Three years previously, a 77-year-old Tanzanian male developed post-anoxic encephalopathy after cardiac arrest and pacemaker implantation. After cardiac arrest, there was residual pseudobulbar and cerebellar dysarthria, spastic-dystonic quadriplegia, and largely preserved cognition. Follow-up computed tomography (CT) scans of the brain showed moderate atrophy. He was using the anticoagulant dabigatran. Nine months before admission, without an obvious precipitating event, he developed jerky movements that hindered breathing and eating, and which resolved after 2 weeks. Two days before admission these same movements recurred, and led to respiratory difficulties that occurred during sleep and when awake. Upon examination he had stable vital signs, was alert, and had labored breathing. His oxygen saturation was 98% when on oxygen. When auscultation was performed, basal crepitations were detected. Apart from the pre-existent neurological abnormalities, there were now inspiratory, involuntary jerky movements of the chest, throat, and neck that were not present during expiration. Inspection and palpation of the flanks and abdomen were normal and the patient did not experience hiccups or movements of the abdominal wall (Video 1). Magnetic resonance imaging of the brain was not possible because of a pacemaker. CT scans of the brain, chest, and diaphragm were normal. A chest X-ray showed bilateral infiltrates; however, the laboratory test results were normal. Clonazepam and sodium valproate were associated with mild improvement. Three days after admission, he died from aspiration pneumonia and respiratory failure.

The working diagnosis was intermittent inspiratory myoclonus of brainstem origin in a patient with post-anoxic encephalopathy. It may have recurred after a previous, self-limiting episode of possible brainstem ischemia or hemorrhage undetected by a CT scan of the brain. Myoclonus was less likely to be cortical because the patient was alert throughout. The fatal outcome was likely due to respiratory insufficiency rather than progressive brainstem involvement. Diaphragmatic flutter or dyskinesia was considered, but our patient had a normal expiration phase and was not tachypnoeic. Belly dancer dyskinesia would, as the name suggests, affect the abdominal wall primarily and is a lower frequency and amplitude-movement disorder. He did not display Lance Adams (post-anoxic) myoclonus at any time after his cardiac arrest. Truncal spinal myoclonus or a voluntary component is...
unlikely. Encephalitis is less likely to be due to the recurrent nature of movement disorders with a symptom-free interval of several months and normal consciousness.

This study's limitations are the short duration of the video and lack of several investigations inherent to the low-resource setting. This is one of few recordings of this phenomenon,\(^5\) stressing the potentially fatal outcome, and originating from an area that is underserved by neurologists.

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**References**

1. Rigatto M, De Medeiros NP. Diaphragmatic flutter. Report of a case and review of literature. *Am J Med* 1962;32:103–109. doi: 10.1016/0002-9343(62)90186-9

2. Illiceto G, Thompson PD, Day BL, Rothwell JC, Lees AJ, Marsden CD. Diaphragmatic flutter, the moving umbilicus syndrome and 'belly dancer’s' dyskinesia. *Mov Disord* 1990;5:15–22. doi: 10.1002/mds.870050105

3. Philips JR, Eldridge FL. Respiratory myoclonus (Leeuwenhoek’s disease). *N Engl J Med* 1973;289:1390–1395. doi: 10.1056/NEJM197312272892603

**Video 1. Jerky, Audible Inspiration Phase.** The expiration phase is largely unaffected.