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More about the Respirator Brain

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Letter to Editor

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Letter to Editor

Wijdicks discusses a historically controversial term: the "respirator brain" [1]. When Walker first used this term, several neuropathologists expressed their skepticism and uncertainty regarding the concept of a respirator brain [2]. The main controversy comes from the issue of considering the term "respirator brain" as an anatomical or functional concept or both [3]. The so-called respirator brain is a dynamic process that may progress until pulmonary and/or cardiac disturbances terminate the patient's life or until resuscitation is stopped. The final brain changes may be arrested at any stage due to various factors, including ischemia, acidosis, and intracranial hypertension [2]. The time on the respirator has a critical influence on the pathological changes in the brain. Shewmon reported a series of brain-dead patients with extended somatic survival. He referred to Repertinger, et al., case of a 4-year-old child diagnosed as brain-dead after bacterial meningitis, maintained on a ventilator for 19 years. The autopsy revealed a 750 g calcified intracranial spherical structure and a calcified shell containing grumous material and cystic spaces with no recognizable neural elements grossly or microscopically [4]. Wijdicks and Pfeifer affirmed that improved organ transplant processes have shortened the required time for brain fixation. The classic description of the "respirator brain" occurred when organ transplant protocols were not fully developed [5]. On the contrary, I described the Jahi McMath case, where after nine months of being diagnosed braindead, I found preservation of intracranial structures, but this is another discussion [3].

The rostrocaudal deterioration syndrome depends on the presence of a supratentorial space-occupying lesion [2]. A supratentorial brain lesion usually produces a rostrocaudal transtentorial brain herniation, resulting in the forebrain and brainstem loss of function. In secondary brain lesions (i.e., cerebral hypoxia), the brainstem is also affected like the forebrain. Nevertheless, some cases complaining of posterior fossa lesions may retain intracranial blood flow and EEG activity. If a posterior fossa lesion does not produce an enormous increment of intracranial pressure, a complete intracranial circulatory arrest does not occur, explaining the preservation of EEG activity, evoked potentials, and autonomic function [3].

The description of the "respirator brain" is not only due to the patient being under mechanical ventilation. The final anatomic and functional findings depend on multicausal: the etiology of the coma, whether the patients suffered or not from a cardiac arrest, thr presence of supratentorial or infratentorial lesions, time on ventilation, and many others. Hence, I fully agree with Wijdicks [1] that "respirator brain" is not a synonym for brain death.

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