

Epileptic seizures provoked by bathing with water at room temperature

Ruzica Kravljanac¹, Milena Djuric¹, Maja Milovanovic²,
Vlada Radivojevic²

¹ Institute for Mother and Child Health Care of Serbia, Faculty of Medicine,
University of Belgrade

² Institute for Mental Health, Belgrade, Serbia

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ABSTRACT – We report two Caucasian boys with seizures induced by bathing in lukewarm water. Different mechanisms of provocation were observed; in one boy a complex partial seizure was provoked by pouring water over the body, while in the other boy, a complex partial seizure with secondary generalisation was provoked by immersion. Since the water was not hot in either of the cases, the pathophysiological mechanism was not clear and the seizures could not be explained as hyperthermic-related events. We suggest that in the ILAE classification of epilepsies and epileptic seizures, bathing epilepsy should be added as a separate category, distinct from “hot-water epilepsy”. [*Published with video sequences*]

Key words: water immersion, seizure, bathing

Bathing epilepsy, synonymously called *water immersion epilepsy*, has been described in children worldwide, with a median age at onset of seven months, presenting with focal seizures and precipitated by bathing in water at “normal” temperature (36-38°C) (Nechay and Stephenson, 2009). Seizures provoked by immersion or bathing in water at room temperature are regarded as a specific type of reflex epilepsy (Jansen *et al.*, 2010). The exact mechanisms of these rare seizures are still unrevealed (Nechay and Stephenson, 2009).

The most frequent type of seizure is complex partial and, very commonly, non-convulsive autonomic. Thus, infants appear pale, apnoic and limp, with staring, frequent

nausea, and ictal vomiting. Eye deviation and asymmetric posturing are also observed (Franzoni *et al.*, 2010). The seizure may occur at any time during bathing and is usually of short duration, between 30 seconds to 3 minutes. Interictal EEG is normal for half of the patients, while in others there are slow waves at temporal regions, rarely spikes and waves. Ictal EEG shows high-amplitude focal or unilateral slow rhythmic activity with secondary generalisation (Ceulemans *et al.*, 2008). Recommendations for treatment include changing of bathing habits, shortening the time of bathing, and intermittent using of clobazam before bathing (Franzoni *et al.*, 2010; Panayiotopoulos, 2010). Disappearance of seizures and an



Correspondence:

Ruzica Kravljanac
Neurological Department,
Institute for Mother and Child Health
Care of Serbia,
6 Radoje Dakic St,
11070 Belgrade, Serbia
<ruzica.kravljanac@gmail.com>
<djrusic@eunet.rs>

improvement of executive disorders were reported in one water epilepsy case with oxcarbazepine (Auvin *et al.*, 2006). Prognosis is excellent.

Case 1

A 3-year-old boy was admitted to our hospital due to three seizures during bathing. The conditions of bathing included water at room temperature and no washing of the head or hair. At onset, the boy protested against bathing, as he usually did, but suddenly he became quiet, looked confused, absent, pale with perioral cyanosis, and stared around. The head suddenly fell down, the muscle tone was decreased and he needed support to avoid falling down. The seizures lasted several minutes. At the end of the attack, the boy cried and slept for half an hour.

He was born from an uneventful pregnancy and delivery. Early psychomotor development was normal. At the age of 10 months, he experienced his first seizure during sleep, which repeated twice later; the boy was absent, pale with perioral cyanosis, and stared for 10 minutes. Results of neurological, neuroimaging, and cardiological examination and biochemical blood analyses were normal. Phenobarbital was introduced and maintained for two years.

At admission, neurological status was normal, psychological assessment showed mildly delayed development, and results of metabolic analyses were normal. Interictal EEG on awakening showed normal background activity with no epileptic discharges, while EEG during sleep showed normal sleep organisation with focal epileptic discharges of spikes and waves above the frontal left region. We tried several times to provoke the seizures in the EEG laboratory, including different techniques and conditions; pouring water on the patient and/or putting the patient in the bath using hot water, cold water, and water at room temperature. When we poured lukewarm water onto his body, imitating bathing at home, the seizure occurred (*video-EEG sequence 1*). A complex partial type seizure was provoked with a duration of nearly two minutes. The attack started with motor arrest and through the course of the seizure, the child became motionless and hypotonic, with staring, a head drop, oral automatisms, and perioral cyanosis. The EEG showed generalised delta waves starting above the left fronto-central region, with progressive slowing of activity and increase of amplitude. At the end of the attack, the child started to cry and regained motor activity. A diagnosis of reflex bathing epilepsy was made and a change in the way bathing was performed was suggested, including the duration of bathing, as well as treatment with diazepam for acute seizure cessation.

Three months after discharge from the hospital, the boy experienced one seizure provoked by bathing and washing hair. At the age of 3 years and 10 months, he had one unprovoked, very short seizure during sleep, stopped by rectal application of diazepam. The boy is still without continuous antiepileptic drugs after 27 months of follow-up.

Case 2

A 9-month-old infant was admitted to our hospital due to two seizures provoked by bathing with water at room temperature. The infant usually enjoyed sitting in the bath filled with water, but his excitement due to bathing was interrupted suddenly by confusion, pallor, staring, perioral cyanosis, hypotonia, and unresponsiveness for one minute.

The infant was the first child in the family, from an uneventful pregnancy and delivery. Neurological and psychological status, results of laboratory, and neuroimaging analyses were normal. Interictal EEG showed normal background activity during awakening with no abnormal discharge. EEG during sleep showed normal sleep organisation with slow high-amplitude delta waves and rare sharp waves above the frontal right region.

The seizure was provoked in the EEG laboratory by imitating the previous condition in which the seizures occurred. The child was sitting in a small tub filled with lukewarm water and was very happy and excited. The crucial moment was when he put his face in the water for a moment. After immersion, for the second time, the seizure started (*video-EEG sequence 2*). The initial seizure manifestations were staring, confusion, non-forced right head deviation, and immobility. At the beginning of the seizure, EEG showed a paroxysmal slowing of the background rhythm, followed by a 2-3 Hz pattern of high-voltage waves, starting above the right temporal region with fast unilateral spreading. After 42 seconds, the EEG showed diffuse dissemination of slow rhythm with a clinical correlate of seizure generalisation. After 68 seconds of seizure onset, diazepam was given rectally and the seizure stopped after one minute. During the last 20 seconds of the course of the seizure, oral and hand automatisms were observed while EEG showed rhythmic repetitive sharp waves on the left temporal and parietal region. We concluded that this infant, with normal psychomotor development, had reflex seizures provoked by immersion as the only type of epileptic seizure. We recommended changing the way bathing was performed, avoiding immersion, as well as rectal application of diazepam for seizure cessation. During 3.5 years of follow-up, the boy had normal psychomotor development with no repeat seizures.

Discussion

In both of our cases, the patients experienced provoked seizures at early age (34 months and 9 months, respectively). Median age at onset of bathing epilepsy in the literature is seven months (Nechay, 2010). Diagnosis is mostly based on clinical grounds, as the circumstances of attack and short period from beginning of bathing to seizure onset are diagnostic cornerstones (Ceulemans *et al.*, 2008). Both of our patients had complex partial seizures with behavioural arrest and sudden reduction in motor activity, the so called “hypomotor seizures” (Nordli, 2006), while the second patient had secondary generalisation. Oralimentary and gestural automatism, observed in both patients, together with EEG features point to temporal lobe involvement. Autonomic functions were clearly altered, as in other published cases (loos *et al.*, 2000; Franzoni *et al.*, 2010).

Interictal EEG is usually normal for this type of epilepsy. In the published cases, ictal EEG showed lateralised or focal rhythmic high-voltage delta activity, with fast generalisation at all electrodes (Ceulemans *et al.*, 2008; loos *et al.*, 2000; Franzoni *et al.*, 2010; Jansen *et al.*, 2010). Ictal EEG in our cases showed initial focal slowing of activity presented by delta rhythm, followed by spreading and generalisation of slow waves.

The seizures provoked by immersion were described many years ago (Keipert, 1969), but the first video of a seizure provoked by water immersion in a Caucasian toddler was presented and published recently (Ceulemans *et al.*, 2008). With regards to differential diagnosis, there are some non-epileptic events which are induced by bathing, such as paroxysms in children with: alternating hemiplegia of childhood, hyperkplexia, paroxysmal extreme pain disorder (familial rectal pain syndrome), attack of episodic ataxia type 1, and shuddering attacks (Nechay, 2010). Ictal video-EEG in our cases excludes the possibility of non-epileptic paroxysmal events.

Based on the follow-up of three and a half years in one case and nearly two and a half years in the other, it was only possible to control seizures by avoiding the provocative aspect of bathing or immersion, without continuous or intermittent antiepileptic drugs. The prognosis of bathing epilepsy is usually favourable. Remission is mostly spontaneous and psychomotor development remains normal (Argumosa, 2002). Brain magnetic resonance was normal in both our patients, and further development and seizure control were favourable.

In conclusion, we report two patients with electroclinical patterns of bathing epilepsy. The video-EEG of our second case adds to a very small number of video-reported cases of seizures provoked

by immersion. Using the present ILAE classification (Engel, 2001; Engel, 2006), many authors have presented cases with seizures provoked by bathing in lukewarm water as hot water epilepsy. With regards to new concepts of epilepsy classification (Berg *et al.*, 2010; Berg and Scheffer, 2011), it is very important that the ILAE recognises and distinguishes bathing seizures/bathing epilepsy as a different condition from hot water epilepsy. Also, further studies are necessary to investigate the different epileptogenic mechanisms of bathing epilepsy. □

Disclosures.

None of the authors has any conflict of interest to disclose.

Legends for video sequences and supplementary figures

Video sequence 1

An epileptic attack of complex partial seizure type, provoked by pouring lukewarm water over the child's body, was recorded by video-EEG monitoring. The attack started at 18:36:31 and finished at 18:38:29, according to the time displayed on the video-EEG. The seizure started as motor arrest and staring, with EEG correlate of rhythmic slow waves at 3-4 Hz over the left hemisphere, which continued for the next 42 seconds. From 18:36:50, biphasic sharp waves were registered above the anterior temporal left region for seven seconds. From 18:37:13 to 18:38:29, the child was motionless, stared, had oral automatisms and perioral cyanosis, and showed occasional generalised atonia with head drop. During this period, the EEG showed generalised delta activity with progressive slowing and increase of amplitude starting approximately one second earlier over the left fronto-central region. Towards the end of the attack, from 18:37:44 to 18:37:59, the short periods of EEG attenuation were recorded interchanging with periods of generalised delta activity. After spontaneous seizure cessation, the child started to cry and regained motor activity. Background activity became normal, consisting dominantly of fast symmetric theta waves. EEG conditions: longitudinal montage, calibration: 70 mV; speed: 10 s/page.

Video sequence 2

A partial seizure with generalisation was provoked in the infant by immersion with lukewarm water during bathing. The seizure onset was characterised by staring, confusion, non-forced right head deviation, and immobility. EEG showed a paroxysmal

slowing of the basic rhythm followed by 2-3 Hz high-voltage delta waves, starting above the right fronto-temporal region. The fast unilateral spreading of slow activity and sharp waves was registered with increasing amplitude and decreasing frequency over the whole right hemisphere. After 42 seconds from the seizure onset, the EEG showed diffuse dissemination of high-amplitude (200-300 mcV) rhythm at 2-3 Hz, with clinical correlation of seizure generalisation. After 68 seconds from seizure onset, diazepam was given rectally and the seizure stopped after one minute. During the last 20 seconds of the course of the seizure, oral and hand automatisms were observed while EEG showed rhythmic repetitive sharp waves over the left temporal-parietal region. A) 08.21.24: seizure onset; B) 08.22.05: generalisation; C) 08.22.32: diazepam application; D) 08.23.30: the end of the seizure. EEG conditions: longitudinal montage, calibration: 70 mcV; speed: 10 s/page.

**Key words for video research on
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Syndrome: reflex epilepsy

Etiology: idiopathic

Phenomenology: staring; autonomic symptoms

Localization: unknown

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