Short communication

14 and 6 Hz positive spikes preceding 3 Hz generalized spike and wave in a 15 year old patient with absence: a case report

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Abstract

A 15 year old girl with a history of childhood absence seizures underwent a prolonged EEG recording. Twelve bursts of 3 Hz generalized spike and wave were recorded during drowsiness and light sleep, all of which were preceded by 14 and 6 Hz positive spikes with a latency of under 1 s. Nine bursts of polyspike-wave, not preceded by 14 and 6 Hz positive spikes, were recorded during deeper sleep (stage 3). The bursts of polyspike-wave were significantly longer when preceded by 14 and 6 Hz spikes than when observed in isolation. The close association between these paroxysmal events in our patient is intriguing, may be coincidental, and has never before been reported.

Keywords: 14 Hz positive spikes; 6 Hz positive spikes; Three per second spike-wave; Generalized discharges; Sleep stage

1. Introduction

14 and 6 Hz positive spiking is an electroencephalographic phenomenon first described by Gibbs and Gibbs in 1951 (Gibbs and Gibbs, 1951) as bursts of arch-shaped surface positive waves at 14 and/or 6 Hz. It is seen best during drowsiness and light sleep in the posterior temporal leads of a monopolar recording using an ipsilateral ear reference (Lombroso et al., 1966). Currently its exact origin within the central nervous system is unknown. It was originally believed to be associated with headache, head trauma, epilepsy, behavioral disorders, vomiting and dizziness (Gibbs and Gibbs, 1963); however, it has since been accepted as a pattern appearing in normal children and adolescents (Lombroso et al., 1966).

We describe a patient with absence seizures whose bursts of 3 Hz generalized spike and wave are preceded by 14 and 6 Hz positive spiking with a consistent latency of less than 1 s. This association was seen only during drowsiness and light sleep, and occurred on two EEGs in 1997, as well as on one done in 1996.

2. Materials and methods

2.1. Subject

A 15 year old girl has had a history of absence seizures since the age of 7. She is the product of a normal 38 week gestation and a normal spontaneous vaginal vertex delivery. At the age of 7, the patient’s mother noticed pauses in her speech, after which she would pick up where she left off, or lose her train of thought. These episodes lasted 2–3 s, during which there was eye fluttering as well as automatisms of fumbling of the hands. There was no impairment of consciousness following the episode and they occurred more often when she was tired. The patient was brought to medical attention when the spells increased in frequency from one to two per week to nearly daily, and began to cluster. A complete neurologic exam was non-focal and a typical episode was produced with hyperventilation for 3–4 min. The patient was subsequently placed on ethosuximide.
2.2. Data acquisition and analysis

A recent 21 channel recording contained 6 bursts of 3 Hz generalized spike and wave activity during drowsiness, all of which were preceded by 14 and 6 Hz positive spiking with a latency of less than 1 s. The patient returned a few days later, with no change in her medication regimen, for a 1 h and 45 min EEG. Electrodes were applied in the standard manner using the international 10–20 system.

All impedances were below 5 kΩ. EEG was recorded using bipolar, transverse, and referential montages on a digital EEG machine (Nihon Kodon Neurofax EEG 2100). Hyperventilation was performed 4 times during the recording for 4 min each time. The patient was permitted to sleep without sedation for 45 min of the recording time. No behavioral changes or absence seizures were noted during the recording.

The record was viewed entirely in the referential montage using an ipsilateral ear reference in addition to an averaged reference. Using a cursor, 14 and 6 Hz positive spikes were measured in duration, regardless of association with generalized discharges, from the first positive peak to the last positive peak. A burst was defined as greater than two spikes or polyspikes occurring in a time frame of less than 300 ms. Latency was measured as the distance from the first positive peak of the 14 and 6 Hz spikes to the first peak of the negative spike of the generalized epileptiform burst, if present. The duration of the epileptiform bursts was measured from the peak of the first negative phase of the first spike to the peak of the negative phase of the last slow wave present in the burst. A side to side predominance of the 14 and 6 Hz positive spikes was estimated visually. If the latency from onset of 14 and 6 Hz spiking to the onset of the epileptiform discharges was less than 1 s, the two events were labeled as associated. Discharges during hyperventilation were measured, but were excluded from counts because the patient was awake, and identifying 14 and 6 Hz spiking was difficult.

3. Results

14 and 6 Hz spiking in isolation occurred 92 times during the course of the recording, 53% in stage 1 sleep, 40% in

![Fig. 1.](image-url)

Fig. 1. (A) A burst of 3 Hz generalized spike and wave during drowsiness preceded by 14 and 6 Hz positive spiking by less than 1 s. The 14 and 6 Hz positive spikes are best seen at the F3, O1, and T6 electrodes. The vertical divisions represent 1 s marks. (B) A burst of generalized polyspike and wave during stage 2 sleep that is not preceded by 14 and 6 Hz positive spiking.
stage 2 and 7% in stage 3 with an average duration of 0.57 s (Fig. 1). It occurred in association with a generalized burst of 3 Hz spike and wave 12 times during the recording, 9 (75%) of which occurred in stage 1 sleep. The remainder occurred in stage 2 sleep. The average latency between the 14 and 6 Hz spiking and the epileptiform discharges was 0.48 ± 0.19 s with an average epileptiform burst duration of 2.7 ± 2.4 s. Bursts of epileptiform activity occurring in isolation 9 times were characterized by brief bursts of poly-spike and slow waves with an average duration of 1.25 ± 0.004 s. Seven of these occurred during stage 3 sleep (78%) and the remaining two occurred during stage 2. The bursts of epileptiform activity were significantly longer in duration when preceded by 14 and 6 Hz spikes than when they occurred in isolation ($P = 0.04$, Student’s t test). The 14 and 6 Hz spiking consisted mainly of 14 Hz activity and had a general left-sided predominance, although there was no correlation with the predominant side of the epileptiform activity. A record from 1 year before was examined in the same fashion and was found to contain no 14 and 6 Hz spiking in isolation, but contained two bursts of 3 Hz generalized spike and wave discharges preceded by 14 and 6 Hz spiking with latencies of 0.5 and 0.6 s during stage 1 sleep. No other bursts of epileptiform activity were noted during sleep.

4. Discussion

14 and 6 Hz positive spiking has long been regarded as a benign pattern seen in normal children (Niedermeyer, 1993). Although its relationship to epilepsy has been reported, it was originally considered to be seen least often in absence seizures (Gibbs and Gibbs, 1963). In this particular patient, it was seen frequently in isolation. We think that its association with generalized spike and wave may not always be coincidental because of the consistently short latencies between the 14 and 6 Hz spiking during drowsiness as well as over time, and the uniformity in the given recording.

The epileptiform discharges seen in isolation are different from those seen in association with 14 and 6 Hz spiking for several reasons. First, they are consistently seen in deeper stages of sleep (78% in stage 3), whereas 14 and 6 Hz positive spikes are usually rare and less likely to be activated. They also differ in morphology, consisting mainly of polyspike components rather than the 3 Hz spike and wave seen in lighter sleep. Lastly, they are significantly shorter in duration than the other bursts. The latter two observations have been made previously in patients with absence, in the absence of 14 and 6 Hz spiking, upon moving from stage 2 to stage 3 sleep, although discharges were noted to be less frequent in stage 1 than in stage 2 (Sato et al., 1973).

Experimental models of absence in the cat have demonstrated that stimulation of the thalamic intralaminar nuclei can produce electrographically similar seizures to those observed in the human. These models have been long known to implicate these structures in the generation of the spike wave complex through the mechanism of alternating excitation and inhibition (Pollen, 1964; Pollen et al., 1964). The exact origin of 14 and 6 Hz positive spikes is unknown in humans, but its close associations with 3 Hz spike and wave in this patient suggests a common pathway or mechanism through the thalamus for both events.

The observation of 14 and 6 Hz positive spikes preceding nearly every burst of generalized spike-wave with a latency of less than 0.5 s during drowsiness has never been reported. Although this phenomenon may be coincidental, it is possible that, in this patient, there may be a common generator or pathway between these two entities.

References