

Bradycardia from flash stimulation

Michael Einspenner¹, Donald G. Brunet¹,
Lysa Boissé Lomax^{1,2}, Allison E. Spiller¹

¹ Department of Medicine, Division of Neurology (Clinical Neurophysiology)

² Division of Respiriology, Queen's University and Kingston General Hospital, Kingston, Ontario, Canada

Received April 20, 2015; Accepted August 26, 2015

ABSTRACT – This case study documents a patient who experienced bradycardia brought on by flash stimulation during a routine outpatient EEG recording. The patient had known photosensitive seizures in the past. During this routine EEG, the patient's heart rate dropped to about 12 beats per minute with the EEG displaying slow-delta-frequency waves with no epileptiform spikes or sharp waves. During immediate follow-up, in our emergency department, the patient had a brief asystolic event, followed by bradycardia. Cardiology examinations were normal. We propose that this response was a photic-triggered reflex vasovagal reaction.

Key words: bradycardia, case study, flash stimulation, syncope

Bradycardia during ictal events, although not common, has been well documented (Van Rijckevorsel *et al.*, 1995; Monte *et al.*, 2007). Bradyarrhythmias leading to asystole, are implicated in sudden unexpected death in epilepsy (SUDEP) (Kahane *et al.*, 1999; Leung *et al.*, 2006). Routine outpatient EEG studies in a clinical setting occasionally record spontaneous seizures. However, seizures are more often recorded that are provoked by activation procedures, such as hyperventilation and/or flash stimulation (Verrotti *et al.*, 2012). We present a case where bradycardia was brought on by flash stimulation during a routine outpatient EEG recording in a patient with known photosensitive seizures.

Case study

A right-handed, 23-year-old male patient had a 30-second episode

of pallor and lethargy at the age of 3 months. This led to an EEG, which showed a solitary sharp transient discharge in the left posterior temporal parietal area that was of uncertain significance. Subsequent to this, but prior to high school, he had three episodes of unconsciousness associated with tonic shaking. The shaking lasted less than 10 seconds. One episode was in connection with immunization, the second was after an injury to his hand, and the third occurred after falling off a chair.

At the age of 16, he had a generalized tonic-clonic seizure. The event was precipitated by exposure to a strobe light in a high school physics class. The seizure was described as a feeling of discomfort in his head, followed by a loss of consciousness. His classmates and teachers witnessed sudden stiffening and falling out of his chair, which evolved into a secondary generalized tonic-clonic

Correspondence:

Michael Einspenner
Clinical Neurophysiology,
Connell-7, Kingston General Hospital,
76 Stuart Street, Kingston,
ON, K7L 2V7, Canada
<einspenm@kgh.kari.net>

seizure. He experienced fatigue after this seizure. During the EEG that was planned following the seizure, he reported symptoms during 12-Hz flash stimulation, characterized by headache, and he requested that the flashing be stopped. He was noted to take a few deep breaths and then seemed to stiffen and turn to the right, with the left arm and left leg adopting a “fencing posture” position with the arm pointing towards the wall. His head was hyper-extended. The EEG at that time showed generalized high-amplitude delta frequency waves, maximum in the anterior regions. Coincident with the left arm and left leg stiffening, the EEG exhibited relatively low voltage in the theta frequency band with some artefact. The EKG channel, although noisy, suggested his heart was slowing before his symptoms.

The next EEG, four years later, was normal. Hyperventilation and flash stimulation were unremarkable with no change in heart rate during flash stimulation. He was taking carbamazepine at that time.

Three years later, in March 2014, a routine EEG recording was performed. Up until flash stimulation, the recording was essentially normal with no change in background activity before, during, or after hyperventilation. With flash stimulation, there was background slowing and a change in heart rate. Just before 1-Hz flash stimulation, his heart rate reduced from 92 to 60 beats per minute (bpm). His baseline heart rate in the early parts of the recording varied between 54 and 60 bpm. Changes in heart rate recorded during increasing frequencies of flash stimulation are shown in *table 1*. With 1-Hz flash stimulation, heart rate was reduced to 48 bpm and background EEG activity remained unremarkable. At 3-Hz, the EEG background was unremarkable with a heart rate of 48 bpm. At 6-Hz flash stimulation, heart rate slowed to 42 bpm with further slowing to 24 bpm. At 8-Hz flash stimulation, his heart rate dropped to about

12 bpm (*figure 1*) and he reported that he was getting an aura typical of his usual seizures (during this EEG, he did not elaborate as to what his aura was, but previous times he reported his aura consisting of one or more of the following: “head hurts” and feels “funny”, light headedness, sweatiness, shaking of his hands, and a feeling of pins and needles). Similar heart rates were recorded at 10-Hz flash stimulation. This was followed by a period of marked sinus bradycardia with heart rates in the range of 36 to 40 bpm and at this point, the patient stated that he was starting to perspire and had some chest discomfort. The flashing was stopped and he improved quickly with a heart rate of 60 bpm after several minutes. During the period of marked bradycardia, his EEG showed mainly generalized low-voltage delta frequency waves, which recovered quickly although the bradycardia was more prolonged. There were no epileptiform spikes or sharp waves. There did not appear to be any obvious photic driving response. During drowsiness, as seen in the early part of the recording, minimal theta frequencies were seen in the fronto-temporal areas, maximal on the left side. Because of these observations, the patient was referred immediately to the emergency department (ED) for follow-up. In addition, an ambulatory EEG with extra heart rate leads was arranged, to be started two days after this EEG.

In the ED, the patient was noted to be pale and diaphoretic, and on the monitor was seen to be bradycardic. Blood tests including those for: glucose, blood count, differential, venous blood gas, creatine kinase, electrolytes and troponin I were normal. Lactate was slightly elevated, as was hydrogen ion. Haemoglobin was slightly depressed. Vital signs were unremarkable, and neurological and cardiovascular examinations showed no significant findings. His blood pressure was hypotensive with a systolic pressure of 93 mm Hg. During this measurement, the heart rate paused briefly and was then 26-38 bpm. Further measurement of vital signs showed a blood pressure of 127/72 and pulse of 42-64 bpm with normal O₂ saturation and normal body temperature. When asymptomatic, he was discharged to go home.

The 24-hour ambulatory recording showed a normal EEG in the waking state and all sleep stages. His heart rate during the waking state was approximately 60 bpm but close examination of the EKG showed minor bradycardia at times. During sleep, his heart rate varied from 34 to 85 bpm. There was one brief period of reduced QRS amplitude which may have been artefactual. Flash stimulation was not carried out.

These findings prompted further investigations by our cardiologists. Echocardiogram showed normal left and right ventricular size and systolic function with no significant valvular abnormalities. LVEF (left ventricular

Table 1. Heart rate (bpm) changes during flash stimulation in a routine EEG in a 23-year-old male.

Flash stimulation frequency (Hz)	Heart rate (bpm)
Pre-stimulation	92 down to 60
1	48
3	48
6	42 down to 24
8	12
10	12 then 36-40
Post-stimulation	60



Figure 1. ECG derivation demonstrating bradycardia during flash stimulation.

ejection fraction) was 56%. A 24-hour holter monitor recording showed an average heart rate of 67 bpm, ranging from 33-165 bpm with a normal sinus rhythm. Physical examination showed nothing remarkable. The cardiology examination also indicated that his bradycardia episode was consistent with vasovagal syncope with cardioinhibitory response. Furthermore, it is highly unlikely that this syncope was related to his seizure disorder since these episodes occurred without any prodrome, and he did not have any features to suggest a seizure disorder when he was in the ED.

This patient was a graduate student (in physics) and a lifetime non-smoker with occasional alcohol use. On his mother's side, his grandfather died of a myocardial infarction. On his father's side, his grandfather, who also had a history of seizures, died of a myocardial infarction.

Discussion

This patient reported seizure-like symptoms mainly when exposed to flashing lights. Interestingly, he may have experienced a photic-responsive generalized tonic-clonic seizure in response to photic stimula-

tion at 16 years of age, and subsequently developed a phobia of photic stimulation resulting in secondary vasovagal syncope to flashing lights. He admitted to feeling anxious when seeing strobe lights. When he was taking carbamazepine, he felt "a blanket of comfort" when he was exposed to strobe lights. Nonetheless, exposure to flash stimulation during routine EEGs caused him to experience aura-like symptoms and marked bradycardia. EEG findings did not show epileptiform activity, rather the appearance of slow delta and theta activity during these periods of flash-induced bradycardia. This slow electrical activity would be consistent with decreased perfusion to the brain from the bradycardia. In addition, spontaneous bradycardia occurred in this patient in the ED with no exposure to flash stimulation. Because of the low frequency of his episodes, lifestyle changes to preclude exposure to strobe lights, and his young age, it was felt that pharmacological and cardiac pacing therapies were contra-indicated (Aydin *et al.*, 2010).

We propose that the bradycardia experienced by this patient was not epileptiform in nature but more likely a triggered reflex syncope provoked, at times, by flash stimulation. Upon testing, low flash rates were relatively effective in initiating bradycardia and in the

interest of patient safety, the flashing was stopped. Higher flash rates, such as are present in overhead lighting and TV and computer monitors, might have been as, or even more, effective at producing bradycardia.

We further propose that patients who experience bradycardia from flash stimulation, but who have no obvious EEG changes other than slowing, are not experiencing epileptiform events, rather a photic-triggered reflex syncope. □

Supplementary data.

Summary didactic slides are available on the www.epilepticdisorders.com website.

Acknowledgements and disclosures.

The authors wish to thank Helen Driver for reviewing this manuscript.

The authors have no conflict of interest to declare.

References

- Aydin MA, Salukhe TV, Wilke I, Willems S. Management and therapy of vasovagal syncope: a review. *World J Cardiol* 2010; 2(10): 308-15.
- Kahane P, Di Leo M, Hoffman D, et al. Ictal bradycardia in a patient with a hypothalamic hamartoma: a stereo-EEG study. *Epilepsia* 1999; 40(4): 522-7.
- Leung H, Kwan P, Elger CE. Finding the missing link between ictal bradycardia, ictal asystole and sudden unexpected death in epilepsy. *Epilepsy & Behav* 2006; 9: 19-30.
- Monte CPJA, De Krom MCTFM CPJA, Weber WEJ, et al. The ictal bradycardia syndrome. *Acta Neurol Belg* 2007; 107: 22-5.
- Van Rijckevorsel K, Saussu F, De Barsey T. Bradycardia, an epileptic ictal manifestation. *Seizure* 1995; 4: 237-9.
- Verrotti A, Beccaria F, Fiori F, et al. Photosensitivity: epidemiology, genetics, clinical manifestations, assessment, and management. *Epileptic Disord* 2012; 14(4): 349-62.

TEST YOURSELF



- (1) What dysfunctions and/or mechanisms can be implicated in SUDEP?
- (2) What sort of neurophysiological response may occur during visual stimulation?
- (3) What other visual stimuli can trigger a photosensitive response?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".