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1           **THE CHALLENGE OF APRAXIA: TOWARD AN**  
2           **OPERATIONAL DEFINITION?**

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5

## 6 **TOP STATEMENT**

7 We report how we determined our sample size, all data exclusions (if any), all  
8 inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data  
9 analysis, all manipulations, and all measures in the study. The relevant information can be  
10 found on page 6.

11 This manuscript is a review. The quantitative data are from previous studies. The  
12 supplement provides the full list of studies that have been used to aggregate the data. The data  
13 have been gathered years ago, before the creation of the TOP requirements.

14

## 15 **ABSTRACT**

16 The diagnosis of limb apraxia relies mainly on exclusion criteria (e.g., elementary motor or  
17 sensory deficits, aphasia). Due to the diversity of apraxia definitions and assessment methods,  
18 patients may or may not show apraxia depending on the chosen assessment method or theory,  
19 making the definition of apraxia somewhat arbitrary. As a result, “apraxia” may be diagnosed  
20 in patients with different cognitive impairments. Based on a quantitative and critical review of  
21 the literature, it is argued that this situation has its roots in the evolution from a task-based  
22 approach (i.e., the use of gold standard tests to detect apraxia) toward a process-based  
23 approach, namely, the deconstruction of the conceptual or production systems of action into  
24 multiple cognitive processes: language, executive functions, working memory, semantic  
25 memory, body schema, body image, visual-spatial skills, social cognition, visual-kinesthetic  
26 engrams, manipulation knowledge, technical reasoning, structural inference, and categorical  
27 apprehension. The coexistence of both approaches in the current literature is a major  
28 challenge that stands in the way of a scientific definition of apraxia. As a step toward a  
29 solution, we suggest to focus on symptoms, and on two complementary definition criteria (in  
30 addition with traditional exclusion criteria): Specificity (i.e., is apraxia explained by the  
31 alteration of cognitive processes specifically dedicated to gesture production?), and  
32 consistency (i.e., is the gesture production impairment consistent across tasks?). Two  
33 categories of limb apraxia are proposed: symptomatic apraxia (i.e., gesture production deficits  
34 that are secondary to more general cognitive impairments) and idiopathic apraxia (i.e., gesture  
35 production deficits that can be observed in isolation). It turns out that the only apraxia subtype  
36 that fulfills exclusion, specificity, and consistency criteria is limb-kinetic apraxia. A century  
37 after Liepmann’s demonstration of the autonomy of apraxia toward language, the autonomy

38 of this syndrome toward the rest of cognition remains an open question, while it poses new  
39 challenges to apraxia studies.

#### 40 **KEYWORDS**

41 *Keywords: apraxia, limb apraxia, motor control, epistemology, neuropsychology.*

42

43

## 1. INTRODUCTION

44 Apraxia is a disorder of intentional movement that cannot be accounted for by primary  
45 motor and sensory deficits, comprehension, attentional, or motivational deficits (Rothi et al.,  
46 1991, 1997). The first clinical description of apraxia is attributed to Jackson (1866) who has  
47 described an automatic-voluntary dissociation in patients who were able to eat and swallow  
48 but not to perform orofacial movements on command. Steinthal (1871) has later coined the  
49 term “apraxia”, defined as a disconnection between the movement and the purpose of the  
50 movement. It is a frequent and disabling condition in patients with left-hemispheric stroke or  
51 dementia (Foundas, 2013; Goldenberg, 1999; Lesourd et al., 2013), and it has already been  
52 described in patients with right-hemispheric stroke or traumatic brain injury (Buchmann et al.,  
53 2020; Goldenberg et al., 2009). It is important for clinicians to assess apraxia (Donovan et al.,  
54 2008) because apraxia scores predict functional outcome (Bickerton et al., 2012; Chestnut &  
55 Haaland, 2008; Foundas et al., 1995). The diagnosis, however, is a complex one because in  
56 the absence of consensus on an operational definition of this syndrome, it relies mainly on  
57 exclusion criteria. More than thirty subtypes of apraxia have been identified depending on the  
58 impaired activity or task used for the diagnosis (e.g., constructional apraxia, dressing apraxia,  
59 gait apraxia, mirror apraxia), the presence/absence of tools (e.g., absence in ideomotor  
60 apraxia, presence in ideational apraxia), the static/active nature of gestures (e.g., movement  
61 sequence in limb-kinetic apraxia, static postures in visuo-imitative apraxia), the input  
62 modality (e.g., tactile apraxia, visuo-imitative apraxia), the type of errors (e.g., spatiotemporal  
63 errors in ideomotor apraxia, content errors in ideational apraxia) and the affected limb (e.g.,  
64 oral apraxia, lid apraxia, trunk apraxia, leg apraxia; for a review, see Petreska et al., 2007).  
65 The current article focuses on limb apraxia, that is, deficits of intentional, purposeful  
66 movements carried out with the upper limbs. Limb apraxia is generally assessed by asking  
67 patients to perform meaningful (e.g., sign of the cross) or meaningless gestures (e.g., thumb

68 on the ear), transitive or intransitive gestures (i.e., tool-related or not tool-related gestures), on  
69 verbal command and on imitation. Beyond these gold standards, however, multiple  
70 assessment methods of limb apraxia have been designed over time (Butler, 2002; Dovern et  
71 al., 2012; Vanbellingen et al., 2010; Wheaton & Hallett, 2007). This diversity in definitions  
72 and assessment has led to widespread confusion because patients may or may not show  
73 apraxia depending on the chosen assessment method (Buchmann, 2020; Butler, 2002). As a  
74 result, the wording “apraxia” may be used to diagnose very different clinical conditions and,  
75 in our experience, it is somewhat obscure for many neuropsychologists and students.

76 This may also explain why research and clinical practice tend to rely on a task-based  
77 approach, that is, the use of gold standard tests (e.g., imitation and pantomime of tool use) to  
78 describe the impairments of patients with different pathological conditions (e.g., Buchmann et  
79 al., 2020) as well as between-task dissociations in individual patients (e.g., Rothi et al., 1991,  
80 1997). Nevertheless, the 21<sup>th</sup> century saw the dislocation of classical taxonomies of limb  
81 apraxia (i.e., limb-kinetic, ideomotor, ideational apraxias) and the creation of new ones (e.g.,  
82 Buxbaum, 2001; Petreska et al., 2007). The goal of this process-based approach (to be  
83 detailed further) is not so much to detect apraxia as to infer the cognitive and neural  
84 underpinnings of action (e.g., Binkofski & Buxbaum, 2013; Buxbaum, 2001; Osiurak, 2014).  
85 This approach has shown that different manifestations of apraxia can be explained either by  
86 praxis-specific cognitive impairments, or by non-specific cognitive impairments (e.g.,  
87 semantic memory, working memory). As a result, the potential causes of apraxia have  
88 increased over years, making “apraxia” an umbrella term with poor operational value. As we  
89 will argue, the coexistence of, and discrepancy between, both a task-based and a process-  
90 based approach in the current literature is a major challenge that stands in the way of a  
91 scientific definition of apraxia. As a step toward a solution, we will suggest to focus on  
92 symptoms, and on two complementary definition criteria: specificity (i.e., is apraxia explained

93 by the alteration of cognitive modules specifically dedicated to gesture production?), and  
94 consistency (i.e., is the gesture production impairment consistent across tasks?).

## 95 **2. AN ILLUSTRATION OF THE PROBLEM**

96 A first step to illustrate the issues that exist in the study of apraxia is to review the  
97 definitions and assessment methods used in the international literature. Although there is  
98 relative consensus on some definitions of apraxia (Rothi et al., 1991, 1997), a critical issue  
99 relates to their operationalization (i.e., which definitions are used by researchers, and do the  
100 definitions predict the tasks used to diagnose apraxia?). With the intention to determine  
101 whether there is consensus on definitions and tasks, we searched for English language  
102 experimental or clinical studies using the following keywords in Pubmed: “apraxia”, “limb  
103 apraxia”, “ideomotor apraxia”, “ideational apraxia”, “apraxia of tool use”, “tool use”, “object  
104 use”. No part of the study procedures or analyses was pre-registered prior to the research  
105 being conducted. We did not include studies focusing on apraxia of speech, constructional  
106 apraxia, orofacial apraxia, gait apraxia, or oculomotor disorders. Note that in this manuscript  
107 we shall now use apraxia and limb apraxia as synonyms. The sample population was not a  
108 criterion for we were interested in definitions and methods rather than results. We created a  
109 corpus of 100 studies covering a period of forty years (from 1974 to 2014, the year we  
110 compiled the data). **The limit of 100 studies was fixed arbitrarily, but the included studies**  
111 **were unselected.** Most studies have been published after Roy & Square’s (1985) and Rothi et  
112 al.’s (1991) influential models of apraxia (1970’s  $n = 1$ ; 1980’s  $n = 2$ ; 1990’s  $n = 21$ ; 2000’s  $n$   
113  $= 55$ ; 2010’s  $n = 21$ ). The full list of publications is available in Supplementary Table 1. We  
114 analyzed this corpus using a three-step method.

115 First, in these 100 studies, we found 127 definitions split into ten categories: a broad  
116 “apraxia” category (33% of the 127 definitions), “ideomotor apraxia” or “ideomotor limb



117 apraxia” (24%), “limb apraxia” (14%), “ideational apraxia” (15%), “conceptual apraxia”  
118 (1%), “manipulatory apraxia” (1%), “limb-kinetic apraxia” (1%), “frontal apraxia” (1%),  
119 “apraxia of tool use” (1%), and no definition of apraxia (9%). Note that this repartition  
120 necessarily reflects the keywords we used. Two studies (Blijlevens et al., 2009; Graham et al.,  
121 1999) have used the term “dyspraxia” (generally used in the developmental literature) to  
122 describe acquired gesture disorders following either stroke or corticobasal degeneration in  
123 adults, which actually corresponds to apraxia. Given the low number of studies, apraxia and  
124 dyspraxia were accepted as synonyms to avoid inflating the number of categories. One study  
125 referred to higher-order impairment of movement but not to apraxia and was therefore  
126 included in the “no definition” category. The further steps of the analysis focused on the most  
127 frequent categories only.

128         Second, we counted the number of definitions that made explicit reference to exclusion  
129 criteria (i.e., the sensory and cognitive domains that should not explain the apraxic symptoms)  
130 and/or inclusion criteria (i.e., the positive, presumably specific signs of apraxia). As regards  
131 exclusion criteria, the definitions emphasized motor disorders (35% of the 127 definitions);  
132 sensory disorders (24%); aphasia (20%); incoordination/ataxia (13%); inattention (9%);  
133 global cognitive impairment (8%); uncooperativeness (7%); perceptual disorders (6%);  
134 amnesia (2%). So, the independence of apraxia toward other sensory, motor or cognitive  
135 domains was rarely made explicit, despite the fact that exclusion criteria are critical to the  
136 diagnosis (Rothi et al., 1991, 1997) – although it should be mentioned that the sensory and  
137 motor dimensions are generally controlled for by assessing the ipsilateral hand in patients  
138 with unilateral stroke. Inclusion criteria referred to the clinical signs or domains used to  
139 define what apraxia is, which encompassed both error types and cognitive processes. There  
140 were seven domains: tool use (i.e., a disorder of tool use or naturalistic action; 49% of  
141 definitions); precision (i.e., a disorder of skilled movement; 28%); prior experience (e.g., a

142 disorder of learned/familiar movements or actions; 18%); intentionality (e.g., apraxia defined  
143 as disorder of purposeful, voluntary movement or action; 18%); spatial-temporal movement  
144 errors (13%); planning (e.g., a disorder of movement or action planning; 9%); content errors  
145 (i.e., unexpected or unrecognizable gesture, no gesture; 2%; for details on error types, see  
146 Hoeren et al., 2014; Kangas & Tate, 2006; Mozaz, 1992). There is substantial conceptual  
147 overlap between these categories (e.g., the operational definition of disorders of skilled  
148 movement generally corresponds to spatial-temporal errors), yet they were sometimes used  
149 independently from each other. [Table 1 provides some examples of definitions](#). Ideomotor  
150 apraxia was more frequently defined as a disorder of skilled gestures than ideational apraxia  
151 (35% of definitions of ideomotor apraxia against 5% for ideational apraxia;  $\chi^2 = 26.3$ ,  $df = 1$ ,  
152  $p < .001$ ), and was characterized by spatial-temporal errors (42% for ideomotor apraxia,  
153 against 0% for ideational apraxia;  $\chi^2 = 50.7$ ,  $df = 1$ ,  $p < .001$ ), while ideational apraxia was  
154 more frequently associated with planning impairments than ideomotor apraxia (37% of  
155 definitions of ideational apraxia against 6% for ideomotor apraxia;  $\chi^2 = 26.7$ ,  $df = 1$ ,  $p < .001$ ).  
156 Ideational apraxia was also more frequently defined as an impairment of tool use than  
157 ideomotor apraxia (84% and 42%, respectively;  $\chi^2 = 36.0$ ,  $df = 1$ ,  $p < .001$ ). This criterion,  
158 however, was the most frequent one for all of the definitions (84% of definitions of ideational  
159 apraxia; 50% for limb apraxia; 42% for ideomotor apraxia; 43% for apraxia) – an unexpected  
160 finding considering that tool use impairments have been classically used to distinguish  
161 between ideational and ideomotor apraxia (Wheaton & Hallett, 2007). Although this  
162 repartition broadly corresponds to the current state of the literature, consensus on inclusion  
163 criteria (i.e., 100% values or approaching) was rare. Furthermore, planning impairments  
164 account for some but not all of tool use impairments (Goldenberg et al., 2007; Goldenberg &  
165 Hagmann, 1998; Hartmann et al., 2005), while the definitions do not reflect the well-known  
166 influence of semantic memory deficits on tool use (Baumard et al., 2016; Bozeat et al., 2000,

167 2002; Hodges et al., 2000). Likewise, error-based approaches are not fully satisfying, on the  
168 one hand, because apraxic patients also commit non-clumsy errors (Kangas & Tate, 2006),  
169 and on the other hand, because spatial-temporal errors have only weak localization power  
170 (Hoeren et al., 2014).

171 Third, we analyzed the 319 tests used in these 100 studies to either diagnose or  
172 investigate apraxia, by grouping them into eight categories on which there is relative  
173 consensus: Pantomime of tool use (i.e., performing tool-related actions without holding the  
174 tool in hand); single tool use (i.e., performing tool-related actions while holding the tool in  
175 hand); real tool use (i.e., using a tool with the usual, corresponding object; for a review, see  
176 Baumard et al., 2014); novel tool use (i.e., using novel tools and objects; e.g., Buchmann &  
177 Randerath, 2017; Goldenberg & Hagmann, 1998); production of meaningful intransitive  
178 gestures (i.e., performing communicative gestures other than pantomime of tool use, single  
179 tool use, or real tool use; for a review, see Bartolo & Cubelli, 2014); imitation of meaningless  
180 gestures (i.e., imitating hand and finger postures without semantic reference; e.g., Goldenberg  
181 & Hagmann, 1997); imitation of meaningful gestures (i.e., imitating pantomime of tool use,  
182 single tool use, real tool use, or meaningful intransitive gestures; e.g., Mengotti et al., 2013);  
183 non-production tasks (i.e., tests in which gesture production is not required such as  
184 picture/gesture naming, recognition, or matching; e.g., see Lesourd et al., 2013). The  
185 frequency of use of these tests was as follows: Pantomime of tool use (24% of the 319 tests),  
186 imitation of either meaningful (16%) or meaningless gestures (13%), single tool use (12%),  
187 real tool use (12%), non-production tests (12%), production of meaningful intransitive  
188 gestures (8%), and novel tool use (3%). In order to determine whether the use of specific  
189 definitions and inclusion criteria predicts the selection of specific tests, Table 2 shows the  
190 tasks used as a function of either the definition or its content (i.e., inclusion criteria). [For](#)  
191 [example, we found 31 definitions of “ideomotor apraxia”. In these studies, we found 22](#)

192 pantomime of tool use tests, 10 single tool use tests, 11 real tool use tests, and so on.  
193 Likewise, we found 23 definitions that explicitly referred to prior experience. In these studies,  
194 we found 17 pantomime of tool use tests, and 3 novel tool use tests. For this table only, we  
195 had to duplicate some tests because one study could include several definitions and tests,  
196 resulting in multiple definition x criterion x task combinations (e.g., if pantomime of tool use  
197 was used in a study defining apraxia as an impairment of both motor planning and skilled  
198 movement, this test was counted once for “planning” and once for “skilled movement”,  
199 except if it was made clear that the test assessed one dimension only). Table 2 is, therefore,  
200 illustrative at most, yet it allows two main conclusions.

201 First, the definitions used – and their content – are not clearly associated with specific  
202 tests: the distribution of tests does not seem to vary as a function of definitions and contents,  
203 while there seems to be a main effect of tasks. As an example, 15 to 33% of tests (depending  
204 on the definitions and contents considered) were pantomime of tool use tests (PTU), and the  
205 absence of definition in studies was not associated with the use of different tests (28% of PTU  
206 tests in that case). This suggests that the definitions have only poor operational value. For  
207 instance, they emphasize mainly tool use impairments, while real tool use tests are rarely used  
208 (i.e., only 7 to 18% of tests were real tool use tests). We also found 0 to 18% of non-  
209 production tests. The assessment of apraxia (i.e., a deficit of gesture production) with non-  
210 production tests is logical in the light of the classical dichotomy between a conceptual system  
211 (i.e., the processes involved in the preparation of gestures) and a production system (i.e., the  
212 dynamic adaptation of gestures to external constraints; Bartolo et al., 2007; Binkofski &  
213 Buxbaum, 2013; Buxbaum, 2001; Cubelli et al., 2000; Rothi et al., 1991; Roy & Square,  
214 1985), yet it adds additional noise. Since gesture recognition deficits are not always  
215 associated with production deficits (Baumard et al., 2019; Moreaud et al., 1998), diagnosing  
216 apraxia (a gesture production deficit) based on the performance in a non-production test may

217 be problematic. Second, the “gold standard” status of pantomime of tool use and imitation  
 218 tests is confirmed for these are the most frequent tasks used – even in the absence of  
 219 definition. Nevertheless, these are not monolithic tests for they assess multiple cognitive  
 220 dimensions, **thus they are useful to detect apraxia, but not to infer the underlying impairments.**  
 221 As we shall emphasize below, this raises a critical issue, namely, the discrepancy between a  
 222 task-based approach (i.e., the use of gold standard tests to diagnose apraxia) and a process-  
 223 based approach (i.e., what we know of the cognitive underpinnings of these tests).

224

225 **Table 1. Examples of definitions of apraxia**

Study	Wording	Definition
Heilman et al. (1974)	“Apraxia”	A defect in purposeful movement which is not caused by weakness or other elemental motor disturbances and not caused by incomprehension of commands (Geschwind, 1975).
Selnes et al. (1991)	“Apraxia”	An acquired disorder of skilled movements not secondary to weakness or poor comprehension.
Belanger et al. (1996)	“Limb apraxia”	An abnormality in the execution of acquired movements not attributable to primary motor (paralysis, paresis, incoordination) or sensory deficits.
Endo et al. (1996)	(Modality-specific) “apraxia”	An acquired inability to demonstrate the use of objects presented to a particular sensory modality, despite the preserved ability to perform it when they are presented through other modalities.
Cubelli et al. (2000)	“Limb apraxia”	Disorders of purposive movements resulting from neurological dysfunction which cannot be explained by elementary or sensory defects, by task comprehension problems, or by object recognition deficits.
Maeshima et al. (2000)	“Ideational apraxia”	A disturbance in performance of complex actions involving the serial ordering of simple movements, which, in isolation, could be executed correctly (Poeck & Lehmkuhl, 1980).
Rumiati et al. (2004)	“Ideational apraxia”	A deficit of performing object-related skilled actions.
Wheaton et al. (2008)	“Ideomotor apraxia”	Performance of pantomiming tool use and communicative gestures with spatial and temporal errors.
Frey et al. (2005)	“Ideomotor apraxia”	Difficulties performing one or more of the following acts even when using the ipsilesional hand: pantomiming tool and/or non-tool-use actions, gesturing to verbal command, imitating movements, and in some instances, actually using tools. Not attributed to elementary motor or sensory deficits.
Imazu et al. (2007)	“Ideomotor apraxia”	The inability to pantomime the use of tools, despite the ability to manipulate the actual tools in a normal manner.
Dawson et al. (2010)	“Ideomotor apraxia”	A disorder of complex object-related action observed in both the contralesional as well as ipsilesional hand, associated with deficient anticipatory planning of hand posture and hand orientation.

226 *Notes.* This selection of definitions illustrates the heterogeneity of terms, phenomena, and concepts, used to define apraxia.

227

228 **Table 2. Apraxia tests used as a function of the definition of apraxia in 100 studies**

Definition	N	PTU	STU	RTU	NTU	PMI	lml	lmf	NP	Total
Apraxia	42	35 (26)	18 (13)	19 (14)	6 (4)	10 (7)	16 (12)	20 (15)	13 (9)	137 (100)
Ideomotor apraxia	31	22 (22)	10 (10)	11 (11)	2 (2)	9 (9)	18 (18)	17 (17)	13 (13)	102 (100)
Ideational apraxia	19	11 (15)	11 (15)	12 (16)	4 (5)	3 (4)	8 (11)	13 (17)	13 (17)	75 (100)
Limb apraxia	18	14 (23)	8 (13)	5 (8)	1 (2)	6 (10)	7 (12)	10 (17)	9 (15)	60 (100)
No definition	12	8 (28)	2 (7)	2 (7)	0 (-)	4 (14)	5 (17)	5 (17)	3 (10)	29 (100)
Content	N	PTU	STU	RTU	NTU	PSG	lml	lmf	NP	Total
Intentionality	23	15 (21)	9 (12)	7 (10)	1 (1)	10 (14)	5 (7)	17 (23)	9 (12)	73 (100)
Planning	12	9 (23)	4 (10)	7 (18)	2 (5)	4 (10)	3 (8)	4 (10)	7 (18)	40 (100)
Prior experience	23	17 (23)	10 (14)	12 (16)	3 (4)	7 (9)	8 (11)	12 (16)	5 (7)	74 (100)
Skilled movement	35	30 (27)	12 (11)	14 (13)	3 (3)	11 (10)	8 (7)	18 (16)	15 (14)	111 (100)
Spatial-temporal errors	17	12 (24)	6 (12)	6 (12)	2 (4)	8 (16)	4 (8)	7 (14)	5 (10)	50 (100)
Content errors	3	3 (33)	1 (11)	1 (11)	1 (11)	1 (11)	1 (11)	1 (11)	0 (-)	9 (100)
Real tool use	62	50 (23)	29 (13)	31 (14)	9 (4)	24 (11)	10 (5)	28 (13)	34 (16)	215 (100)

229 Notes. Contents refer to inclusion criteria (i.e., positive/specific signs of apraxia). How to read the table:  
230 We found 42 definitions of a broad “apraxia” category. In these papers, apraxia was assessed with 137  
231 different tests among which pantomime of tool use ( $n = 35$ , i.e. 26% of 137 tests). [The table shows that](#)  
232 [definitions, and their content, do not clearly predict the tests used in the corresponding studies.](#)  
233 [Pantomime of tool use is generally preferred to other tests, even in the absence of definition.](#) PTU:  
234 Pantomime of tool use; STU: Single tool use; RTU: Real tool use; NTU: Novel tool use; PMI: Production of  
235 meaningful intransitive gestures; lml: Imitation of meaningless gestures; lmf: Imitation of meaningful  
236 gestures; NP: Non-production tasks.

### 237 3. FROM TASK-BASED TO PROCESS-BASED STUDIES

238 In Liepmann’s taxonomy of apraxia (see Goldenberg, 2003), ideational apraxia  
239 corresponded to the disintegration of concepts resulting in defective actions according to their  
240 purpose, as illustrated by the faulty use of single tools. This disorder “manifests in the domain  
241 of action but has its roots in deficits which are not specific to action” (Liepmann, 1929, cited  
242 by Goldenberg, 2003, p.518). Ideo-kinetic apraxia was the separation between the idea of the  
243 movement and its execution, as demonstrated by imitation tests: since the model is given,  
244 errors are caused by the disconnection between the motor cortex and the whole cerebral  
245 cortex. Limb-kinetic apraxia resulted from lesions of the central region and affected any  
246 movement, intentional or routine. These three labels have become canonical, while there has  
247 been substantial evolution in the definition of limb-kinetic apraxia (i.e., deficits of fine,  
248 essentially finger movement skills following precentral lesions; Gross & Grossman, 2008;  
249 Luria, 1978), ideomotor apraxia (i.e., a broad gesture production deficit category  
250 encompassing mainly pantomime of tool use, symbolic gesture production and imitation  
251 deficits; Butler, 2002; Wheaton & Hallett, 2007), and ideational apraxia (i.e., the inability to

252 properly use tools and objects, either with tool/object pairs or in multiple-step, naturalistic  
253 action tests; e.g., Goldenberg & Hagmann, 1998; Hartmann et al., 2005).

254         These categories had significant relevance in Liepmann’s view of motor control but  
255 they no longer correspond to modern views on apraxia. Cognitive models have sustained the  
256 conceptual-production dichotomy but have fractionated the conceptual system (see further). In  
257 doing so, the “ideomotor” category, for example, has partially lost its meaning for it can now  
258 be diagnosed with a variety of tasks (see section 2). Furthermore, imitation may call for  
259 different cognitive mechanisms (Mengotti et al., 2013). Pantomime of tool use is an  
260 ambiguous test (i.e., is it a test of ideomotor or ideational apraxia? Bartolo et al., 2003) that is  
261 not superimposable with real tool use (for a review, see Baumard et al., 2014), given that both  
262 tests tap partly different cognitive mechanisms (see also Goldenberg, 2003, 2003b) and brain  
263 regions (Goldenberg, 2017). The operational definition of ideational apraxia has changed over  
264 time, being first defined as a disorder of gestures performed in sequence (Poeck, 1983), then  
265 as a disorder of the actual use of tools and objects due to semantic memory deficits (Barbieri  
266 & De Renzi, 1988; De Renzi & Lucchelli, 1988). The boundaries between these categories are  
267 sometimes blurred. For instance, patients diagnosed with ideomotor apraxia are slower to  
268 open doors (Chestnut & Haaland, 2008) and in tool use situations calling for precision (e.g.,  
269 bean spooning task; Sunderland & Shinner, 2007). So, the diagnosis does not depend on the  
270 impaired task, but rather on the selected observation criterion, which makes it arbitrary in  
271 essence. To sum up, there is now a gap between the taxonomy, operationalization, and theory  
272 of apraxia, meaning that the same apraxia tests may be used with two different approaches.

273         Although the abovementioned tests have been used in theory-driven studies which goal  
274 was to infer their cognitive underpinnings, their use has also frequently been atheoretical  
275 (e.g., to determine the apraxic/non apraxic status of patients). The high frequency of use of  
276 these tests even in the absence of definition (section 2) is in line with this assumption. The use

277 of gold standard tests to diagnose apraxia, but not necessarily to infer the underlying cognitive  
278 impairments, corresponds to what we hereafter call a task-based approach. This approach is  
279 useful because consensus can be achieved more easily regarding tests than regarding  
280 concepts, yet it raises several issues. The first counterpart of it is a paradox: if several tests  
281 assess exactly the same impairment (i.e., apraxia), then why are several tests necessary?  
282 Furthermore, this approach is mainly descriptive (i.e., patients pass or fail the test) and might  
283 become almost circular in some instances (e.g., studying the praxis skills of patients that have  
284 already been diagnosed with apraxia). Although using gold standards is an effective and  
285 pragmatic way to diagnose apraxia, it necessarily reifies some testing conditions over others,  
286 and makes the clinical definition of apraxia partly arbitrary. It is an issue both for a scientific  
287 approach of apraxia and for clinical practice, because scientific concepts, and their  
288 corresponding operationalization, should not be subject to arbitrary choices. More  
289 importantly, research on apraxia has led to the scientific deconstruction of apraxic symptoms  
290 (i.e., the process-based approach), meaning that the task-based approach, although still vivid,  
291 is scientifically dated.

292 Over the time, a diversity of cognitive impairments have been invoked to explain  
293 apraxic symptoms (i.e., the process-based approach). Clinical dissociations have led to the  
294 creation of new taxonomies and models of apraxia. These works have all endeavored to  
295 precise the nature of the conceptual system, i.e. the cognitive processes involved in the  
296 production of gestures (Figure 1). Impaired pantomime of tool use has been explained by  
297 deficits of working memory (Bartolo et al., 2003; Cubelli et al., 2000), language, semantic  
298 memory (Goldenberg et al., 2003; Rothi et al., 1991; Roy & Square, 1985; Stamenova et al.,  
299 2012), visual-kinesthetic engrams or manipulation knowledge (i.e., implicit, sensorimotor  
300 knowledge containing invariant features of transitive gestures; Buxbaum, 2001; Rothi et al.,  
301 1991), or body schema (Buxbaum, 2001). Performance in single or real tool use has been

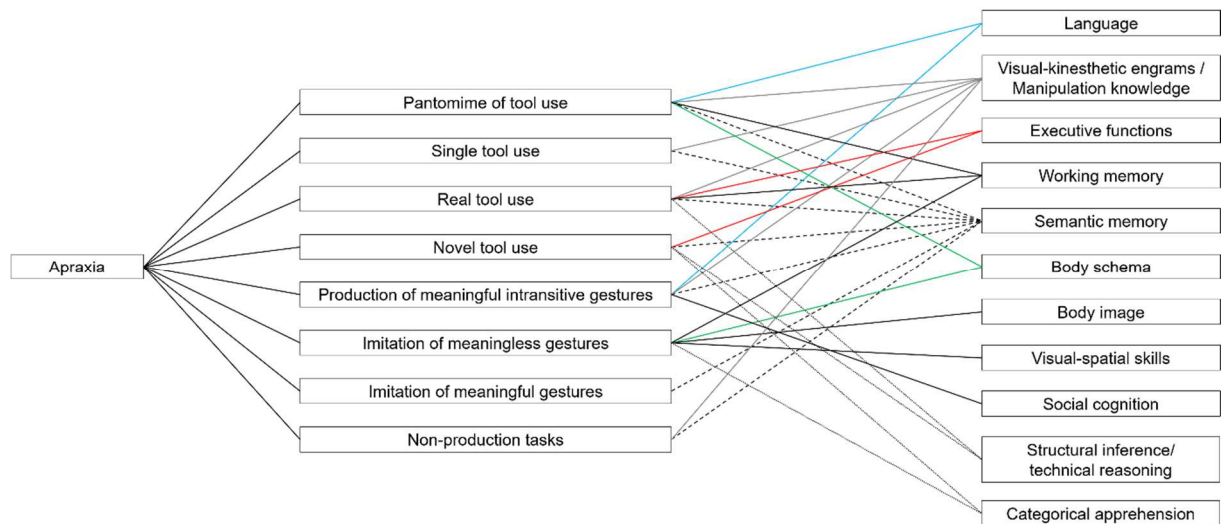


302 explained by deficits of semantic memory (e.g., De Renzi & Lucchelli, 1988), manipulation  
303 knowledge, executive functions and working memory (Giovannetti et al., 2002; Goldenberg et  
304 al., 2007; Hartmann et al., 2005; Jarry et al., 2013; Schwartz et al., 1998), structural inference  
305 or technical reasoning (i.e., broadly, the ability to infer the functions of tools and objects from  
306 the analysis of their sensory characteristics; Goldenberg & Hagmann, 1998; Jarry et al., 2013;  
307 Osiurak, 2014; Osiurak et al., 2010), or categorical apprehension (i.e., a general function of  
308 the left parietal cortex that is to differentiate and combine parts of multi-part objects or body  
309 parts; Goldenberg & Spatt, 2009; Goldenberg, 2009). Novel tool use has been proposed to  
310 depend on the two latter processes, as well as on mechanical knowledge, sometimes  
311 understood as a subcategory of semantic memory (Ochipa et al., 1992). The production of  
312 meaningful intransitive gestures may rely on semantic memory (Mengotti et al., 2013), visual-  
313 kinesthetic engrams (Rothi et al., 1991, 1997), or social cognition (Bartolo & Stieglitz Ham,  
314 2016). Imitation impairments have been proposed to reflect deficits of body image  
315 (Goldenberg, 1995), body schema (Buxbaum, 2001), visual-spatial skills (Goldenberg et al.,  
316 2009), semantic memory (Mengotti et al., 2013), or direct visual-motor conversion  
317 mechanisms (Achilles et al., 2017; Rothi et al., 1991, 1997), depending on the meaningfulness  
318 of the gesture, the body parts involved (e.g., hand or finger configurations), the  
319 presence/absence of visual control (Okita, 2017), and the procedure (e.g., mixed versus  
320 separate lists; Tessari et al., 2006). It is, therefore, no longer possible to consider that imitation  
321 tests assess a disconnection between ideation and production, which could lead one to use it  
322 as an “ideational” test. Finally, non-production tests have been thought to assess manipulation  
323 knowledge (Buxbaum, 2001) or semantic memory (e.g., Baumard et al., 2016; Jarry et al.,  
324 2013). To sum up, one cognitive process may underlie several tasks, and different cognitive  
325 processes may underlie one and the same task.

326 Overall, the task-based and process-based approaches are not compatible. For instance,  
327 using some tests as gold standards to diagnose “apraxia” (i.e., task-based approach) is at high  
328 risk of bias because each actually taps different underlying cognitive impairments (i.e.,  
329 process-based approach). The consequence is that two studies may diagnose “apraxia” in  
330 patients with very different profiles, making it impossible to compare these works on a one-  
331 to-one basis and hence to draw general conclusions on what apraxia is or is not (see also  
332 Butler, 2002; Wheaton & Hallett, 2007). For clinical neuropsychologists, does it make sense  
333 to diagnose “apraxia” in patients with actually very different cognitive impairments ( Bartolo  
334 & Stieglitz Ham, 2016). The counterpart of the process-based approach, however, is that  
335 apraxia has been associated with so many cognitive impairments that it is hard to obtain a  
336 consensus on an operational definition (i.e., inclusion criteria). This is why exclusion criteria  
337 remain critical for the diagnosis. Finally, the process-based approach cannot be fully  
338 independent from the task-based approach, because studying the cognitive processes behind  
339 apraxia mandatorily implies the a priori selection of tasks and observation criteria to define  
340 the apraxic status of patients. Both approaches are, therefore, better viewed as the two faces of  
341 the same, ill-posed problem.

342

343 **Figure 1. The task-based and process-based approaches**



344

345 *Notes.* The task-based approach (on the left) consists in using tests to detect “apraxia” (or subtypes). The  
 346 process-based approach (on the right) consists in inferring the cognitive underpinnings of praxis tests. They are  
 347 not compatible because the same wording “apraxia” could apply to patients with different cognitive impairments.  
 348 Colors and dotted lines for the sake of clarity only.

## 349 **4. TOWARD AN OPERATIONAL DEFINITION OF APRAXIA?**

350 The further sections discuss additional issues that stand in the way of an operational  
 351 definition of apraxia. As a step toward a solution, we will suggest to focus on symptoms and  
 352 to make a distinction between specific and non-specific causes of apraxia on the one hand,  
 353 and between consistent and task-based impairments on the other hand.

### 354 **4.1. DIFFERENT LEVELS OF EXPLANATION**

355 Previous works have resulted in the creation of new apraxia subtypes, in an attempt to  
 356 clarify symptoms of apraxia (e.g., conceptual apraxia; Rothi et al., 1991; Ochipa et al., 1992;  
 357 ideomotor dynamic or ventral apraxia; Buxbaum, 2001). One may therefore consider that  
 358 using a general “apraxia” or “limb apraxia” wording is not an issue because these subtypes  
 359 help to specify the nature of impairments, just as aphasia, agnosia, or amnesia are general  
 360 syndromes encompassing different clinical phenotypes. This approach, however, does not

361 apply well to apraxia. Defining new subtypes of ideomotor or ideational apraxia does not  
362 solve the issues mentioned in previous sections. There have been more than thirty apraxia  
363 subtypes described (Petreska et al., 2007), while there is some consensus on a limited number  
364 of aphasia, agnosia or amnesia syndromes (e.g., Baugh et al., 2017; Eustache et al., 2016;  
365 Romero & Moscovitch, 2015; Sarno, 2017). Since there is no a priori reason for a higher  
366 complexity of apraxia over other neuropsychological syndromes, the multiplication of apraxia  
367 subtypes likely reflects epistemological issues.

368         Apraxia taxonomies have been created with reference to three different explanatory  
369 frameworks: a phenomenological level (i.e., the definition is based on clinical symptoms), a  
370 theoretical level (i.e., the definition is based on the underlying cognitive impairment or  
371 psychological construct), and an anatomical level (i.e., the definition is based on the  
372 underlying brain lesions). For example, limb-kinetic apraxia involves deficits in fine motor  
373 movements (Wheaton & Hallett, 2007); conceptual apraxia is the inappropriate use of tools  
374 and objects caused by loss of knowledge on tools and actions (Ochipa et al., 1992; Rothi et  
375 al., 1991, 1997); and ventral apraxia is the inability to perform tool-related gestures following  
376 temporal lobe lesions (Buxbaum, 2001). The co-existence of these different levels of  
377 explanation, sometimes in the same taxonomies (e.g., dynamic and ventral apraxias;  
378 Buxbaum, 2001), is an issue for the neuropsychological approach of apraxia, the aim of which  
379 is to study the correspondence between these three levels.

380         In its strict form, the cognitive, representational approach tries to achieve full alignment  
381 of the three levels. This is, for example, manifest in the manipulation-based theory of tool use  
382 (Binkofski & Buxbaum, 2013; Buxbaum, 2001; Rothi et al., 1997): Pantomime of tool use  
383 impairment (phenomenological level) is caused by loss of manipulation knowledge about  
384 tool-related gestures (theoretical level) stored in the left parietal lobe (anatomical level).  
385 While this is a serious evidence-based approach, it has also drawn criticism. The heuristic

386 value of these models has been a matter of debate because some of the clinical profiles they  
387 have predicted have not been consistently observed, while unpredicted profiles have been  
388 documented (Cubelli et al., 2000; Stamenova et al., 2012). More importantly, the virtually  
389 infinite expansion of cognitive architectures may result in unfalsifiable theories, in that adding  
390 new modules to explain new profiles cannot lead to refute the existing modules (De  
391 Vignemont, 2007). Furthermore, recent works using new statistical methods have started to  
392 call into question the existence of some cognitive modules (e.g., body image for imitation;  
393 Achilles et al., 2016, 2017). In contrast with the cognitive stance, a rather “structuralist”  
394 stance (De Saussure, 1915; Lévi-Strauss, 1958; Sabouraud, 1995) has posited that the number  
395 of psychological processes in humans is more limited than the number of phenomena,  
396 meaning that there is no systematic symptom-cognitive process matching, and hence, that  
397 some phenomena are an emanation of several processes (e.g., writing requires language,  
398 motor, and praxis functions, while what is written is context-dependent and does not exist at  
399 the theoretical or anatomical level). This assumption is in line with most of the process-based  
400 approach (e.g., pantomime of tool use results from the interaction of multiple cognitive  
401 processes that are not necessarily praxis-specific; see section 3).

402       Beyond the issue of correspondence between levels of explanation, some of these levels  
403 have intrinsic epistemological limitations. The anatomical level, by definition, does not  
404 explain the psychological underpinnings of the symptoms. Establishing a link between faulty  
405 gestures and brain lesions is of high scientific and clinical interest, yet it does not by itself  
406 explain the psychological impairments at the root of the symptoms, just as establishing a link  
407 between Broca’s area and aphasia does not reveal what language is. As a matter of fact,  
408 clumsy spatial-temporal errors have only poor diagnostic and localization power (Hoeren et  
409 al., 2014; Kangas & Tate, 2006), suggesting that the phenomenological level and the  
410 anatomical level are not superimposable. As regards the theoretical level, defining apraxia (or

411 subtypes) as an impairment of particular cognitive mechanisms is logical, especially since  
412 Liepmann has made a distinction between ideation and production (see Goldenberg, 2003). It  
413 may also, however, lead to a circular reasoning, namely, to define the symptom (e.g., the  
414 patient selects the wrong tool) directly by its causal explanation (e.g., semantic memory  
415 deficit). Taken to its logical extreme, this reasoning would hold, for example, that all  
416 manifestations of apraxia are a priori caused by loss of knowledge. For example, “conceptual  
417 apraxia” has initially been referred to as familiar tool use impairments caused by loss of  
418 semantic memory, has been rapidly extended to novel tool use impairments (Ochipa et al.,  
419 1992), and has been sustained in more recent works (Wheaton & Hallett, 2007) in spite of  
420 clinical dissociations between novel tool use and tests of semantic memory (Bozeat et al.,  
421 2002; Bozeat et al., 2000). To conclude, both the theoretical and anatomical levels of  
422 explanation actually correspond to a “transcendentalist stance” of ontology (i.e., the study of  
423 mind-world relationships; Lundh, 2018): not only do they explain but they also define apraxic  
424 symptoms (the phenomena) by dimensions that are external to it (as an analogy, the human  
425 “soul” has been the transcendent of personality until the advent of scientific psychology;  
426 Janet, 1929). It is a critical issue because by definition, a transcendentalist approach will  
427 always define apraxia by non-apraxic impairments, and hence it will always fail to  
428 circumscribe apraxia (an issue further addressed in the next sections). Perhaps a first step  
429 toward an operational definition of apraxia may be to prioritize an “immanent” stance, that is,  
430 to disconnect the definition of symptoms from their interpretation and to emphasize a self-  
431 explanatory, symptom-based definition of apraxia – a condition to the secondary generation of  
432 theoretical interpretations. Otherwise, this would lead to renounce to the autonomy of apraxia  
433 and to assume that apraxia is not a gesture production deficit per se, but merely an emanation  
434 of other cognitive impairments in the domain of gestures.

#### 435 4.2. SPECIFIC VERSUS NON-SPECIFIC CAUSES OF APRAXIA

436 In an era marked by major neuropsychological discoveries in the fields of aphasia and  
437 agnosia, Finkelnburg (1870) interpreted apraxia as a reflect of “asymbolia”, namely, the loss  
438 of conventional knowledge as demonstrated by the inability to perform meaningful gestures,  
439 to identify visual objects or people, or a lack of politeness – what would nowadays refer to  
440 semantic memory deficits and prosopagnosia. So, from the start, the autonomy of apraxia  
441 toward other neurological signs or psychological constructs was a topic of debate. The  
442 independence of apraxia toward aphasia, elementary motor or sensory deficits was only to be  
443 demonstrated by Liepmann in the beginning of the 20th century. The originality of his work  
444 has been to provide apraxia a specific status, an effort that has been continued in  
445 contemporary definitions: Apraxia should not be explained by elementary motor or sensory  
446 deficits, task comprehension problems, object recognition deficits, lack of coordination, or  
447 inattention to commands (Geschwind, 1975; Rothi et al., 1991, 1997; Signoret & North,  
448 1979). So, the clinical diagnosis still relies on Liepmann’s intellectual approach. The process-  
449 based approach, however, has led to a paradox: If any cognitive impairment can explain  
450 movement disorders, then what is the specificity of apraxia? The autonomy of apraxia toward  
451 language has long been demonstrated; it now has to be demonstrated toward other cognitive  
452 processes. This demonstration would imply to remake Liepmann’s approach, with the  
453 difference that the number of cognitive domains to be tested is now such that it would imply  
454 to test the whole cognition – a procedure that is difficult to operationalize in aphasic patients.  
455 To our knowledge, no studies have carefully examined all the potential causes of gesture  
456 production deficits in the same patients. Therefore, most, if not all the published data have  
457 provided only a partial picture of apraxia, meaning that none has demonstrated the autonomy  
458 of apraxia toward other cognitive domains.

459 A first step in this direction would be to make a distinction between non-specific and  
460 specific praxis impairments. A cognitive deficit can be considered praxis-specific if it

461 generates impairments in gesture tasks only, but non-specific if it explains clinical signs or  
462 syndromes other than the defective production of gestures. Impairments of language, visual-  
463 spatial skills, executive functioning, working memory, and social cognition, can be considered  
464 as non-specific, because patients with such impairments may also fail tests in which gestures  
465 are not requested. Subsequently, these processes should not be referred to in a definition of  
466 apraxia (for reasons exposed in section 4.1). Other cognitive processes have a more  
467 ambiguous status. Semantic memory has long been viewed as the main cause of apraxia (Roy  
468 & Square, 1985; Rothi et al., 1991, 1997), especially in the light of category-specific clinical  
469 impairments (i.e., knowledge on man-made objects versus living things; Warrington &  
470 Shallice, 1984). Patients with semantic loss, however, also fail language tests, meaning that  
471 semantic memory cannot be considered praxis-specific. To consider this process praxis-  
472 specific, it would be necessary to demonstrate that patients who have gesture deficits also  
473 have isolated loss of knowledge of actions or objects. That said, naming tools selectively  
474 activates premotor areas (Martin et al., 1996), a brain region that has been more associated  
475 with networks of motor cognition than of semantic memory. Dissociations between semantic  
476 tests and gesture production (Bartolo et al., 2007; Buxbaum et al., 1997; Buxbaum & Saffran,  
477 2002; Negri et al., 2007; Osiurak & Badets, 2016; Silveri & Ciccarelli, 2009) have paved the  
478 way for the visual/kinesthetic or manipulation knowledge hypothesis. [This hypothesis](#)  
479 [assumes that gesture production is made possible by retrieval of stored sensorimotor](#)  
480 [knowledge, or “gesture engrams”. The latter are implicit, non-declarative memories that](#)  
481 [contain invariant, spatial characteristics of the gesture \(e.g., “ample movements of the elbow”](#)  
482 [correspond to a “hammering” gesture; Buxbaum, 2001; Rothi et al., 1991\). Gesture engrams](#)  
483 [may be critical not only to perform tool-related gestures in the absence of tools and objects](#)  
484 [\(i.e., pantomime of tool use\), but also to discriminate and recognize pantomimes. Since they](#)  
485 correspond to praxis-specific knowledge, their loss should selectively hamper praxis tasks.



486 The existence of such representations, however, remains under debate (Buxbaum et al., 2015;  
487 Osiurak & Le Gall, 2015). More importantly, defining apraxia as a (gesture) memory deficit  
488 questions the autonomy of apraxia toward memory deficits; after all, apperceptive agnosia is  
489 generally not interpreted as the loss of stored visual representations. As a matter of fact, this  
490 representational stance of apraxia has been challenged by the observation of patients who can  
491 use, but not recognize the use of the same tools (Baumard et al., 2019; Moreaud et al., 1998).  
492 On ground of this dissociation, we argue that apraxia, by definition, should be diagnosed in  
493 patients who do have gesture production deficits. Other approaches have proposed that  
494 gesture production relies on online processing and reasoning rather than on memory (i.e.,  
495 technical reasoning, Osiurak, 2014; structural inference, Goldenberg & Haggmann, 1998; see  
496 also categorical apprehension; Goldenberg, 2009). This mode of reasoning is independent  
497 from general reasoning skills (Goldenberg et al., 2007), [and has been associated with](#)  
498 [performance in praxis \(tool use and imitation\) tests only](#). Thus, it may be considered specific  
499 to apraxia.

500       Body image (Goldenberg, 1995) or body schema (Buxbaum, 2001) impairments may be  
501 at the root of apraxia as well, especially imitation impairments (for a review, see de  
502 Vignemont, 2010). The status of these processes (specific or not) is [particularly](#) ambiguous.  
503 First, alterations of body image (i.e., knowledge on the structural description of the body) may  
504 also provoke autotopagnosia (i.e., the inability to point at one's own body parts; Schwoebel &  
505 Coslett, 2005; Sirigu et al., 1991) and hence might be considered unspecific, yet the inability  
506 to point at body parts might also fall into the scope of gesture production deficits. [Generally,](#)  
507 [autotopagnosia \(i.e., the inability to point at one's own body parts on demand\) is considered](#)  
508 [different from apraxia, and is interpreted as a consequence of the loss of body image](#). As De  
509 [Vignemont \(2010\) emphasized it, however, seeing autotopagnosia as either a body image or](#)  
510 [body schema deficit is arbitrary because pointing at body parts requires body image \(to](#)

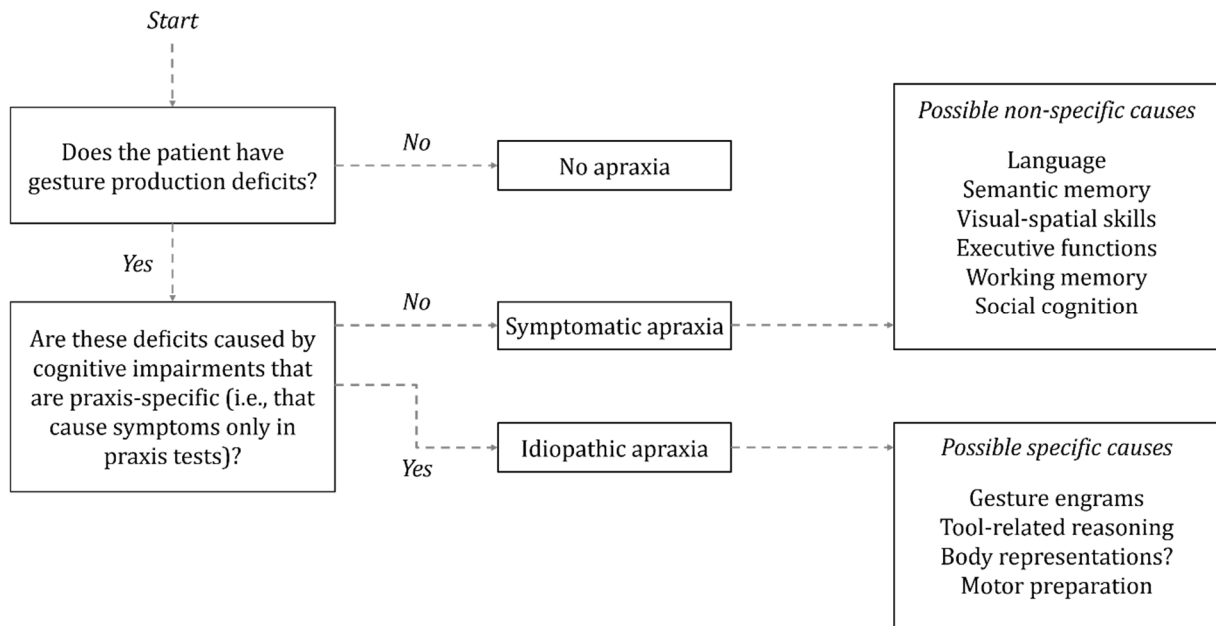
511 represent the target of the movement), and body schema (because it requires a tridimensional  
512 representation of the body posture). With a similar intellectual approach, we suggest that  
513 defining autotopagnosia as a body representation task, but not as a praxis task, is somewhat  
514 arbitrary because after all, pointing at body parts requires to perform intentional, goal-directed  
515 movements. The interdependence between autotopagnosia and apraxia remains to be  
516 systematically addressed. Second, body schema (i.e., online coding of body posture;  
517 Schwoebel & Coslett, 2005) has been associated with apraxia, yet the independence between  
518 both syndromes also remains an open issue. For instance, some “body schema” tests like  
519 repetitive finger movements are very similar to the tapping tasks used to assess limb-kinetic  
520 apraxia, and hence may be considered praxis tasks. In fact, given that any production of  
521 gestures involves the body, it makes it difficult to tell whether gesture production impairment  
522 is caused either by “apraxia” (a gesture production deficit that cannot be explained by other  
523 cognitive impairments), or by body representation disorders (a gesture production deficit that  
524 can be explained by loss of body representations). Therefore, whether body representations  
525 should be “specific” or “non-specific” remains an unsolved issue, because it depends on how  
526 one sees body representations tests. Body image would be “specific” if one accepts  
527 autotopagnosia as a praxis task, but “unspecific” if one does not – because it would mean that  
528 body image is necessary to fulfill a non-praxis task. Body schema is even more difficult to  
529 disentangle from specific praxis processes as it is a representation of the body defined by its  
530 involvement in action (De Vignemont, 2010). Pending future research, we have opted for  
531 considering body representations as a specific process, on the one hand, because body schema  
532 has been included in cognitive models of apraxia published by some colleagues (Buxbaum,  
533 2001) and, on the other hand, because body image deficits have been explicitly proposed to  
534 explain some manifestations of apraxia (e.g., Goldenberg, 1995). Future works may document  
535 associations or dissociations between praxis and other body-related tasks to address this issue.

536 To sum up, only manipulation knowledge, technical reasoning (or similar views), and  
537 perhaps body image and body schema, might be considered specific of apraxia. There is a  
538 possible need for a new vocabulary to distinguish between “pure” apraxia and other forms of  
539 gesture production deficits. Strictly speaking, apraxia with identified non-specific causes  
540 should probably not be defined as “apraxia”, yet, for want of anything better, we suggest to  
541 make at least a distinction between “primary/idiopathic” apraxia and  
542 “secondary/symptomatic” apraxia (Figure 2). The latter may correspond to cases in which  
543 gesture production deficits can be explained by non-specific cognitive impairments. After  
544 having excluded all of the possible non-specific causes, one may finally diagnose  
545 “Primary/idiopathic” apraxia. Note that apraxia is by definition “symptomatic” of brain  
546 lesions, yet we have selected these widely used terms to reflect the specific/non-specific  
547 distinction.

548

549

550 **Figure 2. Symptomatic versus idiopathic apraxia**



551

552 *Notes.* The list of possible causes includes the most studied processes but may not be exhaustive.

### 553 4.3. CONSISTENT VERSUS TASK-DEPENDENT APRAXIA

554 Since apraxia is a gesture production deficit, the operational definition of apraxia should

555 focus on a “consistency” criterion, namely, gesture production deficits should be observed in

556 any task that requires the production of gestures. It turns out that none of the abovementioned

557 specific causes of apraxia may elicit task-independent impairments. The loss of

558 visual/kinesthetic engrams or manipulation knowledge hampers the production of already

559 experienced gestures, but neither the imitation of meaningless gestures, nor the use of novel

560 tools (Buxbaum, 2001; Rothi et al., 1991, 1997). Alterations of technical reasoning or

561 structural inference are thought to provoke impairments in tool use tests only. The loss of

562 body representations has been associated with imitation impairments and pantomime of tool

563 use (Buxbaum, 2001), but not with other testing conditions. Previous works have already tried

564 to define apraxia as the production of spatial-temporal movement errors (see section 2), yet

565 these errors are actually task-dependent (e.g., errors with transitive and intransitive gestures,

566 but not in tasks of fine motor coordination; Kangas & Tate, 2006). Kinematic studies have  
567 found these errors to be task-dependent (Hermsdörfer et al., 2013). In addition, this error-  
568 based definition applies to patients with optic ataxia as well – an impairment of visually-  
569 guided reaching movements and of hand configurations while grasping objects (Rossetti et al.,  
570 2003; for a discussion on the distinction between apraxia and optic ataxia, see Goldenberg,  
571 2009).

572         The only apraxia subtype that fulfills the consistency criterion may be limb-kinetic  
573 apraxia (see section 3; Denes et al., 1998; Gross & Grossman, 2008; Leiguarda et al., 2002;  
574 Leiguarda & Marsden, 2000), also called “innervatory apraxia” (Kleist, 1907). Limb-kinetic  
575 apraxia is characterized by unilateral, clumsy distal movements of the limb contralateral to the  
576 lesioned hemisphere. It is typically consistent across tasks, relatively independent of modality  
577 (e.g., verbal command or imitation) and not subject to automatic-voluntary dissociation.  
578 Typically, the content of the movement is recognizable whereas the execution is characterized  
579 by faulty selection of motor patterns, and interfinger uncoordination (i.e. the simultaneous  
580 activation of agonist and antagonist muscle groups). [This clinical sign can be revealed with](#)  
581 [the coin rotation task, in which the patient is asked to rotate a coin with the thumb, index and](#)  
582 [middle fingers](#) (Mendoza et al., 2009). Limb-kinetic apraxia can be distinguished from  
583 elementary motor disorders (i.e., paresis, parkinsonian syndrome): concurrent motor  
584 weakness is not systematic (see Denes, 1998), and the performance varies as a function of the  
585 length of the sequence of the requested movement. Luria (1978) has made a distinction  
586 between defective selection of motor patterns caused by post-central lesions and sensory  
587 deficits, and faulty seriation of motor inputs following lesions of the premotor cortex. Strictly  
588 speaking, only the latter form passes through the filters of exclusion, specificity ([i.e.,](#)  
589 [abnormal motor preparation hampers gesture production tasks only](#)), and consistency criteria,  
590 meaning that limb-kinetic apraxia could be [the purest and most unequivocal form of apraxia](#)

591 (Table 3). For this reason, we added deficits of “motor preparation” as a possible cause of  
592 idiopathic apraxia in Figure 2.

593         It should be acknowledged that limb-kinetic apraxia is quite different from other forms  
594 of limb apraxia. Limb-kinetic apraxia follows unilateral, pericentral lesions, and affects the  
595 production of finger sequences performed with the contralesional hand. In contrast, other  
596 forms of limb apraxia generally affect both hands following lesions of the left hemisphere (in  
597 right-handed patients). In that case, however, the localization of lesions depends on the task  
598 used to assess apraxia. For example, meaningless imitation and mechanical problem-solving  
599 tests depend on the left parietal lobe (Goldenberg, 2009; Goldenberg & Spatt, 2009), while  
600 tool use tests may be also altered due to semantic loss after temporal brain damage (e.g.,  
601 Hodges et al., 2000). Pantomime of tool use has only poor localization value (Goldenberg,  
602 2017). This clinical variability can be easily understood if one assumes that different tests of  
603 limb apraxia actually assess different cognitive processes. In contrast, limb-kinetic apraxia is  
604 very recognizable, is less task-dependent, and has stronger localization value than other forms  
605 of limb apraxia. It amounts to considering bilateral gesture production deficits as the  
606 expression of cognitive impairments *in* gesture production, and the unilateral deficits  
607 characteristic of limb-kinetic apraxia (interhemispheric disconnection syndromes set apart) as  
608 impairments *of* gesture production. For example, a patient with a semantic deficit may fail to  
609 perform meaningful gestures only, while a patient with limb-kinetic apraxia may fail to  
610 perform any gesture, whether meaningful or meaningless. After all, aphasia (a linguistic  
611 disorder that may or may not affect language production) is not the same thing as dysarthria (a  
612 motor deficit of speech). Of note, these gesture production deficits are not synonyms of  
613 deficits of the production system in cognitive models of apraxia. In these models, the  
614 production system has been the name of different processes like working memory (Roy &

615 Square, 1985; Cubelli et al., 2000), body schema (Buxbaum, 2001), or motor simulation  
616 (Osiurak, 2014), which praxis-specific nature is questionable (see the rationale in section 4.2).

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**Table 3. Application of the specificity and consistency criteria to apraxia subtypes**

Apraxia subtype	Definition	Proposed definition criteria		
		Exclusion	Specificity	Consistency
Limb-kinetic	Contralateral inaccurate or clumsy distal limb movement, especially with movement sequences. Precentral lesions.	✓	✓	✓
Dynamic ideomotor	Alterations of the body schema (coding of the relative position of body parts) resulting in impaired imitation and pantomime of tool use. Lesions of the superior parietal cortex (Buxbaum, 2001).	✓	✓	✗
Representational ideomotor	Loss of gesture engrams preventing patients from recognizing and performing familiar gestures. Lesions of the left inferior parietal lobe (Buxbaum, 2001).	✓	✓	✗
Conceptual	Loss of mechanical and tool knowledge resulting in content errors in tool use situations. Lesions in the left hemisphere (Heilman et al., 1997).	✓	✓ / ✗	✗
Visuo-imitative	Faulty imitation of meaningless movements (Mehler, 1987). Might reflect the loss of body representations (Goldenberg, 1995).	✓	✓ / ✗	✗
Ideomotor	Inability to convert the intention of the movement into its proper execution. Initially assessed under imitation but later extended to a variety of gesture production deficits. Lesions depend on the task.	✓	✓ / ✗	✗
Ideational	Alteration of the “concept” of the gesture, caused by any deficit (action-specific or not). Generally considered as an impairment of tool use or action sequences. Lesions depend on the task.	✓	✗	✗
Ventral	Gesture production deficits reflecting conceptual problems with object knowledge, following temporal lobe lesions (Buxbaum, 2001).	✓	✗	✗

619 *Notes.* Exclusion criteria refer to what apraxia is not, namely, clinical signs that should not better explain the deficit in gesture production  
620 (e.g., elementary motor or sensory deficits, aphasia). We have assumed that the exclusion criteria were systematically met, although this has  
621 not always been extensively discussed in previous literature. Specificity: a cognitive impairment can be considered praxis-specific if it  
622 generates deficits in gesture production tasks only, but non-specific if it explains clinical signs other than the abnormal execution of gestures  
623 (e.g., a semantic deficit can also be demonstrated in tests of language). The specific or non-specific status may depend on how one defines  
624 and explains the apraxia subtype (see section 4.2 and table 2). Consistency: the deficit can be considered “consistent” if it can be documented  
625 in any task that requires the production of gestures, and not only in particular gesture production tasks (e.g., if a patient fails to imitate  
626 meaningless gestures, but not meaningful gestures, then the deficit is not consistent across tasks). Only the most widely used apraxia  
627 categories are displayed. For an exhaustive list, see Petreska et al. (2007). Details are in the text.

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## 5. CONCLUDING REMARKS AND PERSPECTIVES

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The evolution from a task-based toward a process-based approach is a significant scientific progress, while it is also an invitation to remake Liepmann’s initial intellectual approach, that is, to question the autonomy of apraxia again toward the whole cognition. Avoiding arbitrary definitions, and tending toward a consensual, scientific definition of this neuropsychological syndrome, may require to discuss some new operational criteria, among which specificity and consistency (a criterion that has actually driven Liepmann’s work). It should be noticed that idiopathic and symptomatic apraxias are not intended to become new clinical phenotypes, but rather to draw attention on the autonomy of the syndrome. [This](#)



639 epistemological standpoint could prove beneficial for neurological patients, while it may also  
640 raise new issues.

## 641 5.1. ON THE FUNCTIONAL SIGNIFICANCE OF APRAXIA

642 Discussing the incompatibility of task-based and process-based approaches, as well as  
643 narrowing the definition of apraxia, may have significant implications for studies on the  
644 functional significance of this syndrome – a topic on which there is only sparse literature  
645 (Foundas, 2013). In a task-based approach, standard praxis tasks, like pantomime of tool use,  
646 are generally used to predict the functional outcome (e.g., Bickerton et al., 2012; Chestnut &  
647 Haaland, 2008; Foundas et al., 1995; Hanna-Pladdy et al., 2003). This method raises two  
648 issues.

649 First, using pantomimes (i.e., tool-related actions without actual tools and objects) to  
650 predict the ability to use actual tools and objects may sound paradoxical. A significant body of  
651 evidence has indicated a strong association between performance in pantomime and real tool  
652 use in patients with left hemisphere stroke (Foundas et al., 1995; Neiman et al., 2000;  
653 Randerath, 2011). Nevertheless, pantomime of tool use is more difficult than real tool use, and  
654 also correlates with novel tool use tests (for a review, see Baumard et al., 2014). This has  
655 suggested that pantomiming the use of tools has only poor ecological value, and is a non-  
656 routine, creative test (e.g., it is more frequent to use a potato-peeler than to pantomime the use  
657 of it) that calls for semantic memory, working memory, motor simulation, and perhaps social  
658 cognition (Bartolo et al., 2003; Finkel et al., 2018; Goldenberg, 2017; Goldenberg et al., 2003;  
659 Osiurak, 2014). Since both pantomime of tool use, and activities of daily living, call for  
660 cognitive processes that are not praxis-specific, functional recovery may not be a synonym of  
661 apraxia recovery. Actually, if one defines limb apraxia as the inability to pantomime the use of  
662 tools without tools in hands, and since pantomime and real tool use may partly dissociate,  
663 then the functional significance of limb apraxia can only be unclear.

664           Second, as we have argued, different tests of apraxia may call for different  
665 motor/cognitive processes. It means that using one test or the other may result in very  
666 different findings, reflecting task effects as much as actual recovery. Does it make sense to  
667 study apraxia recovery, if the operational definition of apraxia itself is already a challenging  
668 issue? In a task-based approach, it is always unclear which cognitive process may support  
669 functional recovery. Switching from a task-based to a process-based approach may help better  
670 understand which motor/cognitive impairments predict functional recovery. In a process-  
671 based approach, it would be necessary to achieve full alignment between the processes behind  
672 the initial clinical picture, and those behind the tasks used to estimate the functional recovery.  
673 The distinction between idiopathic apraxia and symptomatic apraxia, as well as the  
674 consistency criterion, may be helpful in this regard. For instance, if apraxia is symptomatic,  
675 then the non-gestural portion of activities of daily living may be altered. For example,  
676 semantic loss may prevent a patient from using technical devices, but also from understanding  
677 the pieces of advice given by the customer service. If a patient wants to build a shelf, a  
678 planning deficit will make it difficult for her/him to anticipate that she/he needs wooden  
679 boards, cleats, and screws, and how to find them, whereas manipulating the screwdriver will  
680 not pose her/him any kind of problem. One may assume that limb-kinetic apraxia, being task-  
681 independent, may be associated with more significant functional deficit than other forms of  
682 apraxia – although this necessarily depends on the precision needed to perform the task (e.g.,  
683 eating pickles from a jar Versus wiping a table clean with a sponge).

## 684           5.2. A FINAL WORD ON REMAINING ISSUES

685           It should be acknowledged that the suggestions made in this review intend to be a first  
686 step to improve the definition of apraxia, yet they do not solve all the problems. For instance,  
687 the distinction between limb-kinetic apraxia and motor disorders is sometimes difficult to  
688 demonstrate. Furthermore, limb-kinetic apraxia affects gesture production with specific body

689 segments (i.e., fingers), but it remains unclear how it hampers gesture production tests  
690 performed with other body parts. Said differently, perhaps limb-kinetic apraxia is not  
691 “absolutely constant”, but we have argued that it is at least “more constant” (less task-  
692 dependent) than other forms of limb apraxia. The nature of limb-kinetic apraxia, and how it  
693 affects different gesture production conditions, calls for future studies.

694 More importantly, to further clarify the concept of apraxia, there is probably a need to  
695 create new taxonomies to better describe and understand abnormal gesture production, as well  
696 as a new vocabulary to call different clinical signs and syndromes by different names. This, in  
697 turn, raises the issue of *which* clinical manifestation would deserve the “apraxia” wording at  
698 last. Since limb-kinetic apraxia passes all the filters evoked above, we argue that it is a good  
699 candidate. Such a strict definition of apraxia may lead to reconsider its prevalence. Limb-  
700 kinetic apraxia has been less frequently described than other forms of apraxia because it is a  
701 rare condition, yet this may also reflect the heterogeneity of others forms of apraxia. The  
702 latter, indeed, encompass different clinical conditions, cognitive impairments and brain  
703 lesions. Switching from a task-based to a process-based approach offers new challenges for  
704 studies on the prevalence, semiology, recovery, and treatment of apraxia.

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