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Sensory Integration Deficits in Neurodegenerative Diseases: Implications for Apraxia

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ABSTRACT

Objective. Apraxia is the inability to perform voluntary, skilled movements following brain lesions, in the absence of sensory integration deficits. Yet, patients with neurodegenerative diseases may have sensory integration deficits, so we tested the associations and dissociations between apraxia and sensory integration.

Methods. Forty-four patients with neurodegenerative diseases and 20 healthy controls underwent extensive testing of sensory integration (i.e., localization of tactile, visual and proprioceptive stimuli; agraphesthesia; astereognosis) and apraxia (i.e., finger dexterity, imitation, tool use).

Results. The results showed (1) that patients with Alzheimer's disease, corticobasal syndrome or posterior cortical atrophy were impaired on both dimensions; (2) An association between both dimensions; (3) that when sensory integration was controlled for, the frequency of apraxia decreased dramatically in some clinical subgroups.

Conclusion. In a non-negligible portion of patients, the hypothesis of a disruption of sensory integration can be more parsimonious than the hypothesis of apraxia in case of impaired skilled gestures. Clinicians and researchers are advised to integrate sensory integration measures along with their evaluation of apraxia.

Keywords: dementia, apraxia, neuropsychology, motor control, multisensory integration.

1. INTRODUCTION

1.1. APRAXIA AND NEURODEGENERATIVE DISEASES

Limb apraxia has long been defined as the inability to perform intentional, skilled movements following brain lesions, in the absence of elementary motor or sensory deficits, lack of attention, and comprehension (Rothi et al., 1997). This syndrome is generally assessed with measures of finger dexterity, imitation of meaningless gestures, and tool use (for a review, see Baumard & Le Gall, 2021). Impairment in these tasks has been associated with different subtypes of apraxia like limb-kinetic apraxia (i.e., loss of finger dexterity), ideomotor apraxia (i.e., inability to perform skilled movements on verbal command or imitation), and ideational apraxia (i.e., difficulties using tools). It has also been associated with lesions of somatosensory and temporo-fronto-parietal brain regions (Goldenberg, 2017; Luria, 1978; Reynaud et al., 2016). Apraxia is frequent in neurodegenerative diseases and its presence can be valuable for the differential diagnosis of several diseases (e.g., Buchmann et al., 2020).

The “typical” amnesic form of Alzheimer’s disease (AD) is characterized by severe episodic memory and learning deficits, associated with one or more cognitive impairment, as well as atrophy of the hippocampus, parietal and frontal brain regions (McKhann et al., 2011). Posterior cortical atrophy (PCA) is defined as a progressive decline of neuropsychological functions of the occipital and parietal lobes (e.g., visual-spatial impairments, neglect, agnosia, apraxia), contrasting with the relative sparing of memory (Crutch et al., 2017). Corticobasal syndrome (CBS) combines a parkinsonian syndrome with cortical signs corresponding to frontal/parietal brain atrophy (Armstrong et al., 2013). Finally, patients with semantic dementia (SD) typically have atrophy of the temporal lobes sparing the hippocampal cortex, and causing severe semantic loss but no episodic memory deficits (Gorno-Tempini et al., 2011).

1.2. APRAXIA AND SENSORY INTEGRATION

Exclusion criteria play a critical role in the clinical diagnosis of apraxia, because their presence makes the diagnosis of limb apraxia (i.e., a *specific* impairment of skilled gestures) highly uncertain (Bartolo & Stieglitz Ham, 2016). These criteria have been historically defined for the study of patients with cerebrovascular disease and unilateral brain damage, to distinguish apraxia from sensory and motor disorders. It is a critical issue when it comes to neurodegenerative diseases (ND), because the latter typically have bilateral brain atrophy. Furthermore, sensory integration occurs in multiple brain regions, including elementary sensory areas, the posterior parietal cortex, the superior temporal sulcus, entorhinal cortex, hippocampus, and subcortical structures (e.g., Yau et al., 2015), which are typical lesion sites of several neurodegenerative diseases like Alzheimer's disease, posterior cortical atrophy, and corticobasal syndrome. It means that sensory integration deficits may actually explain apraxia in some patients at least. This is all the more important because during the 20th century, some manifestations of apraxia have been explained by body representation disorders, especially since the body schema is thought to underlie posture adjustment and movement guidance (e.g., Buxbaum, 2001). It turns out that body representations are derived from the integration sensations arising from multiple sensory inputs (Petkova & Ehrsson, 2008). So, if sensory integration is at the root of body representations, and if the latter can be at stake in apraxia, then the question arises as to whether deficits of sensory integration may explain limb apraxia in some patients.

Sensory integration is “*the process by which information from different sensory modalities is combined by the nervous system to form a stable and coherent percept of the world [...], to perceive and understand our environment, enabling us to move and interact with objects in our surroundings*” (Yau et al., 2015, p.1). It relies on a two-step process (Berlucchi & Vallar, 2018). First, sensory inputs are processed within an intrasensory integration system that transforms primary afferent information from individual sensory systems into higher-order

sensory maps. Second, unimodal signals are combined by an intersensory integration system that binds information arising from sources of different modalities. This process allows individuals to detect and locate stimuli (e.g., on the skin, or in the visual field) and is at the root of body representation (e.g., proprioception). It has been studied with different paradigms like single unit recordings, event-related potentials, fMRI, non-invasive brain stimulation methods, and behavioral measures (i.e., accuracy and response time; Stevenson et al., 2014; Yau et al., 2015). In a clinical setting, neurologists and neuropsychologists investigate sensory integration by testing how patients can detect, localize and identify stimuli. Hereafter sensory integration refers to this clinical definition.

1.3. GOALS OF THE STUDY

The goals of this study were twofold. First, to undertake a systematic assessment of sensory integration in patients with Alzheimer's disease (AD), semantic dementia (SD), posterior cortical atrophy (PCA), or corticobasal syndrome (CBS). These diseases were selected because they are typically associated with atrophy of brain regions that are critical to sensory integration or apraxia. The second goal of this study was to explore associations and dissociations between sensory integration and apraxia. We made the hypothesis that sensory integration deficits, an exclusion criterion for apraxia, actually linked to apraxic deficits in some patients with neurodegenerative diseases.

2. MATERIALS AND METHODS

2.1. PATIENTS

Forty-four neurologic patients diagnosed with AD, SD, PCA, or CBS, as well as 20 healthy control participants (HC) gave informed consent to take part in the study (Table 1). To improve statistical power, PCA and CBS cases, which are rare diseases, were pooled into a larger group, corresponding to patients with typically severe atrophy of frontal and/or parietal

lobes (i.e., FPA). Exclusion criteria included having a previous history of neurological or psychiatric condition, having a MMSE score below 10, having a medical condition that precluded reliable and/or ethical data collection (e.g., rheumatologic condition, mood disorders, medical treatment, comprehension impairments). Standard clinical procedures, imaging data, cerebro-spinal fluid biomarker analysis and neuropsychological assessment were used to make a diagnosis with respect to international consensus criteria (Armstrong et al., 2013; Crutch et al., 2017; Gorno-Tempini et al., 2011; McKhann et al., 2011). Based on these criteria, neurologists and neuropsychologists from two memory clinics (in two different cities) identified patients from their active file for participation in the study. If patients gave informed consent, they completed the experimental protocol with the first author. Six patients were finally not included because of diagnosis uncertainty. AD patients had mainly memory and orientation deficits; SD patients had verbal, semantic deficits; PCA patients had global cognitive impairment with mainly visual-spatial and memory deficits; and CBS patients had motor disorders contrasting with relatively normal cognitive functioning. The study was conducted in conformity with the Declaration of Helsinki and approved by local, regional ethical committee (Western Protection to Persons Committee II, n°2012/32).

2.2. SENSORY INTEGRATION

The participants completed the following tests of sensory integration: (1) localizing 20 tactile stimuli, eyes closed (i.e., light touch of the hand or forearm); (2) localizing 20 visual stimuli (i.e., movement of the examiner's index finger). In both tests, half of the stimulations were unilateral and half were bilateral; (3) detecting 20 proprioceptive stimuli (i.e., "up" or "down" passive movement of fingers); (4) Agraphesthesia (i.e., recognition of 10 numbers and 10 meaningless shapes drawn on the surface of the hand's skin); (5) Astereognosis (i.e., blind manipulation and identification of 10 meaningful and 10 meaningless objects; Supplementary Figure 1). In all of the tests, half of the unilateral stimulations were presented in the right hemi-

body or visual field. In tests 1, 2, and 3, patients could answer by moving the hand on the stimulated side, or by a verbal response. In the agraphesthesia and astereognosis tests, patients could name the shapes or objects, or they could point at them on photographs, among three distractors. This design allowed circumventing naming and visual-spatial difficulties. The maximum total sensory integration score was 120. Details are available in Supplementary Table 1.

2.3. APRAXIA

Three tests of apraxia were proposed. First, finger dexterity was assessed with the Purdue Pegboard test (Tiffin, 1968). Patients were instructed to pick and insert small metal rods into two hole lines as fast as possible within a 30-second time limit. There were three testing sessions (i.e., right hand, left hand, both hands simultaneously) each preceded by a short training session. The score was the total number of rods – or pairs of rods, in the bimanual session – inserted in holes. Second, we used an adapted version of a classical test of imitating meaningless hand and finger postures (Baumard et al., 2020, 2023). Performance was coded on a 2-point scale as a function of the precision of imitation. The maximum score was 90. Third, patients were instructed to select and use ten tools with their corresponding recipient object (e.g., screwdriver/screw; Baumard et al., 2016). Each correct use within 60 seconds (per item) was worth 1 point. The maximum score was 10. The three praxis scores were summed up to generate a global apraxia score for each individual.

2.4. STATISTICS

Nonparametric statistics were preferred due to low samples sizes. Between-group comparisons were performed using Kruskal tests and post-hoc pairwise Mann-Whitney U tests. Effect size was measured with Cliff's delta. P-values were adjusted with Holm's correction for multiple tests. The relation between sensory integration and apraxia was tested with Spearman rank order correlations based on the total patient sample, in order to increase statistical power

and avoid data inflation. A single-case methodology in neuropsychology (Crawford & Garthwaite, 2002) helped to determine individual impairments and between-task dissociations. Two types of dissociations were studied: classical dissociations (i.e., score A is pathological but score B is normal), and strong dissociations (i.e., both scores are pathological but score A is significantly lower than score B).

3. RESULTS

3.1. SENSORY INTEGRATION

Control-patient differences are reported in Table 1. Patients had a pathological score ($M = 87.9$, $sd = 17.0$; $U = 705.5$, $p < .001$, $d = 0.68$, large). In more details, FPA patients had large differences from the controls, SD patients had subnormal performance, and AD patients were intermediate. SD patients performed in normal range on all tests, with the exception of the agraphesthesia test. This was probably due to semantic rather than sensory integration deficits because the performance was higher with meaningless items ($M = 15.0$, $sd = 2.6$) than with meaningful items ($M = 8.9$, $sd = 5.0$; $U = 0.0$, $p = .009$, $d = -0.7$, large; see Supplementary Figure 2). Patients with AD had isolated agraphesthesia and astereognosis. The FPA group showed impairment in all tests of sensory integration.

3.2. APRAXIA

The praxis scores are displayed in Table 1. Patients had a lower apraxia score than healthy controls ($M = 103.1$, $sd = 26.2$; $U = 734.5$, $p < .001$, $d = 0.83$, large). Again, FPA patients had large differences from the controls, SD patients had subnormal performance, and AD patients were intermediate. Patients with SD had mainly apraxia of tool use, while other patient groups had difficulties in all three praxis tasks.

3.3. ASSOCIATIONS BETWEEN SENSORY INTEGRATION AND APRAXIA

There was a positive correlation between the global sensory integration and apraxia scores (Figure 1; see also Supplementary Figure 3). The correlation remained significant after controlling for the MMSE score (partial correlation, $r = 0.62$, $p < .001$), suggesting that dementia severity explained a limited portion of the variance. Correlations were found between Astereognosis and the Purdue Pegboard test ($r = 0.46$, $p = .047$), as well as between proprioception and imitation ($r = 0.48$, $p = .034$; Supplementary Table 2). The absence of correlation between sensory integration and tool use suggests that the latter is the best way to assess apraxic impairments that are not related to sensory integration deficits.

3.4. DISSOCIATIONS BETWEEN SENSORY INTEGRATION AND APRAXIA

Based on individual scores, the frequency of apraxia varied after controlling for the sensory integration score. The global apraxia score was impaired in 11 (61%) AD cases, 3 (30%) SD cases, and 11 (92%) FPA cases, in comparison with healthy controls. Nevertheless, using a strict definition of apraxia (i.e., impaired global apraxia score but normal global sensory integration score, which corresponds to the definition of a classical dissociation), apraxia frequency fell to 5 (28%) AD cases, 0 SD cases, and 3 (25%) FPA cases. Using a less strict definition of apraxia (i.e., the apraxia score is abnormal and significantly lower than the sensory integration score, whether the latter is impaired or not, which encompasses both classical and strong dissociations), apraxia was observed in 5 (28%) AD cases, 0 SD cases, and 10 (83%) FPA cases (Supplementary Table 3). So, deficits of sensory integration may have explained apraxia in 33% of AD cases (61 minus 28), 30% of SD cases (30 minus 0), but 9% of FPA cases (92 minus 83).

We also tested for dissociations between the global sensory integration score and each of the three apraxia scores in individual cases. A significant “Purdue Pegboard < Sensory integration” dissociation (either classical or strong) was found in 1 (6%) AD, 0 SD and 6 (50%) FPA cases. The same was found regarding the imitation test in 3 (17%) AD, 0 SD, and 7 (58%)

FPA cases, and with the tool use test in 5 (28%) AD, 2 (20%) SD, and 1 (8%) FPA cases. To sum up, after controlling for sensory integration, AD and SD patients had mainly apraxia of tool use, whereas FPA patients had mainly impaired finger dexterity and visuo-imitative apraxia. Of note, the reverse dissociation (i.e., apraxia score higher than sensory integration score) was found with the Purdue Pegboard test in 2 cases (PCA02, PCA03); with the imitation test in 3 cases (AD14, AD20, SD01); and with the tool use test in 5 cases (AD13, CBS07, PCA02, PCA05, PCA07).

4. DISCUSSION

The first goal of this study was to test sensory integration deficits in patients with neurodegenerative diseases. In our findings, patients with syndromes associated with frontal-parietal brain atrophy (posterior cortical atrophy, corticobasal syndrome) showed severe sensory integration deficits, while patients with polar-ventral temporal lobe atrophy (semantic dementia) showed normal sensory integration – when the meaning of the stimuli was controlled for. Patients with Alzheimer’s disease showed a more nuanced picture, that is, normal elementary sensory integration but impaired graphesthesia and stereognosis. So, the AD group could be qualified as having tactile agnosia, which is the inability to identify objects by touch in the context of spared sensory functions (Zillmer *et al.*, 2008). Future works could examine the diagnostic value of the abovementioned dissociations, and study the associations between sensory integration and cognitive scores, especially since many cognitive models include sensory modules in their architecture. A more fine-grained analysis of tactile agnosia subcategories like ahylognosia/barognosia (i.e., the inability to discriminate textures and weight) and amorphognosia (i.e., the inability to discriminate sizes and shapes) may also be conducted.

The second goal of this study was to test the relationships between sensory integration deficits and apraxia. Limb apraxia is defined by default in that it should be explained neither by elementary motor or sensory deficits, nor by additional cognitive impairments (Rothi et al., 1997). Cognitive models of apraxia include sensory inputs in their architecture. It follows that in presence of sensory integration deficits, the diagnosis of limb apraxia is highly uncertain (Bartolo & Stieglitz Ham, 2016). We found a positive correlation between both dimensions, in line with the strong connections between frontal and parietal areas (Luria, 1978). While sensory integration deficits are classical exclusion criteria of apraxia in stroke patients, they seem to be linked to apraxia in some patients with neurodegenerative diseases. In fact, when sensory integration was controlled for, the frequency of apraxia fell from 61 to 28% in AD, from 30 to 0% in SD, and from 92 to 83% in FPA cases. Recently, Baumard & Le Gall (2021) have proposed to make a distinction between symptomatic apraxia (i.e., gesture production disorders that can be explained by factors not specific to gestures) and idiopathic apraxia (i.e., gesture production disorders that occur in isolation). By controlling sensory integration in a systematic manner, we documented symptomatic apraxia in all groups, but idiopathic apraxia only in the AD and FPA groups, which is consistent with the lesions that typically cause apraxia (Goldenberg, 2017; Luria, 1978; Reynaud et al., 2016). In other words, in a non-negligible portion of patients with neurodegenerative diseases, the hypothesis of a disruption of sensory integration can be more parsimonious than the hypothesis of apraxia. Controlling for dementia severity with cognitive measures other than the MMSE score may also be relevant. Clinicians and researchers are advised to integrate sensory integration measures along with their evaluation of apraxia to control for exclusion criteria and hence better understand the origins of gesture impairments. Dementia severity probably explains a portion of the variance in tests of sensory integration and apraxia, so future studies should control for cognitive scores. Future studies are needed to investigate the relationship between specific sensory integration deficits

and specific apraxia impairments, “*to elucidate the true nature of limb apraxia*” (Bartolo and Stieglitz Ham, 2016, p.6).

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The authors report no disclosure and have no conflict of interest.

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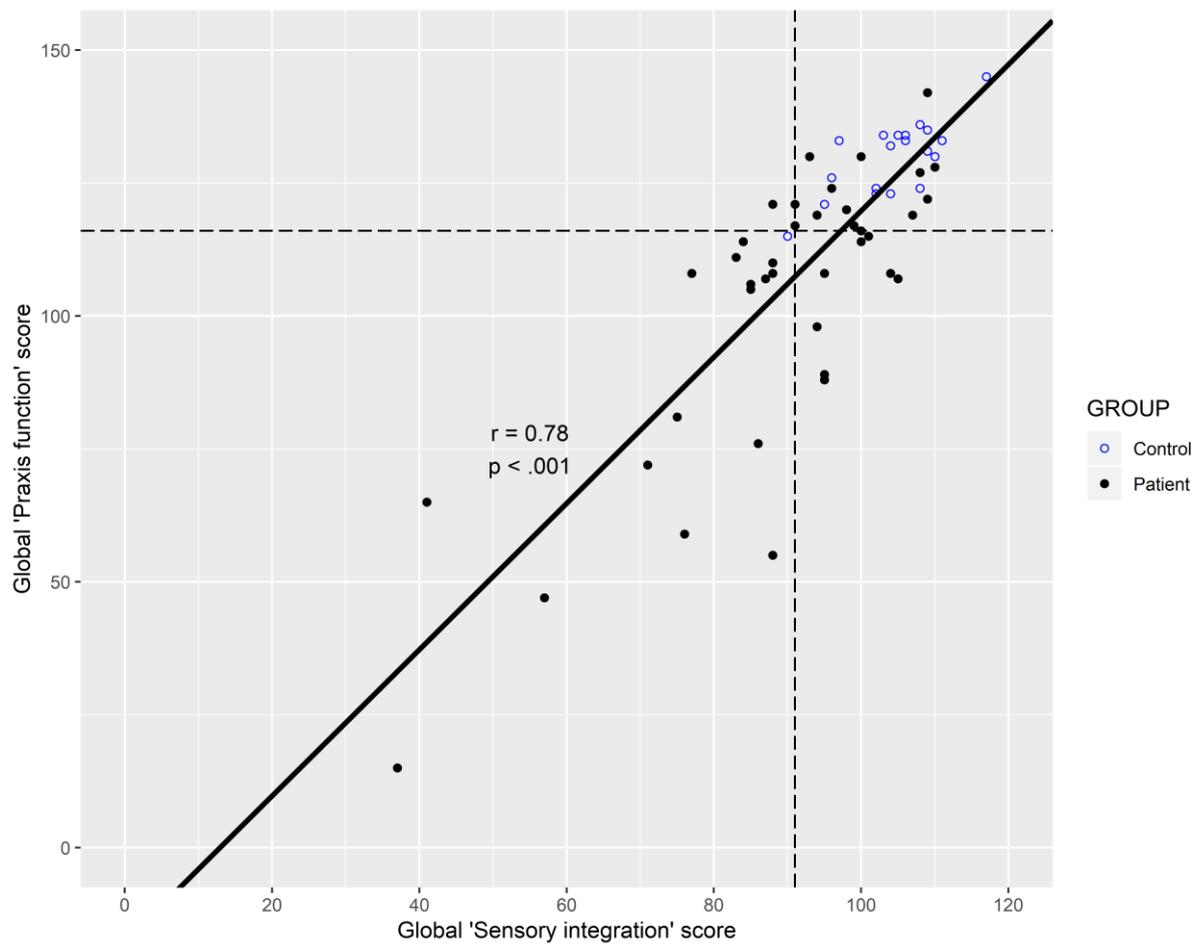
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TABLE 1. DEMOGRAPHIC AND CLINICAL DATA

	Max.	Healthy controls (HC)	Semantic dementia (SD)	Alzheimer's disease (AD)	FPA	PCA	CBS
N	-	20	10	20	14	7	7
Female / Male	-	13 / 7	5 / 5	14 / 6	8 / 6	5/2	3/4
Age	-	71.4 (6.4)	66.5 (8.1)	71.8 (6.3)	66.4 (5.9)	65.6 (5.0)	67.3 (7.1)
Education (years)	-	11.4 (4.4)	11.5 (2.4)	8.5 (4.0)	10.0 (4.8)	10.0 (6.1)	10.0 (3.5)
MMSE score	30	27.7 (1.8)	24.7 (2.7)	20.6 (2.0)	19.5 (8.3)	15.0 (7.1)	27.3 (1.5)
Sensory integration	120	104.1 (6.3)	96.7 (9.5)	90.7 (11.7)	77.3 (22.7)	64.7 (20.7)	88.1 (19.4)
Localization tactile	20	19.1 (1.2)	18.5 (1.5)	17.9 (1.9)	13.4 (4.4)	11.6 (4.0)	15.3 (4.2)
Proprioception	20	19.9 (0.4)	19.7 (0.7)	19.6 (0.7)	16.1 (4.0)	14.7 (4.2)	17.4 (3.5)
Localization visual	20	19.6 (0.6)	19.8 (0.4)	19.5 (0.8)	14.8 (4.5)	11.3 (3.3)	17.8 (2.9)
Agraphesthesia	40	30.8 (3.6)	23.9 (6.8)	23.2 (5.8)	20.9 (10.2)	14.5 (6.8)	26.4 (9.7)
Astereognosis	20	14.7 (2.4)	13.6 (3.1)	11.6 (3.3)	10.0 (3.9)	9.0 (3.6)	10.8 (4.3)
Apraxia		129.8 (6.7)	120.5 (12.0)	112.2 (9.6)	74.8 (29.9)	57.8 (22.9)	91.8 (27.3)
Purdue Pegboard	-	36.3 (3.7)	32.5 (5.8)	29.9 (4.1)^b	18.0 (8.7)^b	18.3 (9.4)	17.7 (8.8)
Imitation	90	83.9 (4.7)	80.8 (6.0)	74.1 (7.4)	48.9 (24.0)^b	32.7 (12.7)	65.2 (21.8)
Tool use	10	9.7 (0.7)	7.2 (3.2)	7.7 (1.7)	7.9 (2.7)^a	6.8 (3.5)	8.8 (1.2)

Notes. Data between brackets are standard deviations. Bold values are significant differences ($p < .05$) in comparison with the HC group (Kruskal tests and post-hoc Mann-Whitney U tests). Small effect sizes are not displayed to avoid exaggerating statistical effects. FPA: patients with frontal-parietal lobe atrophy, corresponding to cases with either posterior cortical atrophy (PCA) or corticobasal syndrome (CBS; see section 2.4. Statistics). Missing data due to scheduling issues or depressive symptoms: ^a $n = 1$, ^b $n = 2$.

FIGURE 1. DEMOGRAPHIC AND CLINICAL DATA



Notes. Correlation between sensory integration and praxis functions. *Notes.* The dashed lines correspond to cut-offs based on a classical single-case methodology in neuropsychology (Crawford & Garthwaite, 2002). Five cases were removed due to missing values. The correlation value without controls was: $r = 0.69, p < .001$