LETTERS TO THE EDITOR

To the Editor

Central Pattern Generators: A Bridge Between Life and Death

Re: Movements in brain death: a systematic review. Can J Neurol Sci. 2009;36(2):154-60.

I read with interest the excellent review of Saposnik et al[1] on movements in brain death. As the Authors stated, for the most part, these movements are considered to be spinal reflexes. Spinal reflex movements and automatisms occurring after brain death have been considered phylogenetically "old motor patterns," which may be set free when the cord is uncoupled from the "younger" input of the brainstem and neocortex[2].

Regarding the pathogenesis of these motor patterns, I suggest to introduce the term central pattern generator (CPG), thus referring to a concept widely present in the literature.

Central pattern generators are genetically determined specialized neuronal networks localized in the brainstem and spinal cord, representing the anatomical substrate of stereotyped inborn fixed motor behaviours which are essential for survival. In humans CPGs are largely under neocortical control. Stereotyped action patterns, expression of genetically determined CPGs, have been described to occur in physiological movements in foetuses and newborns, in physiological sleep, in parasomnias and some epileptic seizures[3].

A cortical inactivation is currently considered underlie the pathophysiology of some of these motor patterns in both seizures and syncope[4].

It is rather surprising that although widely present in the literature, the term CPG never appears in relationship with brain death-associated reflexes and automatisms, which are nevertheless generated by spinal CPGs.

I think that introducing the term CPGs also in the pathogenesis of death-associated motor activity would be conceptually useful, since it would provide a more global vision on neural networks generating motor behaviours occurring in several conditions, building therefore a bridge between life and death.

Francesco Brigo, University of Verona, Verona, Italy

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To the Editor

Common Misconceptions Regarding Neuroimaging in Epilepsy Diagnosis

Unfortunately, in clinical daily practice there are several misconceptions regarding the process of epilepsy diagnosis and the role of neuroimaging within such a process.

As a matter of fact, the diagnosis of epilepsy relies mainly on clinical grounds, is sometimes supported by EEG features, and may integrate neuroradiological findings. The clinical evaluation (and the EEG) is by itself necessary and sufficient to make a diagnosis of seizures/epilepsy and to provide a proper classification (generalized versus focal), but is of little value in determining the etiology, with the exception of some idiopathic epilepsies and some acute symptomatic seizures. On the contrary, neuroimaging by itself has no role at all in the diagnosis or classification of seizures/epilepsy, but is nevertheless essential for an etiological evaluation. In the process of epilepsy diagnosis neuroradiological studies therefore depend on and are subsequent to the clinical evaluation, from both a practical and a conceptual point of view. As a consequence, neuroimaging should always be interpreted on the basis of the clinical picture and, in case of discrepancies between clinical and neuroradiological findings, the former should be considered prominent with respect to the latter. Such a simple methodological concept, too often forgotten in clinical practice, is nevertheless of paramount relevance in order to avoid a misdiagnosis of epilepsy. As a matter of fact, when neuroimaging is not considered within the global diagnostic process, the detection of a brain injury/lesion may lead to an erroneous diagnosis of epilepsy.

As an example of this, in the literature there are several reports[1,2] on patients with psychogenic nonepileptic seizures who were initially misdiagnosed as having epilepsy because of a magnetic resonance imaging evidence of mesial temporal sclerosis, despite the clinical picture clearly pointed to a non-epileptic nature of the events. In these cases a positive neuroradiological test has been erroneously considered able to modify a pre-test probability of seizures which was nevertheless very low, thus forgetting that neuroimaging has a role only in the etiological evaluation and not at all in the diagnosis of epilepsy in the strict sense of the word.

Trusting too much a diagnostic procedure, as well as ignoring its limitations, represents always a mistake, which can have relevant consequences, such as that of a misdiagnosis. Each diagnostic test should always be considered as a part of a wider step-wise diagnostic process, in which the clinical picture plays the main role.

It is therefore important to remember that, although essential for the etiological evaluation, neuroimaging never helps the neurologist to make a diagnosis of epilepsy, which remains a clinical one.

Francesco Brigo, University of Verona, Verona, Italy

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