eight were recorded on paper print-out. Typically a parasagittal montage was utilised, with a band pass of 1-70 Hz (-3dB).

Scalp electrodes were placed according to the 10-20 International system and applied with collodion. Electro-oculogram (EOG) was monitored with electrodes placed above and below the right eye, and adjacent to the outer canthus of the right eye. Blink artifact was distinguished from epileptogenic cortical activity on the basis of their differing topographical distribution, and by the fact that there was a latency difference between the epileptic discharges and the blink artifact during those segments of the record where the two activities were associated.

After informed consent was obtained from the patient and her mother for these studies, eight 4-8 hour recordings were conducted on a childrens neuropsychiatric ward over a three month period. Parts of these recordings were structured such that mental effort was systematically manipulated using

Address for reprint requests: RD Rafal, MD, Roger Williams General Hospital, Department of Medicine, 825 Chalkstone Avenue, Providence, Rhode Island 02908, USA.

Received 26 July 1985. Accepted 1 September 1985

Fig (A) Isolated blink triggered by an epileptiform transient, (B) An epileptiform transient triggered by an isolated spontaneous blink, (C) Recruitment of epileptiform discharge by rhythmic blinking. Calibration 1 s, 100 μ V.

standard academic and psychometric tests of variable difficulty. In addition, observations were made of the effects of social stress as it occurred spontaneously on the ward, for example embarrassment, frustration, infatuation. No manipulations were contrived deliberately to cause the patient pain, embarrassment, or mental stress beyond that which she experienced on a day to day basis on the ward or in classroom type activities.

Results

A clear linkage between blinking and clinical, and electrographic seizures was consistently demonstrated during the period of study. Isolated epileptic transients were occasionally followed by a single blink (fig

A). Just as frequently an isolated spontaneous blink was followed by an epileptic discharge (fig B). Most commonly, however, epileptiform discharges were precipitated by repetitive blinking which resulted in increased sharp activity, and in the recruitment of rhythmic slowing culminating in spike and wave discharges (fig C). Absence seizures often accompanied these discharges. Epileptiform activity could easily be elicited by voluntary blinking on command in the light and in the dark (either blindfolded or in a blackened room), and by reflex blinking on repetitive corneal stimulation with puffs of air from an otoscope bulb. The latency for the first spike from a preceding blink was 100–300 ms. The phenomenon was specific for blinking and could not be elicited by sustained eyelid closure or by other eye movements.

The most effective activator of electrographic seizures was blinking triggered by social stress or cognitive effort. In structured recordings where cognitive stress was manipulative systematically, repetitive blinking triggered electrographic seizures with a probability of 70–90% in all recording sessions. On a digit-span task, the successive increase in the number of digits to be recalled resulted in an increase in blinking; her EEG became increasingly sharper culminating in frank spike-wave discharges, often with clinical absences. Between digit-span trials, both blinking and epileptiform activity were inconspicuous. During a paired associate learning task, blinking and associated epileptiform discharges occurred more often with the difficult to recall association. No verbal response by the patient was necessary to produce the effect: for example, it occurred when she was asked simply to “think of” a colour name for the examiner to “guess”. In addition to stress from cognitive tasks, social stress was an effective activator. The patient had an infatuation with one of the male staff. It was only necessary for him to chat with her casually for her to begin blinking with resulting epileptiform activity and occasional clinical seizures.

Discussion

These observations complement a growing literature linking pathways subserving eye closure response with those recruited by corticoreticular epileptic discharges.^{1–5} To our knowledge this is the first reported case in which blinking specifically, as distinct from eyelid closure in general, has been shown to be an activator of epileptic activity. Since blinking activates the same efferent and afferent cranial nerves as does sustained eye closure, it appears that the trigger linking eye closure to epileptic pathways in our patient was at a supranuclear level, modulating blinking specifically.

The supranuclear control of blinking appears to

have a physiologic substrate separate from that mediating sustained eye closure. In contrasting the electromyographic features of blinks compared to sustained eye closure, Gordon identified motor units in the pretarsal region of the lid which were specifically activated by blinks.⁸ Although it seems unlikely that a complex and independent mechanism for blinking should have evolved simply to moisten and protect the eyes, little is known about the other functions of blinking. Volkmann demonstrated that a decrement in visual perception, similar to that which occurs with saccadic suppression, accompanies blinks.⁹ It has been suggested that blinking may contribute to the regulation of circadian rhythms and other biological clocks by periodically pulsing the mesencephalic (extrageniculate) visual system with red light.^{10 11}

Blinking also functions as a "complex indicator" of phasic responses to stress such as that produced by listening to emotionally laden words.¹² Because blinking, in our patient, provided us both with a direct window on her responses to stress, and also appeared to cause her epileptic discharges, we had a special opportunity to study the relationship between stress and epilepsy. We sought to determine whether blinking was specific in linking pathways activated by stress with those involved in the generation of epileptic activity. We found that stress, related to a variety of mental and environmental factors which produced blinking, was an effective epileptic activator.

This patient bears some resemblance to photosensitive patients with self-induced epilepsy.⁶ Typically, this syndrome occurs in retarded epileptic children with photosensitive epilepsy. Their seizures may be self induced not only by blinking and waving their hands in front of their eyes, but also by sustained, forceful eyelid closure.¹³ Often, the seizure-generating behaviour is reinforced in these patients because the resulting seizures are experienced as pleasurable, or because they serve as an escape from stressful situations. Our patient differs from these individuals in that she was not photosensitive, and because her seizures were induced specifically by blinking rather than by eyelid closure or photic stimulation. Furthermore, her seizure activity behaviour did not appear to be wilful or consciously generated.

In this patient, the well recognised association between blinking and generalised epileptic discharges has been found to be a two-way street; their common

pathways are reciprocally linked such that activation of either facilitates the other.

This work was supported by a Comprehensive Epilepsy Program grant from NINCDS, The Good Samaritan Hospital, Portland, Oregon (Contract No. INS-5-2328). Dr James Schimschock kindly referred this patient to us. We are grateful to Drs Marvin Greenbaum and Joan Kelly who provided psychological supervision for this work, and to Dr Janice Stevens for her guidance in conducting the study. We are grateful to Bonnie Becker and Corrine Hopp who typed the manuscript.

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