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## Cognitive Neuropsychological and Brain Imaging Investigation of Apraxia in Corticobasal Degeneration

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The aim of the study was to characterise the presence of limb apraxia in 18 patients (mean age  $64 \pm 8.8$  years) suffering from probable corticobasal degeneration disease (CBD), from a cognitive and brain imaging perspective.

### Neuropsychological Testing

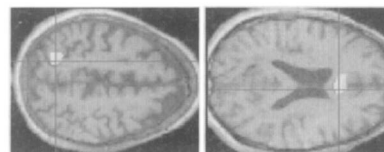
CBD patients and 15 healthy elderly volunteers (CTL1; mean age  $68.4 \pm 8.4$  years) underwent a detailed cognitive neuropsychological assessment of limb apraxia (BEP, Peigneux & Van der Linden, 1998).

*Gesture Reception* : CBD patients made a few errors during (a) discrimination between meaningless and meaningful gestures and (b) meaningful gesture naming ; scores were below CTL1 mean performance ( $ps < .05$ ).

*Gesture Production*: (c) pantomime to verbal command, (d) pantomime to object presentation, (e) actual use of objects, (f) imitation of meaningful gestures and (g) of meaningless gestures were administered to the less affected limb in CBD patients, to both limbs in CTL1 group. Gesture production performance was measured both through the overall frequency of errors (accuracy score) and error correction ability in the second trial (correction score). From a cognitive perspective, accuracy score is a performance measure which reflects the contribution of both praxic-specific components and general cognitive factors to the gestural performance, while the correction score gives more specific information on the integrity of praxic system components. CBD patients' accuracy score was significantly decreased face to CTL1 group in all modalities ( $ps < .05$ ), except actual use of objects, suggesting a production-execution deficit in CBD. At variance, correction scores were significantly decreased for imitation of gestures only ( $ps < .05$ ), irrespective of their meaning, which suggests that specific visuo-imitative deficits might characterise further the presence of apraxia in CBD patients.

### Brain Imaging

CBD patients underwent a positron emission tomography (PET) at rest, on a Siemens 951/31R tomograph with collimated septa extended, using the (18F) fluorodeoxyglucose (FDG) technique. FDG images of brain metabolism in CBD patients with left body side symptoms were flipped so that the hemisphere contralateral to the most affected side was on the left in all CBD cases. Twenty healthy elderly volunteers (CTL2; mean age  $61.3 \pm 1.8$  years) scanned in similar conditions served as control. Data spatial transformations (normalisation on symmetric template, 12mm smoothing) and statistical analyses were run using SPM99b (Wellcome Department of Cognitive Neurology, London). To evidence brain regions specifically responsible for apraxia in CBD, a cut-off score for the presence of apraxia was defined as the minimal correction or accuracy score observed in CTL1 group. With regard to each score, regional metabolism in CBD apraxic patients was compared with half of CTL2 group, as well as regional metabolism in CBD patients without apraxia was compared with the other half of CTL2 group. Interaction between subtractions evidenced brain areas specifically related to the presence of apraxia in CBD. Regarding to accuracy scores, significant metabolic regional differences were found in anterior cingulate area (BA 24;  $p < .001$ ; *Figure, left panel*). At variance, regional metabolism in CBD patients falling below the cut-off correction score was significantly decreased in the superior parietal lobule (BA 7;  $p < .001$ ; *Figure, right panel*).



### Conclusion

In agreement with cognitive analysis, accuracy score which reflects multiple contributions to the gestural performance is associated with hypometabolism in cingulate area, in a region important for both movement generation and selection. With regard to the correction score which reflect more specifically the state of the praxic system, hypometabolism of the superior parietal lobule (LPS) differentiates apraxic from non-apraxic patients. Imitation deficits in CBD might be related to the impaired functioning of LPS, in charge of the regulation and integration of sensorimotor information necessary to process mental transformations of the body which underlie imitation processing.