Pure associative tactile agnosia for the left hand: Clinical and anatomo-functional correlations

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Abstract
Associative tactile agnosia (TA) is defined as the inability to associate information about object sensory properties derived through tactile modality with previously acquired knowledge about object identity. The impairment is often described after a lesion involving the parietal cortex (Caselli, 1997; Platz, 1996). We report the case of SA, a right-handed 61-year-old man affected by first ever right hemispheric hemorrhagic stroke. The neurological examination was normal, excluding major somaesthetic and motor impairment; a brain magnetic resonance imaging (MRI) confirmed the presence of a right subacute hemorrhagic lesion limited to the post-central and supra-marginal gyri. A comprehensive neuropsychological evaluation detected a selective inability to name objects when handled with the left hand in the absence of other cognitive deficits. A series of experiments were conducted in order to assess each stage of tactile recognition processing using the same stimulus sets: materials, 3D geometrical shapes, real objects and letters. SA and seven matched controls underwent the same experimental tasks during four sessions in consecutive days. Tactile discrimination, recognition, pantomime, drawing after haptic exploration out of vision and tactile–visual matching abilities were assessed. In addition, we looked for the presence of a supra-modal impairment of spatial perception and of specific difficulties in programming exploratory movements during recognition.

Tactile discrimination was intact for all the stimuli tested. In contrast, SA was able neither to recognize nor to pantomime real objects manipulated with the left hand out of vision, while he identified them with the right hand without hesitations. Tactile–visual matching was intact. Furthermore, SA was able to grossly reproduce the global shape in drawings but failed to extract details of objects after left-hand manipulation, and he could not identify objects after looking at his own drawings.

This case confirms the existence of selective associative TA as a left hand-specific deficit in recognizing objects. This deficit is not related to spatial perception or to the
1. Introduction

Tactile agnosia (TA) is defined as the inability to recognize objects after tactile exploration, in the presence of relatively intact sensory functions and adequate cognitive abilities. The deficit is modality-specific, as the same object is promptly recognized through other modalities (Saetti, De Renzi, & Comper, 1999). The process of recognition of an object requires various abilities, such as encoding elementary sensory data, integrating the sensory information in order to generate a coherent tactile representation of the object, and associating the tactile representation with semantic knowledge about the object. According to Lissauer (1890), TA may result from impairments at the stage of sensory integration (apperceptive TA) or at a subsequent stage in which tactile representation acquires meaning (associative TA).

The neural correlates of tactile object recognition involve the inferior parietal cortex and the insular cortex (Bohlhalter, Fretz, & Weder, 2002; Caselli, 1997; Crutch, Warren, Harding, & Warrington, 2005; Platz, 1996; Reed, Caselli, & Farah, 1996). Crutch et al. (2005) described a patient with bilateral apperceptive TA due to a left hemisphere lesion extending from the parietal operculum to the superior parietal lobe, which involves the posterior part of the post-central gyrus, the supramarginal gyrus and the anterior part of the angular gyrus in the inferior parietal lobe. The two patients affected by apperceptive TA described by Bohlhalter et al. (2002) showed a lesion in left post-central gyrus and left retro-insular/parietal operculum, and in the posterior parietal lobe. With respect to associative TA, Caselli (1991) described seven patients with brain lesions mainly damaging the left or the right inferior parietal, the posterior temporal, and the posterior insular cortex. Platz (1996) reported a patient with left-hand associative TA, HK, who showed a lesion limited to the right post-central gyrus and supralong marginalgyrus. More recently, a focal lesion involving selectively the trunk and the splenium of the corpus callosum was associated with left-hand associative TA (Balsamo, Trojano, Giamundo, & Grossi, 2009).

Since the first observations of TA, various interpretations and explanation attempts of the deficit have been advanced. The absence of primary somatosensory impairment is a crucial point, which however is hard to disentangle because, as clearly explained by Saetti et al. (1999), ‘the concomitant presence of TA and somesthetic deficits is frequent, due to the fact that the parietal lesion that causes agnosia often extends to the somatosensory cortex and/or its afferent pathways’. Studies that investigated tactile object recognition abilities of brain-damaged patients reported only relatively, rather than completely, intact elementary processing (e.g., touch, pain, temperature, vibration, thermal properties). For this reason, some authors concluded for the impossibility to support the concept of agnosia (Bay, 1944; Campora, 1925). However, Wiebers, Dale, Kokmen, and Swanson (1998) pointed out a disproportion between the severity of the sensory deficit (often mild) and the recognition deficit, suggesting that in these cases sensory difficulties cannot account for defective recognition. Moreover, some cases of ‘pure’ TA have been described (Caselli, 1991; Endo, Miyasaka, Makishita, Yanagisawa, & Sugishita, 1992; Wernicke, 1895). Platz (1996) proposed an alternative concept for TA, which is compatible with the presence of sensory deficits, suggesting that the tactile recognition of an object involves distributed perceptual-motor processes rather than a separate stage following perception.

The determinants of defective tactile object recognition represent an open issue. Some authors highlighted that the exploratory motor procedures of the handling process are directly linked to the extraction of specific properties of objects. In particular, Caselli (1991) suggested that tactile object recognition involves a dynamic reciprocal interaction between exploratory hand and finger movements and the sensory data acquired as a result of those movements. Valenza et al. (2001) described a patient with a praxic disorder, showing dissociation between impaired shape and object recognition when using spontaneous exploratory procedures (active touch) and intact shape recognition when the experimenter guided the exploration (passive modality). In this case, the deficit was attributed to a selective difficulty in the exploration and manipulation of objects (pure tactile apraxia). Crutch et al. (2005), describing a patient with both apraxia and apperceptive TA, demonstrated that the computation of shape properties depends on intact programming of exploratory hand movements. An alternative possibility, however, is that the deficit in tactile object recognition does not allow the subject to manipulate efficiently the stimulus, because of the inability to distinguish a priori the salient features. In line with this view, Platz (1996) suggested that the mild deficit in exploration exhibited by patient HK might be the consequence, rather than the cause of the recognition deficit.

Another hypothesis is that TA could be secondary to a general supra-modal disorder of spatial perception (De Renzi, 1982; Ettlinger, Warrington, & Zangwill, 1957; Semmes, 1965). Different tasks, such as point localization, sense of passive movement and map-following, have been used to test the spatial abilities in patients with difficulty in tactile object recognition. Semmes, Weinstein, Ghent, and Teuber (1963) used a locomotor spatial test, in which patients were required to walk through a set of nine discs placed on the floor, guided by a visually presented map. They showed that the patient recognition performance correlated with the spatial recognition abilities, concluding that TA was
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