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Brief Communication

Stretch syncope: Reflex vasodepressor faints easily mistaken for epilepsy

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ABSTRACT

The pathophysiology of stretch syncope is demonstrated through the clinical, electrophysiological, and hemodynamic findings in three patients. Fifty-seven attacks were captured by video/EEG monitoring. Simultaneous EEG, transcranial (middle cerebral artery) doppler, and continuous arterial pressure measurements were obtained for at least one typical attack of each patient. They all experienced a compulsion to precipitate their attacks. Episodes started with a stereotyped phase of stretching associated with neck torsion and breath holding, followed by a variable degree of loss of consciousness and asymmetric, recurrent facial and upper limb jerks in the more prolonged episodes. Significant sinus tachycardia coincided with the phase of stretching and was followed within 9–16 seconds by rhythmic generalized slow wave abnormalities on the EEG in attacks with impairment of consciousness. Transcranial doppler studies showed a dramatic drop in cerebral perfusion in the middle cerebral arteries during the episodes. The combination of the stereotyped semiology of the attacks, the pseudofocal myoclonic jerking, and the rhythmic generalized slow wave EEG abnormalities with the tachycardia make differential diagnosis from epilepsy challenging.

1. Introduction

Stretch syncope has only rarely been described in the literature [1–3]. Seven of the eight individuals previously reported were adolescent males; one was a teenage female. Their attacks consisted of stereotyped episodes of stretching actions while performing a valsalva maneuver (VM). Hyperextension of the neck (with or without lateral rotation of the head) was associated with abduction/external rotation of the shoulders during the stretch [1,2,4]. Stretching was followed by a variable degree of loss of consciousness.

The pathophysiology of stretch syncope remains obscure although it has been suggested that the cerebral manifestations may be caused by vertebrobasilar insufficiency or occlusion [1–3]. However, it is uncertain whether neck hyperextension or torsion could produce a sufficiently high degree of bilateral vertebral artery stenosis [4].

2. Methods

We describe three male patients with frequent stereotyped attacks. Continuous video/EEG telemetry (Figs. 1–3) was performed in all cases as there was a suspicion that the attacks could be epileptic in nature. All patients underwent further examination with simultaneous video/EEG recordings, transcranial doppler (TCD) of the middle cerebral artery (MCA), and continuous beat-to-beat (Finometer-Finapres medical systems) arterial pressure measurements during stretch attacks. Case 1 precipitated attacks by playing a computer game. Case 2 had a spontaneous attack. Case 3 experienced a milder version of his usual symptoms by deliberately performing a VM while stretching his trunk and laterally flexing his neck.

2.1. Case 1

This 21-year-old man had a 1-year history of several stereotyped episodes per week, triggered by mental exertion. Attacks were preceded by an overwhelming urge to stretch. Stretching would be followed by a sensation of depersonalization, numbness in body and tongue, a tight sensation in his chest, and (with more severe attacks) loss of consciousness or balance and collapse (Fig. 1). Repetitive asymmetric face and upper limb twitching was reported in some attacks. Attacks would leave the patient feeling washed out and unable to concentrate for several hours. The episodes had failed to respond to carbamazepine, levetiracetam, and topiramate. He had a history of irritable bowel syndrome, obsessive–compulsive disorder, agoraphobia, and mild depression. He took citalopram. Neurological and cardiovascular examinations, interictal EEG, and brain MRI were normal.

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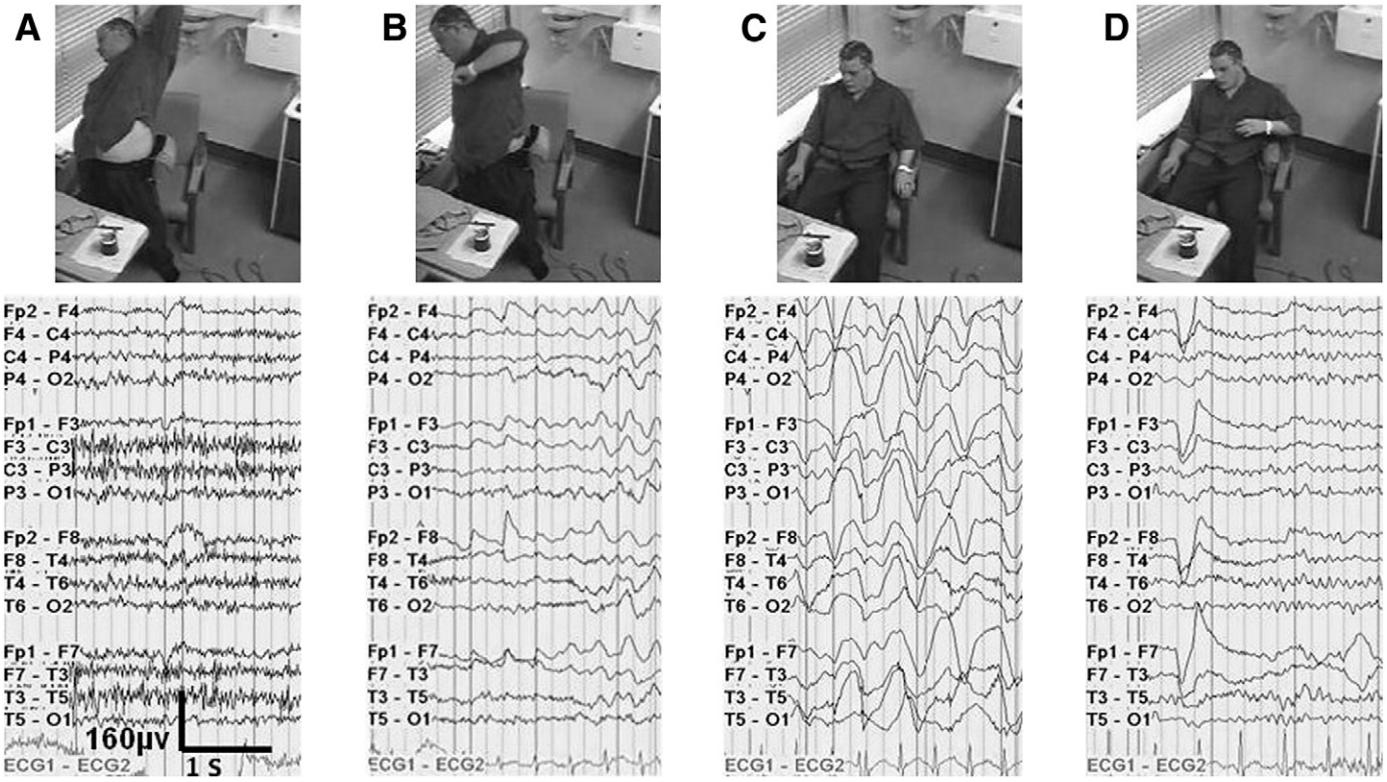

Fig. 1. Case 1: attack captured on video telemetry. (A) Stereotyped stretching actions are consistent at the onset of the patient's attacks. Note that there are no abnormalities on the EEG during the initial and middle phases of the stretching actions. (B) At the end of the stretching maneuver, the generalized slow wave EEG abnormalities emerge (initially in the theta range). The ECG shows sinus tachycardia (160 bpm). The patient is unresponsive at this stage. (C) Although the patient is sitting, the generalized delta slow wave abnormalities continue for several seconds before gradually subsiding. Sinus tachycardia continues. The patient has myoclonic twitches of his head and is deeply unconscious. (D) Recovery phase. He reports feeling very tired. Note the alpha rhythm on the record.

2.2. Case 2

This 20-year-old man had a 1-year history of 10–20 blank episodes per day occurring from sitting or standing. Some attacks were associated with déjà vu phenomena and repetitive asymmetric head and upper limb twitches. He fell to the ground during some of these episodes. The patient also had schizoaffective disorder. He was treated with sodium valproate for the attacks with no improvement. He received sulpiride, clonazepam, and lorazepam for his psychiatric condition. Neurological and cardiovascular examinations, baseline ECG, brain CT and MRI, and standard and sleep deprived EEGs were all normal (Fig. 2).

2.3. Case 3

This 26-year-old man had a 10-year history of blackouts. He experienced brief blank spells that occurred in a seated or standing position, and were characterized by blurring of vision, right arm twitching, decreased awareness, and confusion lasting up to 30 seconds (Fig. 3). His mother described him as “frozen” during these events and noticed occasional body twitches. He could have up to ten attacks per day and he had a compulsion to precipitate episodes. Levetiracetam failed to stop the events. He had a history of childhood asthma. On examination there was left eye esotropia; otherwise, neurological and cardiovascular examinations were normal. EEG, ECG, 24-hour ECG, and MRI were normal.

3. Results

All patients underwent MCA transcranial doppler examination and head-upright tilt-table testing with normal findings. Blood pressures measured during a 15-second VM and MCA mean flow velocities were within the range observed previously in normal volunteers [5] (Mid-15-second VM mean flow velocities for case 1, 10 cm/s; for case 2, 30 cm/s; and for case 3, 32 cm/s). Clinical symptoms were neither observed nor reported by the patients during and around the time the VMs were performed.

All observed attacks started with a fairly stereotyped phase of stretching and breath holding (Figs. 1, 2). Within 9 to 16 seconds, this was followed by a variable degree of impairment of consciousness and asymmetric facial or upper limb jerks in more prolonged attacks. The stretch was associated with tachycardia and hypotension. For cases 1 and 2, the systemic beat-to-beat arterial pressure measurements showed a drop in systolic and diastolic pressures (below 40 mm Hg) toward the end of the stretching actions (see example in Fig. 2). Case 3 had a slightly higher blood pressure during the event (73/56 mm Hg). All three patients showed arrest of the diastolic MCA blood flow on TCD at the time blood pressure fell. The mean flow velocities were too low to be calculated. Systemic hypotension was associated with an increased heart rate (to 160 bpm in case 1, 130 bpm in case 2, and 110 bpm in case 3). The EEG showed rhythmic generalized slow waves when consciousness was impaired.

4. Discussion

Stretch syncope is a diagnostic entity that is clinically difficult to distinguish from epilepsy. All patients described here were erroneously treated with antiepileptic drugs. Clinicians unaware of the typical manifestations of stretch syncope may easily be misled by the stereotyped motor manifestations involving asymmetric facial and upper limb twitching, rhythmic EEG changes, and rapid development of ictal tachycardia seen in the attacks. The patient's clinical history may not