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# Gestural buffer impairment in early onset Corticobasal Degeneration: a single-case study

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## Abstract

*We investigated limb apraxia in a case of a clinically diagnosed Corticobasal Degeneration with early onset (48 years) in a right handed woman. We used a new battery for the assessment of limb apraxia, based on a cognitive model of praxis. This cognitive model predicts five clinical pictures of limb apraxia: scores reported suggested that apraxia of the patients could be described as an impairment of the gestural buffer, that affects all execution tasks and preserves the ability to perform judgement and categorization tasks. This empirical evidence is in the same direction of other studies supporting that production of actions can be impaired while recognition of actions is spared, and recognition of actions can be impaired while production of actions is spared. This case finding and previously reported studies are consistent with models of praxis that draw a functional distinction between representations that underlie perception and representations that underlie production of actions.*

*Keywords:* Corticobasal Degeneration; Limb Apraxia; Praxis; Gestural Buffer; Executive Functions; Semantic System

## 1. Introduction

Corticobasal degeneration (CBD) is considered a degenerative disease with atrophy, gliosis and tau-immunoreactive pathology of both gray and white matter of neocortex, basal ganglia and substantia nigra (Mahaoatra et al., 2004). Clinically, it is characterized by asymmetric akinesis and rigidity, dystonia of the upper limb, apraxia, myoclonus and dementia. CBD is rare with an incidence estimated as being less than 1 per 100.000 per years. It usually presents in the sixth to eighth decades of life and the youngest case with a pathological confirmation had onset at age 45 years.

Cognitive deficits are often associated with CBD (Graham et al., 2003): as in other frontal-subcortical neurodegenerative diseases associated with parkinsonism, like Progressive Supranuclear Palsy and Multiple System Atrophy, impairments on tests of executive functioning are one of the most consistent finding in CBD (Bonelli & Cummings, 2008; Lange et al., 2003; Salmon & Filoteo, 2007). Impaired performances on the Wisconsin Card Sorting Test, Trail-Making Test, letter fluency, and working memory have been almost consistently reported in groups of CBD patients and in individuals. If executive functions based on the dorsolateral portion of the prefrontal cortex are impaired, no findings are available about executive functions based on the orbital portion of the prefrontal cortex (for a anatomical distinction see Stuss & Levine, 2002). This kind of functions involves risk evaluation, reward processing and decision making, etc. (Wallis, 2007). Evaluation of these executive functions is of particular interest because they have been shown to be impaired early in the course of Frontotemporal Dementia (Gregory et al., 2002; Torralva et al., 2007) and in Parkinson Disease (Kobayakawa et al., 2008; Pagonabarraga et al., 2007; Perretta et al., 2005).

Regards to memory impairments in CBD, the distinction between episodic and semantic memory must be taken in account. Findings about episodic memory functioning in CBD are inconsistent (Graham et al., 2003): some studies (group studies and single case studies) showed normal performance on story/list recall while some other studies found deficits. Performance on a list recall task usually improves when patients are given semantic cues, suggesting problems with retrieval, rather than simply with encoding. Trying to explain this phenomenon, common in predominantly frontal-subcortical neurodegenerative diseases, has been proposed that the episodic memory impairment in CBD is due to poor use of strategic processes in encoding and retrieval, arising from the disruption of frontal-subcortical circuits and the subsequent frontal impairment (Pillon & Dubois, 2003). Less findings on semantic memory in CBD are reported. Taking performances in naming and verbal fluency as a semantic index (Hodges, 2006), few studies suggested

that semantic memory may remain relatively preserved (Beatty et al., 1995), at least respect to episodic memory, but further studies are needed to consolidate this finding.

The assessment of grammatical comprehension and reading has been sporadically conducted and is not possible to establish their typical functioning in CBD. An exhaustive review of aphasia reports in CBD patients was made by Graham et al. (2003). This review established that 34% (137 of 399) of patients reported in published studies were noted to be aphasic. For clinically diagnosed cases, 31% (90 of 293), were aphasic, whereas for pathologically confirmed cases 44% (47 of 106) of patients were aphasic. Several studies reported also impairments in numerical cognition (Halpern et al., 2007; McMillan et al., 2006) and in visuospatial functions, usually assessed by tests like the Benton's Judgement of Orientation of Lines (Benton et al., 1978).

However, as previously underlined, apraxia is the symptom most often associated with CBD. Limb apraxia is usually asymmetric and ideomotor: patients are typically impaired at using tools, miming tool use, and imitating mimes of tool use, while recognition of actions is usually relatively preserved (Graham et al., 2003). Rothi et al. (1991; 1997) proposed a cognitive model of limb apraxia, further modified (Cubelli et al., 2000; see Figure 1; for a review of cognitive approaches to limb apraxia see Petreska et al., 2007), that predicted several clinical pictures.

This cognitive model of praxis process presents the following characteristics, summarized in Bartolo et al. (2007), where a specific neuropsychological battery (Limb Apraxia Battery), based on this model, is proposed: (a) a route for the processing of familiar gestures (lexical route) and one for meaningless gestures (non lexical route); (b) the lexical route includes a mechanism for gesture identification (action semantic), which specifies gestures meaning, the context in which they have to be executed, the functional properties of the objects, and all the knowledge related to the objects. It also includes a lexical system (gestural lexicon) that processes learned gestures and constitutes the repertoire of their motor programs; (c) the lexical system is further divided into an "input" stage for gesture recognition and an "output" stage for gesture production; (d) the non-lexical route includes a visuo-motor conversion mechanism, which transforms the visual information into motor action; (e) either the lexical route and the non-lexical route converge in a gestural buffer that holds the motor program information on-line until the gesture is reproduced.

This cognitive model predicts five clinical pictures of limb apraxia, according to the different loci of impairment. A deficit of the action input lexicon should lead to a pantomime agnosia (i.e., a difficulty in the discrimination and comprehension of gestures; a deficit of the action semantics should

lead to a system conceptual apraxia without ideomotor apraxia; a deficit of the action output lexicon should lead to a conceptual apraxia, with spared gesture-meaning association; a deficit of the visuo-motor conversion mechanism should lead to conduction apraxia; a deficit of the gestural buffer should lead to both ideomotor and ideational apraxia (i.e., impairment in all execution tasks with preserved ability to perform judgement and categorization tasks).

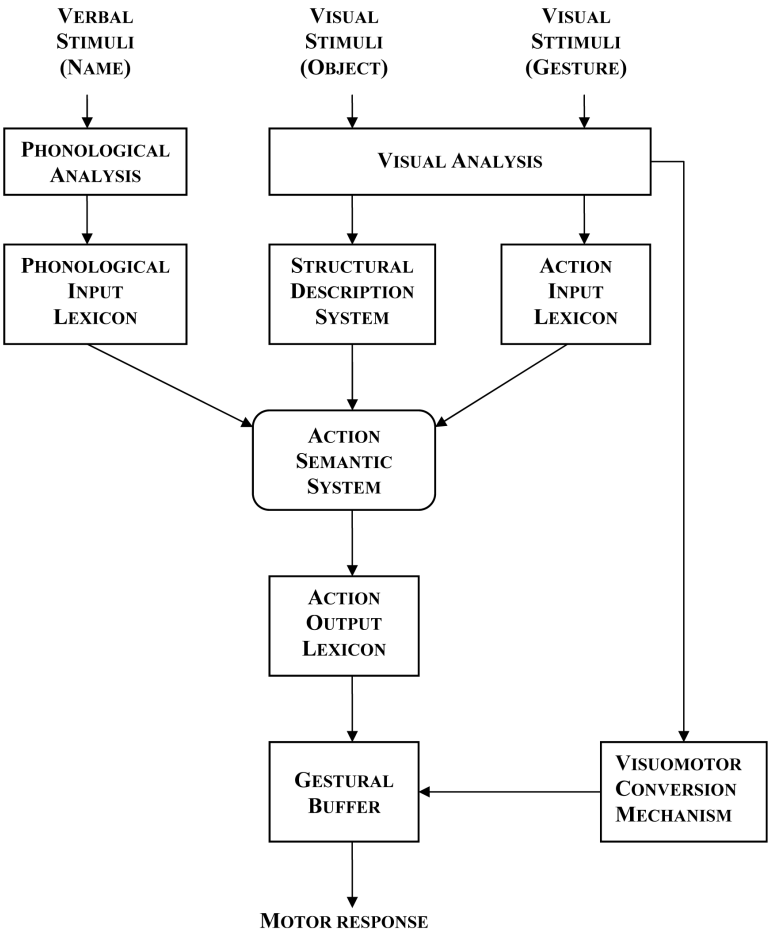


*Figure 1. Outline of the cognitive model of limb praxis (Cubelli et al., 2000), with permission*

Finally, other deficits of praxis are reported in CBD patients apart from the ideomotor one: limb kinetic apraxia, ideational apraxia, buccofacial apraxia, truncal apraxia. Constructional apraxia (evidenced on tests of copying and drawing) and handwriting impairment are also common in CBD, related to the prominent limb apraxia displayed by most patients. Thus, apraxia is common in CBD and typically of ideomotor type but is not universal: it's present in up to 70% of patients with clinically diagnosed CBD (Leiguarda et al., 1994; Soliveri et al., 2005; Zadikoff & Lang, 2005).

In this case-report we describe a patient, with a clinically diagnosed CBD, that showed a clear pattern of ideational and ideomotor apraxia, that can be framed within one of the clinical pictures of the cognitive model of limb apraxia (Cubelli et al., 2000).

## 2. Methods

### *2.1. Case report and cognitive assessment*

N.C. (original patient initials are substituted by fictitious ones), a right-handed, 50-year-old italian housewife, with 8 years of formal education, presented to our attention in July 2006. She had no family history of neurological disease. During the summer of 2004 she developed progressive clumsiness in her left arm, particularly the hand. In February 2005 she underwent brain magnetic resonance imaging (MRI) with gadolinium that resulted normal. Further she developed myoclonous movements; the husband described her memory less efficient than before during daily life activities, and her mood lower than usual. Over the next year her motors symptoms worsened, involving both arms and legs. Mood and memory disturbances became evident. When she underwent to our attention in July 2006, the neurological examination showed global bradikynesia, rigidity of left limbs, bilateral focal myoclonous, stimulus sensitive more evident on the left limbs, right foot dystonia, gait apraxia and alien limb phenomenon on the left arm. She also presented impairments in two point discrimination, graphaestesia and stereognosis on the left side. D antibody testing was in a normal value. Cerebrospinal fluid examination showed no olygoclonal bands and increased levels of Tau protein (t-Tau liquor: 420 pg/ml, normal value 121-294; p-Tau liquor: 119 pg/ml, cut-off: 61). Positron emission tomography with 18FDG and single-photon-emission computed-tomography scanning with Tc99m-neurolite showed bilateral reductions in resting levels of glucose metabolism and blood flow in the posterior frontal, inferior parietal and superior temporal re-

gions with a right hemisphere prevalence. It was performed a brain MRI with gadolinium, that showed an aspecific periventricular anterior hyperintensity on the right hemisphere.

At the time of testing, the patient was alert, better oriented in space than in time: 4/5 and 3/5, respectively, at the MMSE orientation items (Mini Mental Status Examination: Folstein et al., 1975). MMSE global score was 22/30, one point below the cut-off. Her language production was not fluent, with correct syntax, a mild anomia, and adequate content. Her autobiographical memory, as measured with tests currently used for clinical purposes, was preserved. Results of the neuropsychological examination are reported in Tables 1, 2.

Language examination showed: a not fluent speech, with a minimal syntactic simplification; an impaired grammatical comprehension as assessed with the Token Test, with many errors in the second half of the test; an impairment in category and letter fluency; an almost preserved oral naming in Boston Naming Test-Short Form and in an another similar test; an impaired oral repetition, with many errors for long words (e.g. "extraterritorial") and for sentences with subordinates. Episodic memory was assessed by word-list recall: immediate recall was impaired while delayed recall was above the cut-off point; semantic memory, assessed by naming test, resulted normal. Verbal and spatial span resulted impaired. The performance in a modified version of the Stroop Test (Caffarra et al., 2002) was impaired, due to a strong interference effect; also the score in the Frontal Assessment Battery (Dubois et al., 2000) was markedly under the cut off point: in this task must be underlined the presence of a prehension behaviour. In contrast, the performance in a test of cognitive estimates of times and weights (Nichelli et al., 2002) was not impaired. The assessment of abstract reasoning skills and visuospatial functions showed impaired performances in Raven's Coloured Progressive Matrices and the Benton's Judgement of Line Orientation Test, respectively. In September 2007, one year after N.C. arrived in our Neurology Unit, the clinical conditions markedly worsened. She became unable to walk, developed a clear dementia, with visual hallucinations, a phenomenon not so rare in parkinsonian taupathies (Diederich et al., 2007).

*Table 1. Scores reported in tests used in neuropsychological assessment of patient*

| Cognitive Tasks | Cut-off | Range | Scores* |
|---|---|---|---|
| **Memory** | | | |
| Verbal Span (Spinnler & Tognoni, 1986) | > 3.75 | | **3.05** |
| Spatial Span (Corsi Test) (Spinnler & Tognoni, 1986) | > 3.50 | | **2.12** |
| Rey Auditory Verbal Learning Test: Immediate Recall (Carlesimo et al., 1996) | > 28.53 | 0 - 75 | **26.8** |
| Delayed Recall | > 4.69 | 0 - 15 | 5.84 |
| **Language** | | | |
| Boston Naming Test: Short Form (Mack et al., 1992) | | 0 - 30 | 25 |
| Oral Naming (Gainotti et al., 1976) | > 95% | 0 - 16 | 16 |
| Token Test (Spinnler & Tognoni, 1986) | > 26.5 | 0 - 30 | **18** |
| Category fluency (Spinnler & Tognoni, 1986) | > 7.25 | | **5.10** |
| Letter fluency (Spinnler & Tognoni, 1986) | > 17.35 | | **9.7** |
| Benton's Judgement of Line Orientation Test (Benton et al., 1978) | > 19 | 0 - 30 | **16** |
| **Abstract Reasoning** | | | |
| Coloured Progressive Matrices (CPM 47) (Spinnler & Tognoni, 1986) | > 18.96 | 0 - 36 | **12.5** |
| Weigl Sorting Test (Spinnler & Tognoni, 1986) | > 4.50 | 0 -15 | 8.1 |
| **Executive Functions** | | | |
| Frontal Assessment Battery (Dubois et al., 2000) | > 13.5 | 0 - 18 | **5** |
| Stroop: Time Interference Effect (Caffarra et al., 2002) | < 36.9 | … | **186.5** |
| Stroop: Error Interference Effect (Caffarra et al., 2002) | < 4.24 | … | 2.65 |
| Time and Weight Estimation Test (Nichelli et al., 2002) | | | |
| A: Times | > 19 | 0-30 | 22 |
| B: Weights | > 19 | 0-30 | 25 |
| A + B | > 39 | 60 | 47 |
| \| B – A \| | < 8 | 0-30 | 3 |

* Scores in bold are under cut-off point, indicating an impaired performance.
Scores are corrected for age and level of education, when needed.

*Table 2. Scores reported in tests for assessment of apraxia in patient MB*

| Tests for apraxia assessment | v.n. | Range | Score* |
|---|---|---|---|
| Ideomotor Apraxia: Right Upper Limb (Spinnler & Tognoni, 1987) | > 16 | 0 - 20 | **13.75** |
| Ideomotor Apraxia: Left Upper Limb (Spinnler & Tognoni, 1987) | > 16 | 0 - 20 | **10** |
| Buccofacial Apraxia | > 16 | 0 - 20 | **15.75** |
| Movement Imitation Test: Right Upper Limb (De Renzi & Lucchelli, 1988) | | | |
| Preliminar trial | | 0 - 6 | 6 |
| Finger movements | | 0 - 36 | 8 |
| Hand/Limb movement | | 0 - 36 | 20 |
| Total | > 62 | 0 - 78 | **34** |
| Movement Imitation Test: Left Upper Limb (De Renzi & Lucchelli, 1988) | | | |
| Preliminar trial | | 0 - 6 | 5 |
| Finger movements | | 0 - 36 | 3 |
| Hand/Limb movement | | 0 - 36 | 5 |
| Total | > 62 | 0 - 78 | **13** |

* Scores in bold ar under cut-off point, indicating an impaired performance.
Scores are corrected for age and level of education, when needed.

### *2.2. Apraxia assessment*

The profile that emerged from the neuropsychological examination revealed several cognitive deficits, with a marked limb apraxia. She failed in tests of either ideomotor (Spinnler & Tognoni, 1987) and ideational apraxia. The Movement Imitation Test (De Renzi & Lucchelli, 1988) was performed first with the right upper limb, than with the left one. Left limb resulted more affected than the right one, confirming the asymmetry of limb apraxia in CBD. The score in a test of buccofacial apraxia (Spinnler & Tognoni, 1987) was around the cut-off point, so the presence of buccofacial apraxia was questionable. The right limb apraxia strongly affected constructional praxis and handwriting, not possible to assess with formal tasks like the Copy of the Complex Rey Figure. After this preliminary assessment of praxis (see Table 2), a deepest exam was made with a new battery for the assessment of limb apraxia (Limb Apraxia Battery: italian version in Bartolo et al., 2008; english version in Bartolo et al., 2007). Because of the alien limb phenomenon registered in her

left arm and because the general difficulty to complete a wide neuropsychological examination, the Limb Apraxia Battery was performed only with the right upper limb. Scores and types of errors in gesture production are reported in Table 3. She presented an impairment of all levels of production of gestures (production and imitation), more for the left than the right hand as evidenced by the Movement Imitation Test, and no deficit at the level of gesture reception (recognition and identification). The most common error across different subtests was the production of a not recognizable movement.

## 3. Discussion

Having presented cognitive impairments of patient N.C., it's possible to discuss some points of interest presented by this case. (1) N.C. presented an early onset of clinical manifestations (48 years of age), even if younger cases of clinically diagnosed CBD individuals are reported (DePold Hohler et al., 2003). (2) Even if previously reported studies suggested that almost all cognitive functions may be affected in CBD patients, N.C. quickly developed a neuropsychological profile characterized by impaired performance in, at least, one task for each cognitive domain that was possible to assess (praxis, language, executive functions, visuospatial functions, reasoning). (3) The third point of interest involves the description of the apraxic deficit. As reported before, N.C. failed in either tests of ideomotor and ideational apraxia, so her deficit could be described as ideational plus ideomotor apraxia. Which clinical picture better describes praxis impairment exhibited by patient N.C.? The scores reported in the tests of the Limb Apraxia Battery (Bartolo et al., 2008) suggest that apraxia of patient N.C. could be described by the latter clinical picture, where a deficit of the gestural buffer causes an impairment in all execution tasks. This empirical evidence is in the same direction of other studies (Johnson-Frey, 2004; Rothi et al., 1991; for a review Mahon & Caramazza, 2005) that reported findings supporting that production of actions can be impaired while recognition of actions is spared, and recognition of actions can be impaired while production of actions is spared. Also Cubelli et al. (2000), presenting the cognitive model of limb apraxia, reported three cases of a specific gestural buffer impairment. Theses cases are so consistent with models of praxis that draw a functional distinction between the representations that underlie perception and the representations that underlie production of actions (e.g., Cubelli et al., 2000; Rapcsak et al., 1995; Rothi et al., 1991; 1997; Rumiati et al., 2001) and are against models that assume that motor production processes are necessary to successfully recognize actions.

*Table 3. Scores reported in the tests of the Limb Apraxia Battery (Bartolo et al., 2008), performed only with the right upper limb*

| Levels of Processing | | Types of Gesture | Tasks (input) | Score | Range | Cut-off | Errors |
|---|---|---|---|---|---|---|---|
| PRODUCTION LEVEL | LEXICAL SEMANTIC ROUTE | Intransitive | 1. Verbal (denomination) | 10 | 0 - 16 | ≥ 14 | 6: 2 M; 4 NR |
| | | | 2. Verbal (description) | 8 | 0 - 16 | ≥ 14 | 8: 3 M; 5 NR |
| | | | 3. Visual | 7 | 0 - 16 | ≥ 11 | 9: 2 M; 1 O; 6 NR |
| | | | 4. Imitation | 13 | 0 - 16 | ≥ 15 | 3: 2M; 1 NR |
| | | Transitive | 5. Use | 11 | 0 - 16 | ≥ 15 | 5: 2 G; 2 O; 1 M |
| | | | 6. Imitation | 8 | 0 - 16 | ≥ 15 | 8: 2 G; 2 O; 1 M; 3 NR |
| | | Pantomime | 7. Verbal | 3 | 0 - 16 | ≥ 12 | 13: 3 PLO; 3 D; 7 NR |
| | | | 8. Visual | 4 | 0 - 16 | ≥ 12 | 12: 3 PLO; 2 D; 7 NR |
| | | | 9. Tactile | 6 | 0 - 16 | ≥ 11 | 10: 3 PLO; 7 NR |
| | | | 10. Imitation | 4 | 0 - 16 | ≥ 15 | 12: 1 PLO; 11 NR |
| | Visuomotor Conversion Mechanism | Meaningless | 11. Imitation | 2 | 0 - 16 | ≥ 15 | 14: 2 P; 2 M; 10 NR |
| RECEPTIVE LEVEL | INPUT LEXICON | Intransitive | 12. Recognition | 37 | 0 - 40 | ≥ 35 | |
| | | Pantomime | 13. Recognition | 36 | 0 - 40 | ≥ 35 | |
| | ACTION SEMANTIC | Intransitive | 14. Identification | 16 | 0 - 16 | ≥ 13 | |
| | | Pantomime | 15. Identification | 16 | 0 - 16 | ≥ 15 | |

Errors:
Production of intransitive gestures: M = movement/sequence; O = orientation; NR = not recognizable.
Production of transitive gestures: G = grasping; O = orientation; M = movement; NR = not recognizable.
Production of meaningless gestures: P = position; M = movement; NR = not recognizable.
Production of pantomimes: PLO = parts (of body) like objects; D = distance; NR = not recognizable.

## 4. Conclusions

Patient N.C. presented an early onset of a clinically diagnosed corticobasal degeneration. Her neuropsychological profile is of particular interest for the rapid development of a diffuse cognitive impairment. Her limb apraxia may be explained by an impairment of the gestural buffer, as proposed by a cognitive model of praxis.

## References

Bak, T.H., Crawford, L.M., Hearn, V.C., Mathuranath, P.S., & Hodges, J.R. (2005). Subcortical dementia revisited: similarities and differences in cognitive function between progressive supranuclear palsy (PSP), corticobasal degeneration (CBD) and multiple system atrophy (MSA). *Neurocase*, 11, 268-273.

Bartolo, A., Cubelli, R., & Della Sala, S. (2008). Cognitive approach to the assessment of limb apraxia. *The Clinical Neuropsychologist*, 22, 27-45.

Bartolo, A., Drei, S., Cubelli, R., & Della Sala, S. (2008). Approccio cognitivo alla valutazione della aprassia degli arti. Presentazione di una nuova batteria. *Giornale Italiano di Psicologia*, 2, 435-462.

Beatty, W.W., Scott, J.G., Wilson, D.A., Prince, J.R., & Williamson, D.J. (1995). Memory deficits in a demented patient with probable corticobasal degeneration. *Journal of Geriatric Psychiatry & Neurology*, 8, 132-136.

Benton, A.L., Varney, N.R., & Hamsher, K.D. (1978). Visuospatial Judgement. A clinical test. *Archives of Neurology*, 35, 364-367.

Caffarra, P., Vezzadini, G., Dieci, F., Zonato, A. & Venneri, A. (2002). Una versione abbreviata del test di Stroop: dati normativi nella popolazione italiana. *Nuova Rivista di Neurologia*, 12, 111-115.

Carlesimo, G.A., Caltagirone, C., & Gainotti, G. (1996). The Mental Deterioration Battery: normative data, diagnostic reliability and qualitative analyses of cognitive impairment. *European Neurology*, 36, 378-384.

Cubelli, R., Marchetti, C., Boscolo, G., & Della Sala, S. (2000). Cognition in action: testing a model of limb apraxia. *Brain and Cognition*, 44, 144-165.

DePold Hohler, A., Ransom, B.R., Chun, M.R., Troster, A.I., & Samii, A. (2003). The youngest reported case of corticobasal degeneration. *Parkinsonism & Related Disorders*, 10, 47-50.

De Renzi, E., & Lucchelli, F. (1988). Ideational Apraxia. *Brain*, 111, 1173-1185.

De Renzi, E., Motti, F., & Nichelli, P. (1980). Imitating gestures: a quantitative approach to ideomotor apraxia. *Archives of Neurology*, 37, 6-10.

Diederich, N.J., Leurgans, S., Fan, W., Chmura, T.A., & Goetz, C.G. (2008). Visual hallucinations and symptoms of REM sleep behavior disorder in Parkinsonian tauopathies. *International Journal of Geriatric Psychiatry*, 23, 598-603.

Dubois, B., Slachevsky, A., Litvan, I., & Pillon, B. (2000). The FAB: a frontal assessment battery at bedside. *Neurology*, 55, 1621-1628.

Folstein, M.F., Folstein, S.E., & McHugh, P.R. (1975). Mini Mental State. A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatry Research*, 12, 189-198.

Graham, N.L., Bak, T.H., & Hodges, J.R. (2003). Corticobasal Degeneration as cognitive disorder. *Movement Disorders*, 18, 1224-1232.

Gregory, C., Lough, S., Stone, V., Erzinclioglu, S., Martin, L., Baron-Cohen, S., et al. (2002). Theory of mind in patients with frontal variant frontotemporal dementia and Alzheimer's disease: theoretical and practical implications. *Brain*, 125, 752-764.

Halpern, C., Clark, R., Moore, P., Cross, K., & Grossman, M. (2007). Too much to count on: impaired very small numbers in corticobasal degeneration. *Brain and Cognition*, 64, 144-149.

Hodges, J.R. (2006). Alzheimer's centennial legacy: origins, landmarks and the current status of knowledge concerning cognitive aspects. *Brain*, 129, 2811-2822.

Johnson-Frey, S.H. (2004). The neural basis of complex tool use in humans. *Trends in Cognitive Sciences*, 8, 71-78.

Kobayakawa, M., Koyama, S., Mimura, M., & Kawamura, M. (2008). Decision making in Parkinson's disease: Analysis of behavioral and physiological patterns in the Iowa gambling task. *Movement Disorders*, 23, 547-552.

Lange, K.W., Tucha, Q., Alders, G.L., Preier, M., Csoti, I., Merz, B., et al. (2003). Differentiation of parkinsonian syndromes according to differences in executive functions. *Journal of Neural Transmission*, 110, 983-995.

Leiguarda, R., Lees, A.J., Merello, M., Starkstein, S., & Marsden, C.D. (1994). The nature of apraxia in corticobasal degeneration. *Journal of Neurology, Neurosurgery & Psychiatry*, 57, 455- 459.

Leiguarda, R., Merello, M., & Balej, J. (2000). Apraxia in corticobasal degeneration. In: Litvan, I., Goetz, C.G., Lang, A.E. (eds). *Corticobasal Degeneration*. Philadelphia: Lippincott Williams & Wilkins.

Mack, W.J., Freed, D.M., Williams, B.W., & Henderson, V.W. (1992). The Boston Naming Test: shortened version for use in Alzheimer's disease. Journal of Gerontology. *Journal of Gerontology*, 47, 154-158.

Mahaoatra, R.K., Edwards, M.J., & Schott, J.M. (2004). Corticobasal degeneration. *Lancet Neurology*, 3, 736-743.

Mahon, B.Z., & Caramazza, A. (2005). The orchestration of the sensory motor systems: clues from neuropsychology. *Cognitive Neuropsychology*, 22, 480-494.

McMillan, C.T., Clark, R., Moore, P., & Grossman, M. (2006). Quantifier comprehension in corticobasal degeneration. *Brain and Cognition*, 62, 250-260.

Monelli, R., & Cummings, J.L. (2008). Frontal-subcortical circuits. *The Neurologist*, 14, 100-107.

Pagonabarraga, J., Garcia-Sanchez, C., Llebaria, G., Pascual-Sedano, B., Gironell, A., & Kulisevsky, J. (2007). Controlled study of decision making and cognitive impairment in Parkinson's disease. *Movement Disorders*, 22, 1430-1435.

Perretta, J.G., Pari, G., & Beninger R.J. (2005). Effects of Parkinson Disease on two putative nondeclarative learning tasks: probabilistic classification and gambling. *Cognitive & Behavioural Neurology*, 18, 185-192.

Petreska, B., Ariani, M., Blande, O., & Billard, A.G. (2007). Apraxia. A Review. In: von Hofsten, C. (ed.). *From Action to Cognition. Progress in Brain Research*. Amsterdam: Elsevier.

Pillon, B., & Dubois, B. (2000). Memory and executive processes in corticobasal degeneration. In: Litvan, I., Goetz, C.G., Lang, A.E. (eds). *Corticobasal Degeneration*. Philadelphia: Lippincott Williams & Wilkins.

Rapcsak, S.Z., Ochipa, C., Anderson, K.C., & Poizner, H. (1995). Progressive ideomotor apraxia: Evidence for a selective impairment of the action production system. *Brain and Cognition*, 27, 213-236.

Rothi, L.J., Ochipa, C., & Heilman, K.M. (1991). A cognitive neuropsychological model of limb praxis. *Cognitive Neuropsychology*, 8, 443-458.

Rothi, L.J., Ochipa, C., & Heilman, K.M. (1997). A cognitive neuropsychological model of limb praxis and apraxia. In: Rothi, L.J., Heilman, K.M. (eds.). *Apraxia, the Neuropsychology of Action*. Hove, UK: Psychology Press.

Rumiati, R.I., Zanini, S., Vorano, L., & Shallice, T. (2001). A form of ideational apraxia as a selective deficit of contention scheduling. *Cognitive Neuropsychology*, 18, 617–642.

Salmon, D.P., & Filoteo, J.V. (2007). Neuropsychology of cortical versus subcortical dementia syndromes. *Seminars in Neurology*, 27, 7-21.

Soliveri, P., Piacentini, S., & Girotti, F. (2005). Limb apraxia in corticobasal degeneration and progressive supranuclear palsy. *Neurology*, 64, 448-453.

Spinnler, H., Tognoni, G., & Gruppo Italiano per lo Studio Neuropsicologico dell'Invecchiamento (1987). Standardizzazione e taratura italiana di test neuropsicologici. *The Italian Journal of Neurological Sciences*, 6, Suppl. 8.

Stuss, D., & Levine, B. (2002). Adult clinical neuropsychology: lessons from studies of the frontal lobes. *Annual Reviews of Psychology*, 53, 401-433.

Torralva, T., Kipps, C.M., Hodges, J.R., Clark, L., Bekinschtein, T., Roca, M., et al. (2007). The relationship between affective decision-making and theory of mind in the frontal variant of frontotemporal dementia. *Neuropsychologia*, 45, 342-349.

Wallis, J.D. (2007). The orbitofrontal cortex and its contribution to decision making. *Annual Reviews of Neuroscience*, 30, 31-56.

Zadikoff, C., & Lang, A.E. (2005). Apraxia in movement disorders. *Brain,* 128, 1480-1497.