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# One century after Liepmann's work on apraxia: Where do we go now?

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## Introduction

The term apraxia derives from Greek and means “without action”. It has been introduced by a German philologist and philosopher, Heymann Steinthal, in 1871. By this term he aimed to denote the difficulty observed in aphasic patients in the use of everyday objects. Nevertheless, this term has been rapidly extended to difficulty in producing gestures with or without objects and required in different modalities (verbal command, visual input, imitation). Hugo Liepmann could be considered the father of the neuropsychology of apraxia, as he identified apraxia as a disorder independent from other cognitive deficits. Almost 20 years ago, Georg Goldenberg wrote an eminent article on Cortex (2003) on his life and works on apraxia and provided an extensive overview of Liepmann’s legacy until the 2000s.

Hugo Liepmann was born in Berlin in 1863. He obtained a doctoral degree in philosophy before graduating in medicine. He was Wernicke’s pupils for four years (1895–1899) before moving to Dalldorf where he directed the psychiatric hospital and started his work on apraxia. At the end of his life he was affected by Parkinson’s disease. More than one hundred years after his original studies on apraxia, Hugo Karl Liepmann’s ideas are still very influential for neuropsychology. The main contribution of Liepmann consisted in the formulation of the first theoretical model of apraxia aiming to explain how intended actions can be performed in the human brain. In a very first diagram depicting the transformation of an intended movement into a motor action, Liepmann considered that the idea of the movement had no specific localization. In this first diagram, he considered that the *intended movement* (i.e. the idea of the movement) was stored in the “whole brain cortex” (vertritt den Gesamtkortex; 1908). This term was subsequently replaced by the terms *movement formulae* and adopted in the further models. Evidence from lesion studies (Liepmann, 1905) led him to propose that movement formulae could be finally localized in a posterior region corresponding to the left temporo-parieto-occipital junction (Liepmann, 1920). For Liepmann, any purposeful action requires movement

formulae, which can be considered as a spatio-temporal mental image of the action. These movement formulae are not “motoric”, strictly speaking, and must be distinguished from kinetic or motor memories that specify the innervatory patterns. Movement formulae can be visual or acoustic and provide the individual with a global idea of the intended action. Interestingly, even if Liepmann (1908) considered that the failure to create these movement formulae, i.e., ideational apraxia<sup>1</sup>, could manifest itself in actions, these formulae were not specific to actions. As mentioned above, he initially assumed that they were generated by the whole brain, but he gave, over the years, a greater importance to the left hemisphere and in particular to the inferior parietal lobe (IPL) in the generation of these movement formulae, suggesting that only the left hemisphere could support the realization of actions from memory. Since to Liepmann (1908) movement responds to an internal mental imagery, the actual interaction with objects may compensate the loss of mental imagery in patients with apraxia. In agreement with this view, actual tool use was generally less impaired than pantomime of tool use or than actions performed under imitation modality (Liepmann, 1908).

Liepmann distinguished the movement formulae from motor memories that he thought were supported by central motor regions. For him, the loss of motor memories led to limb-kinetic apraxia also commonly called motor apraxia (1908). Contrary to ideational apraxia and ideo-motor apraxia (see below), this variant of apraxia can be found even in routine actions. Finally, he also posited that some patients could suffer from ideo-motor apraxia, which refers to the failure of the transition from movement formulae to motor memories because of the interruption of fibers from the whole cerebral cortex to the motor cortex (after lesions to the white matter below the IPL). In this case and contrary to ideational apraxia, patients are not able to perform appropriate gestures even by imitation, that is, when the demonstration provides the movement formula. Faulty imitation demonstrates the inability of the patient to transpose the correct idea of the action into appropriate motor programs. Finally, Liepmann contributed

to the autonomy of apraxia from other cognitive disorders, such as language, in showing that apraxia and aphasia could dissociate in brain-damaged patients.

This special issue brings together 15 contributions from some of the most influential research groups on apraxia nowadays. Besides presenting these contributions, we will try to determine if and how these findings diverge from the main conclusions drawn by Liepmann, leading us to introduce what could be the next steps in the future research on apraxia.

First of all, we will focus on the concept of movement formula, a central notion in Liepmann's thought that will allow us to introduce the contributions of the authors of this SI on the gesture-engram hypothesis. Then, Liepmann defended the idea that apraxia is an autonomous disorder and not the consequence of another deficit, yet, we know that other deficits can coexist, therefore a paragraph on this point has been devoted to understand how apraxia can be disentangled from other diseases. Our editorial ends with a paragraph on imitation deficits before concluding our Editorial.

### **The movement formulae**

As explained above, even if Liepmann considered that apraxia – and particularly ideational apraxia – can occur after damage to different locations of the posterior cerebral cortex, he nevertheless considered that, in right-handed patients, the left IPL could play a specific role in the creation of movement formulae (1920). Thus, when this brain region is damaged, apraxia is more likely to be observed. Several contributions to this special issue are consistent with the important role played by left IPL and particularly the left supramarginal gyrus (SMG) in apraxia. **Dressing et al. (2021)** investigated the clinical course of recovery of apraxia in left brain-damaged patients. They examined 90 patients during the acute (about 5 days) and chronic (about 14 months) stage after left hemisphere stroke on imitation of meaningless postures and production of pantomime of tool use on visual presentation. They found that errors in both tasks persisted at the chronic stage. More importantly, they reported

that chronic pantomime production errors persisted after lesions to the left SMG, leading them to suggest that this brain region is essential for pantomime of tool use, and more generally, tool use. This interpretation is perfectly in line with a recent comprehensive review on pantomime of tool use (and tool use), which was based on six meta-analyses (behavioral, brain lesions and neuroimaging; Osiurak et al., 2021). **Pastore-Wapp, Nyffeler, Nef, Bohlhalter, and Vanbelligen (2021)** also came up with a similar conclusion, through their scoping review on non-invasive brain stimulation (NIBS) in healthy subjects and patients with stroke. They confirmed that NIBS over left IPL interferes, in healthy subjects, with gesture processing and that excitatory anodal transcranial direct current stimulation over left IPL improves limb apraxia in left brain-damaged patients. In the same vein, **Valério et al. (2021)** reported the case of a patient, FP, who had lesions to the left IPL and who had severe difficulties in producing pantomimes of tool use or in using tools in isolation. **Stoll et al. (2022)** investigated in 58 left brain-damaged patients and 51 right brain-damaged patients the selection and application of familiar and novel tools and reported that the left IPL was involved in the application of tools, and particularly the application of novel tools. Finally, **Matheson, Garcea, and Buxbaum (2021)** conducted an fMRI experiment in which participants made conceptual judgments about images of tools embedded in scenes that either suggested their use (i.e., use contexts) or that they would simply be moved (i.e., move contexts). By using a representational similarity analysis, they found that category information predicted patterns in the left SMG more strongly in the *use contexts* than in the *move contexts*. Taken together, all these studies confirm the contribution of the IPL in apraxia as proposed by Liepmann more than one century ago.

### **The gesture-engram hypothesis**

The key role of the left IPL/SMG in gesture performance in right handers – and notably tool use – is in line with Liepmann’s thought. Over the last century, several neo-associationist and cognitive models have assumed that the left IPL might store gesture engrams, which

characterize the key parameters of movements that are associated with the use of a tool (e.g., an ample oscillation of the elbow and a power grip for a hammer; Buxbaum, 2001; Cubelli, Marchetti, Boscolo, & Della Sala, 2000; Heilman, Rothi, & Valenstein, 1982; Rothi, Ochipa, & Heilman, 1991). The integrity of these engrams can be assessed with manipulation-knowledge tasks in which the patient has to match two tools that have the same manner of manipulation or to match a tool with the correct posture/gesture. Two contributions of this special issue question the validity of the gesture-engram hypothesis. As mentioned above, **Valério et al. (2021)** reported the case of a patient, FP, who had lesions to the left IPL and who showed severe difficulties in producing pantomimes of tool use or in using tools in isolation. However, this patient was not impaired on a manipulation-knowledge task. Interestingly, they described the opposite case, LS, who could produce appropriate pantomimes in the presence of impaired performance on the same manipulation-knowledge task. **Lesourd, Naëgelé, Jaillard, Detante, & Osiurak (2020)** documented the case of VF, a left-handed patient, left-lateralized for language, who showed a severe apraxia after damage to the right hemisphere. Although she could use tools “correctly”, she showed a great number of hand posture errors when manipulating them (e.g., holding a hammer by grasping the handle with the thumb oriented toward the handle and not the head of the hammer). In a way, this patient could be characterized as exhibiting a pure “gesture engram” deficit because these engrams are thought to contain the information useful for correct postures during tool use. Surprisingly, her performance on manipulation-knowledge tasks was not severely impaired. As a consequence, this case of patient cannot be explained by the gesture-engram hypothesis.

**Stoll et al. (2022)** rightly stressed that Liepmann did not conceive his movement formulae (Liepmann, 1908) as “invariant cognitive units” (i.e., the motor engrams as defined above). Instead, for him, the praxis network was based on the interplay of many brain regions, even if lesions to the left IPL could impair the praxis network more severely (Liepmann, 1900; 1908;

1920; 1925). This “hub” hypothesis of the left IPL is in line with the working-memory interpretation proposed by **Stoll et al. (2022)** or by **Matheson et al. (2021)**. The corollary is that other cognitive aspects can participate in the production of purposeful actions even if they are not *a priori* considered as dedicated to the domain of action, as Liepmann thought. This is confirmed, for instance, by the study of **Rounis, Halai, Pizzamiglio, and Lambon Ralph (2021)**, who investigated a cohort of 41 left brain-damaged patients on different gestural tasks, and who found by using a principal component analysis that a specific component is associated with semantic control. This is also in line with **Stoll et al. (2022)**, who also found a specific role of semantic memory and ventral structures in object selection.

### **The autonomy of apraxia from other disorders**

Since Liepmann's time, one of the problems to be solved in order to consider apraxia as an autonomous disorder has been the link between praxis and language, which is a long-standing question in the scientific literature. It has been repeatedly hypothesized that tool-use/praxis skills and language skills could have co-evolved (Greenfield, 1991; Holloway, 1969, Uomini, 2009; Roby-Brami, Hermsdörfer, Roy, & Jacobs, 2012; Corballis, 2012), suggesting a strong link between praxis and language. It has been also debated whether aphasia causes other communication disorders like dysarthria, dysgraphia and apraxia (to note that also Steinthal, 1881, considered that apraxia was an exaggeration of aphasia) or whether a motor-sequencing disorder is at the basis of other gestural disorders including those required for the articulation of language (Helm-Estabrooks, 1984).

As apraxia is found in patients with acquired lesions to the left hemisphere, the same hemisphere damaged in aphasia, the co-existence of apraxia and aphasia in these patients is not uncommon. In 1905, Liepmann administered a detailed study protocol consisting of transitive and intransitive gestures performed on verbal command and imitation to 42 patients with right hemispheric lesions and 47 patients with left hemispheric lesions (41 of whom suffered from



right hemiplegia). Results showed that no patient with a right hemispheric lesion was apraxic, providing a strong argument on the role of the left hemisphere in praxis processing. Of the 47 patients with left hemisphere damage, 23 had no apraxia or aphasia, while 14 had signs of apraxia and aphasia. Crucially, the remaining 10 cases showed a clear double dissociation, in particular, 6 of them were apraxic but not aphasic, whereas the other 4 showed the opposite profile, they were aphasic but not apraxic. This result was confirmed by Papagno, Della Sala and Basso (1992), who tested language and gesture processing in 699 patients. While 540 patients had both apraxia and aphasia, 10 apraxic patients were not aphasic and 149 aphasic patients were not apraxic. These results are in line with the idea that apraxia and aphasia are two independent disorders and that most patients show signs of apraxia and aphasia because the cortical regions involved in language and praxis overlap; therefore, language and gesture processing after a stroke in the left hemisphere are both likely to be damaged (Roby-Brami et al., 2012). In line with this, Vingerhoets, Alderweireldt, Vandemaele, Cai, Van der Haegen, Brysbaert, and Achten (2013) showed that the brain regions involved in language and gesture overlap in the supplementary motor area, dorsal and ventral premotor cortex, dorsolateral prefrontal cortex, and posterior parietal cortex. They considered that the “*non-overlapping parts might hamper language but not praxis or vice-versa, in agreement with the clinical observations of double dissociation*” (Vingerhoets et al., 2013, p. 181), suggesting that language and gesture processing differ to some extent. In another study, Mengotti, Corradi-Dell’Acqua, Negri, Ukmar, Pesavento and Rumiati (2013) tested 57 left-brain-damaged patients on tasks assessing language and gesture processing (among others, imitation of meaningful and meaningless gestures). Voxel-based lesion-symptom mapping analysis showed that praxic and linguistic performances were associated when the gesture to be imitated was meaningful, and were dissociated when the gesture to be imitated carried no meaning. More specifically, damage to the angular gyrus affected imitation of meaningless gestures

independently of the patients' performance on linguistic tests, whereas damage to the supramarginal gyrus affected not only imitation of meaningful gestures but also patients' performance on linguistic tasks.

In this issue, **Schmidt, Achilles, Fink, and Weiss (2022)** investigated 91 left brain-damaged patients on several tests assessing apraxia and aphasia. By applying a principal component analysis, they found a specific component related to language functions, confirming Liepmann's works that praxis and language could dissociate. However, **Kroliczak et al. (2021)** explored praxis and language networks and their links to handedness. In a fMRI study, they asked 125 healthy participants (52 right handers, 31 ambidextrous, and 42 left handers) to plan and execute pantomimes of tool use *versus* to perform a subvocal fluency task. They found that the atypical organization of praxis was present in the three groups and was even more frequent than the atypical organization of language. They also reported a non-symmetrical relationship between praxis and language organization in that a possession of atypical organization of language increased the likelihood for praxis to be atypical too, whereas the inverse relationship was weaker. This link is also influenced by handedness. Indeed, atypical lateralization of language and/or praxis was more frequent in left handers than in right handers. Unlike the findings of **Schmidt et al. (2022)**, the outcomes of **Kroliczak et al. (2021)** suggest that it remains debatable whether praxis and language are fully independent even if they might be supported by distinct neurocognitive substrates (see also **Mauri, Zanin, Aggujaro, Molteni, & Luzzatti, 2021**).

From his work, Liepmann was also able to demonstrate that apraxia can be dissociated from aphasia and also from asymbolia, a concept used at that time to describe the general ability to generate symbols. Thus, apraxia is an autonomous disorder, which shouldn't be confused with other "cognitive" disorders. There is perhaps here an ambiguity in Liepmann's thought. Indeed, as stressed above, he conceived the creation of movement formulae to be based on

mental processes that are not necessarily dedicated to actions even if the impairment of these mental processes could manifest itself in actions. In a way, even if the distinction drawn with language skills is not inconsistent with this idea, we can legitimately wonder whether apraxia is a gestural deficit strictly speaking or whether the manifestation of other cognitive deficits does appear in the gestural performance. In recent years, several studies have reported signs of apraxia in patients with no acquired lesions, such as individuals with autism (Stieglitz Ham et al., 2010; Stieglitz Ham & Bartolo, 2012) or schizophrenia (Walther, Vanbellingen, Muri, Strik, & Bohlhalter, 2013). **Walther, Mittal, Stegmayer, and Bohlhalter (2020)** provided a comprehensive review of apraxia in schizophrenia, which stressed that gesture deficits (mainly spatial and temporal errors) could be found in about 25% of schizophrenia patients. They emphasized that schizophrenia patients generally do not produce incomprehensible movements or no movements at all, which diverges from the severe difficulties that can be sometimes observed in left brain-damaged patients. Nevertheless, the errors in schizophrenia patients remain far more frequent than in healthy controls and cannot be explained by other cognitive or psychiatric disorders. Evidence also indicates that these patients tend to recruit the praxis cerebral network less than healthy controls when asked to perform familiar or novel hand gestures. Taken together, these findings suggest that the gesture deficit in schizophrenia is not an apraxia-like deficit but a genuine apraxia. Walther et al. drew a parallel here with patients with Parkinson's disease whose gestural deficits have not always been considered as signs of apraxia but rather as the impact of other motor-cognitive disorders in gestural performance. For them, similar to schizophrenia, a substantial portion of patients with Parkinson's disease are apraxic, a conclusion that is confirmed by the review of **Heilman (2020)**. In particular, Heilman addressed the issue of the assessment of apraxia in Parkinson's disease. Given that apraxia can be assessed in the absence of other motor impairments, the presence of rigidity and bradykinesia in Parkinson's disease is a challenge for the evaluation of apraxia in this population. However,

the levodopa treatment allows patients to reduce action tremor and at the same time allows the evaluation as well as the identification of the three forms of apraxia as described by Liepmann: limb-kinetic, ideomotor and ideational apraxia. Another deficit that has been observed in Parkinson's disease concerns what Heilman calls "synergetic apraxia", which is the ability to make alternate movements with each hand. All in all, the co-existence of other cognitive or motor impairments does not prevent to isolate the praxis defect.

It is true that the neuroanatomical models developed by Liepmann targeted the cerebral cortex (Liepmann, 1900). However, Liepmann stressed that the creation of movement formulae is an active process (see above), which can also be sometimes disturbed in healthy individuals if they are distracted, for instance. This was confirmed several decades later by some experimental studies, which have shown that healthy participants can produce spatiotemporal errors when asked to pantomime the use of tools under pressure (Rumiati & Humphreys, 1998) or can grasp tools in a non-functional way if they have to perform a concomitant semantic task (i.e., dual-task paradigm; Creem & Proffitt, 2001). Even if damage to the posterior cerebral regions can generate severe praxic difficulties, some difficulties can also occur even in the absence of damage to these specific regions. Thus, the findings that apraxia can be found in patients with subcortical lesions or without acquired lesions are not really at odds with Liepmann's thought. Instead, these findings suggest that the study of apraxia could have (too) long been oriented towards the investigation of patients that are reputed to exhibit gestural deficits, such as patients with left brain damage or Alzheimer's disease. Unfortunately, this could have led neuropsychologists to minimize the investigation of apraxia in other populations that are not known to show severe apraxia, although gestural deficits do exist when assessed. This is the case for schizophrenia and Parkinson's disease, but this is also true for frontotemporal dementia, a disease in which signs of apraxia can be found as reviewed by

**Yliranta and Jehkonen (2020)**. This is also consistent with the findings of **Stoll et al. (2022)**, which stressed that right brain-damaged patients can be impaired when asked to use novel tools.

**Baumard and Le Gall (2021)** questioned whether apraxia is an idiopathic disorder. They argued that among the numerous variants of apraxia, only the limb-kinetic (or motor) apraxia could meet the specificity and consistency criteria that allows us to view it as idiopathic. The other variants could be instead labelled as symptomatic apraxia to the extent that the deficits could be secondary to more general cognitive impairments. According to us, this conclusion, has to be taken with caution for several reasons. First of all, only 1% of the studies selected by Baumard and Le Gall was on limb-kinetic apraxia, therefore, the amount of information we can gather on this syndrome is weak to allow us to conclude that such a disease represents the only pure form of apraxia. Next, limb-kinetic apraxia concerns fine movements rather than complex actions. Furthermore, the arm affected in limb-kinetic apraxia is the one contralateral to the lesion site, while in the other forms of apraxia, ideational and ideo-motor, the disease affects (also) the ipsilateral limb, which is the most studied limb, as the contralateral one might be affected by hemiplegia. A final point is about the methodological possibilities that we have nowadays to set apart the role of other deficits in the praxis disorder. One of the clearest examples is apraxia in Parkinson's disease, where motor defects and praxis deficits might co-exist (see **Heilman, 2021**).

Overall, the presence of other deficits in the praxis disease might be a false problem. The challenge would be to set apart the level of clinical analysis from the level of cognitive analysis, as apraxia can appear in the presence of different clinical syndromes, yet this would not prevent from identifying apraxia as an independent disorder.

## **Imitation**

Liepmann considered that imitation tasks could be useful to distinguish ideational apraxia from ideo-motor apraxia. As explained above, for him, patients with ideational apraxia can

improve on imitation because the demonstration provides them with the correct movement formula. By contrast, faulty imitation can be found in patients with ideo-motor apraxia because these patients are unable to transpose the correct idea of the action into appropriate motor programs. De Renzi and collaborators largely contributed to the investigation of ideo-motor apraxia, primarily for their methodological approach. He took care of the meaning of the gesture, by considering meaningful and meaningless actions, as well as the portion of the limb used in the imitation task (the whole limb or just the fingers). Finally, he considered gestures performed as single actions or sequences of actions (De Renzi, Motti & Nichelli, 1980). According to Liepmann's model, in right-handed patients an intended action is generated in the left parietal lobe (movement formulae). To execute the action, the information is sent from the left parietal lobe to the left premotor cortex. To perform the gesture with the left hand, the information crosses the corpus callosum to reach the right premotor cortex and finally the right motor areas. Following this model, deficits in left parietal or frontal regions or a deficit that disconnects the parietal from the frontal region would cause praxis deficits at the same extent. This first point has been rejected by a series of studies carried out in the 1980's. In particular De Renzi and collaborators (1983) found that, with respect to healthy individuals, patients with parietal lesion were more affected in an imitation task than patients with frontal lesions. Furthermore, patients with lesions in the parietal lobe were less likely to recover from imitation deficit after 3-6 months than patients with lesions in regions sparing the parietal cortex (Basso et al., 1987). This conception of imitation has been then challenged by Goldenberg's works in which he demonstrated that the inability to imitate meaningless postures, particularly hand postures, requires body representations (Goldenberg, 1995, 1999), thereby drawing a direct link between autotopagnosia and visuo-imitative apraxia (Goldenberg & Karnath, 2006). In this special issue, several contributions stressed that this form of imitation involves ventro-dorsal and/or dorso-dorsal structures (**Dressing et al. 2021; Schmidt et al., 2022; Stoll et al., 2022**).

Previous findings showed that the ability to imitate meaningful (MF) and meaningless gestures (ML) varies if these gestures are presented in pure lists (i.e. a list with only MF and another with only ML) or in a mixed list (i.e. a list containing both ML and MF gestures). In particular, it was found that the imitation via pure lists of gestures allowed patients to use the dedicated route, which is the semantic for MF and the direct route for ML gestures (Bartolo et al., 2001; Cubelli et al., 2006; Tessari et al., 2007). In the same vein, **Mauri et al. (2021)** investigated the imitation of MF *versus* ML gestures in 32 left brain-damaged patients and 25 healthy controls by means of pure and mixed lists of gestures. Single-case analyses showed that patients were generally impaired on the imitation of MF gestures in both the pure and the mixed lists but they showed better scores on ML gestures in the pure list, meaning that the direct route was intact in these patients. This finding goes against the hypothesis that, at the presence of a deficit in the semantic route, patients can capitalize on the intact direct route to imitate MF gestures. This result rather suggests that the deficit at the semantic route interferes with the direct route and prevents the processing of MF gestures along the direct route. All these findings, although they are difficult to reconcile with Liepmann's view, mainly for the lack of a fine methodological approach during the times of Liepmann, are not against the idea, still kept for granted, that imitation deficits can be a symptom of ideo-motor apraxia.

### **Where do we go now?**

Interestingly, more than a century after the initial systematic works on apraxia by Liepmann, it appears that most of the conclusions he drew are relatively consistent with the current literature. Of course, many of his ideas remained close to intuitions given that he did not possess the technologies and the data to support them. Nevertheless, the idea that left IPL might be a specific hub for purposeful actions as suggested by **Stoll et al. (2022)** or **Matheson et al. (2021)** is remarkably close to the way Liepmann conceived the creation of movement formulae. Perhaps the main "*criticism*" that can be addressed to Liepmann's work was to focus

too much on the clinical. This criticism is extended by **van Elk (2021)** to the community of researchers working on apraxia. The author stressed that the literature on apraxia has been too long disconnected from the advances made in other disciplines. The fact that gestures carry semantic information, are used to communicate and / or express social and emotional information indicates the urgency of interacting with scientists who are specialists in other fields. One example is in the domain of tool use. Even if many papers on apraxia start by stressing that humans are tool users, it is very rare to see direct connections with other phenomena. For instance, cumulative technological culture refers to the accumulation in the complexity and/or efficiency of tools over generations (Dean, Vale, Laland, Flynn, & Kendal, 2014). This phenomenon is thought to be supported by a high-fidelity transmission mechanism and an innovative mechanism (Legare & Nielsen, 2015). Many studies in archaeology, anthropology, neuroscience, developmental psychology, or comparative psychology have been developed to investigate the (cognitive) origins of this phenomena. Given that this phenomenon targets tool-use skills, one would think that the literature on apraxia would be able to contribute to this topic, yet it remains largely absent from the debate. Neuropsychologists have not responded to the call of some cognitive archeologists about the requirement of having strong neurocognitive models to think how tool-use skills could have evolved in our lineage (Wynn & Coolidge, 2014; Wynn, Haidle, Lombard, & Coolidge, 2017). To date only few studies exists on the link between what we know from the neuropsychology on apraxia and this topic (Osiurak, Lesourd, Navarro, & Reynaud, 2020; Osiurak & Reynaud, 2020). However, these are centered on the key role of technical reasoning in tool-use skills.

Many other contributions could be made from other research groups with other viewpoints, as in the predictive processing framework of tool use proposed here by **Van Elk (2021)**. It is obvious that such contributions would be of greatest utility to help scientists better understand how tool-use skills but also some cultural phenomena have evolved across human



evolution. For example, teaching, which describes any behavior that facilitates learning for others (Kline, 2015), may be critical to the faithful transmission of content and thus to the emergence of a cumulative culture (Tomasello et al., 2005). Pantomime could be a proto-language, i.e. an early form of teaching that focuses the learner's attention on the salient part of the demonstration (Morgan et al., 2015). The wealth of neuropsychological literature on pantomime but also more generally on meaningful/communicative gestures, could provide new insights into the cognitive origins of teaching or proto-language. Thus, our answer to the question “where do we go now?” is “let’s go to the past”, meaning that the research on apraxia could significantly benefit from attempting to make new connections with all the literature interested in the evolution of complex motor acts.

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