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Apraxia: Clinical Types, Theoretical Models, and Evaluation

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1. Introduction

Apraxia is traditionally defined as a disorder of skilled movement that cannot be attributed to elementary sensorimotor deficit, aphasia or severe mental deterioration (De Renzi, 1989). This negative definition has led to integrate within the same framework a multitude of relatively different clinical manifestations, which have little in common with the kind of deficits to which it was originally applied (e.g., gaze apraxia, gait apraxia, trunk apraxia). These forms probably concern automatic movements and, therefore, will not be treated here. It is now largely admitted that some clinical signs are particularly useful for the diagnosis. First, the disorder affects the two sides of the body, even though the brain lesions are generally unilateral and more particularly located in the left (dominant) hemisphere. Second, the errors made by apraxics vary depending on the conditions of testing. For instance, apraxics can succeed in many circumstances, but fail when the movement must be executed to the clinician's request. In this frame, three categories of movement are regarded as relevant to the evaluation: Imitation of meaningless postures, pantomime production (i.e., demonstration of the use of a tool without the tool in hand) and actual tool use. Apraxia has been, and is still, subject to intense debate notably about its autonomy from elementary sensorimotor deficits and from higher-level cognitive processes. As a result, neurologists and neuropsychologists alike are commonly uncertain about the good way of assessing and interpreting it. In this chapter, we propose to address different issues relative to the notion of apraxia in light of recent developments made in the field.

The first part of the chapter will introduce two authors who made a significant, historical contribution to the notion of apraxia, namely Hugo Karl Liepmann and Norman Geschwind. Then, in the second part, we will present the cognitive models of apraxia that have emerged since 1980 (Buxbaum, 2001; Rothi, Ochipa, & Heilman, 1991; Roy & Square, 1985). Third, we will discuss recent data collected by Georg Goldenberg that raise a certain number of controversies about the cognitive models mentioned above (Goldenberg, 1995, 2003, 2009). In the sake of clarity, our discussion will deal with apraxic manifestations

assessed only with tasks of imitation of meaningless gestures, pantomime production and actual tool use. Besides discussing the psychological bases of apraxia, lesion sites associated with the different forms or apraxia will also be treated all along the paper. We hope that this chapter will help clinicians and students to better understand what apraxia is and how to evaluate it.

2. Historical background

2.1 Liepmann (1908, 1920)

We owe the first description of apraxia to Jackson (1866), who observed a motor intentional deficit in aphasic patients. Those patients were unable to move the tongue or lips on command, but could carry out these movements in an automatic movement sequence such as swallowing or eating. Jackson observed that this automatic-voluntary dissociation was not restricted to muscles of the facial region, since some of those aphasic patients were also unable to move their right, non-plegic hand on command while the same actions could be performed correctly in a spontaneous way. Although the first observations of apraxic patients are credited to Jackson, it was Steinthal (1871; see also Gogol, 1873) who coined the term apraxia. He described the case of an aphasic patient who attempted to write by holding a pencil upside down, or manipulated a fork and a knife as if he had never used them before. Steinthal stressed that it was not the ability to perform movements of extremities which was defective, but rather the relationship between the movements and the manipulated object, thereby suggesting that the absence of action (i.e., "a-praxia") might result from a perceptual deficit affecting object use recognition.

These observations led authors to hypothesize that apraxia might be a single neurological syndrome to be distinguished from agnosia, aphasia or asymbolia. Nevertheless, clinical and experimental evidence was still not enough to support this hypothesis. In fact, many authors thought at that time that it was hard to differentiate between apraxia and elementary sensorimotor deficits such as paresis. Independence of apraxia from pure motor deficits was supported by the observation of a 48-year-imperial councillor, the patient MT, by Liepmann (1900). MT was aphasic and showed clear-cut apraxia of the right hand. The deficit affected the movements of right extremities as well as movements of the head, the face and the tongue. MT was however able to perform properly, with his left hand, gestures on verbal command and upon imitation, including tool use. Verbal comprehension, visual recognition and global intellectual functioning were largely preserved. Liepmann proposed the term "motor apraxia" to describe this particular impairment.

In 1908, Liepmann published a study including 42 right brain-damaged (RBD) patients and 47 left brain-damaged (LBD) patients. He found apraxia in 20 out of 47 LBD patients. He also stressed that it did not occur at all in the right hands of the group of RBD patients. Liepmann proposed to call "sympathetic apraxia" this kind of apraxia which accompanies right hemiplegia. Moreover, 14 out of 20 LBD patients with apraxia were aphasic, but 6 were not. So, he argued that the left cerebral hemisphere is dominant not only for language but also for motor control. He also suggested that apraxia often accompanies aphasia, but is independent of it. In the meanwhile, Pick (1905) reported the case of an aphasic patient who was able to understand simple instructions, name objects and explain their functional use, but could use a knife to comb his hair, put a match to the mouth in an attempt to smoke it or

encounter severe difficulties to demonstrate how to use a key or a pair of scissors. Pick interpreted this disorder as a sign of motor apraxia given that the patient showed intact knowledge of functional uses of objects.

On the basis of these findings, Liepmann (1908, 1920) made the first clinical and anatomical synthesis of apraxia. He thought skilled movements to be supported by the creation of movement formulae in the whole posterior cortex. Movement formulae are constituted by acoustic or visual images of the action. To perform skilled movements, movement formulae produced by the posterior brain have to be associated via cortical connections with the innervatory patterns stored in the left sensorimotor region. When the left hand has to carry out the movement, the information is transferred via the corpus callosum to the right sensorimotor region. In sum, there is only one mechanism for skilled movements, which can nevertheless be impaired at three levels, thus producing three distinct forms of apraxia (Figure 1).
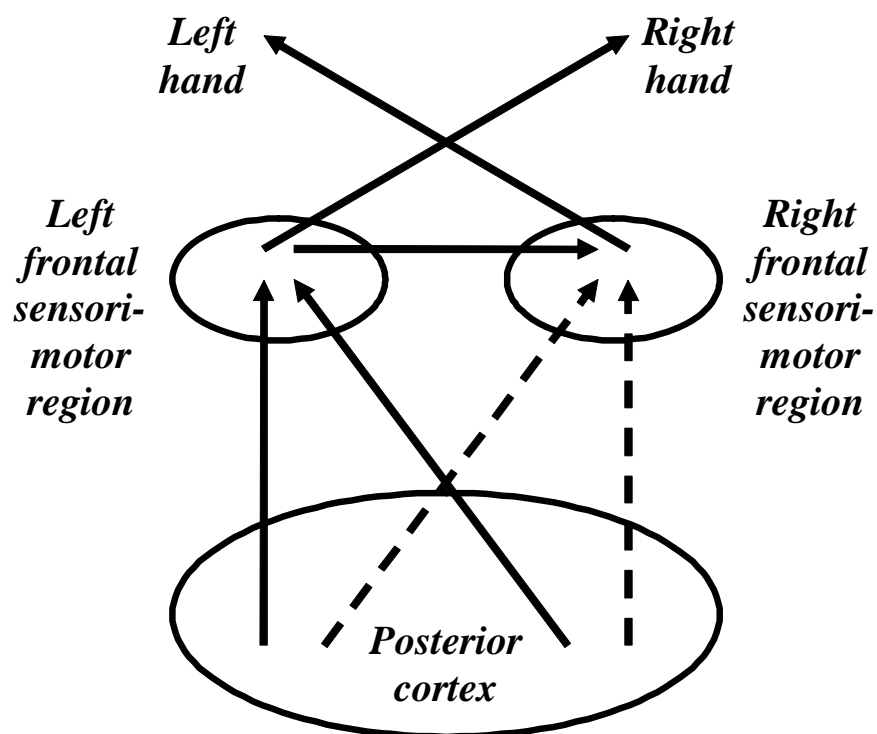

Fig. 1. First synthesis of Liepmann (1908) displaying the mechanism of apraxia. Explanations are given in the text. Adapted from Goldenberg (2009).

First, patients with "Ideational Apraxia" (IA or motor apraxia for Pick) fail to generate movement formulae and, therefore, show impairment in the real and pantomimed use of tools. Patients may however perform normally when provided with the idea of the gesture, such as when asked to carry out movements upon imitation. It was first thought to be a severe form of ideomotor apraxia (see below), but Liepmann confirmed its independence in 1920 and associated this disorder with lesions of the whole posterior cortex and, more particularly, the left posterior regions. Second, in "IdeoMotor Apraxia" (IMA or motor apraxia in the princeps study of 1900), movement formulae are intact but can no longer

guide the innervatory patterns. In short, the patient knows what he has to do, but does not know how to do it. Patients with IMA show predominantly difficulties to perform meaningful gestures on verbal command and on imitation. Actual tool use is partly spared because of the adaptation of movements to external constraints. This form of apraxia was associated with the destruction of fibres connecting posterior brain regions to the frontal sensorimotor regions (callosal, frontal and parietal). Given that these fibres pass below the parietal cortex, Liepmann suggested that deep lesions of the inferior parietal lobe and the supramarginal gyrus may cause IMA (see Goldenberg, 2009). Third, "Motor Apraxia" (melo-kinetic apraxia in the paper of 1908) corresponds with the impairment of innervatory patterns. Contrary to IA and IAM that affect movements of both hands, motor apraxia is unilateral and occurs regardless of the conditions of evaluation (no automatic-voluntary dissociation). Motor apraxia is associated with lesions of frontal sensorimotor regions.

2.2 Geschwind (1965, 1975)

The disconnection approach was partly initiated by the description of the imperial counsellor MT by Liepmann (1900). The problem inherent to this description is that, unlike the patient MT, unilateral non-sympathetic apraxia affects more frequently the left upper limb than the right one. Geschwind (1965) developed his theoretical model of apraxia on this basis. Geschwind and Kaplan (1962) discussed the case of a patient with an extensive infarction of corpus callosum, who was able to write correctly with the right hand but not with the left hand, which did not show an elementary sensorimotor disturbance. Moreover, the patient could perform gestures on verbal command (pantomime) with the right hand but frequently failed to carry them out with the left hand. Importantly, he could imitate the movement made by the examiner as well as use objects correctly with either hand. The hypothesis that the failures of this patient resulted from a general conceptual disturbance was ruled out by the fact that the patient could pretend to use objects with the right hand. Instead, given that the difficulties occurred when the patient was asked to perform actions to command, Geschwind and Kaplan interpreted these disturbances as the effects of disconnection of the right motor cortex from the speech area and suggested that the extensive infarction of the corpus callosum must be regarded as the cause of the symptoms.

Geschwind (1965) stressed that this case did not represent the difficulties which usually accompany left unilateral apraxia. Indeed, impairment is generally not limited to gesture on verbal command and also occurs when patients are requested to imitate or to actually use tools. This is consistent with the first description of the clinical manifestation of extensive disconnection of the corpus callosum by Liepmann and Maas (1907). Their patient, Ochs, had a right hemiplegia. He could not write with his left hand and failed to perform many actions on verbal command and did not improve on imitation. Ochs also frequently mishandled objects placed in his left hand. So, Geschwind (1965, p. 606) emphasized that "the designation of "apraxic" is an inadequate one unless the stimulus conditions are specified…Rather than use the term "apraxia" it is therefore preferable to specify the stimulus-response combinations which fail".

A few years later, Geschwind (1975) proposed a neo-associationist model which diverted from Liepmann's theory in that he replaced the movement formulae by the verbal command which elicits motor actions by using a neural substrate similar to that used by Wernicke to language processing (Figure 2). Sensorial centres are directly linked to motor centres and

apraxia is viewed as an interruption of the translation of sensorial stimulations into motor inputs. The Wernicke area is connected to the left motor association area by the arcuate fasciculus, and the left motor association area is linked to the left primary motor area. When a person is requested to perform a gesture on verbal command with the right hand, this pathway is used. If a person has to use the left hand, information is transferred from the left motor association area to the right motor association area and then to the right primary motor area. For Geschwind, lesions in the region of the arcuate fasciculus and supramarginal gyrus disconnect Wernicke area from motor association areas. So, patients with lesions in this area can comprehend verbal commands but show difficulties in carrying out movements to command with either hand. Gesture-to-command but not imitation requires left hemisphere language processing. So, given that many LBD patients have no right hemisphere lesion, they should be able to imitate, but cannot. To account for this discrepancy, Geschwind suggested that the left arcuate fasciculus is also dominant for visuomotor connections. Finally, callosal lesions produce a unilateral apraxia of the left hand, as described by Geschwind and Kaplan (1962) and Liepmann and Maas (1907).
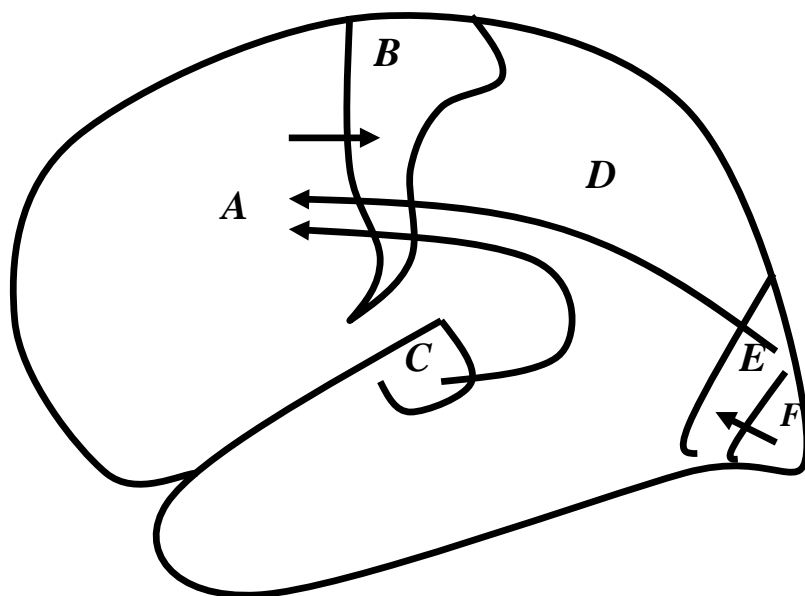

Fig. 2. Neo-associationnist model of Geschwind (1975). A, motor association cortex; B, primary motor cortex; C, Wernicke area; D, arcuate fasciculus; E, visual association cortex; F, visual cortex. Adapted from Geschwind (1975).

Tool use impairments, central to IA, are not directly concerned by this model whose primary focus is IMA. Liepmann and Maas (1907) observed that Ochs failed in some very simple object manipulations, yet could button his clothing blindfolded. They attributed this to the ability of the isolated sensory and motor cortex to do over-learned tasks without the mediation of vision. This observation corroborated previous ones indicating that LBD patients with apraxia are commonly better at demonstrating the real use of tools than at pantomiming. In line with this, Geschwind (1965) suggested that for the use of tools the pathway from primary somesthetic to primary motor cortex may be via association cortex, as in the case of connexions of other modalities to the motor system. Sparing of this

pathway, as it is generally the case in LBD patients, might leave object handling totally unaffected. Given that much knowledge about tools is acquired visually, visuo-motor connections might inhibit somesthetic-motor connections, so that patients may nevertheless show some difficulties to demonstrate the actual use of tools to the sight of tools. But, as Liepmann and Maas (1907) stressed, these difficulties might be considerably reduced if patients are asked to use tools blindfolded.

3. Cognitive models of apraxia

In 1980s, there has been a renewal of interest for the study of apraxia with the emergence of cognitive models which intend to describe the different levels of processing involved in gestural production, leaving aside the issue of neuroanatomical locations. All these models share a common feature inspired from the disconnection approach of Geschwind: There would be several possible routes for producing gestures, and even the same gesture. This is the famous multiple-routes-for-action hypothesis, which diverts from Liepmann for whom there was only one mechanism. Nevertheless, unlike Geschwind, cognitive models do not conceive of apraxic manifestations as resulting from disconnections between sensorial and motor centres. They assume, as Liepmann thought, that there are several processing stages which translate sensorial codes into motor codes. In broad terms, the production of a gesture would be generally based upon conceptual processing (indirect, lexical route). But, the system would be flexible, so that conceptual processing could be bypassed (direct, non-lexical route). We discuss in more detail these models in the following lines.

3.1 Roy and Square (1985)

The model of Roy and Square (1985) is thought to rely on the operation of conceptual and production processes. The conceptual system provides an abstract representation of action relevant to limb praxis. Three types of knowledge are incorporated in this system. The first is *knowledge of objects and tools in terms of the action and functions they serve*. This kind of knowledge may have internalized linguistic referents (knowing that a knife is a piece of cutlery which can be used with a fork to eat). The second is *knowledge of actions independent of tools or objects but into which tools or objects may be incorporated*. This kind of knowledge is decontextualized, that is, associated with any particular object. People can use this practical knowledge about objects based on perceptual attributes to use tools in an unusual way (a shoe would make a good hammer). The third is *knowledge relevant to the seriation of single actions into a sequence*. Impairment of the conceptual system would lead to difficulties in pantomiming the use of tools (IMA) as well as in the actual use of objects (IA). Object substitution (e.g., using a pencil as a comb) would be one of the main manifestations of patients with defective conceptual knowledge.

While the conceptual system encompasses knowledge for action, the production system provides the mechanisms for movements. At one level, action may be directed by generalized programs representing actions and which might be of ecological importance (hammering, stirring). These programs are not specific to any particular unit (hand, foot) but can guide any of these units in the production of the action. Importantly, all of the information relevant to action would not be "in the head". Much information is also "out there" in the environment, thereby suggesting that the environment could also control the

movement through bottom-up processing. Roy and Square (1985) suggested that errors in performing the sequence of movements in the sequence (omission, repetition) as well as clumsiness may be caused by impairment of the production system.

3.2 Rothi, Ochipa, and Heilman (1991)

Geschwind (1965, 1975) was opposed to the idea of specific cerebral areas in which movement formulae are stored. Nevertheless, he never tested this hypothesis. For him, the left arcuate fasciculus is dominant for visuomotor connections. And, given that these fibres pass below the left parietal cortex, deep lesions of the parietal lobe cause IMA. The corollary is that patients with damage to arcuate fasciculus and parietal lobe should encounter difficulties not only to produce gestures but also to recognize the gestures performed by others (only one form of IMA). By contrast, if the connection between the visual association cortex and the motor association cortex is relayed by movement formulae contained in the left parietal lobe, then lesions anterior to this cerebral region should cause impaired gesture production without impairment in gesture recognition.

The study of Heilman, Rothi, and Valenstein (1982) aimed at testing these predictions. They examined 20 LBD patients who were classified into four groups according to the locus of lesion (anterior vs. posterior) and whether or not they were apraxic. Apraxia was assessed by asking patients to perform 15 gestures on verbal command (12 pantomimed acts and 3 meaningful gestures such as hitchhiking). Some patients did not have a CT scan because their cerebral infarctions occurred before the advent of CT. So, they classified the subjects as fluent or non-fluent based on spontaneous speech. Because patients with fluent aphasia have generally posterior lesions and those with non-fluent aphasia have commonly anterior lesions, they used this indicator to determine the locus of lesions. Four groups were thus formed: 1, apraxic patients with anterior lesions/non-fluent aphasia; 2, apraxic patients with posterior lesions/fluent aphasia; 3, non-apraxic patients with anterior lesions/non-fluent aphasia; 4, non-apraxic patients with posterior lesions/fluent aphasia. All the patients were asked to perform a gesture discrimination test consisted of 32 trials, each containing three separate videotaped pantomimed acts. Patients were instructed to discriminate the gesture corresponding to the verbal description of the action. Only one of the three gestures was correct.

The results indicated that apraxic patients with posterior/fluent aphasia (Group 2) performed worse than the three other groups (Groups 1, 3 and 4), thereby suggesting that gesture production can be dissociated from gesture recognition and that there would be two different forms of IMA: The posterior form (impairment in both gesture recognition and gesture production: Group 2) and the anterior form (impairment in gesture production only: Group 1). These findings confirmed the hypothesis of movement formulae, which was reformulated "visuo-kinesthetic motor engrams" by Heilman et al. (1982), as well as the possible involvement of left inferior parietal lobe (supramarginal gyrus and angular gyrus) in the storage of these so-called engrams. In a subsequent study, Rothi, Heilman, and Watson (1985) asked 13 LBD patients to carry out a gesture discrimination test in which the correct gesture had to be associated with the drawing of the object. Besides corroborating the data obtained by Heilman et al. (1982), this study ruled out the possibility that the posterior type of IMA resulted from aphasia since the material used was visual and not verbal.

On the basis of these works as well as other observations of dissociation collected in the neuropsychological literature, Rothi et al. (1991) proposed a cognitive model of apraxia detailing the different processing stages required for gesture production and recognition (Figure 3). The different processing modules are presented in the following lines.
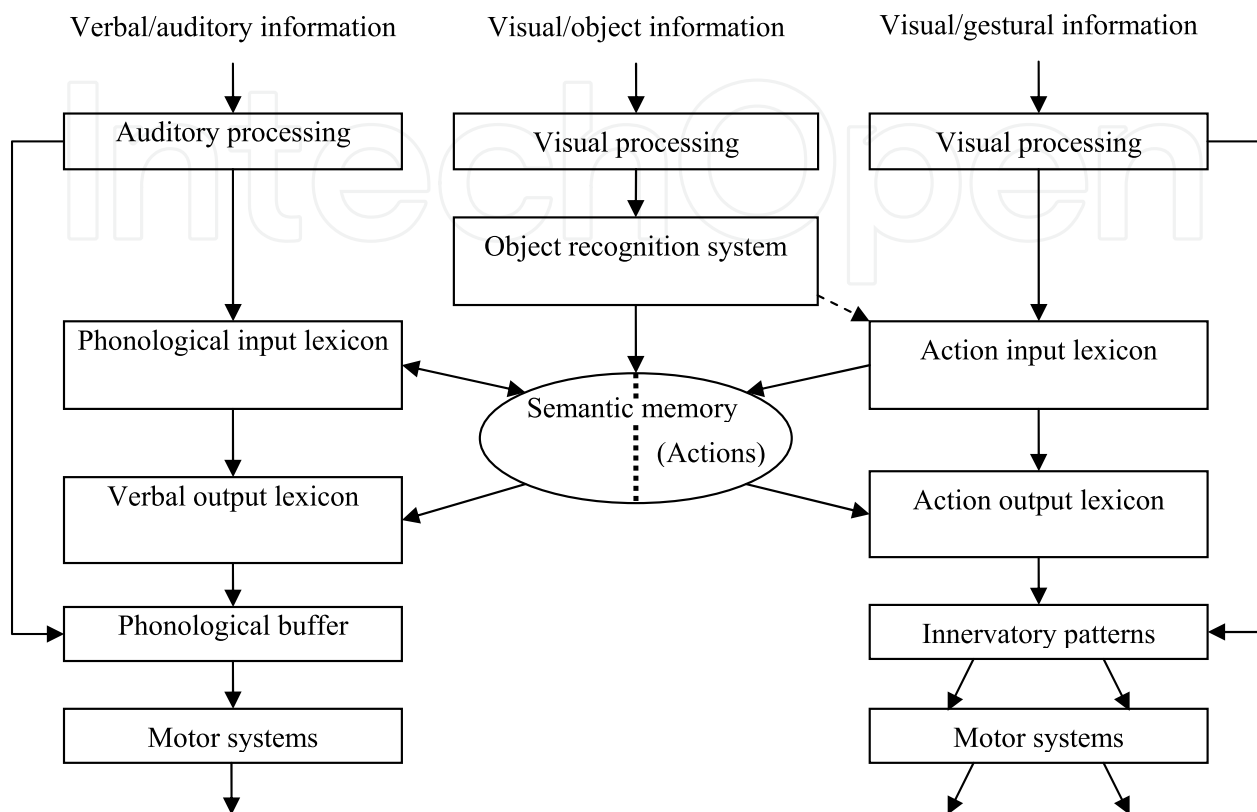

Fig. 3. Cognitive model of apraxia of Rothi et al. (1991). Note that the model is integrated into a larger cognitive architecture thought to explain language deficits. Adapted from Rothi et al. (1991).

The action-lexicon. Rothi et al. (1991) proposed the term "lexicon" to refer to the movement formulae of Liepmann (1908) and the visuokinesthetic motor engrams of Heilman et al. (1982). The model posits that input and output processing of praxis require division of the action-lexicon into an input action-lexicon (devoted to gesture recognition) and an output action-lexicon (devoted to gesture production). This distinction is supported by the fact that some patients are significantly better for recognizing or producing pantomimes on verbal command than for imitating them (Ochipa, Rothi, & Heilman, 1994). Indeed, spared gesture recognition in the presence of impaired imitation may be explained by dysfunction after access to the input action-lexicon. Moreover, given that pantomime production on verbal command is less impaired than pantomime imitation, the model assumes that spoken language might gain access to the output action-lexicon without having to be processed by the input action-lexicon.

Input modality selectivity. Rothi et al. (1991) posited that there would be selective input into the action-lexicons according to modality. This proposal is based, for example, on the observation of some patients who are able to perform gestures to command correctly, but who cannot produce visually-presented gestures (optic apraxia; see Assal & Regli, 1980). On

the basis of these findings as well as other somewhat similar findings, the model suggests the existence of separate input systems for visually presented gestural information (imitation), visually presented objects (pantomime or actual tool use), and auditory presented verbal information (gestures to command).

Non-lexical action processing or direct route. In addition to a lexically based system, the model assumes that there would be a non-lexical action processing system available for the imitation of meaningful (symbolic and transitive) as well as meaningless gestures. This hypothesis is notably supported by the observation of a patient who had a selective deficit of imitation (Mehler, 1987).

Action semantics. The different stages of action processing were mainly isolated from observations of patients with IMA encountering difficulties in pantomime production or imitation. To account for IA, Rothi et al. (1991) stressed that actual tool use is dependent upon the interaction of conceptual knowledge related to tools, objects and actions, what they called action semantics. This proposal is consistent with that of Roy and Square (1985) that tool use is supported by a conceptual system (see above).

Buxbaum (2001)

For Rothi et al. (1991), damage to action lexicons should be accompanied by impairments in gesture production and comprehension and, more generally, in tasks requiring knowledge about tool manipulation. However, knowledge about tool function should not be impaired notably because this kind of knowledge would be supported by action semantics.

Buxbaum and Saffran (2002) examined this prediction with 7 aphasic patients with IMA and 6 aphasic patients without IMA. Apraxia was assessed by asking patients to perform gestures to command and on imitation (15 pantomimes and 5 symbolic gestures). Patients performed a picture-matching task in which they had to select among three tools the two tools that were the more similar to one another. In the manipulation condition, matching had to be done on the basis of similar manipulation (e.g., a typewriter and a piano are tapped with the fingertips). In the function condition, matching had to be done on the basis of similar function (e.g., a radio and a record player are used for listening music). The results indicated that apraxic patients performed worse the manipulation condition than non-apraxic patients. The opposite pattern was obtained for the function manipulation. All of the 7 apraxics and only 1 non-apraxic had lesions of the left frontoparietal cortex. The two groups did not differ in temporal lobe involvement (see also Buxbaum, Veramonti, & Schwartz, 2000b).

On the basis of these findings, Buxbaum (2001) proposed an updated version of Rothi et al.'s (1991) model, consisting of three systems (Figure 4). The first system, the dorsal action system, includes a dynamic representation of the body forming the basis for calculation of several frames of reference centred upon the body parts. Therefore, patients with "dynamic apraxia" are impaired at producing pantomime, but can use tools correctly when they are given in hand. Imitation is also defective. This apraxia could be observed after damage in the dorsal frontoparietal cortex, with involvement of the superior parietal lobe particularly likely (Buxbaum, Giovannetti, & Libon, 2000a; Heilman et al., 1982; Heilman, Rothi, Mack, Feinberg, & Watson, 1986).
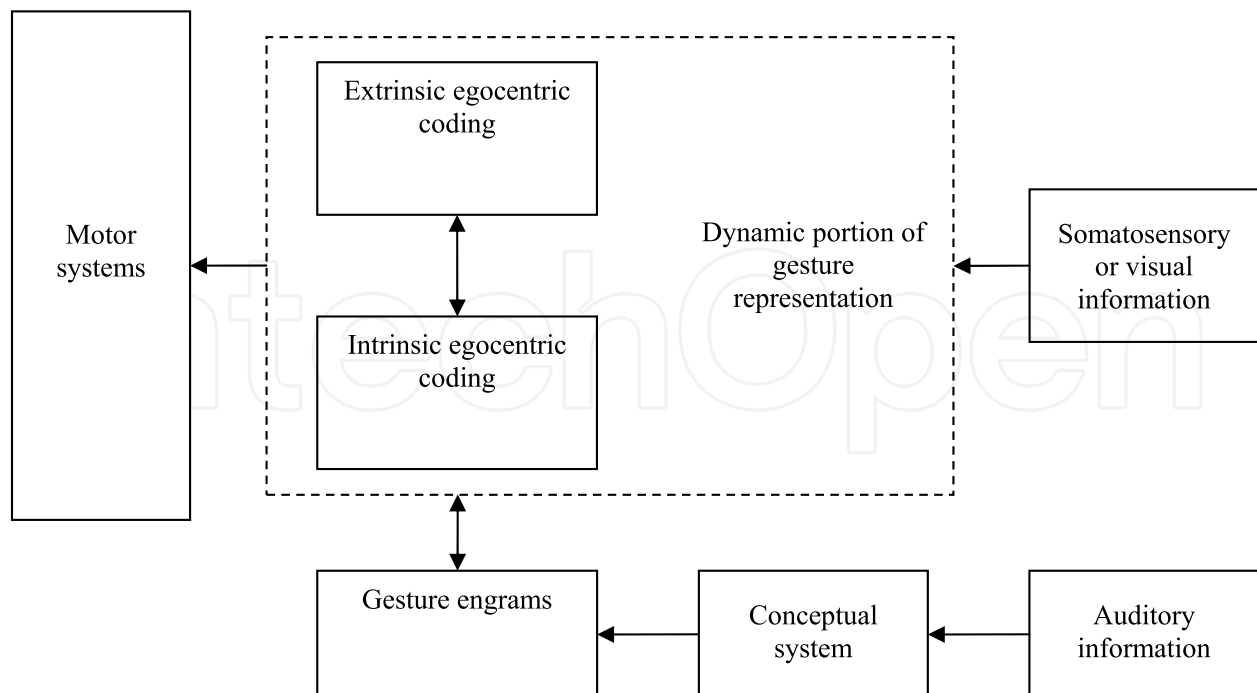
Fig. 4. Model of Buxbaum (2001). Adapted from Buxbaum (2001).

The second system, the ventral system, would include declarative, conceptual knowledge about tool function. Ventral apraxia is accompanied with difficulties with actual tool use, and more particularly in action errors revealing conceptual problems with tool knowledge (misuse, tool substitution). Gesture recognition as well as imitation would be spared. This apraxia could also be revealed by matching tasks involving knowledge about tool function (see above). Temporal lobe lesions would be critical for the occurrence of ventral apraxia (Hodges, Bozeat, Lambon Ralph, Patterson, & Spatt, 2000; Sirigu, Duhamel, & Poncet, 1991).

The third system, the central praxis system, would be specifically involved in pantomime production and recognition as well as matching tasks requiring tool manipulation (see above). This system includes gesture engrams (the action lexicons of Rothi et al., 1991) which are thought as existing at the confluence of the ventral and dorsal streams because they contain representational features (ventral system), which are themselves dependent on dynamic spatiomotor processes (dorsal system). Damage to the left inferior parietal lobe would be critical for representational apraxia (Buxbaum, Kyle, Grossman, & Coslett, 2007; Buxbaum, Kyle, & Menon, 2005; Buxbaum & Saffran, 2002; Heilman et al., 1982). For Buxbaum (2001, p. 452), gesture engrams have to be viewed as containing "the features of gestures which are invariant and critical for distinguishing a given gesture from others. For a hammering movement, for example, a broad oscillation from the elbow joint is critical, as is a clenched hand posture, and these and other similar gestural features are construed as forming the 'core' of the gesture representation".

4. Recent contribution to apraxia

The multiple-routes-for-action hypothesis is central to cognitive models of apraxia. This way of conceiving action is very heuristic by allowing the formulation of a great number of hypotheses to account for patients' difficulties. Unfortunately, this proposal is subject to a

severe limitation in the interpretation of disorders. How, indeed, to demonstrate that a deficit is associated with a single process if it can always be explained by impairment at other levels? By contrast, recent works have indicated that a limited number of processes might be involved in praxis, and that they would not be parallel, but rather orthogonal. In broad terms, each process might have a specific function that could not be supported by another process. These works will be detailed in the following lines.

4.1 The role of conceptual knowledge for actual tool use

Cognitive models based on the multiple-routes-for-action hypothesis generally assign specific information to each form of conceptual knowledge. There is, however, considerable confusion about the precise role of each of these different forms, given that impairment of each of theses forms could be easily compensated. For instance, a patient with impaired conceptual knowledge about tool function may nevertheless maintain the ability to actually use tools by activating gesture engrams (Buxbaum, 2001).

In general, cognitive models distinguish, at a first level, sensorimotor, non-conceptual knowledge, about tool manipulation (i.e., visuokinesthetic motor engrams, action lexicons, gesture engrams) from conceptual/semantic knowledge[1] about tool function (Buxbaum, 2001; Rothi et al., 1991). At a second level, there is also a distinction between conceptual knowledge about the prototypical[2] use of tools (for which purpose, in which context and with which object) and knowledge containing practical information about tools and objects based on perceptual attributes (e.g., a shoe would make a good hammer) (Buxbaum, 2001; Hodges et al., 2000; Rothi et al., 1991; Roy & Square, 1985). Different tasks have been developed to assess the integrity of each of these two forms. As mentioned above, conceptual knowledge about prototypical use can be examined with picture-matching tasks in which subjects are instructed to match a tool with its usual object, its typical location or another tool that can be used to achieve the same purpose (e.g., Bozeat, Lambon Ralph, Patterson, & Hodges, 2002; Goldenberg & Spatt, 2009; Hodges, Spatt, & Patterson, 1999; Hodges et al., 2000; Osiurak et al., 2009). By contrast, practical knowledge can be evaluated by asking patients to solve mechanical problems (Goldenberg & Hagmann, 1998; Goldenberg & Spatt, 2009; Hartmann, Goldenberg, Daumüller, & Hermsdörfer, 2005) or to use familiar tools in a non-conventional way (e.g., screwing a screw with a knife) (Osiurak et al., 2007, 2008, 2009). Note that in line with the multiple-routes-for-action hypothesis, damage to one of these forms of knowledge might be compensated by the other one, and vice versa. For example, a patient showing difficulties in determining the usual function of a hammer (prototypical knowledge) might nevertheless be able to demonstrate how to use it by activating practical knowledge based on perceptual attributes. In other words, the impairment of each of these forms should not necessarily be associated with tool use disorders.

This prediction has however been invalidated by several studies indicating the existence of a strong association between practical knowledge and actual tool use. For example,

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[1] Semantic memory is usually defined as a system that stores and retrieves information about the meaning of words, concepts and facts (Tulving, 1972; Warrington, 1975).

[2] In this chapter, we use the terms "conventional", "usual", "familiar" and "prototypical" interchangeably.

Goldenberg and Hagmann (1998) found a significant correlation in LBD patients between familiar tool use and mechanical problem solving (see also Goldenberg & Spatt, 2009; Hartmann et al., 2005; Silveri & Ciccarelli, 2009). It has also been shown that mechanical problem solving skills are often disrupted in patients with cortico-basal degeneration, who are known to exhibit severe difficulties in activities of daily life involving the use of tools[3] (Hodges et al., 1999; Spatt, Bak, Bozeat, Patterson, & Hodges, 2002). Recently, Osiurak et al. (2009) asked 20 LBD patients, 11 RBD patients and 41 healthy control subjects to perform a familiar tool use task (screwing a screw with a screwdriver) as well as a task requiring the non-conventional use of tools (screwing a screw with a knife). The findings indicated that only LBD patients encountered difficulties in the two tasks as well as a strong correlation between the two tests. While a clear-cut relationship appears to be drawn between actual tool use and practical knowledge, such a relationship has not been observed between actual tool use and prototypical knowledge. Indeed, many studies have shown that brain damage can impair actual tool use and knowledge about the prototypical use of tools (assessed with picture-matching tasks) independently from each other (Bartolo, Daumüller, Della Sala, & Goldenberg, 2007; Bozeat et al., 2002; Buxbaum, Schwartz, & Carew, 1997; Forde & Humphreys, 2000; Goldenberg & Spatt, 2009; Hodges et al., 2000; Lauro-Grotto, Piccini, & Shallice, 1997; Negri et al., 2007; Osiurak et al., 2008, 2009; Silveri & Ciccarelli, 2009).

Taken together, these findings rule out the prediction formulated above that the specific impairment of each form of conceptual knowledge (prototypical versus practical) should not cause tool use disorders. More particularly, these findings provide convincing evidence for the hypothesis that any situations involving the use of tools (actual use of familiar tools, non-conventional use of familiar tools and mechanical problem solving) might require practical but not prototypical knowledge. This conclusion raises the question as to the role of conceptual knowledge about the prototypical use for tool use.

To account for the discrepancy between these results and the predictions derived from the multiple-routes-for-action hypothesis, Buxbaum et al. (1997) suggested that prototypical knowledge may be neither necessary nor sufficient for actual tool use. Such an account is nevertheless delicate since it requires explaining why the human brain would possess knowledge that is not relevant for action. There is another way to interpret these findings, but this implies ruling out the multiple-routes-for-action hypothesis by considering that each form of knowledge has an assigned function. In this way, Osiurak et al. (2008, 2009, 2010, 2011) have suggested that practical knowledge – what they called "technical reasoning" – would be specifically devoted to the formation of representations about the possible physical actions on the world (see also McCloskey, 1983; Penn, Holyoak, & Povinelli, 2008). By contrast, prototypical knowledge, as any semantic knowledge, would enable people to determine the usage of tools, that is, in which context, in which location or with which object a given tool is commonly employed. This second form of knowledge would be particularly useful for adapting oneself to social usages in knowing, for example, that a toothbrush is appropriate for cleaning teeth and not shoes or that Japanese people use sticks and not forks and knives to eat.

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[3] It is noteworthy that we do not refer here to the difficulties observed in actual tool use because of motor apraxia, but those difficulties that are due to conceptual impairment and which appear later in the disease (see below).

In line with this proposal, it can be predicted that patients with a selective semantic deficit and, as a result, impaired prototypical knowledge, should meet difficulties to demonstrate how tools presented in isolation (pantomime, use of tools in isolation) are usually used. Recently, we brought evidence for this prediction by describing the case of the patient MJC who had a severe semantic impairment (matching picture tasks) following closed head injury (Osiurak et al., 2008). This patient was unable to demonstrate the use of tools presented in isolation. Very interestingly, MJC used almost systematically the desk to show the use of tools, as she attempted to bring out mechanical relationships from the desk and the tools. For instance, she used a key to for scrapping the chamfered edge of the wooden desk or a screwdriver as a gimlet, adding that "one can make a hole with it". Nevertheless, MJC performed normally when asked to use tools presented with their corresponding objects and was even able to use tools in a non-conventional way to the extent that these tools were present with a given object (see above). Somewhat similar strategies were also reported by Sirigu et al. (1991) who described a patient (FB) with bitemporal lobe lesions caused by herpetic encephalitis. FB was unable to recognize many familiar tools and objects, but could describe how these tools could be manipulated. For instance, when asked to identify a nail clipper, he said that "it can attach several sheets of paper together. You turn the piece on the top and tip it back…You press and it maintains them" (Sirigu et al., 1991, p. 2566). Recent works on patients with semantic dementia have also shown a strong association between semantic picture matching tasks and the use of isolated tools (Hodges et al., 2000; Silveri & Ciccarelli, 2009).

In sum, these findings reveal that the actual use of tools presented in isolation – and therefore production of pantomimes to command or to the sight of tools – poses serious problems to patients with semantic impairment[4]. Interestingly, these patients tend to employ a compensatory strategy consisting in using the immediate environment in order to bring out possible actions which are, as a result, not necessarily the actions usually carried out with the tools. This can be easily explained by the preservation of practical knowledge. Of course, we are not saying that the human brain would contain semantic, prototypical knowledge whose main purpose would be to enable patients to show how an isolated tool can be used. As discussed above, prototypical knowledge may be particularly relevant for adapting oneself to social situations, but also to retrieve tools that are not immediately present to the senses. After all, knowledge about the fact that nails can generally be found in a workshop enables people to seek the tool necessary for completing the current action in the appropriate place. But, in the clinical context, the involvement of prototypical knowledge might be more pronounced when patients are asked to demonstrate the use of isolated tools. Note that this also requires examining whether the performance improves when the corresponding object is given. If such improvement occurs along with difficulties in semantic picture matching tasks, then the hypothesis of a selective semantic impairment can be reasonably formulated.

The evaluation of semantic disorders we propose here differs from the classical, cognitive approach which focuses on the type of errors committed by patients to specify the nature of the deficit (e.g., content errors would be specific to semantic impairment). However, we think that this way of addressing apraxia is delicate because it is generally very hard to

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[4] Note that the distinction we make here between the use of tools in isolation versus with the corresponding object does not appear in the cognitive models discussed above.

distinguish content errors from other types of errors. Our way of assessing the impairment is simpler and is based on the conjunction of the deficits observed in a patient on several different tasks (Table 1). Moreover, it is noteworthy that semantic impairment could be found more frequently in patients with bilateral or left temporal lobe lesions, that is, in patients with cortical degeneration (semantic dementia or Alzheimer's disease) or after vascular lesions of these areas.

Task	Production system (motor apraxia)	Conceptual system		
		Prototypical knowledge	Practical knowledge	Topographical knowledge about body parts
Sequence of finger movements	Impaired	Normal	Normal	Normal
Functional picture matching	Normal	Impaired	Normal	Normal
Use of isolated tools	Impaired manipulation	Impaired	Impaired	Normal
Use of tools with corresponding objects	Impaired manipulation	Normal	Impaired	Normal
Selection of the correct tools and use of it with a given object	Impaired manipulation	Normal	Impaired	Normal
Mechanical problem solving	Impaired manipulation	Normal	Impaired	Normal
Imitation of meaningless hand postures	Quasi-normal	Normal	Normal	Impaired
Imitation of meaningless finger postures	Impaired	Normal	Normal	Normal

The distinction we make here between production system versus conceptual system is not based on the different cognitive models presented in this chapter since none of them suggests that the conceptual system would contain prototypical knowledge, practical knowledge and topographical knowledge about body parts. Rather, this distinction corresponds to a synthesis of the works presented here.

Table 1. Examination of apraxia

With regard to the neuroanatomical loci of damage associated with impaired practical knowledge, it is interesting to note that many works have pointed out that anomalous tool use is generally present in patients with large bilateral or left fronto-parieto-temporal lobe lesions (Fukutake, 2008; Rumiati, Zanini, Vorano, & Shallice, 2001), even though it is not systematic (Halsband et al., 2000). In this study, five patients with circumscribed lesions of the left parietal cortex did not encounter any difficulty to use tools. In fact, De Renzi and Lucchelli (1988) had already indicated that IA was not systematically present in patients with parietal lobe lesions. More recently, Goldenberg and Hagmann (1998) examined the ability to use tools in LBD patients. They did not find any association between tool use performance and specific brain areas. In a more recent study, Goldenberg and Spatt (2009) asked 38 LBD patients to perform a familiar tool use test as well as a mechanical problem

solving task. The correlational analysis between the scores and the lesional sites indicated that left parietal and frontal, but not temporal areas played a significant role in the two tasks. In total, it appears that the left parietal lobe might store practical knowledge, although more extended lesions of the left hemisphere and even of the right hemisphere could be necessary for the emergence of clinically observable difficulties. So, impaired practical knowledge might be more frequent in diseases causing lesions in these areas (stroke, some degenerative diseases such as the cortico-basal degeneration at advanced stage or Alzheimer's disease).

4.2 Imitation of meaningless gestures

The multiple-routes-for-action hypothesis assumes that there would be a non-lexical, direct route between early visual centres and motor centres. This proposal has been supported by observations of patients with a visuo-imitative apraxia, that is, a specific or at least more pronounced deficit of imitation of meaningless gestures (Mehler, 1987; Ochipa et al., 1994). Several studies have however questioned the existence of this direct route.

Goldenberg (1995) asked 35 LBD patients, 20 RBD patients and 20 healthy subjects to imitate meaningless hand postures as well as to replicate the gestures on a life-sized manikin. All the postures involved the face. The results indicated that LBD patients performed dramatically worse than RBD patients and controls on both tests. In another study, Goldenberg and Hagmann (1997) reported two patients (LK and EN) with left inferior parietal lobe lesions (angular gyrus) who were also severely impaired at imitating these postures as well as replicating on the manikin. On the basis of these findings, Goldenberg and Hagmann (1997; see also Goldenberg, 1997) suggested that imitation of meaningless hand postures is not based on a direct route between perception and action, but might be rather mediated by general topographical knowledge about the human body.

Goldenberg and Hagmann (1997) also developed a task of imitation of meaningless finger postures and showed that EN but not LK failed this test. This distinction was quite surprising given that until this work no neuropsychologist had focused on a possible distinction between imitation of hand versus finger postures. In fact, the evaluation was generally based on a composite score obtained from the two conditions (e.g., De Renzi, Motti, & Nichelli, 1980). Goldenberg (1999) examined 26 LBD patients, 21 RBD patients and 17 healthy subjects on imitation as well as matching of meaningless hand and finger postures. He found that LBD patients performed worse than RBD patients for hand posture imitation and matching, whereas the opposite pattern was observed for finger postures. So, he concluded that imitation and matching of hand postures would be based on topographical knowledge about body parts and would involve the left parietal lobe. Indeed, given that the hand is in contact with the face, the patient does not see the posture he/she is carrying out. So, the ability to execute such gestures needs knowledge about body parts and notably the face (knowing that the extremity of the hand must be in contact with the nose implies being able to distinguish the nose from other parts of the face). By contrast, imitation and matching of finger postures would be based on visuo-spatial skills supported by the right posterior cerebral hemisphere. Indeed, the reproduction of finger postures requires visual guidance in order to represent the position of each finger relative to the others. The involvement of topographical knowledge about body parts is here largely limited. These conclusions were corroborated by more recent neuroimaging studies conducted in healthy subjects (Hermsdörfer et al., 2001; see also Goldenberg, Laimgruber, & Hermsdörfer, 2001).

Besides challenging the relevance of the multiple-routes-for-action hypothesis and notably the existence of the non-lexical, direct route, these findings more generally challenged the interest of proposing a unique cognitive model of apraxia accounting for tool use and imitation impairment. Note also that the works by Goldenberg, as those mentioned above about the distinction between the use of tools in isolation versus with the corresponding object, shed a new light on the evaluation of apraxia, in stressing that even if situations may appear quite similar, subtle modifications in the material used can lead to observe very different disorders. Consequently, the distinction demonstrated by Goldenberg between finger versus hand postures must be taken into consideration in the clinical assessment of apraxia (Table 1). Finally, it is noteworthy that the association observed by Goldenberg between imitation of meaningless hand postures and the left inferior parietal lobe is inconsistent with the cognitive models discussed above. Indeed, in those models, the left inferior parietal region is thought to contain gesture engrams. Yet, other studies have confirmed the relationship obtained by Goldenberg. For instance, Haaland, Harrington, and Knight (2000) found that patients who were impaired at imitating meaningless hand postures generally had left inferior parietal lobe lesions (supramarginal gyrus and angular gyrus). Moreover, they also reported that errors concerning the position of the hand relative to the face were only present in patients with parietal lobe lesions whereas the errors concerning the position of the fingers relative to the others were rather present in patients with frontal damage and in only 60% of the patients with parietal lobe lesions (see also Goldenberg & Karnath, 2006).

4.3 Pantomime production, actual tool use and gesture engrams

The cognitive models discussed above, with the exception of Roy and Square's (1985), suggest that knowledge about tool manipulation are supported by gesture engrams. Quite surprisingly, damage to these engrams would not cause significant difficulties in actual tool use, because physical constraints inherent to tools and objects could be sufficient to guide the utilization. The corollary is that damage to gesture engrams mainly affects the production of pantomimes. If it can be thought that the clinical tasks, even if they are often not ecological, may be relevant for revealing impairment, it can nevertheless seem somewhat surprising to consider that the major role of these representations would be to support pantomime production, namely, a situation people meet very rarely in real life. In order to solve this theoretical curiosity, it has been suggested that damage to gesture engrams might cause subtle manipulation errors during actual tool use and that these errors might occur conjointly with the errors observed in pantomime production (Clark et al., 1994; Rothi et al., 1991). In line with this prediction, Clark et al. (1994; see also Poizner, Clark, Merians, & Macauley, 1995) asked 3 apraxic LBD patients to carry out the action "slicing bread" in 4 conditions: No cues (verbal command only), object present (bread), tool present (knife) and both object and tool present (bread and knife). Gestures were recorded and submitted to kinematic analyses. Results indicated that patients showed disturbances in planning the movement of the hand in space across the 4 conditions. These findings were thought as providing evidence for the existence of gesture engrams.

These results are however to be considered with caution because of several methodological limitations. For example, the size of the patient sample was relatively weak and could not allow the use of statistical correlations between the errors observed in the 4 conditions. More

recently, Hermsdörfer, Hentze, and Goldenberg. (2006; see also Goldenberg, Hentze, & Hermsdörfer, 2004) replicated this experiment by asking 9 apraxic LBD patients to carry out the action "sawing a piece of wood" in 3 conditions: Pantomime (visual presentation of the saw), pantomime with a bar shaped like the handle of the saw, and actual sawing. Gestures were recorded and submitted to kinematic analyses. Results revealed that, patients executed large proportions of their pantomiming movements in an incorrect direction away from the appropriate anteroposterior direction. The availability of the handle-like bar did not improve performance. During actual use, patients moved with moderately decreased velocity. However, this deficit was not related to the errors in movement direction characteristic of pantomiming, suggesting that pantomime and actual tool use are dictated by different requirements and constraints. In broad terms, unlike Clark et al. (1994), manipulation errors during actual tool use do not occur conjointly with the errors observed in pantomime production.

On the basis of these findings, it appears that the gesture engram hypothesis fails to explain how people use tools or produce pantomimes. Again, that these models primarily focus on the ability to perform pantomimes may appear somewhat surprising as people carry out such actions quite occasionally in real life as compared to actual tool use. In fact, in line with the distinction made above between prototypical versus practical knowledge, another interpretation can be offered with regard to pantomime production. The demonstration by pantomime, like any other tool use situation, requires technical reasoning (see above) to create a representation of the action (the use of the tool). Unlike actual tool use, however, people have to carry out actions, while some of the components (the tool and/or the object) are not available to the senses. So, in a way similar to the use of isolated tools (see above), people have to form representations of these components from semantic/conceptual memory, putting high demands on storage and elaboration because the performance can be guided and controlled only with reference to these representations (Goldenberg, Hartmann, & Schlott, 2003; Roy & Hall, 1992). Moreover, in the context of pantomime, after the representation of the use is produced, it is still necessary to keep it in mind to convert into a pantomime the shape, movement and position of the acting hand via an affordance-perception process (Bartolo, Cubelli, Della Sala, & Drei, 2003; Goldenberg et al., 2003; Roy & Hall, 1992). In short, the demonstration by pantomime would be a non-routine, creative task, consisting in the creation and the temporary storage of a representation of the use which then guides the movement. Note also that in line with the gesture engram hypothesis, disturbance in pantomime production should be accompanied by lesions of the left inferior parietal cortex. However, it has been demonstrated that it remains relatively hard to specify precise brain localisations associated with the performance of pantomime production (Alexander, Baker, Naeser, Kaplan, & Palumbo, 1992; Goldenberg, 2003; Goldenberg et al., 2003; see also Goldenberg, 2009).

To sum up, performance in pantomime tasks remain hard to interpret because of the involvement of a high number of cognitive processes. This may explain why pantomime production is frequently impaired after lesions in various areas of the left hemisphere (Goldenberg, 2003). Moreover, even if the use of three-dimensional motion recording systems or the development of procedure based on video-recording and multiple-judge techniques have allowed neuropsychologists to improve the measurement of movements in an objective way, these procedures remain very far from the reality of clinicians who

evaluate patients' performance on the basis of their own experience and intuition (Le Gall, 1992: Poeck, 1986). So, we advise employing pantomime tasks with caution and parsimony and we favour the use of other tasks involving the actual use of tools or imitation of meaningless postures.

4.4 The independance of motor apraxia

So far, we have focused on the conceptual facet of apraxic manifestations. In this section, we put an emphasis on "motor apraxia" also called "melo-kinetic apraxia" or "limb-kinetic apraxia", corresponding with the other facet of the syndrome (production).

Kleist (1907) was the first to describe the loss of hand and finger dexterity resulting from inability to isolate individual innervation. This lack of dexterity is generally confined to hand and finger movements contralateral to the lesion, regardless of the hemisphere which is damaged. The deficit can be distinguished from paresis because of preservation of power and sensation. Nevertheless, movements are awkward. Fruitless attempts commonly precede erroneous movements, which are frequently contaminated by extraneous movements. Importantly, the deficit is consistent, showing the same degree in activities of daily life. In other words, there is no voluntary-automatic dissociation. For Kleist (1907), this disorder reveals damage to innervatory patterns acquired with experience. He called this deficit "innervatory apraxia".

Denny-Brown (1958) provided an original way of conceiving of motor apraxia in distinguishing "frontal, magnetic apraxia" from "parietal, repellent apraxia". The first type is characterised by prominence and persistence of instinctive grasping of the hands, the mouth and even the feet when they make contact or even merely when they are close to any object. Object manipulation can be impaired because patients do not open the hand wide enough when they take hold of an object. The magnetic, exploratory aspect of this behaviour in relation to the environment would be managed by the parietal cortex, released by frontal and temporal lobe lesions. The second type is characterized by avoiding reaction and levitation of the extremities. Similarly, object manipulation may be impaired because of overextension when patients attempt to pick up of grasp objects. The repellent bias to behaviour would be determined by a strip of cerebral cortex in the premotor region and released by parietal lobe damage. These two types of apraxia are unilateral and can generate problems in bimanual activities or in tasks requiring the coordination of the two "hemi-bodies". Denny-Brown stressed the functional independence of these forms of apraxia from conceptual skills. He reported, for instance, the observation of a patient unable to correctly grasp a pair of scissors, but could cut up a piece of paper if it was helped to correctly grasp the pair of scissors.

Interestingly, this observation points out the severe difficulties that patients with motor apraxia can encounter when they have to carry out distal movements involving a precise positioning of the fingers. In another study, Sirigu et al. (1995) also reported disturbances in the ability to adequately grasp tools to actually use them in a patient with bilateral superior parietal lobe lesions. Nevertheless, when the examiner helped this patient to correctly grasp the tool, the demonstration was performed properly.

Motor apraxia has received a resurgence of interest in recent years with the study of patients with cortico-basal degeneration. This disease is characterized by an akinetic-rigid syndrome

accompanied by asymmetric, lateralizing cortical signs including alien limb behaviour, sensory loss and apraxia (Gibb, Luthert, & Marsden, 1989; see also Zadikoff & Lang, 2005). Imaging studies have revealed the presence of frontoparietal cortical atrophy, which is most notable contralateral to the most severely affected side. Two types of apraxia are present in patients with cortico-basal degeneration: Motor apraxia and IMA. Some patients can also show signs of IA, but this kind of apraxia would appear later in the disease (Gibb et al., 1989; Leiguarda, Lees, Merello, Starkstein, & Marsden, 1994). While a significant number of patients have a bilateral IMA (pantomime to command or on imitation; see Zadikoff & Lang, 2005), IMA is generally unilateral at early stages of the disease. This questions the independence of IMA from motor apraxia given that this latter is specifically unilateral.

As Kleist (1907) suggested, the severity of motor apraxia is more pronounced in distal than proximal movements, and can be readily observed in tasks requiring the coordination of finger movements in actual tool use or imitation of gestures (Leiguarda & Marsden, 2000; Zadikoff & Lang, 2005). One effective way of assessing motor apraxia is to ask patients to carry out sequence of finger movements (1-4-2-4). Nevertheless, this task as any other task is certainly not supported only on the production system and can also be failed, for example, by patients with visuo-spatial deficits (see above). So, the best way to determine the presence of motor apraxia remains to examine whether impairment is constant across the tasks. For example, in order to isolate motor apraxia from other disorders during tool use, we advise first examining whether the patient does not have impaired practical knowledge by asking him/her first to select among several tools (hammer, key, saw) the appropriate one to be used with a given object (nail). If the choice is correct, then this implies that the patient is still able to correctly reason about the physical properties of tools and objects and, as a result, to form an appropriate representation of the action. And, if the patient shows severe difficulties to manipulate the tool to show its use with the object, then it is very likely that the patient has a motor apraxia. In sum, we agree with Foix (1916) who challenged the idea that tool use impairment is based on damage to motor representations. For him, IA was due to a general intellectual deficit and motor apraxia was the only form of apraxia. For us, IA was rather due to the inability to do technical reasoning (see above).

5. Conclusion

As mentioned above, the study of apraxia has been subject to intense debate, particularly with regard to its independence from other cognitive processes. The emergence of cognitive models has contributed to the idea that damage to sensorimotor representations (gesture engrams) would be central to apraxia and particularly IMA. Recent findings have however shed a new light on this issue, suggesting that the debate is far from being resolved. As Foix (1916) and Morlass (1928) thought, there might be only one form of apraxia, that is, motor apraxia, the other types of apraxia being nothing else than the manifestations of conceptual disorders in gesture production. After all, when a patient fails an episodic memory test by pointing the wrong word, this gestural error is not characterized as apraxic. Likewise, a patient who is impaired at performing the Tower of London Test may also be considered as expressing manipulation errors. Yet, nobody would consider that this patient is apraxic. So, the question remains to understand what the arguments are leading neuropsychologists to still think that the difficulties observed in actual tool use is necessarily due to damage to sensorimotor representations.

The framework we offer here allows renewing the assessment of disorders which are generally viewed as apraxia. We have given examples concerning the evaluation of practical versus prototypical knowledge. Importantly, the evaluation we advise lies in the principle that it is necessary to escape from the assessment of the quality of movements per se, and rather to view disorders as the manifestation of a deficit that is constant across the tasks. This is not to say that the observation of the gestures executed by patients do not provide any information concerning the disease. If the difficulties are indeed constant across several gestural tasks, then it is very likely that the patient has a motor apraxia (Table 1). But, expected from motor apraxia, the focus must not be placed on the movement executed but rather on the nature of the task which is failed (actual tool use, imitation).

6. Acknowledgments

7. References

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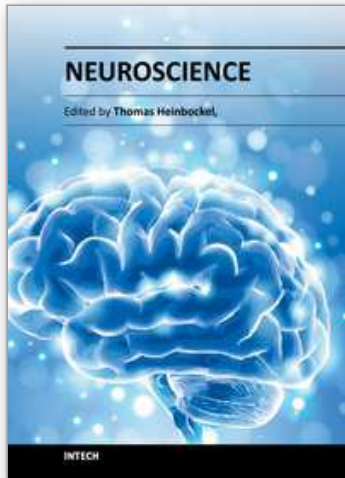
Neuroscience

Edited by Dr. Thomas Heinbockel

If one asks what neuroscience is, the answer can be found in this book. Neuroscience embraces not only anatomical and physiological studies but also cell biology, computer science, and biochemistry. Equally important for neuroscientific research are other disciplines, such as psychology, psychiatry, neurology and additional recent ones, such as neuroeconomics and social neuroscience. This book comprises chapters on diverse topics in neuroscience ranging from cellular, computational, cognitive, and clinical neuroscience. Individual chapters focus on recent advances in specific areas including social neuroscience, which is a relatively new field that studies the neural basis of social interactions. Other chapters focus on technological developments such as optical tools to study the function of the brain. All chapters represent recent contributions to the rapidly developing field of neuroscience and illustrate the range of research conducted under the umbrella of the truly interdisciplinary neurosciences.

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