

# Fasciculations in brain death

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**Objectives:** Brain death is the colloquial name for human death determined by tests showing irreversible cessation of the clinical functions of the brain. Spontaneous and reflex movements have been described in brain death. The aim of this report is to describe a brain-dead patient with unusual motor movements.

**Design and Setting:** The patient was followed and her motor movements were videotaped.

**Patient:** We report the presence of extensive and long-lasting fasciculations in a patient who fulfilled the criteria for brain death.

**Measurements and Main Results:** We describe and show on videotape a brain-dead patient with rare motor movements.

**Conclusion:** We suggest that fasciculations outlined in this study has to be accepted as motor symptoms in brain death patients. (Crit Care Med 2010; 38:2377–2378)

**KEY WORDS:** brain death; fasciculation, motor movements

A previously healthy 72-yr-old woman had a sudden, severe headache, vomited, and collapsed unconscious at home. She was taken to our intensive care unit within 2 hrs, where she was intubated and ventilated. She had no history of abnormal movements including fasciculations, previous neurologic disease, toxin exposure, and was not on any concomitant pharmacologic treatment. Neurologic examination disclosed deep coma with no motor responses to noxious stimuli to the sternum or the limbs. Brainstem reflexes including pupillary responses to light, oculocephalic, oculovestibular, corneal, pharyngeal, and tracheal reflexes were absent. Her admission blood pressure was 130/95 mm Hg and temperature was 36.5°C. Routine blood tests including electrolytes were within normal limits. A computed tomography scan of the head disclosed a massive subarachnoidal hemorrhage in the basal cisterns. She was intubated and treated with intravenous mannitol 20% (1 g/kg followed by 0.5 mg/kg every 4 hrs). An electroencephalogram using

eight scalp electrodes was isoelectric. There was no spontaneous respiration for 10 mins off the ventilator during the apnea testing. The patient met the criteria for brain death as established by the American Academy of Neurology guidelines (1). Twelve hours after the first examination, her neurologic status remained the same except increased deep tendon reflexes in the lower extremities. Another electroencephalogram remained unchanged. At that time, fine, rapid, and vermicular twitching movements were noticed in the muscles of the extremities, chest, and abdomen (see Supplemental Digital Content 1, <http://links.lww.com/CCM/A177>, which shows fasciculations and finger jerks in a patient with brain death). These movements appeared every 2–3 mins during 4 hrs. They were rhythmic and repetitive twitches lasting for 5 secs. The movements were extensive enough to cause digital jerks and clinically characterized as fasciculations (see Supplemental Digital Content 2, <http://links.lww.com/CCM/A181>; and Supplemental Digital Content 3, <http://links.lww.com/CCM/A182>). No other movements or responses were present. Sixteen hrs later, fasciculations stopped and the deep tendon reflexes were absent. Cardiac arrest occurred 48 hrs after admission.

We are indebted to relatives of our case who agreed with videotape recording and written informed consent. This study and the videotaping were approved by the ethics committee of our hospital.

## DISCUSSION

Brain death is a state that includes irreversible cessation of all cortical functions

and brainstem reflexes resulting from organic brain lesions. The cardinal diagnostic criteria for brain death require profound coma with total unresponsiveness, apnea despite induced hypercapnia, absence of all reflexes subserved by the brainstem and cranial nerves, presence of an underlying cause sufficient to produce the clinical findings, exclusion of reversible metabolic or toxic factors, and irreversibility (2). Occasionally, diverse types of spinal reflexes and automatisms are encountered in brain death such as plantar withdrawal responses, muscle stretch reflexes, abdominal contractions, slow turning of the head to one side, Lazarus sign (shoulder adduction, crossing both arms on the chest, moving hands to the neck, and finally falling to the bed), respiratory-like movements, facial myokymia, finger jerks, periodic limb movements, and automatic stepping (3, 4). The most controversial issue related to the determination of brain death is the occurrence of motor movements that suggest some retention of brain function. However, such motor movements are generated by the spine, and the evidence of brain death in such cases comes from a consistent clinical documentation of brain death and confirmation by isoelectric electroencephalogram or cerebral angiography (5). Although by definition the diagnosis of brain death requires the irreversible loss of cerebral function, including the brainstem, it appears that all functions are not immediately lost. Several functions including complex spinal automatisms have been reported in patients with brain death and these spinal reflexes can be maintained for up to several hours or days (3).

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Freitas et al (6) reported one brain-dead patient with coarse fasciculation in the upper limbs and pectoralis muscles that were stopped after approximately 30 secs. Our patient had generalized, extensive, and long-lasting fasciculations in the muscles of the extremities, chest, and abdomen. To our knowledge, this is the second report describing fasciculations in a brain-dead patient. We suggest that these fasciculations result from involuntary motor discharges arising from previously intact spinal cord anterior horn cells. Besides fasciculations, we observed that our patient had hyperactive deep tendon reflexes. Deep tendon reflexes are usually absent but may be retained. Because deep tendon reflexes are integrated at a purely spinal cord level, their presence or absence is not directly relevant to brain functioning. Jørgensen et al (7) reported the presence of deep tendon reflexes of the limbs in one third of a series of 63 patients with brain death. The presence of hyperreflexia and fasciculation together might result from incomplete ischemia of the spinal cord. Kasdon et al (8) described four patients with spasticity and fasciculations in the upper and lower extremities after surgical decompression for cervical spondylosis and stated that three mechanisms by which cervical

spondylosis might cause fasciculations in the lower extremities are vascular insufficiency, cord traction, and denervation.

Motor movements in brain death are not indicative of brain functioning but may be misinterpreted as such by unwary physicians. It is best to refer to these movements as “spinal reflexes due to hypoxic stimulation of spinal cord neurons” to clarify that they are neither voluntary nor represent evidence of residual brain function (4). Clinical evidence from animal studies suggests that in the absence of cortical inhibitory and modulatory afferents to the spinal cord, neurons could allow for activation of basic spinal cord programs, causing the reflex movements seen in brain-dead humans (4, 9).

The purpose of the present study is to describe a brain-dead patient with unusual movements, something that has been very rarely reported in the medical literature. Although fasciculations are seen most frequently in association with disorders resulting from degeneration of the anterior horn cells of the spinal cord and the motor nuclei of the brainstem, in such conditions as amyotrophic lateral sclerosis and spinal cord injury, we suggest that physicians should recognize these symptoms outlined here and the

presence of these motor movements do not preclude the diagnosis of brain death.

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