Leiguarda et al reply: We appreciate Okuda and Tachibana’s comments about our apraxia in corticobasal degeneration because it enables us to clarify the status of limb-kinetik apraxia, a point which is particularly relevant for the clinical interpretation of the apractic disorders in patients with corticobasal degeneration.

Limb or melenokinetik apraxia (originally called “innervatory apraxia” by Kleist’) was considered by Liepmann to be a form of limb-kinetik apraxia due to the loss of “Kinetik-neuroanthropische innervatory engrams” secondary to “sensorimotor” damage. The disorder is characterised by clumsiness in the performance of fine motor acts by the hand contralateral to the hemispheric damage. All types of movements including gestures, regardless of whether the patient creates or imitates them, become “uncouth, clumsy, inexpert, and preceded by fruitless attempts which only bring the wrong muscles into play.”

The status of limb-kinetik apraxia has been debated for over three decades. Most authorities have refused to consider symptoms connected with limb-kinetik apraxia as apractic.6 Geschwind disregarded it completely7 and Rothi et al failed to include limb-kinetik apraxia in their neuropsychological model of limbus.8 Brain concluded that it is simply a partial symptom of pyramidal tract lesion,9 a view also shared by Ajuriaguerra and Tissot,10 Hecaen and Rondot,11 and Mesulam.12 De Renzi in particular contends that “limb-kinetik apraxia has never been described with sufficient accuracy to be distinguishable from a mild form of apraxia”13 to gain acceptance by neurologists.14 In support, monkeys with lesions restricted to the corticospinal tract show similar errors.15

This may be an extreme view of limb-kinetik apraxia. Liepmann’s definition of apraxia may be summarised as a deficit in the performance of purposeful skilled movements, in the absence of elementary motor (waxing, abnormal posture, or tone) or sensory deficits, or of impaired comprehension or memory.16 The disruption of movement seen in lesions of the corticospinal pathway, or as seen in Parkinson’s disease, can sometimes be fully explained by weakness, akinesia, abnormal posture, or tone. There is additional breakdown of the movement pattern or formula—Liepmann’s innervatory engraving—that suggests a higher motor disorder or apraxia. This is exactly what is seen to a pronounced degree in corticobasal degeneration, particularly in the initially affected limb. To this extent we agree with Okuda and Tachibana.

We deliberately employed standardised tests for ideomotor and ideational apraxia, however, and concentrated on the less affected limb. We did not explore the contentious topic of limb-kinetik apraxia, because it is such an uncertain area.

Nevertheless, we agree that patients with corticobasal degeneration characteristically exhibit a higher order motor deficit in their more affected limb, which we would be happy to call limb-kinetik apraxia if others would allow the term! Our study also shows that many patients with corticobasal degeneration likewise fail the ideomotor apraxia, a failure that we do not think can be explained by limb-kinetik apraxia alone.


Transient epileptic amnesia—a clinical update and a reformulation

In relation to the interesting article by Kapur1 on transient epileptic amnesia, I would like to remind your readers of some of the published work related to such findings. Thus experimental studies, using either intracarotid sodium amylobarbitone or electrical stimulation, for diagnostic purposes on epileptic patients, have shown associations between the temporal lobe of the hemisphere dominant for speech and both memory and consciousness.2,13 It is important to keep this in mind when discussing the anatomical and pathophysiological basis of amnesic phenomena, transient or otherwise.

EA SERAFETINIDES


The Editor is grateful to the following, who assisted in the assessment of papers during the past year.

The Editor is grateful to the following for reviewing books in 1994.

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