because it and affected limb. We tests

This is suggests a degree of movement
due to apraxia, abnormal movements, and a corticobasal
development. Rondot,6 which only authors have

ments, performance of the apraxic acts by the hand contra-

teral to the hemispheric damage. All types of movements including gestures, regardless of whether the patient creates or imitates them, become "uncouth, clumsy, inex- expert, and preceded by fruitless attempts which only bring the weak muscles into play."1

The status of limb-kineti c apraxia has been debated for over three decades. Most authors have refused to consider symptoms connected with limb-kineti c apraxia as apractic.6 Geschwind disregarded it completely7 and Rothi et al.5 to include limb-kineti c apraxia in their neuropsychological model of limb praxis.8 Brain con-

cluded that it is simply a partial symptom of pyramidal tract lesion,8 a view also shared by Ajuriaguerra and Tissot," Hecaen and Rondot,2 and Mesulam." De Renzi in par-

cular contends that “limb-kineti c apraxia has never been described with sufficient accuracy to be distinguishable from a mild form of paresis to gain acceptance by neurologists.”20 In support, monkeys with lesions restricted to the corticospinal tract show similar errors.21

This may be an extreme view of limb-kineti c 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 (weak muscles, abnormal posture, or tone) or sensory deficits, or of impaired comprehension or memory.22 The disruption of movement seen in lesions of the corticospinal pathway, or as seen in Parkinson’s disease, can similarly be fully explained by weakness, akinesis, abnormal posture, or tone. There is additional breakdown of the movement pattern or formula—Liepmann’s innervatory engraving—that sug-

gests a higher motor disorder or apraxia. This is exactly what is seen to a pronounced degree in corticobasal degeneration, partic-

ularly in the initially affected limb. To this extent we agree with Okuda and Tachibana.23

We deliberately employed standardised tests for ideomotor and ideational apraxia, however, and concentrated on the less affected limb. We did not explore the con-

tentious topic of limb-kineti c 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 limbs. This would be happy to call limb-kineti c apraxia if others would allow the term! Our study also shows that many patients with corticobasal degeneration likewise suffer from ideational apraxia, a failure that we do not think can be explained by limb-kineti c apraxia alone.

Leiguarda et al reply:

We appreciate Okuda and Tachibana’s comments about our apraxia in corticobasal degeneration1 because they enable us to clarify the status of limb-kineti c apraxia, a point which is particularly rele-

vant for the clinical interpretation of the apractic disorders in patients with corti-

cobasal degeneration.

Limb or melokinetic apraxia (originally called “innervatory apraxia” by Kleist) was considered by Liepmann to be a form of limb kinesia. The loss of “Kinetikorringer- ing engravings” secondary to “sen-
somotor” damage.2 The disorder is characterised by clumsiness in the perfor-

mance of fine motor acts by the hand contra-
teral to the hemispheric damage. All types of movements including gestures, regardless of whether the patient creates or imitates them, become “uncouth, clumsy, inex-

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Transient epileptic amnesia--a clinical update and a reformulation.

E A Serafetinides

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