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Anosognosia for apraxia: Experimental evidence for defective awareness of one's own bucco-facial gestures



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ABSTRACT

Anosognosia is a multifaceted, neuro-psychiatric syndrome characterized by defective awareness of a variety of perceptuo-motor, cognitive or emotional deficits. The syndrome is also characterized by modularity, i.e., deficits of awareness in one domain (e.g., spatial perception) co-existing with spared functions in another domain (e.g., memory). Anosognosia has mainly been reported after right hemisphere lesions. It is however somewhat surprising that no studies have thus far specifically explored the possibility that lack of awareness involves apraxia, i.e., a deficit in the ability to perform gestures caused by an impaired higher-order motor control and not by low-level motor deficits, sensory loss, or failure to comprehend simple commands. We explored this issue by testing fifteen patients with vascular lesions who were assigned to one of three groups depending on their neuropsychological profile and brain lesion. The patients were asked to execute various actions involving the upper limb or bucco-facial body parts. In addition they were also asked to judge the accuracy of these actions, either performed by them or by other individuals. The judgment of the patients was compared to that of two external observers.

Results show that our bucco-facial apraxic patients manifest a specific deficit in detecting their own gestural errors. Moreover they were less aware of their defective performance in bucco-facial as compared to limb actions. Our results hint at the existence of a new form of anosognosia specifically involving apraxic deficits.

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Abbreviations: AHP, Anosognosia for Hemiplegia; AA, Anosognosia for Apraxia; BFA, bucco-facial apraxia; A+, apraxic patients; A-, left hemisphere brain damaged non apraxic patients; RBD, right hemisphere brain damaged patients.

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1. Introduction

The term *anosognosia*, initially coined to indicate the denial of motor deficits contralateral to a brain lesion (Anosognosia for Hemiplegia – AHP, Babinski, 1914), refers to a multifaceted syndrome where patients who have suffered strokes, traumatic brain injury, degenerative diseases or neuropsychiatric disorders show complete or partial lack of awareness of a variety of neurological and cognitive deficits (e.g., hemianopia, blindness, hemianesthesia, neglect, aphasia, amnesia) (Prigatano, 2010).

Clinical reports and experimental studies on AHP (Cocchini, Beschin, Fotopoulou, & Della Sala, 2010; Moro, 2013; Moro, Pernigo, Zapparoli, Cordioli, & Aglioti, 2011; Ramachandran, 1994) and Alzheimer's Disease (Mograbí & Morris, 2013) show distinct types of anosognosia where implicit and emergent residual forms of awareness are present. Moreover, patients may exclusively deny their own paralysis but recognize deficits in other patients (1st person deficit) or fail to recognize motor impairment both in themselves and in other subjects (1st and 3rd person deficit) (Marcel, Tegnèr, & Nimmo-Smith, 2004; Moro et al., 2011).

Despite the steady increase of interest in anosognosia, no studies have thus far specifically set out to investigate the possible existence of anosognosia for apraxia (AA). The term apraxia refers to a wide spectrum of disorders with in common an inability to perform skilled or learned purposeful gestures. Although sometimes co-existing with motor or sensory deficits or language disorders, apraxia is not explained by any of these (Zadikoff & Lang, 2005). Conceptual and production components of gestural organization may be differentially affected, leading to ideational (i.e., defective action and object-use knowledge) or ideomotor apraxia (i.e., defective action execution in gesture pantomime and imitation) (Leiguarda & Marsden, 2000). In limb apraxia, imitation of transitive gestures (e.g., hammering a nail) is more impaired than the imitation of intransitive gestures (e.g., waving goodbye) (Buxbaum, Kyle, & Menon, 2005). Among the body-part defined subtypes of apraxia, bucco-facial apraxia (BFA) refers to an inability to voluntarily control facial, lingual, pharyngeal and masticatory actions (e.g., protruding tongue, blinking eyes) on purpose but not in ecological situations, when the movements are automatically performed. Neuropsychological and neuroanatomical results indicate that BFA and limb apraxia are at least partially independent (Raade, Rothi, & Heilman, 1991). While limb apraxia appears to be more commonly associated with left frontal and parietal brain damage (Pazzaglia, Smania, Corato, & Aglioti, 2008), BFA follows lesions in left prefrontal areas, the central operculum, the insula, the centrum semiovale, their subcortical projections and the basal ganglia (Pramstaller & Marsden, 1996).

In this study we investigated the existence of a specific form of AA related to the possibility that subjects presenting with apraxia also show defective awareness of their difficulties. With this aim we asked patients with or without BFA to judge the correctness of bucco-facial and limb related gestures performed by themselves or by a gender-matched model. The patients' responses were compared with the evaluations provided by their therapist and caregiver. The comparison between discrepancies in judgment of actions

performed by themselves or others allowed us to distinguish deficits in awareness from non-specific difficulties in action recognition.

2. Methods

2.1. Participants

Fifteen brain damaged patients recruited from the Neuro-rehabilitation Units at the IRCCS Santa Lucia (Rome) and the Sacro Cuore Hospital (Negrar, Verona) gave their informed consent for their participation in the study. The procedures were approved by the two local Ethics Committees and the study was carried out in accordance with the guidelines of the Declaration of Helsinki. All the patients were right-handed (Briggs & Nebes, 1975) and had suffered from vascular lesions at times varying between 1 and 27 months before the assessment. They were divided into three groups depending on their symptoms and side of lesion: i) patients presenting with BFA (A+); ii) left brain damaged non-apraxic patients (A-); iii) right brain damaged non-apraxic patients (RBD). The groups were comparable in terms of age and education but differed in onset-assessment intervals with A- and RBD in more acute phases with respect to A+.

The contralesional upper limb motor deficit was assessed by means of the Medical Research Council Scale (Florence et al., 1992). Clinical and demographical data are reported in Table 1.

2.2. Preliminary neuropsychological screening

A battery of standardized tests was used for neuropsychological screening. This involved general cognitive abilities (Raven, Court, & Raven, 1988), Verbal and Visual Memory (Spinnler & Tognoni, 1987), executive functions (non-verbal subtests of the Frontal Assessment Battery – Appollonio et al., 2005) and spatial attention (Line Bisection – Wilson, Cockburn, & Halligan, 1987). Verbal comprehension and denomination subtests of the Aachen Aphasia Test (Luzzatti, Willmes, & De Bleser, 1996) were used to assess language deficits.

As shown in Table 1, the patients' scores rule out the possibility that cognitive disorders play any major role in the experimental results. Verbal Comprehension was spared in all the patients.

2.3. Assessment of apraxia

The presence of BFA was ascertained by means of the Upper and Lower Face Apraxia test (Bizzozero et al., 2000). In this test 29 and 9 actions are selected to evaluate lower and upper face gestures respectively, according to the territory of the cranial nerves involved. Each action is scored 1 (correct) or 0 if there are errors in execution (i.e., amorphous movements, protracted pauses, additional movements, conduits d'approche or incomplete action). These scores are then weighted taking into account relative difficulty (cut-off: lower face = 400.04, upper face = 38.43).

The test for Upper Limb Ideomotor Apraxia (TULIA – Vanbellingen et al., 2010) consists of 48 items, including

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