

Convulsive syncope: a Stokes-Adams case

Síncope convulsivo: un caso de Stokes-Adams

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A 26-year-old male with two-days prior cocaine consumption, presented with a 5-day history of new-onset focal nonmotor dyscognitive epileptic events. After three days of treatment with oral phenytoin (PHT) 100 mg t.i.d., a convulsive status epilepticus developed at the same time a third-degree atrioventricular block was registered (Figures 1 and 2).

As we know, cocaine consumption and PHT (specially in infusions >50 mg/min or previous heart disease) are both related with adverse cardiovascular effects, specially arrhythmias^{1,2}. Our case exemplifies the lethal combination of both, and the narrow neurological and cardiologic assessment of syncope *versus* epilepsy in patients with stereotypic paroxysmal episodes^{3,4}.

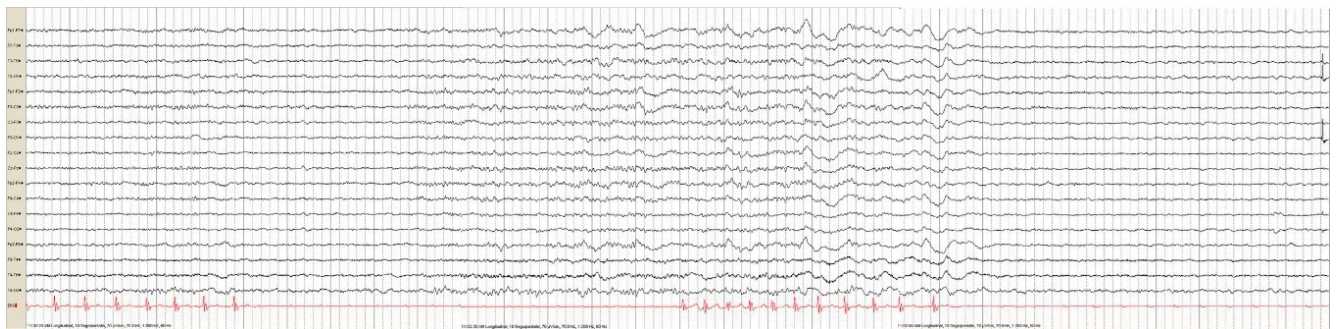


Figure 1. 6 consecutive epochs (11:02:28 to 11:03:28) are shown. Montage: Bipolar, longitudinal, double-banana. HF: 70 Hz, LF: 1 Hz, NF: 60 Hz, Sens: 70 μ V/cm; 13 seconds after a 10-second ventricular pause, 2–3 Hz generalized polymorphous slow waves are registered with greater expression in the anterior regions. Following this, a new ventricular pause of 9 seconds appears, which will continue in the following figure.

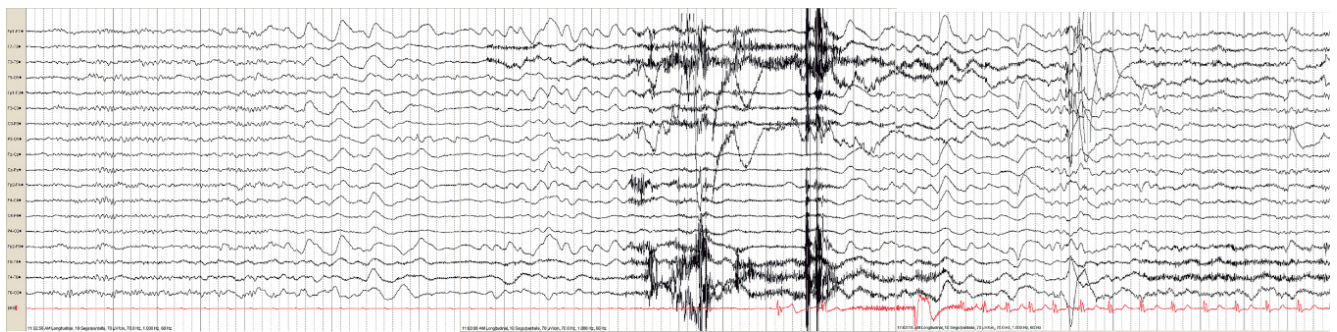


Figure 2. The last ventricular pause described in Fig. 1 continues for 17 more seconds (26 in total). Synchronous generalized slow waves are again registered. On the 24th second, generalized electrodecrement with muscle artifact due to a generalized tonic seizure of 4 seconds of duration. This event ends with the recovery of the heart rhythm and subsequent slow waves in delta range before recovering its alpha rhythm.

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