



## Letter to the Editor



We read with interest the case report *Non-convulsive status epilepticus with right arm apraxia* [1]. The authors describe a patient with an old left temporoparietal lesion from a lobar hematoma who suddenly developed a motor disturbance of his right arm during a nonconvulsive status epilepticus (NCSE). NCSE was verified by EEG showing continuous focal rhythmic theta-delta activity. Before admission, the patient noted that his performance of everyday routine tasks such as dressing, or preparing a meal was impaired and he was worried because his right arm did not obey him as usual. On examination, the patient was unable to demonstrate meaningful movements with his right arm on command like drawing, combing, or using common objects. The condition was classified as apraxia; it was accompanied by an aphasia, and stopped after iv treatment with clonazepam and levetiracetam. Apraxia was only present in the patient's right arm, whereas the left arm performed flawlessly. Apparently, the authors describe a case of ictal apraxia, a unique phenomenon which is particularly interesting for epileptologists with interest in ictal cognitive and motor phenomena.

Limb apraxia (LA), the term which the authors supposedly mean differs from other, conceptually unrelated forms of apraxia, such as apraxia of gait, dressing, touch, lid opening, or constructional abilities. Originally described more than a century ago [2] LA denotes an acquired disorder of motor control. The syndrome is defined as inability to perform limb actions on request and/or imitation despite normal elementary sensorimotor functions, task comprehension or object recognition [3,4]. Typically, lesions producing LA involve the left (dominant) hemisphere praxis network which stores, selects, adapts and controls skilled limb movements. The deficit of LA is located at the executive level of the motor hierarchy. It causes a loss of control of subordinate motor systems in both hemispheres; as a result, LA always involves both limbs. In contrast to bilateral LA from left hemisphere damage, unilateral apraxia is rare condition resulting from lesions of the corpus callosum and usually affects only the left upper limb. In 'callosal apraxia' the right hemisphere generates apraxic movements because it is disconnected from left hemisphere motor programs [5,6].

It appears that this patient's motor impairment has little to do with a LA as known from numerous studies and agreed definitions. Most notably, the unilateral appearance argues strongly against this diagnosis. Also, relevant clinical features are poorly described or lack completely. We receive no detailed information on the interictal motor or cognitive status, although the patient had amyloid angiopathy with a large focal lesion. Basic motor functions during the NCSE remain unreported, similar as systematic apraxia testing. Based on a limited description the reader can only guess how to label the observed clumsiness and distorted movements correctly. Potential alternative diagnoses for this case's movement

disorder are an upper limb ictal monoparesis [7] or a paroxysmal alien limb syndrome [8,9]. Monoparesis and LA can easily be differentiated by a standard motor examination. First, motor functions of both upper limbs need to be assessed to exclude clumsiness, weakness, spasticity, bradykinesia, or ataxia and to detect a possible left-right asymmetry. Next, LA is tested by asking the patient to show hand movements and positions, symbolic gestures, or object use, either by verbal command or by imitation, and always separately for both limbs [10]. LA cannot be tested in a paretic hand. For this reason, patients with left brain damage are tested on their left-hand praxis abilities to confirm or exclude LA (which is, by definition, bilateral). In the presence of a co-existing aphasia and during a NCSE, additional tests should make sure that the patient has intact comprehension of complex speech, attentional functions and imagery of object pantomime to follow commands like 'show me how you would use a hammer'. Describing the movements of a patient as amorphous or awkward conveys little information about the underlying disturbance. Rather, patients with LA make specific errors which can be classified as spatial (e.g. configuration), temporal, content, or substitution errors of motor performance and which allow for a qualitative evaluation.

LA represents a unique behavioral-motor syndrome which has expanded our understanding of the motor system and its cognitive control. It remains unclear whether it can be observed during seizures, how 'ictal LA' is characterized and whether it has any credit as semiological manifestation. However, ictal LA may help to further elucidate and classify abnormal motor behavior during seizures. NCSE is difficult to diagnose when it presents with minimal or no changes in EEG beyond focal slowing. Like other cognitive impairments LA has localizing value and is clinically diagnosed. These facts highlight the value of a careful clinical examination during NCSE. Due to its duration, NCSE may allow for brief cognitive bedside testing [11], much more than other epileptic conditions. In EEG, epileptologists have developed high standards to discriminate true brain signals from artifacts that mimic epileptiform abnormalities [12]. Using adequate clinical assessment procedures and background knowledge about its definition, it should be a straightforward task to differentiate ictal LA (and other cognitive syndromes) from diagnostic artifacts in patients with seizures. This will reduce the risk of erroneous diagnoses and misleading concepts.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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