Modeling the Neural Correlates of Imitation from a Neuropsychological Perspective

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Abstract

Imitation is a fundamental mechanism by which humans learn and understand the actions of others. This thesis addresses the low-level neural mechanisms underlying the imitation of meaningless gestures, using tools from computational neuroscience. We investigate how the human brain perceives these gestures and translates them into appropriate motor commands. In addition, we take a relatively unexplored neuropsychological perspective, which looks at imitation following a brain lesion. The analysis of how imitation breaks down in apraxia, a complex disorder of voluntary movement, enables us to reverse engineer brain function through the identification of those building blocks that are preserved.

To better understand the phenomenon of apraxia, we develop a neurocomputational model of imitation that proposes potential neuroanatomical correlates, such as the flow of information across the two brain hemispheres. The model accounts for the pattern of errors observed in apraxic patients with disconnected brain hemispheres. To validate the predictions of our model, we further analyze the experimental errors and uncover a goal-dissociation, where a goal is defined as the spatial relation between two body parts. The experimental observations suggest that the imitation deficit in apraxia arises from an incorrect coordination between the reproductions of multiple goals. A prediction of this hypothesis was validated on three apraxic patients.

The collected body of kinematic and neuropsychological data allowed us to refine our neurocomputational model of imitation, and to propose a biologically plausible mathematical model for the execution stage of the imitation. The model controls movement by following nonlinear dynamics, and precisely reproduces both the spatial and temporal aspects of unconstrained and natural three-dimensional reaching movements. Importantly, the model is stable and robust against external perturbations.

Overall, our computational models and neuropsychological experiments contribute to a better understanding of how the brain performs the imitation of meaningless gestures; that is, by first decomposing the gesture into imitation goals, and then reproducing these goals through the association of different sensory modalities.

Keywords: computational neuroscience, nonlinear dynamical systems, imitation of meaningless gestures, apraxia, brain lesion, human motor control,

reaching movements.

Résumé

L'imitation est un mécanisme fondamental qui permet à l'homme d'apprendre et comprendre les actions d'autrui. Cette thèse étudie les mécanismes neuronaux de bas niveau qui sous-tendent *l'imitation de gestes sans signification*, au moyen d'outils issus des neurosciences computationnelles. Nous nous interrogeons sur la manière dont le cerveau humain perçoit ces gestes et les traduit en des commandes motrices appropriées. Cela d'une perspective neuropsychologique relativement inexplorée, qui observe l'imitation suite à une lésion du cerveau. L'analyse de la manière dont l'imitation échoue dans les cas d'apraxie -une défaillance complexe du mouvement volontaire- nous permet de réaliser une rétro-ingénierie des fonctions du cerveau, grâce à l'identification des modules fondamentaux qui ont été préservés.

Pour mieux comprendre le phénomène de l'apraxie, nous développons un modèle neurocomputationnel de l'imitation qui propose des corrélats neuroanatomiques potentiels, tels que le flux d'information entre les deux hémisphères du cerveau. Le modèle rend compte du type d'erreurs observées chez des patients aux deux hémisphèes du cerveau déconnectées. Pour valider les prédictions de notre modèles, nous analysons ensuite les erreurs expérimentales et révélons une dissociation d'objectifs, au cours duquel un objectif est défini comme un rapport spatial entre deux parties du corps. Les observations expérimentales suggèrent que le déficit d'imitation dans l'apraxie (très peu compris) provient d'une mauvaise coordination entre la reproduction des différents objectifs. Une prédiction de cette hypothèse a été validée sur trois patients apraxiques.

L'ensemble des données cinématiques et neuropsychologiques réunies nous a permis d'affiner notre modèle neurocomputationel d'imitation et de proposer un modèle mathématique pour la partie qui concerne la réalisation de l'imitation. Le modèle contrôle le mouvement en suivant une dynamique non-linéaire et reproduit précisément à la fois les aspects temporels et spatiaux de mouvements d'atteinte tridimensionnels sans contrainte et naturels. Il est important de noter que le modèle reste stable et robuste face aux perturbations extérieures.

Dans l'ensemble, nos modèles computationnels et nos expériences neuropsychologiques contribuent à une meilleure compréhension de la manière dont le cerveau imite des gestes sans signification; d'abord en décomposant le mouvement en objectifs d'imitation et ensuite en les reproduisant à travers l'association

de différentes modalités sensorielles.

Mots Clés: neurosciences computationnelles, systèmes non-linéaires dynamiques, imitation de gestes sans signification, apraxie, lésion cérébrale, contrôle moteur humain, mouvements d'atteinte.

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Chapter 1

Introduction

A man may be attracted to science for all sorts of reasons. Among them are the desire to be useful, the excitement of exploring new territory, the hope of finding order, and the drive to test established knowledge. [...] Though the result is occasional frustration, there is good reason why motives like this should first attract him and then lead him on.

The Structure of Scientific Revolutions. Thomas S. Kuhn.

1.1 Motivations

Even before the eyes open, your hand reaches to find the alarm clock and embrace your loved one. And this is only the beginning of a day in which your hand will faithfully execute thousands of movements in a remarkably efficient way. You don't even have to think about the movements you make, they come in a very natural and primarily automatized way. But if you do think about it, you would find the mechanisms and principles underlying human reaching only deceptively simple. In order to control human motion, the brain performs a highly complex task: coordinate the activity of all the muscles in your body. The function and constraints optimized by the brain are extremely difficult to grasp, as they stem from multiple sources and have been optimized since the day you were born. Accordingly, many optimization functions have been proposed by the field of human motor control, from minimizing the smoothness of the trajectory, work load, torques to muscle tension. The difficulty to find an appropriate optimization function is confirmed by the problems met by roboticists while controlling the arms of a humanoid robot. These topics relate to the problem of *how the Central Nervous System (CNS) controls human motion.*

Furthermore, a remarkable feature about the human brain is that in addition to controlling movement execution, it is able to relate its own movements to the movements of others. The establishment of such a correspondence function between the motor outputs of the self and others has boosted human evolution,

since the capacity to understand and reproduce the motor acts of others is at the core of the ability to learn from others. Learning from imitation is a fundamental cognitive mechanism for the transmission of knowledge and skills, observed as early as in infancy. While primates can imitate the *goal* of an action, only humans are capable of imitating the *means* of an action. The imitatee and imitator usually have different physical embodiments, which results in an ambiguous correspondence between their actions. Moreover, dissimilarities due to the viewing perspective need to be overcome through transformations of frames of reference. These topics relate to the *neural and computational principles underlying imitation*.

The first part of this thesis addresses the identification and modeling of the neural principles and computational mechanisms underlying visuo-motor imitation. The second part of the thesis provides insights on how the Central Nervous System (CNS) controls human motion, in particular on how the imitation is executed within the context of reaching a specific body posture.

1.2 Approach

To investigate these questions, we strive to use a broad base of techniques and scientific tools, such that we are not limited to a particular technique or tool. We thus follow both an analytic and a synthetic approaches.

The analytic approach consists of collecting *kinematic and behavioral* data of a human that executes a visuo-motor imitation task, and exhibits either normal or defective behavior. In particular, we study how the motor act breaks down in the case of a subject that suffers from a brain lesion deficit such as *apraxia*. Apraxia is a disorder of the high-level control of voluntary movement, not due to basic sensory and motor deficits, that often causes faulty imitation. We attempt to use apraxia so as to reverse engineer the neural processes underlying visuo-motor imitation. In addition to brain lesion studies, our analytic approach relies on observations that stem from other neuroscience fields such as neuroimaging and neurophysiology. Each of these fields sheds light on different but equally important and complimentary aspects of neural imitation, as will be shown below.

While our analytic approach outlines the characterizing principles of neural imitation, our synthetic approach aims to use these principles to construct an artificial system that reproduces the observed behavior. A system built from scratch, that behaves as the original system, can have a very strong explanatory power. However, as there are many systems that can be built to behave as the original system, the model needs to make predictions that can be validated with additional data. Whenever possible, we will propose experiments that can confirm or reject our models. In our synthetic approach, we use *computational modeling* and *simulation methods* grounded in the fields of *dynamical systems*

and *machine learning*. To be more specific we use biologically-plausible machine learning techniques, namely artificial neural networks. To actually implement the models in simulation presents many advantages over cognitive box diagram models. On the one hand, it enforces a greater precision, as computational mechanisms are detailed and not hidden behind labeled black-boxes. On the other hand, simulated models tend to be more complete than diagram models: usually if an important aspect of the real system is neglected, the model will simply not work. Nevertheless, computational models present some drawbacks: it may be difficult to choose the appropriate level at which to model (e.g. behavioral, neurophysiological) and constructing a good model is actually hard, even speculative when one does not know enough facts about the modeled system.

We merge our analytic and synthetic approaches in the following way. The architecture of our neural model is inspired by findings from neuroimaging that identify the brain areas involved in imitation, and the flow of information across these brain areas. For the implementation details of our neural models, we use evidence from neurophysiology. Finally, kinematic and behavioral data of normal and impaired imitation is used to decipher the underlying neural principles, and subsequently to validate the models. In this thesis, we aim towards a multidisciplinary approach and integrative neuroscience, as we believe that the only way to understand the human brain is by tackling it from multiple possible directions.

1.3 Open Questions

We first need to define the type of imitation we are addressing. We are mainly interested in *meaningless imitation*, i.e. reproduction of arbitrary postures of the body (e.g. postures of the hand relative to the head), as opposed to meaningful imitation (e.g. a gesture for hitchhiking, lighting a candle). In tasks of meaningless imitation the subject is asked to imitate the *means* of an action, whereas in tasks of meaningful imitation he is asked to imitate the *goal* of an action. When humans imitate, they concentrate on reproducing the goal rather than the means. However, if the action has no apparent goal, the reproduction of purely intransitive or meaningless gestures is more accurate. Interestingly, ethological studies show that the imitative strategies adopted by monkeys are strictly goal-directed, indicating that humans are endowed with a more evolved cortical structure for imitation that is capable of processing intransitive actions. Accordingly, apraxia studies have provided strong evidence that different neural processes underly these two imitation mechanisms. We focus on meaningless imitation as it is the generic process that enables us to imitate and learn an action, before a semantic label is attached to the action. Furthermore, we believe that modeling semantics at least for now belongs to the philosophical rather than the scientific domain.

We will try to address the following three key questions:

Brain Pathways of Imitation:

- **What** information subserves the human ability to imitate, or in mathematical terms, what are the variables processed (e.g. distance between parts of the body, orientation of body segments)?
- **How** is this information represented and processed in the brain in terms of frames of reference, dimensionality and transformations? How is a visual stimulus to imitate translated into appropriate muscle activations?
- **Where** in the brain is this information represented and processed? What are the brain areas involved in imitation and how does the information flow across these areas?

The neuropsychological and modeling investigations of these fundamental questions further open several very interesting related questions:

Apraxia:

- Is the imitation deficit in apraxia characterizable? Apraxia is still an unexplained brain disorder of voluntary movement, defined only by the absence of purely sensory and motor deficits. Understanding apraxia is crucial for the devise of efficient rehabilitation therapies that could help brain lesion patients.
- In the case of a characterizable imitation deficit in apraxia, can we characterize this deficit by looking at the structure of apraxic errors?

Computational motor control:

- Can we devise a biologically-plausible controller that accounts for the kinematics of natural 3-dimensional reaching movements? Very few existing controllers address unconstrained human movements, i.e. movements that are not a straight line or in a plane. The majority of these controllers are not able to adapt to perturbations during the movement, as they usually preplan the trajectory.
- At what level does the Central Nervous System (CNS) control hand movements? Motor control would be simpler in an intrinsic space (e.g. joint or muscle space), but would render interaction with the extrinsic environment extremely

difficult. For example, the desired trajectory and external perturbations (e.g. obstacles in the environment) have an univocal representation in the extrinsic task space (e.g. Cartesian space) but many possible expressions in the intrinsic spaces.

1.4 Overview of the Dissertation

This thesis collects a number of papers published in relevant scientific journals and peer-reviewed conferences. This format was preferred to a more traditional presentation, as the publications contribute to distinct non-overlapping interconnected areas.

Chapter 2: Apraxia: A Review

We start in Chapter 2 by presenting a thorough and extensive review on apraxia, a disorder of the high-level control of voluntary movement following a brain lesion (i.e. not due to basic sensory and motor deficits). Specifically, we focus on the fact that some apraxic patients show patterns of impaired imitation, which we find to be of particular interest and will provide crucial information for our modeling studies.

As apraxia is a very broad and still poorly understood disorder, the chapter is divided as follows. We start by presenting the current taxonomies and relevant cognitive and neuropsychological models. We then address the neuroanatomical correlates of apraxia, its relation to aphasia (a language disorder) and the current techniques for the analysis of apraxic errors. Finally we speculate on the reasons that may explain why the investigation of apraxia has encountered so many difficulties. We suggest that a more systematic investigation of the apraxic errors and computational modeling may be the solution needed to understand apraxia.

Chapter 3: A Neurocomputational Model of an Imitation Deficit following a Brain Lesion

From our review of apraxia in Chapter 2, we concentrate on a fascinating seminal study of the imitation of meaningless gestures, following a callosal brain lesion that disconnects the brain hemispheres. This neuropsychological case study is reported on in Goldenberg, Laimgruber & Hermsdörfer (Neuropsychologia, 30:1432-1443, 2001). The patient's imitation of hand postures relative to the head was preserved when the task required only

the left hemisphere, but was severely impaired when the task recruited the right hemisphere. To better understand the deficit underlying this patient, in Chapter 3 we propose a simple neurocomputational model of visuo-motor imitation that hypothesizes a brain pathway for the neural information flow and accounts for the scores found in the clinical examination reported.

The model's functional architecture is grounded in brain imaging studies and the model's implementation details take inspiration from monkey brain neurophysiological studies. Specifically, the model consists of self-organizing maps that process sensory information. Imitation is achieved by associating the activities of these sensory maps through supervised learning, i.e. by learning the correspondence function between the visual stimulus, proprioceptive and tactile information. The assumptions we make on the flow of information across the two brain hemispheres, on the sensory and motor representations involved and on the underlying deficit (i.e. uncertainty in the transfer of information between hemispheric-lateralized neural networks) is able to explain the pattern of errors observed. The model predicts inhomogeneities in the precision and time required to process the imitation task when related to different parts of the face, as well as a correlation between the processing time and severity of the lesion. It also predicts different errors depending on the frames of reference underlying the sensory and motor information used.

Chapter 4: Revisiting callosal apraxia: the right hemisphere can imitate the orientation but not the position of the hand

The validation of the model predictions proposed in Chapter 3 requires specific information about the precise errors, additional to their synthetic description, i.e. the success rates provided in Goldenberg et al. (2001). For this reason we contacted the authors and requested access to the experiment's original data. In Chapter 4 we decompose the visual stimulus into a set of geometric variables to reproduce, and re-analyze the patient's errors with respect to these geometric variables.

The results uncover an important goal dissociation, where the posture of the hand in extrinsic coordinates is replicated correctly by both hemispheres, but the position of contact between the hand and face is reproduced solely by the left hemisphere. This finding speaks against the hypothesis of a direct route from perception to motor execution and blurs the frontier between meaningless and goal-directed imitation. To be more specific, it

shows that meaningless gestures with no apparent goal can be reduced to the reproduction of several spatial goals, such as the orientation of the hand or the hand's position of contact with the face. By extracting the distinctive features of a movement and reconstructing them on the new body, this strategy would effectively overcome the correspondence problem, e.g. problems related to the differences in size, shape and perspective between the demonstrator and imitator (Nehaniv & Dautenhahn, 2001; Alissandrakis, Nehaniv, & Dautenhahn, 2002; Goldenberg & Karnath, 2006). We also show that gestures using natural hand postures are perfectly reproduced and may thus be uninformative for diagnosing apraxia. Finally, we propose that the core deficit underlying imitation in callosal apraxia may be an incorrect coordination between multiple imitation goals, such as the orientation of the hand or position of contact between face and hand.

Chapter 5: Variability and Position Errors in Apraxic Imitation: a Short Report

The results that stem from the re-analysis of Goldenberg's experimental data in Chapter 4 reveal that the patient makes imitation errors predominantly for the position of contact between the face and hand. In Chapter 5 we investigate whether these position errors arise because of an impaired spatial representation of the body, i.e. the patient does not know precisely where his or her face is. To address this question we conduct extended versions of Goldenberg's clinical experiment (in collaboration with the Geneva and Vaud University Hospitals). We also address two complimentary questions. First we assess the error variability to see whether the underlying deficit is random or characterizable. Second we investigate whether motor control is entirely preserved, for which we develop an integrated software that allows to record kinematic data using motion sensors.

Data collected from three left parietal patients confirms that the deficit underlying meaningless imitation in apraxia is characterizable, and that the position errors do not arise from a faulty neural representation of the body. The latter result validates one of the predictions of our hypothesis of incorrect coordination between multiple imitation goals.

Chapter 6: Movement curvature planning through force field internal models

In Chapter 5 we collected kinematic data for the imitation of meaningless gestures, that we model mathematically in Chap-

ter 6. As the majority of human motion studies and control models for reaching have essentially focused on straight-line movements, the reproduction of the kinematics of natural self-oriented movements was problematic. Indeed, the movements involved in imitating Goldenberg's visual stimuli follow highly curved trajectories.

We thus develop a nonlinear dynamical system model for reaching that can generate both straight and highly-curved movements with a unique neural controller. The trajectory of the end-effector is the result of a nonlinear dynamical system that creates an attractor at the target position. The system encapsulates body- and environment-related constraints in the form of a repulsive force field. As an example, this local repulsive force field shapes the trajectory to avoid both the subject's body and joint limits. Our model is asymptotically and globally stable and reproduces with high accuracy both the spatial and temporal features of unconstrained reaching movements. The model's biological plausibility and neural correlates are discussed. Finally, we suggest that embodiment, i.e. the geometry of the human body, should be considered as the main cause for the curvature of movements.

Chapter 7: Discussion

This chapter summarizes and discusses the principal contributions of the thesis, with an emphasis on the open questions presented earlier in this chapter. The model presented in Chapter 3 is revised and extended in the light of additional neuropsychological data presented in Chapters 4 and 5. We also make explicit the relation between the neurocomputational model presented in Chapter 3 and dynamical system model proposed in Chapter 6.

We further discuss the main limitations of our work. Several promising research directions are outlined, and concrete and tangible routes for tackling the open issues are suggested.

Chapter 2

Apraxia: a Review

This work was previously published in:

Biljana Petreska, Michela Adriani, Olaf Blanke and Aude Billard. Apraxia: a review. *Progress in Brain Research*, 164:61–83, 2007.

This chapter describes the neuropsychological point of view taken in this thesis. We follow a reverse-engineering approach of the brain, where we look at how imitation falls apart following a brain lesion. Indeed, the contrast of preserved versus impaired functions can help to identify the neural building blocks. A suitable disorder for investigating imitation is *apraxia*.

Abstract

Praxic functions are frequently altered following a brain lesion, giving rise to apraxia — a complex pattern of impairments that is difficult to assess or interpret. In this chapter, we review the current taxonomies of apraxia and related cognitive and neuropsychological models. We also address the questions of the neuroanatomical correlates of apraxia, the relation between apraxia and aphasia and the analysis of apraxic errors. We provide a possible explanation for the difficulties encountered in investigating apraxia and also several approaches to overcome them, such as systematic investigation and modeling studies. Finally, we argue for a multidisciplinary approach. For example, apraxia should be studied in consideration with and could contribute to other fields such as normal motor control, neuroimaging and neurophysiology.

2.1 Introduction

Apraxia is generally defined as "a disorder of skilled movement not caused by weakness, akinesia, deafferentation, abnormal tone or posture, movement disorders such as tremor or chorea, intellectual deterioration, poor comprehension,

or uncooperativeness" (Heilman & Rothi, 1993). Apraxia is thus negatively defined, in terms of what it is not, as a higher order disorder of movement that is *not* due to elementary sensory and/or motor deficits. This definition implies that there are situations where the effector is moved with normal skill (Hermsdörfer et al., 1996). Puzzling parts of apraxia are the *voluntary-automatic dissociation* and *context-dependence*. On the one hand, apraxic patients may spontaneously perform gestures that they cannot perform on command (Schnider, Hanlon, Alexander, & Benson, 1997). This voluntary-automatic dissociation can be illustrated by an apraxic patient who could use his left hand to shave and comb himself, but could not execute a specific motor action such as opening the hand so as to let go of an object (Lausberg, Göttert, Münssinger, Boegner, & Marx, 1999). In this particular case, focusing on the target of the movement rather than on the movement itself increased his chances of a successful execution. On the other hand, the execution of the movement depends heavily on the context of testing (De Renzi, Faglioni, & Sorgato, 1982). It may be well preserved in a natural context, with a deficit that appears in the clinical setting only, where the patient has to explicitly represent the content of the action outside of the situational props (Jeannerod & Decety, 1995; R. C. Leiguarda & Marsden, 2000).

Several authors agree that although apraxia is easy to demonstrate, it has proven difficult to understand. Research on apraxia is filled with confusing terminology, contradictory results and doubts that need to be resolved (De Renzi et al., 1982; Goldenberg, Hermsdörfer, & Spatt, 1996; Koski, Iacoboni, & Mazziotta, 2002; Laeng, 2006). Inconsistencies between similar studies may be explained by differences in the methodological and statistical approaches for the apraxia assessment (i.e. types of gestures used and scoring criteria), chronicity and aetiology of damage and brain lesion localization tools (Haaland, Harrington, & Knight, 2000). Therefore, it still stands that our understanding of the neural and cognitive systems underlying human praxis is not well established.

The chapter is structured as follows. We first review existing types of apraxia as well as important current and historical models of the apraxic deficit. We then consider the inter- and intra-hemispheric lesion correlates of apraxia. Two other sections are dedicated to the relationship between praxis and language and to the analysis of apraxic errors. We finally discuss the current state-of-the-art in apraxia, and argue for a multidisciplinary approach that encompasses evidence from various fields such as neuroimaging or neurophysiology.

2.2 Types of Apraxia

This section reviews the current taxonomies of apraxia. Some of the frequently observed types of apraxia have inspired the apraxia models described in the following section, others still challenge them.

Ideational apraxia was historically defined as a disturbance in the conceptual organization of actions. It was first assessed by performing purposive sequences of actions that require the use of various objects in the correct order (e.g. preparing a cup of coffee) (Poeck, 1983). It was later accepted that ideational apraxia is not necessarily associated to complex actions, but is a larger deficit that also concerns the evocation of single actions. In this view, complex sequences of multiple objects are simply more suitable to reveal the deficit, possibly because of the heavier load placed on memory and attentional resources (De Renzi & Lucchelli, 1988). Nonetheless, the term *conceptual apraxia* was introduced to designate content errors in single actions, excluding sequence errors in multi-staged actions with tools[1] (Ochipa, Rothi, & Heilman, 1992; Heilman, Maher, Greenwald, & Rothi, 1997). In theoretical models, ideational and conceptual apraxia correspond to a disruption of the conceptual component of the praxis system, i.e. action semantics memory, described in more detail in Section 2.3 (De Renzi & Lucchelli, 1988; Graham, Zeman, Young, Patterson, & Hodges, 1999). Patients with ideational apraxia are not impaired in the action execution *per se*, but demonstrate inappropriate use of objects and may fail in gesture discrimination and matching tasks. For example, a patient was reported to eat with a toothbrush and brush his teeth with a spoon and a comb. His inability to use tools could not be explained by a motor production deficit that would characterize ideomotor apraxia (defined below). Interestingly, although he was able to name the tools and point to them on command, he could not match the tools with the objects, hence suggesting a loss of knowledge related to the use of tools.

Ideomotor apraxia is considered to be a disorder of the production component of the praxis system, i.e. sensorimotor action programs that are concerned with the generation and control of motor activity (Rapcsak, Ochipa, Anderson, & Poizner, 1995; Graham et al., 1999). It is characterized by errors in the timing, sequencing and spatial organization of gestural movements (R. Leiguarda, 2001). Since the conceptual part of the praxis system is assumed to be intact, patients with ideomotor apraxia should not use objects and tools in a conceptually inappropriate fashion and should not have difficulty with the serial organization of an action (De Renzi et al., 1982). Ideational and ideomotor apraxia have been assessed by testing the execution of various types of gestures: transitive and intransitive (i.e. with or without the use of tools or objects), meaningless non-representational (e.g. hand postures relative to head) and meaningful representational (e.g. waving good-bye), complex sequences with multiple objects, repetitive movements, distal and proximal gestures (e.g. imitation of finger and hand configurations), reaching in peri-personal and body-centered space (e.g. targets in near space or on the patient's body), novel movements (i.e. skill acquisition) or imagined movements. These gestures can also be executed under different modalities such as: verbal command, imitation, pantomime and tactile

[1] Conceptual apraxia is often observed in Alzheimer's disease.

or visual presentation of objects.

The use of various gestures and different modalities to assess apraxia has helped to uncover many interesting *functional dissociations* that are listed below. For example, apraxia was shown to be modality-specific, i.e. the same type of gesture was differentially impaired according to the modality of testing (De Renzi et al., 1982). One dissociation, named *conduction apraxia*, is the syndrome of superior performance on verbal command than on imitation (Ochipa, Rothi, & Heilman, 1994). The opposite pattern has also been observed: very poor performance on verbal command that improved on imitation or when seeing the object (Heilman, 1973; Merians et al., 1997). The extreme occurrence of conduction apraxia, namely the selective inability to imitate with normal performance on verbal command was termed *visuo-imitative apraxia* (Merians et al., 1997). In some cases of visuo-imitative apraxia, defective imitation of meaningless gestures (e.g. fist under chin) contrasts with preserved imitation of meaningful gestures (e.g. hitchhiking) (Goldenberg & Hagmann, 1997; Salter, Roy, Black, Joshi, & Almeida, 2004). A surprising case of double dissociation from this kind of visuo-imitative apraxia was described in Bartolo, Cubelli, Della Sala, Drei, and Marchetti (2001), where the patient showed impairment in meaningful gesture production (both on imitation and verbal command) and normal performance in imitation of meaningless gestures, suggesting that the patient was able to reproduce only movements he did not identify or recognize as familiar. Similarly, the apraxic patients in Buxbaum, Sirigu, Schwartz, and Klatzky (2003) responded abnormally to familiar objects (e.g. a key, a hammer or a pen) but normally in recognizing the hand postures appropriate for novel objects (e.g. parallelopipeds differing in size and depth). These two studies argue that the reproduction of a gesture may be constrained by its degree of familiarity, indicating that current models of apraxia would need some refinement.

Furthermore, the representation of transitive and intransitive actions may be dissociable. In Watson, Fleet, Gonzalez-Rothi, and Heilman (1986), bilateral apraxia was observed only for transitive (e.g. hammering) but not intransitive (e.g. hitchhiking, waving goodbye) movements.[2] Whereas transitive gestures are constrained by the shape, size and function of objects, intransitive actions are related to socio-cultural contexts (Cubelli, Marchetti, Boscolo, & Della Sala, 2000; Heath, Roy, Black, & Westwood, 2001). The isolated disturbance of transitive hand movements for use of, recognition and interaction with an object, in the presence of preserved intransitive movements, was named *tactile apraxia* and usually appears in the hand contralateral to the lesion (Binkofski, Kunesch, Classen, Seitz, & Freund, 2001).

As mentioned in Section 2.1, contextual cues strongly influence the execution of actions. Some studies have systematically manipulated the contextual cues in order to assess their relative importance. For example, patients with

[2]These patients had lesions in the left supplementary motor area (SMA).

impaired pantomime of motor actions showed no deficit in the comprehension of the use of tools or in manipulating the tools (Halsband et al., 2001). Graham et al. (1999) also observed dramatic facilitation in the demonstration of tool use when the patient was given the appropriate or a neutral tool to manipulate.[3] Interestingly, the patient could not prevent himself from performing the action appropriate to the tool he was holding, rather than the requested action. In another study however, gesture execution improved when the object of the action, but not the tool, was given (Clark et al., 1994). Hence, the addition of visual and somaesthetic cues may improve certain aspects of apraxic movements, since it provides mechanical constraints and supplementary information that facilitates the selection of an adequate motor program (Hermsdörfer, Hentze, & Goldenberg, 2006). Nonetheless, there is the case of a patient that performed much worse when he was actually manipulating the tool than on verbal command[4] (Merians et al., 1999).

Dissociations that concern the nature of the target were also observed. For example, the left brain damaged patients in Hermsdörfer, Blankenfeld, and Goldenberg (2003) had prolonged movement times and reduced maximum velocities when the movements were directed toward an allocentric target without visual feedback, but performed normally when the target was their own nose. Also, a clear dissociation was found in Ietswaart, Carey, and Della Sala (2006) between impaired gesture imitation and intact motor programming of goal-directed movements, hence arguing against the interpretation of impaired imitation as a purely executional deficit (see Section 2.3).

A particular type of apraxia is *constructional apraxia*, originally described by Kleist as "the inability to do a construction" and defined by Benton as "the impairment in combinatory or organizing activity in which details must be clearly perceived and in which the relationship among the component parts of the entity must be apprehended" (Laeng, 2006). Constructional-apraxic patients are unable to spontaneously draw objects, copy figures and build blocks or patterns with sticks, following damage not only to the dominant but also non-dominant hemisphere. Hence, constructional apraxia appears to reflect the loss of bilaterally distributed components for constructive planning and the perceptual processing of categorical and coordinate spatial relations (Platz & Mauritz, 1995; Laeng, 2006).

Apraxia can also be observed in *mental motor imagery tasks*. Motor imagery is considered as a means of accessing the mechanisms of action preparation and imitation, by sharing a common neural basis (Jeannerod & Decety, 1995). Apraxic patients were deficient in simulating hand actions mentally and in imagining the temporal properties of movements[5] (Sirigu, Daprati, Pradat-Diehl, Franck, & Jeannerod, 1999). Other apraxic patients showed a deficit in

[3] The subject had clinically diagnosed corticobasal degeneration.
[4] Ibid.
[5] These patients had posterior parietal lesions.

generating and maintaining internal models for planning object-related actions (Buxbaum, Johnson-Frey, & Bartlett-Williams, 2005). These findings support the notion that the motor impairments observed in apraxic patients result from a specific alteration in their ability to mentally evoke actions, or to use stored motor representations for forming mental images of actions.

Apraxia may also be appropriate to reveal the role of *feedback* during the execution of a movement. Some apraxic patients were impaired in reaching and aiming movements only in the condition without visual feedback (Ietswaart, Carey, Della Sala, & Dijkhuizen, 2001; Ietswaart et al., 2006) and performed worse during pointing with closed eyes (Jacobs et al., 1999; Hermsdörfer et al., 2003). Interestingly, the patients in Haaland, Harrington, and Knight (1999) overshot the target when feedback of the hand was removed, and undershot the target when the feedback of the target was unavailable. Importantly, these patients continued to rely on visual feedback during the secondary adjustment phase of the movement and never achieved normal end-point accuracy when visual feedback of the hand position or target location was unavailable. These findings also suggest that ideomotor limb apraxia may be associated with the disruption of the neural representations for the extra-personal (spatial location) and intra-personal (hand position) features of movement (Haaland et al., 1999).

The importance of feedback signals was demonstrated in one of our own apraxic patients (unpublished data). We reproduced a seminal study of imitation of meaningless gestures[6] by Goldenberg, Laimgruber, and Hermsdörfer (2001) on an apraxic patient with left-parietal ischemic lesion. We observed that the patient relied heavily on visual and tactile feedback. He often needed to bring his hand in the field of vision and corrected the hand posture by directly comparing it with the displayed stimulus to imitate. He also used tactile exploration when searching for the correct spatial position on his face. He showed many hesitations and extensive searching which led to highly disturbed kinematic profiles of the gesture (shown in Fig. 2.3c, d), but often correct final postures.

Apraxia can also be defined in relation to the selectively affected effectors: *orofacial apraxia* or *buccofacial apraxia*, *oral apraxia*, *upper and lower face apraxia*, *lid apraxia*, *limb apraxia*, *leg apraxia*, *trunk apraxia*, etc. Oral apraxia, for example, is defined as the inability to perform mouth actions such as sucking from a straw or blowing a kiss. It should not be confounded with *apraxia of speech* (also called *verbal apraxia*), which is a selective disturbance of the articulation of words (Bizzozero et al., 2000). Motor planning disorders in children are denominated *developmental dyspraxia* (Cermak, 1985). Apraxia can also designate a praxic ability impaired in an isolated manner such as: *gait apraxia*, *apraxic agraphia*, *dressing apraxia*, *orienting apraxia* and *mirror apraxia* (i.e. inability to reach to objects in a mirror (Binkofski et al., 2003)). When the side of brain lesion and affected hand are considered, the terms *sympathetic* and

[6]Hand postures relative to the head, an example is shown in Fig. 2.3a.

crossed apraxia are used. Apraxia can sometimes be related to the specific neural substrate that causes the disorder, for example following subcortical lesions in corticobasal degeneration (Pramstaller & Marsden, 1996; Jacobs et al., 1999; Merians et al., 1999; Hanna-Pladdy, Heilman, & Foundas, 2001; R. Leiguarda, 2001) or following lesions of the corpus callosum (Watson & Heilman, 1983; Lausberg et al., 1999; Lausberg, Davis, & Rothenhäusler, 2000; Goldenberg et al., 2001; Lausberg & Cruz, 2004). *Callosal apraxia* for example is particularly appropriate for disentangling the specific hemispheric contributions to praxis.

An extensive list of the types of apraxia and their definitions, including types that were not mentioned above, can be found in Table 2.1.

2.3 Models of Apraxia

Contemporary neuropsychological views of apraxia arise from Liepmann's influential work that dates from more than a hundred years ago. Liepmann proposed the existence of an idea of the movement, "movement formulae", that contains the "time-space-form picture" of the action (Rothi, Ochipa, & Heilman, 1991). He believed that in right-handers, these movement formulae are stored in the left-parietal lobe, endorsing the view of a left hemispheric dominance for praxis (Faglioni & Basso, 1985; R. C. Leiguarda & Marsden, 2000). To execute a movement, the spatiotemporal image of the movement is transformed into "innervatory patterns" that yield "positioning of the limbs according to directional ideas" (Jacobs et al., 1999). Liepmann distinguished between three types of apraxia that correspond to disruptions of specific components of his model (Faglioni & Basso, 1985; Goldenberg, 2003). First, a damaged movement formula (i.e. faulty integration of the elements of an action) would characterize "ideational apraxia". Second, failure of the transition from the movement formula to motor innervation (i.e. inability to translate a correct idea of the movement into a correct act) is defined as "ideomotor apraxia". According to Liepmann, faulty imitation of movements is a purely executional deficit and proves the separation between the idea and execution of a movement, since in imitation the movement formula is defined by the demonstration (Goldenberg, 1995, 2003; Goldenberg & Hagmann, 1997). Finally, loss of purely kinematic (kinaesthetic or innervatory) inherent memories of an extremity is the "limb-kinetic" variant of apraxia.

Another historically influential model is the disconnection model of apraxia proposed by Geschwind (1965). According to this model the verbal command for the movement is comprehended in Wernicke's area and is transferred to the ipsilateral motor and premotor areas that control the movement of the right hand (Clark et al., 1994; R. C. Leiguarda & Marsden, 2000). For a left-hand movement, the information needs to be further transmitted to the right association cortex via the corpus callosum. The model postulates that the

Table 2.1 Taxonomy of apraxia

Type of Apraxia	Definition
Ideational apraxia	Initially used to refer to impairment in the conceptual organization of actions, assessed with sequential use of multiple objects. Later defined as conceptual apraxia.
Conceptual apraxia	Impairment in the concept of a single action, characterized by content errors and the inability to use tools.
Ideomotor apraxia	Impairment in the performance of skilled movements, characterized by spatial or temporal errors in the execution of movements.
Limb-kinetic apraxia	Slowness and stiffness of movements with a loss of fine, precise and independent movement of the fingers.
Constructional apraxia	Difficulty in drawing and constructing objects. Impairment in the combinatory or organizing activity in which details and relationship among the component parts of the entity must be clearly perceived.
Developmental dyspraxia	Disorders affecting the initiation, organization and performance of actions in children.
Modality-specific apraxias	*Localized within one sensory system*
Pantomime agnosia	Normal performance in gesture production tests both on imitation and on verbal command, but poor performance in gesture discrimination and comprehension. Patients with pantomime agnosia can imitate pantomimes they can not recognize.
Conduction apraxia	Superior performance on pantomime to verbal command than on pantomime imitation.
Visuo-imitative apraxia	Normal performance on verbal command with selectively impaired imitation of gestures. Also used to designate the defective imitation of meaningless gestures combined with preserved imitation of meaningful gestures.
Optical/visuomotor apraxia	Disruptions to actions calling upon underlying visual support.
Tactile apraxia	Disturbance of transitive hand movements for use of, recognition and interaction with an object, in the presence of preserved intransitive movements.
Effector-specific apraxias	
Upper/lower face apraxia	Impairment in performing actions with parts of the face.
Oral apraxia	Inability to perform skilled movements with the lips, cheeks and tongue.
Orofacial/buccofacial apraxia	Difficulties with performing intentional movements with facial structures including the cheeks, lips, tongue and eyebrows.
Lid apraxia	Difficulty with opening the eyelids.
Ocular apraxia	Impairment in performing saccadic eye movements on command.
Limb apraxia	Used to refer to ideomotor apraxia of the limbs frequently including the hands and fingers.
Trunk (or axial) apraxia	Difficulty with generating body postures.
Leg apraxia	Difficulty with performing intentional movements with the lower limbs.
Task-specific apraxias	
Gait apraxia	Impaired ability to execute the highly practised, co-ordinated movements of the lower legs required for walking.
Gaze apraxia	Difficulty in directing gaze.
Apraxia of speech (or verbal)	Disturbances of word articulation.
Apraxic agraphia	A condition in which motor writing is impaired but limb praxis and nonmotor writing (typing, anagram letters) are preserved.
Dressing apraxia	Inability to perform the relatively complex task of dressing.
Dyssynchronous apraxia	Failure to combine simultaneous preprogrammed movements.
Orienting apraxia	Difficulty in orienting one's body with reference to other objects.
Mirror apraxia	A deficit in reaching to objects presented in a mirror.
Lesion-specific apraxias	
Callosal apraxia	Apraxia caused by damage to the anterior corpus callosum that usually affects the left limb.
Sympathetic apraxia	Apraxia of the left limb due to damage to the anterior left hemisphere (the right hand being partially or fully paralysed).
Crossed apraxia	The unexpected pattern of apraxia of the right limb following damage to the right-hemisphere.

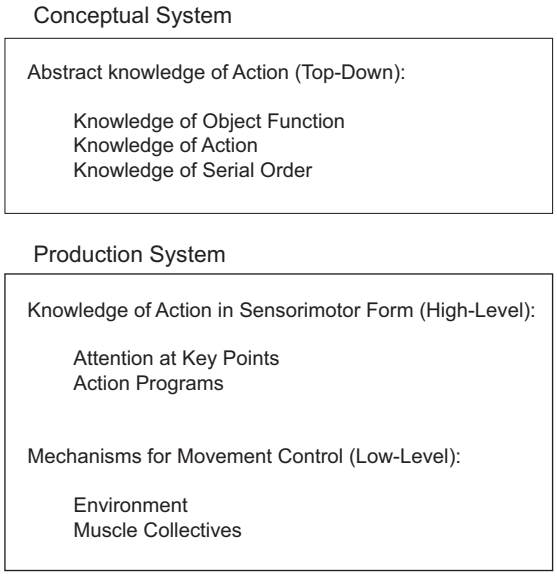

Figure 2.1: Roy and Square's cognitive model of limb praxis. Adapted with permission from Roy and Square (1985).

apraxic disorder follows from a lesion in the left and right motor association cortices, or a disruption in their communication pathways. However this model cannot explain impaired imitation and impaired object use since these tasks do not require a verbal command (Rothi et al., 1991).

Heilman and Rothi (1993) proposed an alternative representational model of apraxia, according to which apraxia is a gesture production deficit that may result from the destruction of the spatiotemporal representations of learned movements stored in the left inferior-parietal lobule. They proposed to distinguish between dysfunction caused by destruction of the parietal areas (where the spatiotemporal representations of movements would be encoded), and the deficit which would result from the disconnection of these parietal areas from the frontal motor areas (Heilman, Rothi, & Valenstein, 1982). In the first case, posterior lesions would cause a degraded memory trace of the movement and patients would not be able to correctly recognize and discriminate gestures. In the second case, anterior lesions or disconnections would only provoke a memory egress disorder. Therefore patients with a gesture production deficit with anterior and posterior lesions should perform differently on tasks of gesture discrimination, gesture recognition and novel gesture learning.

Roy and Square (1985) proposed a cognitive model of limb praxis that involves two systems, i.e. a conceptual system and a production system (illustrated in Fig. 2.1). The "conceptual system" provides an abstract representation of the action and comprises three kinds of knowledge: (1) knowledge of the functions of tools and objects, (2) knowledge of actions independent of tools and objects and (3) knowledge about the organization of single actions into sequences. The "production system" incorporates a sensorimotor representation

of the action and mechanisms for movement control. Empirical support for the division of the praxis system into a conceptual and a production component is provided by a patient who could comprehend and discriminate transitive gestures she was unable to perform (Rapcsak et al., 1995). This model predicts three patterns of impairment (Heath et al., 2001). First, a deficit in pantomime but not in imitation would reflect damage to the selection and/or evocation of actions from long-term memory. Second, a deficit in imitation alone would indicate a disruption of the visual gestural analysis or translation of visual information into movement. Finally, concurrent impairment in pantomime and imitation is thought to reflect a disturbance at the latter, executive stage of gesture production and was the most frequent deficit pattern observed in Roy et al. (2000) and Parakh, Roy, Koo, and Black (2004).

None of these models predict a number of modality-specific dissociations observed in neurologically impaired patients, such as preserved gesture execution on verbal command that is impaired in the visual modality when imitating (Ochipa et al., 1994; Goldenberg & Hagmann, 1997). To account for these dissociations, Rothi et al. (1991) proposed a cognitive neuropsychological model of limb praxis, which reflects more appropriately the complexity of human praxis (illustrated in Fig. 2.2a). This multi-modular model has input that is selective according to the modality, a specific "action semantics system" dissociable from other semantics systems, an "action reception lexicon" that communicates with an "action production lexicon" and a separate nonlexical route" for the imitation of novel and meaningless gestures[7] (Rothi et al., 1997).

Although this model is widely used to explain data from multiple neurological studies, it has difficulties concerning several aspects. First, it does not consider the existence of a selective tactile route to transitive actions (Graham et al., 1999). For example, the model fails to explain data from a patient profoundly impaired in gesturing in the verbal and visual modalities, but not with the tool in hand (Buxbaum, Giovannetti, & Libon, 2000). Second, imitation of meaningless gestures is assumed to test the integrity of a direct route from visual perception to motor control. However, Goldenberg et al. (1996) have shown that this route is far from direct and involves complex intermediate processing steps. For example, apraxic patients that are impaired in reproducing gestures on their own bodies are also impaired in replicating the gestures on a life-sized mannikin (Goldenberg, 1995). Hence, general conceptual knowledge about the human body and the spatial configuration of body parts seems necessary for performing an imitation task (Goldenberg, 1995; Goldenberg et al., 1996; Goldenberg & Hagmann, 1997). The belief that imitation is a rather simple and straightforward visuomotor process is misleading as one would have to resolve the "body correspondence problem"[8] to transpose movements from bodies with

[7]The vocabulary was borrowed from the literature of language processing.

[8]Here we give a shortened version of the informal statement of the body correspondence problem. Given an observed behavior of the model, i.e. a sequence (or hierarchy) of subgoals, find and execute a sequence of actions using one's own (possibly dissimilar) embodiment which

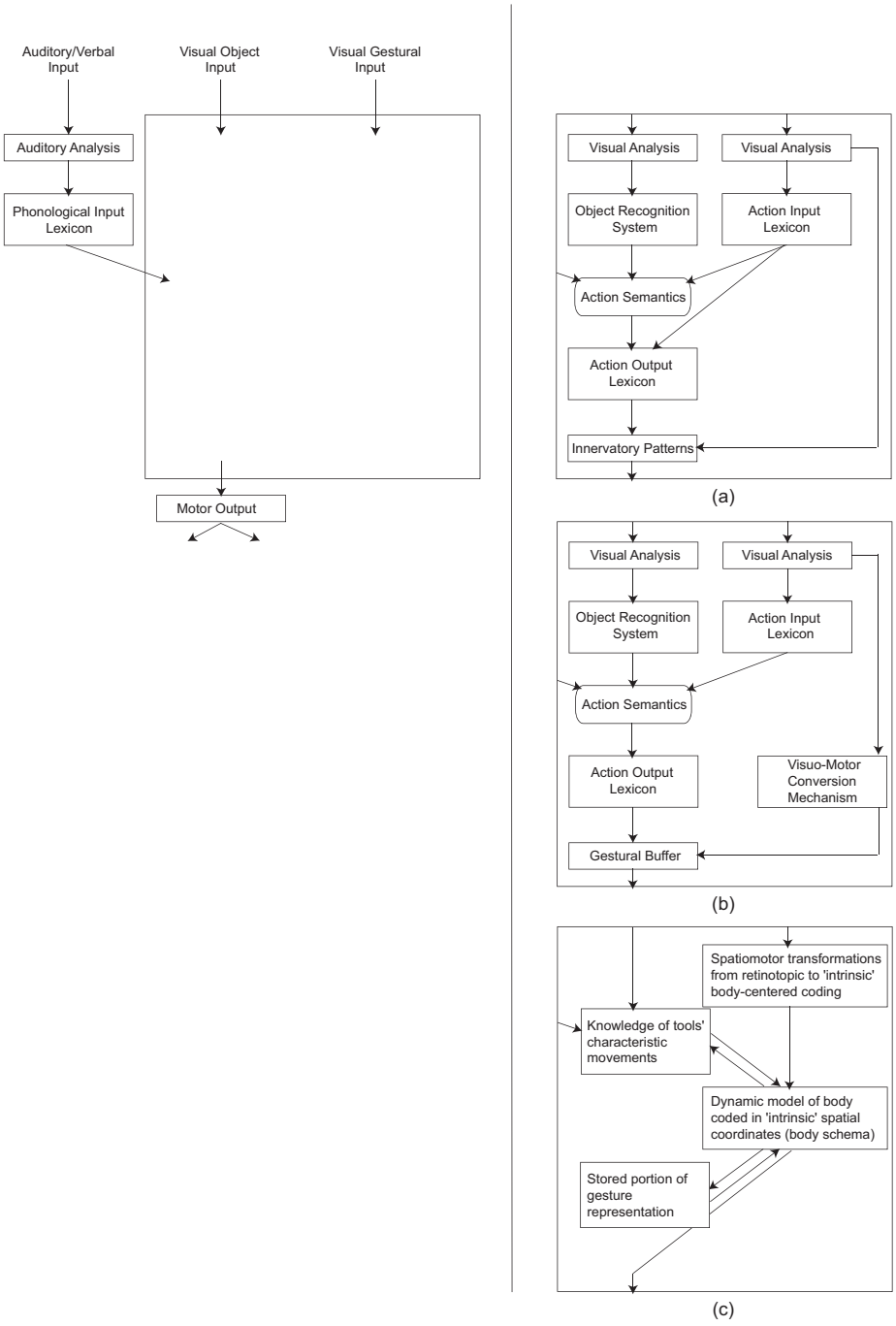

Figure 2.2: A cognitive neuropsychological model of limb praxis. The three components on the right are interchangeable with the empty box in the complete model on the left. Under (a) Rothi et al.'s original model of limb praxis. Under (b) the previous model revised by Cubelli et al. and under (c) the model extended by Buxbaum et al. For a detailed description see the text. Adapted respectively with permission from Rothi et al. (1997), Cubelli et al. (2000) and Buxbaum et al. (2000).

different sizes and different owners, which are in addition represented in different perspectives (Goldenberg, 1995).

To account for the last observation, Cubelli et al. (2000) have revised Rothi et al.'s cognitive neuropsychological model of limb praxis (illustrated in Fig. 2.2b). They have added "a visuomotor conversion mechanism" devoted to transcoding the visual input into appropriate motor programs. They have also suppressed the direct link between the "input" and "output action lexicon", leaving only an indirect link through the "action semantics system", as no empirical evidence was found of a patient able to reproduce familiar gestures with obscure meaning, but not unfamiliar gestures (see Fig. 2.2a, b). Finally, they have also added a "gestural buffer" aimed at holding a short-term representation of the whole action. The model predicts five different clinical pictures (for definitions of the different apraxic disorders please refer to Table 2.1): (1) a deficit of the "action input lexicon": *pantomime agnosia* (i.e. a difficulty in the discrimination and comprehension of gestures), (2) a deficit of the "action semantics system": conceptual apraxia without ideomotor apraxia, (3) a deficit of the "action output lexicon": conceptual apraxia with spared gesture-meaning associations, (4) a deficit of the "visuomotor conversion mechanism": conduction apraxia (not observed in their study) and (5) a deficit of the "gestural buffer": both ideomotor and ideational apraxia (i.e. impairment in all execution tasks with preserved ability to perform judgment and categorization tasks).

Buxbaum et al. (2000) further extended Rothi et al.'s cognitive neuropsychological model of limb praxis, based on their observation of a patient who performed particularly poorly on tasks that required a spatial transformation of the body. According to their model (illustrated in Fig. 2.2c), a unitary set of representations named "body schema" calculates and updates the dynamic positions of the body parts relative to one another. Importantly, this dynamic body-centered representation of actions is a common processing stage between the "lexical" and "nonlexical route" and hence subserves both meaningful and meaningless actions. Note that at the level of the "lexical route", there is an additional interaction with the stored representations of learned actions.

Existing models of apraxia still fail to account for additional empirical evidence such as, for example, the differential performance in imitation of hand postures and imitation of finger configurations shown in Goldenberg and Hagmann (1997) and Goldenberg and Karnath (2006). Furthermore, in a study of ideomotor apraxia, Buxbaum, Kyle, and Menon (2005) provided data which is compatible with the influential "mirror neuron hypothesis". Apraxia models cannot easily be reconciled with this hypothesis which, based upon neurophysiological observations from the monkey brain, postulates a "mirror neuron system" underlying both action recognition and action execution (Rizzolatti & Craighero, 2004). Mirror neurons are a special class of visuomotor neurons, initially discovered in area F5 of the monkey premotor cortex (see Fig. 2.4), which

leads through the corresponding subgoals (Nehaniv & Dautenhahn, 2002).

discharge both when the monkey does a particular action and when it observes another individual doing a similar action (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Rizzolatti & Luppino, 2001; Rizzolatti, Fogassi, & Gallese, 2002). Hence, the "mirror neuron system" is believed to map observed actions onto the same neural substrate used to execute these actions. As the same representations appear to subserve both action recognition and action production tasks, it would not be surprising if the perception of a movement is constrained by its executional knowledge. Related to apraxia, the "mirror neuron hypothesis" questions the separation of the "input" and "output lexicon" (Koski et al., 2002).

2.4 Contributions of the left and right brain hemispheres

Although most apraxia studies show a left-brain hemisphere dominance for praxis, the studies arguing for a significant involvement of the right hemisphere are numerous. Left-brain damage usually affects both hands, whereas right-brain damage affects only the left hand, suggesting that the left hemisphere is not only fully competent for processing movement concepts but also contributes to the generation of movements in the right hemisphere. Apraxic deficits following left hemisphere lesions are also more frequent (De Renzi, Motti, & Nichelli, 1980; Weiss et al., 2001); however, in some rare cases, severe apraxia was observed following right hemisphere lesions (Marchetti & Della Sala, 1997; Raymer et al., 1999). The concept of crossed apraxia was introduced to describe patients with this opposite pattern of limb apraxia that cannot be explained by handedness. Callosal lesions are most suitable for investigating the issues of hemispheric specialization of praxis. For example, split-brain patients were apraxic with their left hands, also suggesting a left hemisphere dominance for processing skilled movement (Watson & Heilman, 1983; Lausberg et al., 1999; Lausberg, Cruz, Kita, Zaidel, & Ptito, 2003), but both hemispheres appeared to contain concepts for skill acquisition (Lausberg et al., 1999) and object use (Lausberg et al., 2003).

In kinematic studies (described in more detail in Section 2.7), only left-brain damaged patients were impaired in imitation of meaningless movements (Hermsdörfer et al., 1996; Weiss et al., 2001), as well as in pointing movements (Hermsdörfer et al., 2003); whereas right-brain damaged patients had deficits in slow-paced tapping and initiation of aiming movements (Haaland & Harrington, 1996). Hence, the left hemisphere was associated with movement trajectory control (Haaland, Prestopnik, Knight, & Lee, 2004), sequencing and ballistic movements (Hermsdörfer et al., 2003) and the right hemisphere was related to on-line control of the movement (Hermsdörfer et al., 2003) and closed-loop processing (Haaland & Harrington, 1996).

A left-right dichotomy was also observed for imitation and matching of hand and finger configurations (Goldenberg, 1999). Left-brain damaged patients had more difficulties with imitation than matching and vice versa. In addition, the left hemisphere seemed fully competent for processing hand postures, but needed the right hemisphere's contribution for processing finger postures (Goldenberg et al., 2001; Della Sala, Faglioni, Motto, & Spinnler, 2006). It was concluded that the left hemisphere mediates conceptual knowledge about the structure of the human body and that the right hemisphere is specialized for visually analyzing the gesture (Goldenberg, 2001; Goldenberg et al., 2001).

Finally, several studies observed similar impairment scores following left- and right-brain lesions, arguing for a bi-hemispheric representation of skilled movement (Haaland & Flaherty, 1984; Kertesz & Ferro, 1984; Roy, Black, Winchester, & Barbour, 1992; Roy et al., 2000; Heath et al., 2001). The less frequent, nevertheless well-detected incidence of limb apraxia following right-brain lesion, was attributed to the sensitivity and precision of the assessment methodology. In addition, right-hemisphere lesions often led to severe face apraxia (Bizzozero et al., 2000; Della Sala, Maistrello, Motto, & Spinnler, 2006). Hence, a model of widespread praxis, distributed across both hemispheres, may be more appropriate than the unique left-lateralized center previously hypothesized. Moreover, it seems that the degree of left-hemisphere dominance varies within subjects and with the type of movement (Haaland et al., 2004), raising the issue of overlap between the contributions of the right and left hemispheres to specialized praxic functions.

2.5 Intra-hemispheric lesion location: a distributed representation of praxis?

Several studies have failed to find a consistent association between the locus of the lesion within a hemisphere and the severity of apraxia (Basso, Luzzatti, & Spinnler, 1980; Kertesz & Ferro, 1984; Alexander, Baker, Naeser, Kaplan, & Palumbo, 1992; Schnider et al., 1997; Hermsdörfer et al., 2003). Moreover, areas involved in apraxia can also be damaged in non-apraxic patients (Haaland et al., 1999; Buxbaum et al., 2003). However, apraxic deficits are most frequent following parietal and frontal lesions, but were also observed in patients with temporal, occipital and subcortical damages (De Renzi & Lucchelli, 1988; Goldenberg, 1995; Hermsdörfer et al., 1996; Bizzozero et al., 2000).

More specifically, ideomotor apraxia and motor imagery deficits were observed following lesions in the left inferior parietal and the left dorsolateral frontal lobes (Haaland et al., 2000; Buxbaum, Johnson-Frey, & Bartlett-Williams, 2005). For example, several studies suggested that Brodmann areas 39 and 40 (i.e. angular and supramarginal gyri of the inferior-parietal lobule) are critical in

visuo-imitative apraxia (Goldenberg & Hagmann, 1997; Goldenberg et al., 2001) and ideomotor limb apraxia (Haaland et al., 1999; Buxbaum et al., 2003). In addition, the superior-parietal lobe appeared crucial in integrating external visual and intra-personal somaesthisic information (Heilman, Rothi, Mack, Feinberg, & Watson, 1986; Haaland et al., 1999). Goldenberg and Karnath (2006) subtracted the lesion overlay of unimpaired from impaired patients and associated disturbed imitation of hand postures with lesions in the inferior-parietal lobe and temporo-parieto-occipital junction, whereas disturbed imitation of finger postures could be related to lesions in the inferior frontal gyrus. Interestingly, parts of the middle and inferior frontal gyri, in the vicinity of Brodmann areas 6, 8 and 46, were involved in all of the ideomotor apraxics in Haaland et al. (1999). Furthermore, premotor lesions (including lesions to the supplementary motor area) particularly affected bimanual actions in Halsband et al. (2001) and transitive actions in Watson et al. (1986).

It has been difficult to disentangle between the specific contributions of the parietal and the frontal cortices, as lesions in these areas lead to similar deficits (Haaland et al., 1999, 2000). For example, target and spatial errors were related to posterior lesions only (Haaland et al., 2000; Halsband et al., 2001; Weiss et al., 2001; Goldenberg & Karnath, 2006), but internal hand configuration errors were present in patients with anterior and posterior lesions (Haaland et al., 2000; Goldenberg & Karnath, 2006). Importantly, only patients with posterior lesions, and not anterior lesions, had difficulties in discriminating between correctly and incorrectly performed actions and in recognizing pantomimes or appropriate hand postures (Halsband et al., 2001; Buxbaum, Kyle, & Menon, 2005).

Apraxia can also develop following subcortical lesions (Pramstaller & Marsden, 1996; Graham et al., 1999; Jacobs et al., 1999; Merians et al., 1999; Hanna-Pladdy et al., 2001). In this case, it is not clear whether the apraxia originates from lesions in the basal ganglia, which are extensively connected to the superior-parietal lobe and premotor and supplementary motor areas (Jacobs et al., 1999; Merians et al., 1999), or from the surrounding white matter (i.e. fronto-parietal connections) (Pramstaller & Marsden, 1996).

Failure to find clear correlations between specific lesion loci and different apraxic deficits argues for a widespread cortical and subcortical representation of praxis, distributed across specialized neural systems working in concert (R. C. Leiguarda & Marsden, 2000; Hermsdörfer et al., 2003). However, we believe that a selective damage to one of these systems may produce a particular pattern of errors tightly related to a subtype of apraxia.

2.6 Praxis and language?

Apraxia is most often seen in association with aphasia (i.e. loss of the ability to speak or understand speech), which renders the assessment of apraxia very

difficult. Indeed, one has to provide evidence that the patient has understood the commands so that the motor deficit cannot be attributed to aphasia (De Renzi et al., 1980). Historically, gestural disturbance in aphasics was considered to be a manifestation of damaged abstract knowledge. This idea of a common impaired symbolic function underlying aphasia and apraxia was supported for a long time (Kertesz & Hooper, 1982). However, several large-scale studies failed to find correlations between subtypes of apraxia and aphasia (Goodglass & Kaplan, 1963; Lehmkuhl, Poeck, & Willmes, 1983; Buxbaum, Kyle, & Menon, 2005). Moreover, clear evidence of a double dissociation between apraxia and aphasia was presented in Papagno, Della Sala, and Basso (1993). For example, some patients were able to verbalize a desired movement but could not perform it (Goodglass & Kaplan, 1963), whereas other patients were able to pantomime actions they were unable to name (Rothi et al., 1991). Hence, it seems that many aspects of language and praxis are subserved by independent, possibly contiguous neuronal processes, but concomitant deficits may also appear because of shared neuroanatomical substrates (Kertesz & Hooper, 1982). Nevertheless, the question of how language is related to praxis is a fascinating one and needs further study, as it can give some insight into the existence of a supramodal representation of knowledge, or alternatively shed light onto the communication mechanisms between the praxic- and language-specific representations of knowledge.[9]

2.7 The analysis of apraxic errors

There are extensive quantitative analyses of the severity of apraxic errors in single case studies and in large samples of brain-damaged patients. Qualitative analyses however are less numerous and nonstandardized, but nonetheless essential for precisely understanding the nature of apraxia. Performances are usually classified in a limited number of response categories such as:[10] temporal errors, spatial errors, content errors, substitutive errors, augmentative errors, fragmentary errors, associative errors (i.e. the correct movement is replaced by another movement that shares one feature), parapraxic errors (i.e. correct execution of a wrong movement), wrong body part errors (e.g. patients that execute a correct movement with the head instead of the hand), body part as tool errors (i.e. a body part is used to represent the imagined tool) and perseveration errors (Lehmkuhl et al., 1983; Poeck, 1983; De Renzi & Lucchelli, 1988; Platz & Mauritz, 1995; Lausberg et al., 1999, 2003; Halsband et al., 2001; Weiss et al., 2001). Perseveration and body parts as tool errors should be accorded some special interest in future studies, as they are prominent in apraxia and their occurrence is far from being elucidated (Poeck, 1983; Raymer, Maher,

[9] Some authors have posited that an action-recognition mechanism might be at the basis of language development (Rizzolatti & Arbib, 1998).

[10] This list is not extensive. Terminologies can vary a lot across different authors.

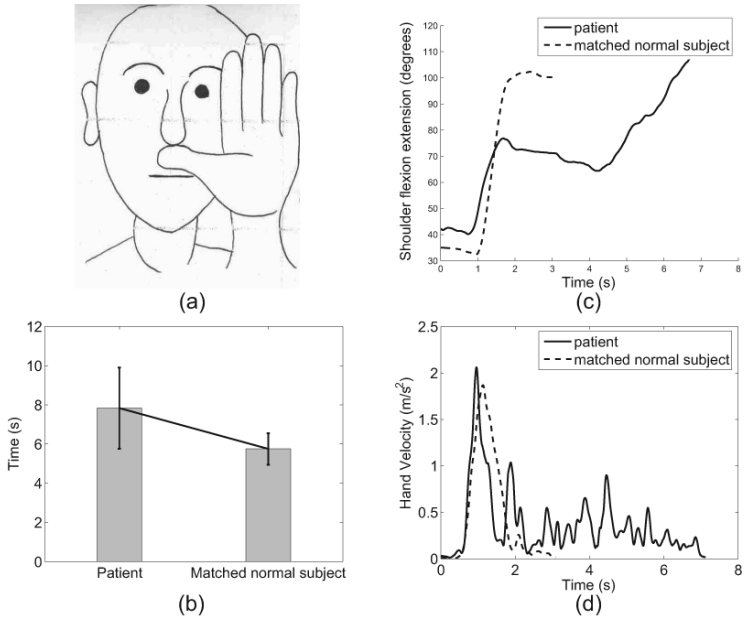

Figure 2.3: An example of the abnormal kinematics of an apraxic movement. A patient with left ischemic lesions was tested in a study of imitation of meaningless gestures. The stimulus to imitate for this movement is shown under (a) and represents a hand posture relative to the head. Under (b), the movement times of the patient are longer than those of a matched normal subject (including replacement of the hand in the initial condition). Under (c), the trajectory of the shoulder flexion-extension joint angle of the patient (shown in solid line) contains several irregularities, which are the result from multiple hesitations and changes of directions, whereas the matched normal subject shoulder flexion-extension trajectory (dashed line) is smooth. The speed profile of the patient (solid line) is shown under (d) and contains multiple peaks with reduced maximum velocities that contrast with the simple bell-shaped velocity profile of the matched normal subject (dashed line).

Foundas, Heilman, & Rothi, 1997; Lausberg et al., 2003). For example, even though normal subjects also commit body part as tool errors,[11] only subjects with brain lesion cannot correct their error after re-instruction (Raymer et al., 1997).

A significant step forward in the analysis of apraxic errors was the use of quantitative 3D kinematic motion analysis. These techniques allowed to show many abnormalities in the kinematic features of apraxic movements such as: deficits in spatial accuracy, irregular velocity profiles, reduced maximum velocities, reduced movement amplitudes, de-coupling of the relationship between instantaneous wrist velocity and trajectory curvature, improper linearity of the movement, wrong orientation of the movement in space and/or deficient joint coordination (Poizner, Mack, Verfaellie, Rothi, & Heilman, 1990; Poizner et al., 1995; Poizner, Merians, Clark, Rothi, & Heilman, 1997; Clark et al., 1994; Platz & Mauritz, 1995; Rapcsak et al., 1995; Merians et al., 1997; Haaland et al., 1999; Binkofski et al., 2001; Hermsdörfer et al., 2006). An example of an

[11] There is a hierarchical organization in the performance of actions with increasing difficulty. Children first acquire the ability to actually use objects, then to demonstrate the action with similar substitute objects, then with dissimilar substitute objects, then to use body parts as substitutes, and finally to perform pantomimes with holding imagined objects. This note was taken from Lausberg et al. (2003).

apraxic movement with abnormal kinematics is shown in Fig. 2.3. Based on kinematic studies it could be concluded that ideomotor limb apraxia impaired the response implementation but not the preprogramming of the movement (Haaland et al., 1999) and decoupled the spatial and temporal representations of the movement (Poizner et al., 1990, 1995). Importantly, the kinematic abnormalities observed were often spatial and not temporal, the longer movement times in the apraxic group could be interpreted as an artifact of the longer distance traveled (Haaland et al., 1999; Hermsdörfer et al., 2006). However, several authors have advised against systematically interpreting the irregular kinematics as an indicator for deficient motor programming or deficient motor implementation (Platz & Mauritz, 1995; Haaland et al., 1999). For example, no correlation could be found between the kinematic abnormalities and apraxic errors in Hermsdörfer et al. (1996). Indeed, movements with degraded kinematics frequently reached a correct final position, while, on the contrary, kinematically normal movements often led to apraxic errors. The abnormal kinematic profile of the gesture probably arose from several corrective and compensatory strategies that the patient used to cope with the apraxic deficit (Goldenberg et al., 1996; Hermsdörfer et al., 1996). For example, hesitant and on-line controlled movements generated multi-peaked velocity profiles in our study (see Fig. 2.3d). Hence, according to the authors, the basic deficit underlying apraxia may concern the mental representation of the target position. Consistently with this hypothesis, it was found that apraxic patients relied more than normal subjects on on-line visual information in aiming movements (Ietswaart et al., 2006).

2.8 Discussion

We have shown in the preceding sections that apraxia has proven very difficult to assess and understand. Here we will try to provide some hypotheses on why these difficulties might arise and we propose several ways to overcome these.

2.8.1 The complex nature of apraxia

Apraxia designates the impairment of the human praxis system following brain lesion and has to deal with the high complexity and wide range of human praxic functions. Therefore studies of apraxia have separately tackled the faulty execution of many types of gestures (e.g. transitive and intransitive, meaningful and meaningless, peripersonal and body-centered, etc.) of various endeffectors (e.g. mouth, face, leg, limb) in different types of modalities (e.g. visual, auditive, tactile presentation and imitation). The high dimensionality of varying parameters has led to a lack of systematicity in the apraxia assessment and

terminologies used. This has also rendered the coherent interpretation of the disorder rather arduous.

It follows that there is a great need to discriminate between different types of actions, as they appear to be differentially impaired in apraxia and hence may involve distinct underlying mechanisms (see Section 2.2). Indeed, it is very likely that the mechanisms of imitation and execution of movements vary according to the type of action that is imitated or executed (Schnider et al., 1997; Goldenberg, 1999; Goldenberg & Karnath, 2006). This suggests that different categories of actions require the use of separate systems at some stage of processing, but the level of separation between the representations underlying actions of different types, or even different actions of the same type, is not at all clear yet.

We will principally argue that it is important to better understand what a particular gesture or execution modality implies in terms of brain resources and processes when compared to another gesture/execution modality. For example, a transitive action, i.e. an action that involves an object, is very different from an intransitive action in the sense that it provides supplementary tactile input as a result from the interaction with the object. This tactile sensory input then needs to be integrated with the representation of the action that relies also on other types of sensory inputs such as visual and proprioceptive. Moreover, executing a transitive action in a pantomime condition is also different from executing it with the object in hand, since the action has to be retrieved without the help of tactile input produced by the object. Indeed the movement is somehow modified, for example movement amplitudes in normal subjects were larger in the pantomime condition when compared to actual sawing (Hermsdörfer et al., 2006).

The distinction between meaningful and meaningless gestures would also need some clarification. The reproduction of a recognized meaningful gesture on the one hand, appears entirely based on the internal representation of the gesture. Indeed, the knowledge of a learned skilled act is preferably retrieved from motor memory rather than being constructed *de novo* (Halsband et al., 2001). On the other hand, the reproduction of a meaningless gesture involves a close visual tracking of the imitatee's body configuration and was modeled by a "visuomotor conversion mechanism" or a "body schema" (see Fig. 2.2b, c). To summarize, a meaningful gesture seems to be, to a certain extent, assimilated to a goal that guides the action from memory, whereas a meaningless gesture is defined as a particular configuration of the body in space and time, with no external referents (Goldenberg, 2001). Hence, imitation of meaningless gestures might be used to test the comprehension and replication of changing relationships between the multiple parts and subdivisions of the refined and complex mechanical device, which is the human body (Goldenberg, 2001). Furthermore, a preserved imitation of meaningless gestures is crucial for the apraxic patient as it might be useful for relearning motor skills. The double dissociation ob-

served between the imitation of meaningless and meaningful gestures argues for completely separate processing systems, and is still not accounted for by any of the existing apraxia models previously described. However, meaningless actions involve novel motor sequences that must be analyzed and constructed from the existing movements (Koski et al., 2002) and both meaningless and meaningful gestures appear to engage the body schema, i.e. a dynamic model for coding the body (Buxbaum et al., 2000). Hence, meaningless and meaningful actions may also share some overlapping conceptual representations.

These examples show that there are some common and some distinct processes involved in the different types of movements and modalities used for testing apraxia. Identification of the overlap of these processes would provide a clearer framework for interpreting the patient's performance and would simplify the analysis of the lesion correlates. The choice of the testing condition is crucial, as well as identification of the processes inherent to the chosen condition. However this is a difficult task, since correlations can be found between some very different and even dissociated types of movements.[12] For example, kinematic measures of pointing movements were correlated to gesture imitation, suggesting that the kinematic deficits observed during pointing movements are generalized to more global aiming movements, including movements for imitating hand gestures (Hermsdörfer et al., 2003). Accordingly, gesture imitation is believed to depend upon some of the same cognitive mechanisms as reaching and grasping (Haaland et al., 2000), however the level and extent of interplay is not clear. To make the picture even more complex, the underlying representations may be componential, for example with separate hand posture representations for transitive gestures (Buxbaum, Kyle, & Menon, 2005). This leads us to two questions that urge to be answered: (1) What are the basic motor primitives from which all movements are constructed? and (2) Which are the motor components that are related to specific movements?

2.8.2 Beyond the complex nature of apraxia

One way to cope with the complex nature of apraxia is to be even more *precise* and *systematic* in assessing the apraxic disorder. Ideally, the full range of praxic functions, related to different effectors, including mouth, face and foot should be tested in a complete set of modalities (Koski et al., 2002). Moreover, we find it unfortunate that qualitative measures of the errors, such as kinematic measures of the movement trajectory (refer to Section 2.7), are frequently missing or given in a purely statistical fashion (e.g. 25% of errors in Condition A). As such, these measures do not suffice to understand why the patient succeeds at the execution of some actions, but not other similar actions. For example,

[12] Surprisingly, single finger tapping was a better predictor of the severity of apraxia than goal-directed grasping and aiming (Ietswaart et al., 2006). Single finger tapping is almost never used to assess apraxia.

in one study the patient was able to evoke some actions (using a razor and a comb) fairly consistently, yet others (hammering and writing) were never produced (Graham et al., 1999). In another study, the same gestures were not always congruently disturbed across the different modes of execution, namely on imitation and on verbal command (Jacobs et al., 1999). We believe that it is this inability to distinguish between different types of errors related to different types of gestures that has prevented us so far from discovering the precise neuroanatomical correlates of apraxia, on top of the difficulty to accurately identify the brain lesion. Hence, the typology and analysis of apraxic errors need to be improved. We encourage extensive categorization of the errors and their characterization via kinematic methods. In addition, the errors should be reported *in relation* to the exact movement and not only specific condition tested.

We also suggest that studies that assess apraxia should more often integrate tasks of motor learning, as patients with apraxia may also be deficient in learning new motor tasks (Heilman, Schwartz, & Geschwind, 1975; Rothi & Heilman, 1984; Platz & Mauritz, 1995; Lausberg et al., 1999). The main motivation in understanding apraxia is to help the apraxic patients in their everyday lives through the development of efficient rehabilitation methods and training programs.[13] Assessing the exact expression of the apraxic deficit, and especially the patient's motor learning abilities, would help to choose an appropriate therapy for the patient. Efficiently targeting the movements and praxis components specifically affected in each patient would accelerate the process of improving his or her praxic faculties. For the moment, apraxia in relation to motor learning is an under-investigated line of research.

Furthermore, we believe that modeling research may prove very helpful to gain some insight into the details and potential implementation of the processes underlying human praxis. When a roboticist searches for an algorithm for his robot to manipulate objects, he or she has to provide with all the different input signals and implement in practice all the necessary computations and processing resources. For example, the differences and similarities between reaching to body-centered versus peripersonal cues would become evident through the development of corresponding algorithms, as they would be explicitly computed. According to Schaal and Schweighofer (2005), computational models of motor control in humans and robots often provide solid foundations that can help us to ground the vast amount of neuroscientific data that is collected today. Thus, biologically inspired modeling studies such as E. L. Sauser and Billard (2006) and Hersch and Billard (2006) seem to be very promising approaches in the understanding of the nature of gestures and in emphasizing the differences and similarities of their underlying processes.

Although neuropsychological models are essential for the understanding of apraxia, they do not address the question of the precise neural representation of

[13] According to Platz and Mauritz (1995), only patients with ideomotor apraxia and not ideational and constructional apraxia could benefit from a task-specific sensorimotor training.

the action and how this representation can be accessed. In a neurocomputational model, one has to take into account the computational principles of movement that reproduce the behavioral and kinematic results of the patient, as well as propose a biologically plausible implementation of the black-box components of apraxia models. In this view, we have a developed a simple neurocomputational model described in Petreska and Billard (2006), that accounts for the callosal apraxic deficit observed in a seminal experimental study of the imitation of meaningless gestures (Goldenberg et al., 2001). Our model combines two computational methods for unsupervised learning applied to a series of artificial neural networks. The biologically inspired and distributed representations of sensory inputs self-organize according to Kohonen's algorithm (Kohonen, 2001) and associate with antihebbian learning (Gerstner & Kistler, 2002). The appropriate transformations between sensory inputs needed to reproduce certain gestures are thus learned within a biologically plausible framework. It is also possible to impair the networks in a way that accounts for the performance of Goldenberg et al.'s apraxic patient in all of the conditions of the study. The model also suggests potential neuroanatomical substrates for this task.We believe that the development of neurocomputational models is a good way to probe our understanding of apraxia and is compatible with the view of integrating knowledge from different lines of research, a point that we will defend in the following section.

2.8.3 Toward a multidisciplinary approach

We believe that apraxia can be best dismantled by adopting a multidisciplinary approach. Future models of apraxia will need to encompass knowledge and data from studies of *normal human motor control*, *human brain imaging* and *monkey brain neurophysiology*. Fortunately, several authors have already attempted to combine different sources of evidence: by considering apraxia in the neurophysiological framework (e.g. R. C. Leiguarda and Marsden (2000)) or by validating a model of apraxia using neuroimaging methods (e.g. Hermsdörfer et al. (2001); Peigneux et al. (2004); Chaminade, Meltzoff, and Decety (2005); Mühlau et al. (2005)).

Normal human motor control has been extensively studied via behavioral, psychophysical, kinematic or computational methods for decades, giving rise to several principles of movement, such as: spatial control of arm movements (Morasso, 1981), maps of convergent force fields (Bizzi, Mussa-Ivaldi, & Giszter, 1991), uncontrolled manifold concepts (Scholz & Schöner, 1999), t-coupling in the perceptual guidance of movements (Lee, Craig, & Grealy, 1999) and inverse and forward internal models (Wolpert & Ghahramani, 2000). Studies of motor control have also inspired several models for reaching like: minimum jerk trajectory control (Flash & Hogan, 1985), vector-integration-to-endpoint model

(Bullock & Grossberg, 1988), minimum torque change model (Uno, Kawato, & Suzuki, 1989) and stochastic optimal feedback control (Todorov & Jordan, 2002); for a review refer to Desmurget, Pélisson, Rossetti, and Prablanc (1998). Proposed models for grasping (e.g. schema design (Oztop & Arbib, 2002)) are reviewed in Jeannerod, Arbib, Rizzolatti, and Sakata (1995), and models for sensorimotor learning (e.g. the modular selection and identification for control model (Haruno, Wolpert, & Kawato, 2001)) in Wolpert, Ghahramani, and Flanagan (2001). In addition, it was also shown that the amplitude and direction of pointing movements may be independently processed (Vindras, Desmurget, and Viviani (2005) or that the kinematics and dynamics for reaching may be separately learned (Krakauer, Ghilardi, & Ghez, 1999). Investigation of apraxia can only benefit from taking into account the rich knowledge of the computational processes of movement used by the brain; and obviously, apraxia models would need to be compatible with the current general theories of movement control.

Progress in describing the contribution of specific brain regions to human praxis through the study of brain-damaged patients has been limited by the variability in the size, location and structures affected by the lesion (Koski et al., 2002). *Human brain imaging studies*, particularly positron emission tomography (PET) and functional magnetic resonance (fMRI) overcome this difficulty to a certain extent and have an essential role in resolving the neuroanatomical correlates of human functions. Despite the evident difficulties and limitations to study movements with neuroimaging, numerous studies have addressed the question of the representation of human praxis, making significant contributions to the understanding of the neural substrates underlying visuomotor control; for a review see Culham, Cavina-Pratesi, and Singhal (2006). In order to give an idea of the number of praxis functions that have been addressed with brain imaging technologies, we will mention some of them: observation of meaningful and meaningless actions with the intent to recognize or imitate (Decety et al., 1997), hand imitation (Krams, Rushworth, Deiber, Frackowiak, & Passingham, 1998), visually guided reaching (Kertzman, Schwarz, Zeffiro, & Hallett, 1997; Desmurget et al., 1999; Grefkes, Ritzl, Zilles, & Fink, 2004), object manipulation and tool-use (Binkofski et al., 2001; Johnson-Frey, Newman-Norlund, & Grafton, 2005), real and/or imagined pantomimes (Moll et al., 2000; Choi et al., 2001; Rumiati et al., 2004) and sequential organization of actions (Ruby, Sirigu, & Decety, 2002). The areas specialized for the perception of body parts and postures have been consistently identified[14] (Peigneux et al., 2000; Downing, Jiang, Shuman, & Kanwisher, 2001). Most importantly, several brain imaging studies have been conducted in relation to apraxia (Hermsdörfer et al., 2001; Peigneux et al., 2004; Chaminade et al., 2005; Mühlau et al., 2005) with the

[14]Interestingly, these occipital and visually specialized areas are not only modulated by the visual presentation of body configurations, but also when the person executes a limb movement (Astafiev, Stanley, Shulman, & Corbetta, 2004), indicating a bi-directional flow of the information.

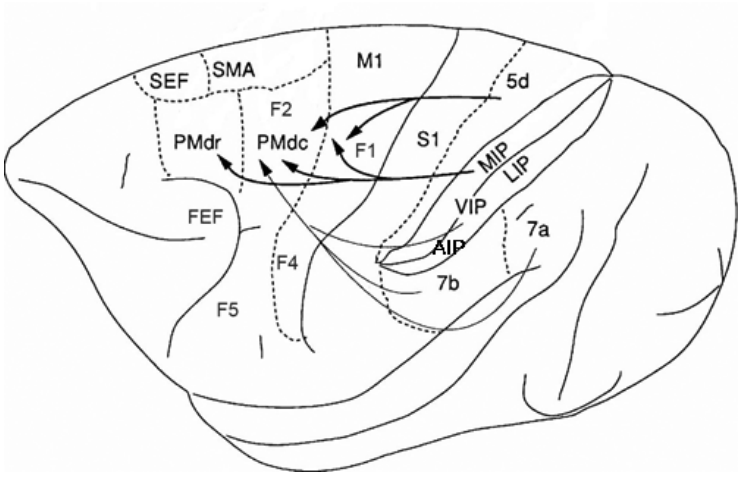

Figure 2.4: Schema of the monkey brain areas and their connectivity. Adapted with permission from Wise et al. (1997).

intent to test the neuroanatomical hypothesis of the neuropsychological models previously described.

Neurophysiological studies allow the investigation of brain processes at the neuronal level and are essential to the understanding of the principles of neural computation. Certainly the monkey brain differs from the human brain, however this discrepancy can be overcome to some extent through the search of homologies (Rizzolatti et al., 2002; Arbib & Bota, 2003; Orban, Van Essen, & Vanduffel, 2004; Sereno & Tootell, 2005). Sensorimotor processes such as reaching and grasping for example, have been extensively studied: several parallel parietofrontal circuits were identified, each subserving a particular sensorimotor transformation (Kalaska, Scott, Cisek, & Sergio, 1997; Wise, Boussaoud, Johnson, & Caminiti, 1997; Matelli & Luppino, 2001; Battaglia-Mayer, Caminiti, Lacquiniti, & Zago, 2003). Without going into the details of the representations used in each of these functionally distinct parietal and frontal areas (illustrated in Fig. 2.4), we will mention those that seem relevant for understanding apraxia. For example, LIP-FEF neurons discharge in relation with eye movements and are sensitive to the direction and amplitude of eye saccades (Platt & Glimcher, 1998), VIP-F4 neurons construct a representation of the "peripersonal space" confined to the head (Duhamel, Colby, & Goldberg, 1998), MIP-F2 neurons have a crucial role in the planning, execution and monitoring of reaching movements (Eskandar & Assad, 1999; Simon, Mangin, Cohen, Le Bihan, & Dehaene, 2002; Raos, Umiltá, Gallese, & Fogassi, 2004) and finally AIP-F5 neurons mediate motor responses selective for hand manipulation and grasping movements (Cohen & Andersen, 2002). Furthermore, multiple space representations appear to coexist in the brain that integrate multisensory inputs (such as visual, somatosensory, auditory and vestibular inputs) (Graziano & Gross, 1998). For example, neurons in area 5 appear to combine visual and somatosensory signals in order to monitor the configuration of the limbs (Graziano, Cooke, &

Taylor, 2000) and the receptive fields of VIP neurons respond congruently (i.e. with matching receptive fields) to tactile and visual stimulation (Duhamel et al., 1998). It is very interesting that the modality-specific activities are spatially aligned: the visual receptive field corresponding to the arm or the face may shift along with that body part when it is passively moved (Graziano, Hu, & Gross, 1997). In addition, neurophysiological data can give us insight into how the arm posture modulates the activity of somatosensory neurons (Tilery, Soechting, & Ebner, 1996) and how it affects the neurons that compute the trajectory of the hand (Scott, Sergio, & Kalaska, 1997). It should be noted that several sensorimotor transformations are needed in order to grasp an object, the motor command being in hand coordinates and the object's location in gaze coordinates. To compute these transformations, the brain appears to use multiple bodycentered frames of references (Graziano & Gross, 1998): the frames of references underlying VIP area neurons appear to be organized along a continuum from eye to head coordinates (Duhamel, Bremmer, BenHamed, & Graf, 1997; Avillac, Denève, Olivier, Pouget, & Duhamel, 2005) and direct transformations from head to body-centered representations are possible in the posterior-parietal cortex (Buneo, Jarvis, Batista, & Andersen, 2002; Buneo & Andersen, 2006) with an error estimate of the target position computed in a common eye reference frame (Batista, Buneo, Snyder, & Andersen, 1999; Cohen & Andersen, 2002). Finally, it was also shown that tools may be integrated into the "body schema" at the neuronal level (Iriki, Tanaka, & Iwamura, 1996; Maravita, Spence, & Driver, 2003).

To conclude, we strongly believe that this multidisciplinary approach should be *bidirectional*. Not only apraxia can be interpreted in the neuropsychological and neurophysiological frameworks, but these research domains would also benefit from taking into consideration observations from apraxia. For example, one could learn enormously on how the normal human praxis system functions by looking at how it is affected by apraxia.

Chapter 3

Neurocomputational Model of an Imitation Deficit Following a Brain Lesion

This work was previously published in:

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This chapter focuses on one particular case study of callosal apraxia by Goldenberg et al. (2001) that raised our interest while reviewing apraxia. This fascinating study shows that the pattern of errors depends on the brain hemisphere that processes the imitation task. Here we develop a neurocomputational model of imitation and simulate a callosal lesion that helps to better understand what might be causing the error pattern observed. Furthermore, the model predictions will be used to guide our neuropsychological investigation of apraxia in Chapter 5.

Abstract

This paper investigates the neural mechanisms of *visuo-motor imitation* in humans through convergent evidence from neuroscience. In particular, we consider an imitation deficit following a *callosal brain lesion*, based on the rational that looking at how imitation is impaired can unveil its underlying neural principles. We ground the functional architecture and information flow of our model in brain imaging studies and use findings from monkey brain neurophysiological studies to drive the choice of implementation of our processing modules. Our neural model of visuo-motor imitation is based on self-organizing maps with associated activities. Patterns of impairment of the model, realized by adding uncertainty in the transfer of information between the networks, account for the scores found in a clinical examination of imitation (Goldenberg et al., 2001). The model also allows several interesting predictions.

3.1 Introduction

Apraxia is generally defined as the inability to perform voluntary movements that cannot be explained by elementary motor, sensory or cognitive deficits (not caused by weakness, ataxia, akinesia, deafferentation, inattention to commands or poor comprehension). A standard test for clinical examinations of apraxia is the *imitation of meaningless gestures* which is believed to test the integrity of a direct route from visual perception to motor control, not mediated by semantic representations or verbal concepts (Poeck & Kerschensteiner, 1971). Goldenberg has shown that knowledge about body parts is also relevant, as apraxic patients were unable to map body configurations to their own body nor to a mannikin (Goldenberg, 1995). Kinematic studies of apraxia show spatial parapraxias (i.e. normal kinematic profiles with abnormal final positions) that seem to arise from a basic deficit that concerns the mental representation of the target position (Hermsdörfer et al., 1996).

Goldenberg's study. A seminal study of the imitation of meaningless gestures examines a patient with a callosal brain lesion or disconnected hemispheres (Goldenberg et al., 2001). The patient was asked to imitate a set of visual stimuli that present different positions of the hand relative to the head (see Fig. 3.1A). To disentangle the contribution of each hemisphere the patient was tested tachistoscopically (i.e. the stimulus was presented either to the left or right visual field) in a left- or right-hand imitation condition. As shown on the Fig. 3.1B the pattern of errors varies as a function of the visual field to which the stimuli were displayed and the hand used to execute the imitative movement. The schema in Fig. 3.1C shows the hypothesized non-uniform information flow across the two hemispheres in the different conditions, related to regions in the brain and based on brain imaging and lesion studies (Decety et al., 1997; Mühlau et al., 2005; Haaland et al., 1999). The stimulus is visually processed in the hemisphere contralateral to the visual field (due to optic chiasm) and the motor command is prepared in the hemisphere contralateral to the hand. The arrows show the necessary transfer of information between the two hemispheres, thus a possible source of spatial errors in the imitation (as the patient suffers from disconnected hemispheres). Imitation was perfect only in the right visual field-right hand condition, indicating a lateralization of the processing to the left hemisphere and a necessary computational process in the brain area shown in dark grey.

3.2 Neurocomputational Model of Imitation

In this paper, we investigate the impaired imitation of meaningless gestures, namely hand postures relative to the head as the one shown in Figure 3.1A. This

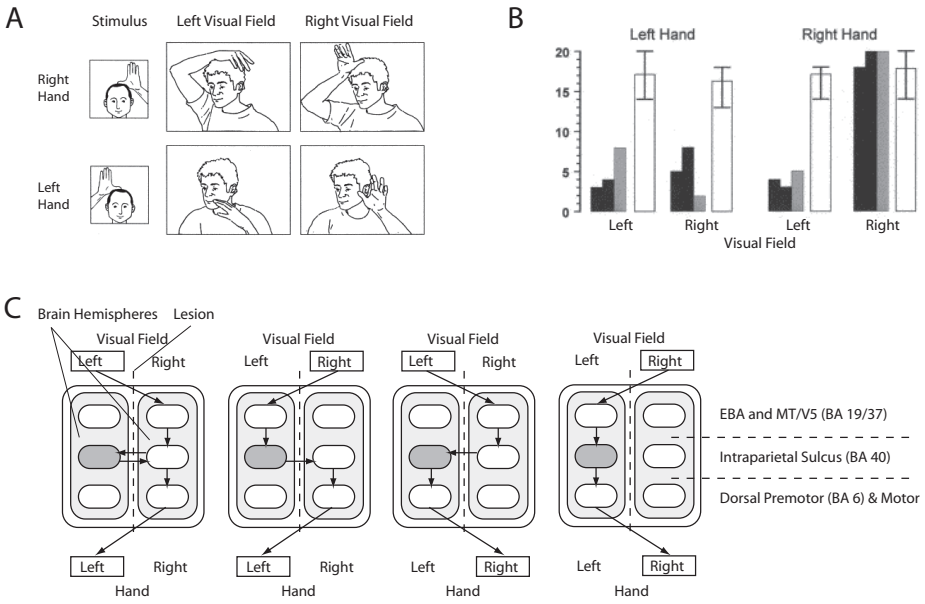

Figure 3.1: **A** *Goldenberg's experiment* of the imitation of meaningless gestures, an example of a visual stimulus to imitate and errors made by the patient. **B** The patient's score of success in the four conditions (several trials, control data in white), taken from (Goldenberg et al., 2001). **C** *Schema of the information flow* across the left and right brain hemispheres in the four conditions, see the text for explanation.

work follows from a general effort in our group to decipher the neural mechanisms of visuo-motor imitation (E. L. Sauser & Billard, 2006; Billard, 2002). In order to model the behavioral data reported in Goldenberg's study, we developed a neural network architecture that accounts for the transformations required to translate the observation of the visual stimulus to imitate to the corresponding tactile and proprioceptive information that will guide the imitative gesture. We simulate a callosal lesion by impairing the transfer of information between the networks and observe the occurrence of spatial parapraxias. Next, we describe the model.

3.2.1 Description of the model

The model is composed of three neural networks, see Fig. 3.2: a *face visual network* in Brodmann Area BA 19/37 at the level of the occipito-temporal junction, a *face somatic network* in area BA 40 in the parietal cortex and a *hand position network* probably in dorsal premotor area BA 6. As is the case in the imitation of meaningless gestures we have implemented a visuo-motor route mediated by somatic knowledge of body parts. The face visual network receives geometrical properties of the visual stimulus to imitate (such as the position and angle of the hand relative to the nose). The face somatic network receives input from the face visual network and somatic input from tactile sensors of the face. The hand position network receives visuo-somatic input from the face somatic

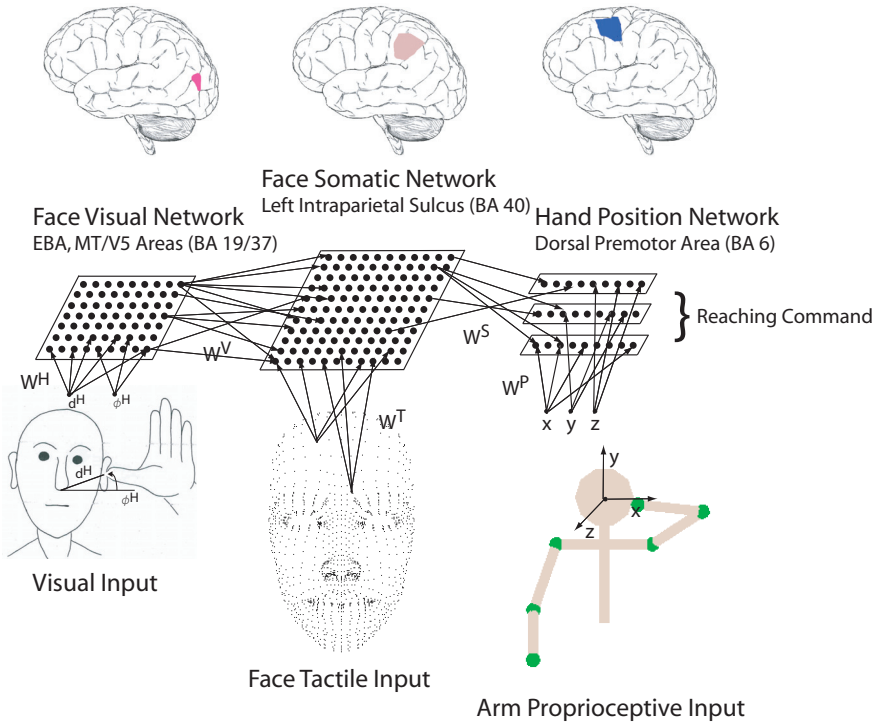

Figure 3.2: Schema of the neurocomputational model. The model is composed of three neural networks that receive visual, tactile and proprioceptive input: a *face visual network* that corresponds to Brodmann Area BA 19/37 at the level of the occipito-temporal junction, a *face somatic network* that corresponds to area BA 40 in the intraparietal cortex and a *hand position network* in area BA6 in the dorsal premotor cortex.

network and proprioceptive input from the arm. The neurons in our model are leaky integrator neurons in order to account for variations of the membrane potential in time and to have integrating properties.

Face visual network. The face visual network encodes geometrical properties of the stimulus to imitate. The network receives the two-dimensional input $\vec{x}^H$ composed of the distance $d^H \in \mathbb{R}[0, 9]$ and angle $\phi^H \in \mathbb{R}[0, 2\pi]$ of the hand relative the nose (shown on Fig. 3.2). We decided to use these two properties as they univocally define the stimulus to imitate and are quantities easy to process visually. It is certain that the brain uses also other quantities when imitating a hand position relative to the head (position relative to the eye may be more appropriate in some cases), however we decided to limit the number of visual properties for simplicity. It was important that the visual and somatic networks rely on completely different representations.

The membrane potential m_i of the visual neuron i is governed by a first order differential equation modulated by a gaussian input:

$$\tau^V \frac{d}{dt} m_i^V = -m_i^V + \mathrm{e}^{-\left(\frac{|\vec{w}_i^H - \vec{x}^H|^2}{2\sigma_V^2}\right)} \tag{3.1}$$

where τ^V is a time constant, $\vec{w}_i^H$ are the synaptic weights that connect the

neuron i to the input $\vec{x}^H = \{d^H, \phi^H\}$ and σ_V corresponds to the "sensitivity" of the neuron to the input (a neuron with a large σ_V responds to a larger interval of inputs values).

The firing rate is a sigmoid function of the membrane potential with slope a and offset b:

$$g(m_i^V) = \frac{1}{(1 + \mathrm{e}^{a(-m_i^V + b)})} \tag{3.2}$$

Face somatic network. The face somatic network is a somatotopically organized network principally processing tactile information from the face. It receives input $\vec{x}^T \in \mathbb{R}^{N^T}[0,1]$ from $N^T = 1500$ tactile sensors non-uniformly distributed on the face (with a preponderant number of sensors around the eyes, nose and mouth). It also receives visual input from the face visual network described previously. The membrane potential m_j^S of a somatic neuron with index j is equal to:

$$\tau^S \frac{d}{dt} m_j^S = -m_j^S + \sum_{k=1}^{N^T} w_{jk}^T \mathrm{e}^{(-\frac{|\vec{x}^P - \vec{r}_k^T|^2}{2\sigma_T^2})} + \sum_{i=1}^{N^V} w_{ji}^V g(m_i^V) \tag{3.3}$$

where τ^S is a time constant, w_{ik}^T is the synaptic weight of the neuron to the tactile sensor with index k and w_{ij}^V is the synaptic weight to the visual neuron with index i, N^T and N^V are the numbers of tactile sensors and visual neurons respectively, $\vec{r}_k^T \in \mathbb{R}^3$ is the position of the tactile sensor k in space, $\vec{x}^P \in \mathbb{R}^3$ is the center position of the hand-face contact and σ_T is the width of the contact. Note that the face somatic network integrates inputs of different types, namely somatic input from the tactile sensors and visual input preprocessed by the face visual network.

Three layers of the *hand position network* encode proprioceptive information from the arm. Each layer encodes a different coordinate of the position of contact $\vec{x}^P \in \mathbb{R}^3$ of the hand and face, expressed in head-centered Cartesian coordinates. Our motivations were the following: there is no "real" proprioceptive information from the face and we hypothesized that this information could be learned from correlations between the face tactile sensory activity and arm proprioceptive activity during reaching movements toward the face. A "positional code" may well be used in the brain where different coordinates are processed in segregated neural substrates, possibly in Cartesian coordinates (Lacquaniti, Guigon, Bianchi, Ferraina, & Caminiti, 1995). The frame of reference is centered in the head to maximize the invariance of the positions of the tactile sensors (which would not be the case in a body-centered frame of reference because of the rotation of the head).

The neurons in the hand position network each have a preferred coordinate value c_k, preferred values were uniformly distributed in a volume that contains the head $\mathbb{R}^3$[-8,8]. The membrane potential m_k^P integrates over the proprioceptive

tive input $\vec{x}^P$ and the visuo-somatic input $g(m_j^S)$ from the face somatic network (the vectorial notation expresses the three layers of the hand position network):

$$\tau^P \frac{d}{dt}\vec{m}_k^P = -\vec{m}_k^P + \mathrm{e}^{-(\frac{|\vec{x}^P - \vec{c}_k|^2}{2\sigma_P^2})} + \sum_{j=1}^{N^S} \vec{w}_{kj}^S g(m_j^S) \tag{3.4}$$

where w_{kj}^S are the weights between a somatic neuron j and a position neuron k, σ_P is the width of the receptive field of the position neuron and N^S is the number of neurons in the face somatic network. The activation function is the same as in equation 3.2. The output of the hand position network is decoded using a weighted average of the firing rates of N^P position neurons, which corresponds to the position $\vec{p}$ on the face:

$$\vec{p} = \frac{\sum_{k=1}^{N^P} \vec{c}_k g(\vec{m}_k^P)}{\sum_{k=1}^{N^P} g(\vec{m}_k^P)} \tag{3.5}$$

The decoded activity of the hand position network is used as a target for the imitation of a visual stimulus.

3.2.2 Training the weights

The synaptic weights between the networks and their sensory inputs (i.e. weights W^H between the face visual network and the extracted visual parameters and weights W^T between the face somatic network and the face tactile input) have been trained with Kohonen's algorithm (Kohonen, 2001). Thus our networks are self-organizing maps (SOMs) whose weights preserve the topology of the input. The unsupervised learning algorithm consists of randomly choosing a sensory input $\vec{x}$ and determining the "winning neuron" with index j^* whose weights are closest to the input. It then updates the synaptic weights of the "winning" neuron and neurons in its neighborhood by the following rule:

$$\Delta\vec{w}_i(j^*) = \epsilon \cdot \mathrm{e}^{-\frac{|i-j^*|^2}{2\sigma_K^2}} [\vec{x} - \vec{w}_i] \tag{3.6}$$

where ϵ is the learning rate, $\vec{w}_i$ are the synaptic weights of the neuron with index i and σ_K corresponds to the size of the neighborhood[1]. After training, stimuli close in the input space are also close in the two-dimensional neural space and more frequent inputs yield larger neural activities.

The synaptic weights between the networks (i.e. weights W^V between the visual and the face somatic network, and weights W^S between the face somatic and the hand position network) were trained with a presynaptic gating anti-hebbian learning rule:

[1]The weights were initialized with random values between 0 and 1 and the parameters ϵ and σ_K were decreased at each step according to the functions given in Table 3.1.

$N^V = 400$	$\tau^V = 35ms$	$\sigma_V = 0.6$	$\sigma_{VK} = 8$	$\epsilon^V = 1$
$N^S = 1225$	$\tau^S = 35ms$	$\sigma_S = 0.3$	$\sigma_{SK} = 22$	$\epsilon^S = 1$
$N^P = 3\text{x}100$	$\tau^P = 35ms$	$\sigma_P = 0.3$		
			$\sigma = l\sigma$	$\epsilon = n\epsilon$
$a = 15$	$\eta = -0.02$			
$b = 0.5$			$l^V = 0.97$	$n^V = 0.98$
			$l^S = 0.9996$	$n^S = 0.99999$

Table 3.1: Parameter values.

$$\Delta w_{i,j} = \eta \cdot x_j[2\sum w_{i,k}x_k - m_i] \tag{3.7}$$

where $w_{i,j}$ is the synaptic weight between a presynaptic neuron x_j and a postsynaptic neuron with membrane potential m_i and η is the learning rate. The learning process associates correlated activities of two networks. The connecting weights learn a mapping between the neural activity of one input and one output network for a given stimulus. In other words the weights organize in order to have the sensory activity in the input network represent the sensory activity in the output network. Both W^V and W^S were trained during the same process of self-observation, which simulates sensory input during reaching movements toward the face in front of a mirror. For example, the activity in the face visual network is associated to the somatic activity due to touching the face and is associated with a position in space through proprioceptive information from the arm. In the end, the presentation of the visual stimulus to imitate alone yields the corresponding neural activities in the face somatic and position networks, thus guiding a correct imitative action. The values used for the parameters of the model were selected by trial and error and are shown in Table 3.1[2].

3.2.3 Simulation of the lesion

In order to simulate the lesion of the corpus callosum (i.e. impaired transfer of information across the two hemispheres) we have taken into account two observations. First, some of the visual information must cross the corpus callosum, since the patient imitates correctly some of the stimuli processed in a different hemisphere than the motor command. Second, interestingly enough, time is a very important variable. If the patients are given "unlimited time", they imitate correctly (Zaidel & Sperry, 1977). To model the observation that some of the information crosses, we introduce a probability of information transfer ρ. The impairment function is either applied at the level of the connection (model 1) or at the level of the neuron input (model 2). To model the improvement of the patient's performance with time we hypothesized an integration factor greater

[2]The inputs selected in the learning processes form a random uniform distribution in the input space. For a faster convergence all the time constants were set to 1. The Kohonen algorithm was run 100 iterations for the face visual network, 9000 iterations for the face somatic network and the anti-hebbian learning process was iterated 5000 times.

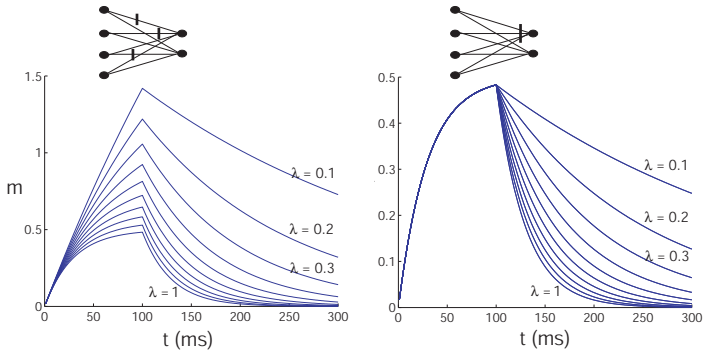

Figure 3.3: The dynamics of the membrane potential of one neuron in model 1 where the lesion impairs the connection (left) and in model 2 where the lesion impairs the neuron input (right). Input $I = 0.5$ was applied during 100ms and τ was set to 30ms.

than the decay factor. We added a constant $\lambda \in \mathbb{R}[0, 1]$, which slows down the membrane decay. The dynamics of the membrane potential m of one neuron for the two models is then expressed by:

$$1)\ \tau \frac{d}{dt} m = -\lambda m + W f(I) \quad 2)\ \tau \frac{d}{dt} m = -\lambda m + f(WI) \quad m < f(WI) \Rightarrow \lambda = 0 \tag{3.8}$$

where W is the weights matrix, I is the membrane input and f is the impairment function such as $f(x) = x$ with probability ρ and $f(x) = 0$ otherwise; see Figure 3.3. Therefore even if the neuron receives bits of information from time to time, the membrane potential is no more precisely tuned to the input but continues to integrate. As the face somatotopic network is situated in the left parietal cortex, we impair the connecting weights W^V in the "left visual field" conditions and the weights W^S in the "left hand" conditions.

3.3 Results

To analyze the performance of our impairment models we have trained the weights once, then quantified the spatial parapraxias as the distance E between the desired end-target position $\vec{r}$ and the position $\vec{p}$ computed from the hand position network under different patterns of impairment[3].

A property of the model is to always converge to the correct response given unlimited time no matter how impaired is the transfer of information, as long as some information does transfer ($\rho > 0$) and λ is small. As shown in Figure 3.4, even for a probability of information transfer as small as $\rho = 0.1$ at the level of the connection, the model converges over time to the correct position given

[3]For a simpler analysis of the results we have impaired all of the connections equally, but our implementation allows variations of the percentage of impairment, as well as of the lesion location and size.

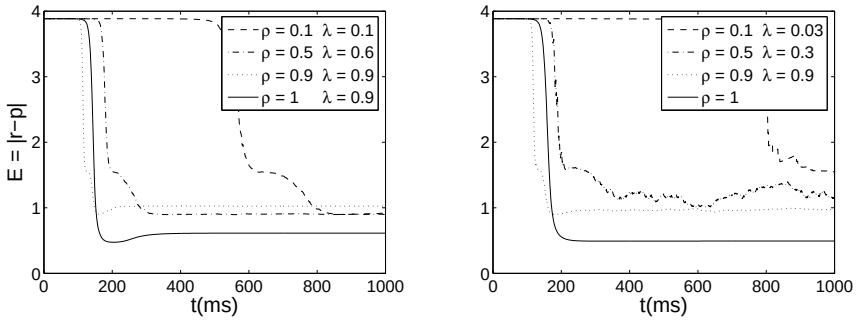

Figure 3.4: The error in imitation computed as the distance between the desired and simulated end-target positions for different values of ρ and λ, according to model 1 (on the left) and model 2 (on the right). The choice of the parameters for the activation function and τ ($\tau = 35ms$) was motivated by the observation that the patient required 180ms of visual stimulus presentation time to be able to imitate. The starting position is the same throughout the trials and corresponds to the origin of the head-centered coordinate axis. We observe that more severe lesions necessitate longer processing time.

a sufficiently small λ (0.1 in model 1 and 0.03 in model 2). The presence of λ deteriorates the performance in the unimpaired situation ($\rho = 1$) in model 1 (see Fig. 3.4) as the neuron membrane "overintegrates" in the first model, as shown on Figure 3.3. Another drawback is that small values of λ render adaptation to a novel stimulus slower. However a longer decay time presents the advantage of having a "fading memory" of the stimulus, the stimulus remains represented in the brain after the presentation time, which is compatible with the occurrence of perseveration errors in experimental studies. Several predictions can be made on the basis of these models. With severe lesions, the patient needs more time to perform a correct imitation (see Fig. 3.4). This suggests that it is possible to obtain a measure of severity of the lesion based on the time needed by the patient to perform the imitation. Small λ values would enable a correct imitation even at very high impairment rates, but would decrease the reaction time.

We compared the simulation results to the scores in Goldenberg's study with some adaptation. As we consider only end-target spatial errors, but not hand posture errors (e.g. orientation of the hand or finger configuration), we took the upper bound of the score used in the original study (2 points for a correct imitation). We replicated the same experimental conditions (i.e. same visual stimuli, 180ms of stimulus presentation and weights impairment coherent with the four conditions). A set of values could explain the scores in the Goldenberg study (see Fig. 3.5). The second model gives slightly better results (not significant).

The representation of different parts of the face in the "face somatic" network is non-uniform: face parts, such as the eyes or mouth, are overrepresented in contrast to the cheek or chin. This is due to the non-uniform distribution of the tactile sensors. Therefore we observe inhomogeneities in the imitation precision and processing times (see Fig. 3.5). Interesting predictions can be made from focal rather than diffuse lesions (i.e. stroke vs degenerative lesions). If only

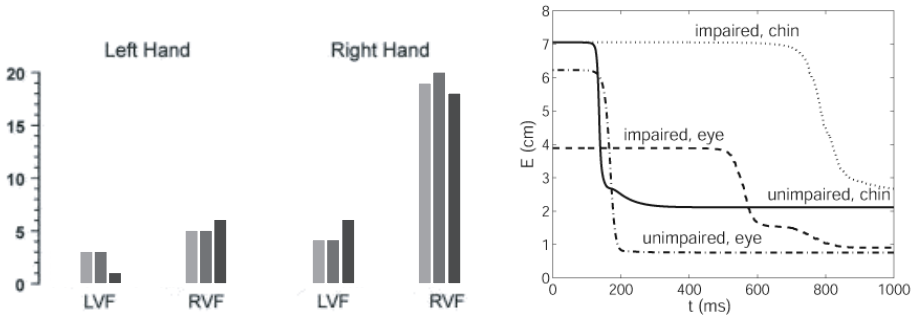

Figure 3.5: Left, comparison between the results from Goldenberg's study (light grey histograms) and the simulation results using the impairment model 2 (dark grey, $\tau = 30ms$, $\rho = 0.5$, and $\lambda = 0.3$) and model 1 (black, $\tau = 30ms$, $\rho = 0.4$, and $\lambda = 0.4$) respectively. The imitation was considered correct if the error distance was lower than 2.5/1.3. Right, inhomogeneities in the precision and processing times of imitation gestures related to different parts of the face, dependent on their representation in the face somatic network (in our case the eye has a larger representation than the chin).

one part of the information transfer in weights W^V that connect the visual and somatic networks is impaired, then one should observe deficits in imitation solely in some parts of the face. Specific local impairment of the weights W^S that connect the somatic and position networks could provoke errors limited to only one coordinate. For example, if the brain really uses a Cartesian representation in a head-centered frame of reference, then the final position of the hand would be shifted only along a single coordinate axis around the head. Spatial errors made by stroke patients should be used to test the plausibility of the model. However, because of brain reorganization, one needs to look at the impairment in imitation immediately after the lesion. As our model has learning properties, the model could possibly account for some of the effects of brain organization.

3.4 Conclusion

We presented a neural network architecture that could reproduce the deficits in the visuo-motor imitation of meaningless gestures, reported in Goldenberg's seminal study (Goldenberg et al., 2001). We modeled two types of lesions that would affect either the integrative computation of the neuron or the connectivity across the neurons, leading to different predictions. Furthermore, the model makes hypotheses on the types of representation underlying imitation, for which there is as yet no neurological evidence. Further behavioral studies will be required to validate or invalidate the model's hypotheses and predictions.

Chapter 4

Revisiting Callosal Apraxia: the Right Hemisphere Can Imitate the Orientation but not the Position of the Hand

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Biljana Petreska, Aude Billard, Joachim Hermsdörfer and Georg Goldenberg. Revisiting callosal apraxia: the right hemisphere can imitate the orientation but not the position of the hand. *Neuropsychologia*, accepted.

In the previous chapter we proposed a neurocomputational model of a seminal study of imitation of meaningless gestures (Goldenberg et al., 2001). Our model could account for the pattern of errors made by an apraxic patient with callosal lesions (disconnected brain hemispheres). Importantly, the model made several interesting predictions that could only be validated or invalidated against the exact errors that the patient made, rather than the synthetic statistical description given in Goldenberg et al. (2001). Therefore, we contacted the authors of the modeled study, who kindly agreed to share their original data. In this chapter we present the results of our analysis of the data.

Abstract

Callosal disconnection can reveal asymmetrical contributions of the two brain hemispheres to praxis. In this paper, we revisit a study of a patient with callosal disconnection (Goldenberg et al 2001, Neuropsychologia, 30: 1432-1443), who perfectly imitated meaningless hand positions when imitation was controlled only by the left hemisphere, but was severely impaired when the the right hemisphere was in charge of motor control. We decomposed the imitation task into a set of geometric variables that were to be reproduced. These geometric variables include the orientation of the hand and the position of contact between the hand and face. Whereas orientation of the hand in extrinsic coordinates

was replicated correctly by both hemispheres, only the left hemisphere reproduced correctly the position of contact between the hand and the face. This goal dissociation as well as several partial perseveration errors speak against the hypothesis of a direct route from perception to motor replication of gestures, as interruption of a direct route would probably impair all the features of the gesture. We speculate that incorrect coordination between the reproductions of multiple goals may be the core deficit underlying callosal apraxia.

4.1 Introduction

Apraxia refers to a disorder of the high-level control of voluntary movement and is described in terms of the absence of certain deficits rather than in terms of its mechanisms, which are still poorly understood (Petreska, Adriani, Blanke, & Billard, 2007). Specifically, apraxia is defined as "a disorder of skilled movement not caused by weakness, akinesia, deafferentation, abnormal tone or posture, movement disorders such as tremor or chorea, intellectual deterioration, poor comprehension, or uncooperativeness" (Heilman & Rothi, 1993). Since the apraxia's symptoms exclude elementary sensory and motor deficits, apraxia is particularly appropriate for studying the neural processes underlying specific sensori-motor transformations, such as visuo-motor imitation. Case studies of impaired imitation following apraxia, such as the one revisited here, offer a valuable route to deciphering the neural functions and mechanisms of imitation (Goldenberg & Hagmann, 1997; De Renzi et al., 1982; Ochipa et al., 1994; Merians et al., 1997; Tessari, Canessa, Ukmar, & Rumiati, 2007). In particular, they have revealed important dissociations between the imitation of meaningless and meaningful gestures, and elements of the hemispheric specialization of praxis (e.g. left hemisphere dominance); see Petreska et al. (2007) for a review.

This paper revisits a case study of imitation of meaningless gestures following callosal lesions or disconnected brain hemispheres (Goldenberg et al., 2001). This seminal study examined a patient during reproduction of hand postures relative to his face (shown in Fig. 4.1). In order to disentangle the contributions of each brain hemisphere, the stimuli to imitate were presented tachistocopically; that is, the stimuli were presented for a very short period of time either in the left or right visual field. In both cases, the patient was requested to imitate alternatively with either the left or right hand (see Fig. 4.2A). A quantitative analysis of the patient performance revealed that only the imitation of hand postures was preserved in the *"right visual field - right hand"* condition; that is, when the information flow was processed solely by the left hemisphere. This observation suggests that the visuo-motor imitation of meaningless gestures requires key competences located in the left hemisphere (see Fig. 4.2B).

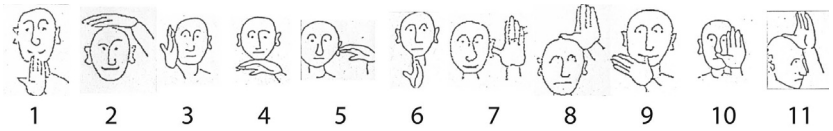

Figure 4.1: The visual stimuli used in the tachistocopic examination of imitation of meaningless gestures.

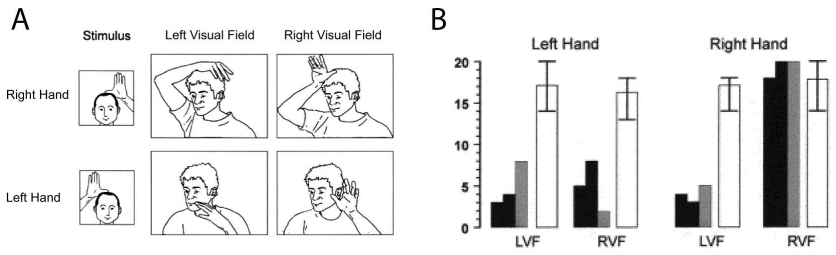

Figure 4.2: **A**, Tachistocopic examination of the imitation of meaningless gestures. The patient is asked to imitate a visual stimulus in four conditions: the stimulus is presented either in the left or right visual field and imitated with either the left or right hand. **B**, The patient's score of success in the four conditions (two trials) compared to control data (in white). Note that there is only one condition where the imitation is preserved, namely the *right visual field-right hand condition*. Note also that the performance is above zero in all the conditions, indicating that some information does cross between the two brain hemispheres. These figures are adapted from Goldenberg et al (2001).

In a previous work, we proposed a neuro-anatomical model for the information flow across hemispheres during the imitation of meaningless gestures (Petreska & Billard, 2006). The model made predictions on the brain areas and neural processes that support this information flow, based on evidence from brain imaging and brain lesion studies (see Fig. 4.3). According to this model, a gesture demonstrated within one visual hemifield will be first processed visually in the contralateral hemisphere, possibly in the "Extrastriate Body Area" situated at the occipito-temporal junction in Brodmann Area (BA) 19/37. This area is specialized in the visual processing of pictures of human body or body parts (Peigneux et al., 2000; Downing et al., 2001; Astafiev et al., 2004). A further crucial station on the way from visual perception to the motor replication of gestures is located in the left parietal cortex. PET and fMRI brain neuroimaging studies have demonstrated that left parietal activation (BA 40, near the intraparietal sulcus) is induced by the observation or imitation of meaningless gestures (Decety et al., 1997; Hermsdörfer et al., 2001; Peigneux et al., 2004; Mühlau et al., 2005), which provides the empirical data for the present investigation. These functional imaging studies are complemented by lesion studies that also suggest a left-hemisphere dominance for the imitation of meaningless gestures (Goldenberg, 1996; Hermsdörfer et al., 1996; Weiss et al., 2001), where the inferior parietal lobe is a common denominator to all the observed lesions (Haaland et al., 2000; Goldenberg & Karnath, 2006; Tessari et al., 2007). Finally, neural activation is consistently observed in a motor brain area contralateral to the hand, namely the dorsolateral premotor cortex (BA 6) (Decety et al., 1997; Hermsdörfer et al., 2001; Peigneux et al., 2004; Mühlau et al.,

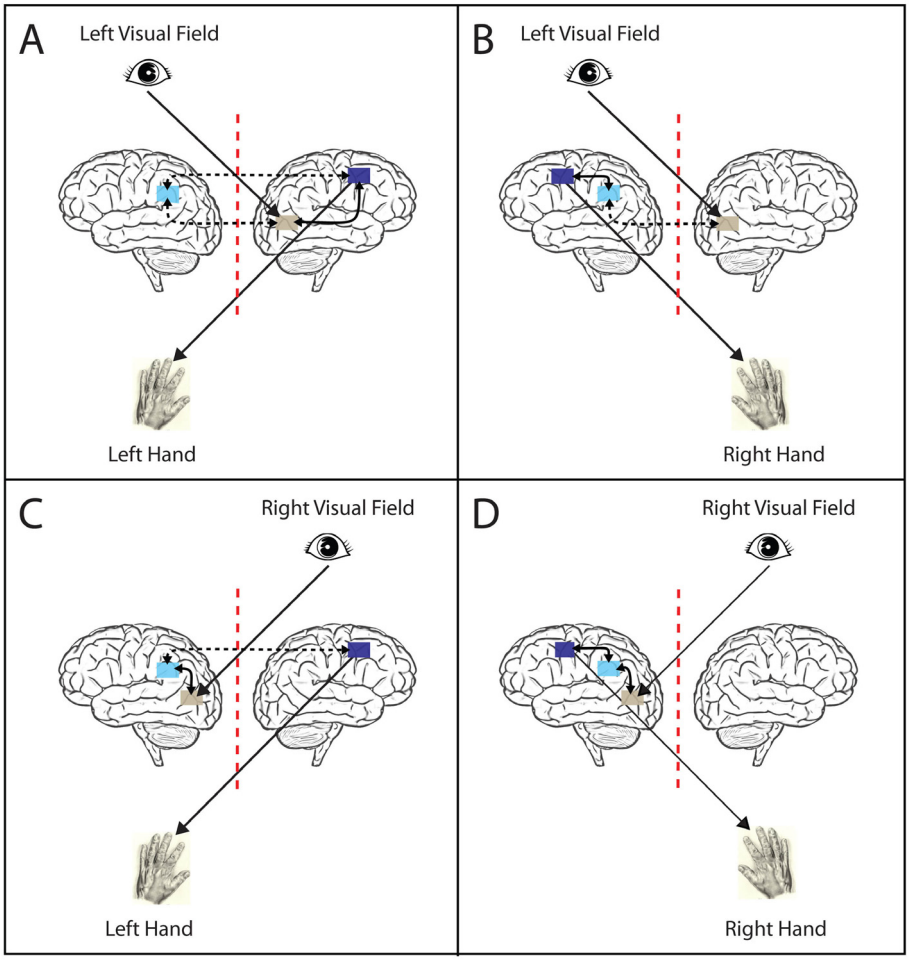

Figure 4.3: A neuro-anatomical model of the neural processes underlying imitation of meaningless gestures. The information flow is shown for the 4 tested conditions. First, the stimulus to imitate is processed visually by the contralateral "Extrastriate Body Area" located at the level of the occipitotemporal junction (BA 19/37). Second, the stimulus is translated into its motor counterpart with the participation of the left intraparietal sulcus (BA 40). Finally, the dorsolateral premotor cortex (BA 6) contralateral to the hand implements the motor component of imitation, possibly through motion attractors.

2005). This brain area may implement the motor command of the hand in the form of a motion attractor, where the imitation is guided by a representation of the target hand posture (Petreska & Billard, 2009).

However, the statistical analysis of the patient's errors in Goldenberg et al. (2001) is not sufficient to pinpoint the exact nature of the contribution of the left hemisphere (see Fig. 4.3). While we know that there is an indispensable left-lateralized process at the level of the intraparietal sulcus, we do not know for what this process is specialized.

To better delineate the function of this neural process, we re-analyzed the patient's errors by defining a set of geometric variables that the brain must reproduce in order to correctly perform the imitation task. We thus exploit the geometric nature of the stimuli used in the experiment and study the pattern of

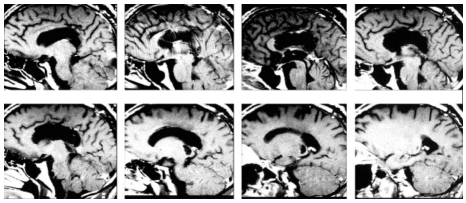

Figure 4.4: T1 weighted MRI images. Sagittal slices are ordered from right-to-left demonstrating damage to the corpus callosum and to the left posterior thalamus. Reprinted from Goldenberg et al (2001) by permission of Elsevier.

errors using objective criteria (e.g. distance between the hand and the position of contact with the face, orientation of the hand). Indeed, the visual stimuli used do not require action-related semantic knowledge. We hypothesized that a finer analysis of the error pattern for each condition and geometric variable would allow us to: (a) better understand the role that each hemisphere plays in the process of imitation of meaningless gestures, (b) explain the fact that imitation of only some stimuli is impaired (see Fig. 4.3B) and (c) highlight the elements of the gesture, if any, that are incorrectly reproduced. By considering the errors with respect to the specific stimulus imitated, we could disentangle a possible deficit underlying callosal visuo-imitative apraxia.

4.2 Methods

4.2.1 Case Report

The patient, PU, had suffered a spontaneous bleeding from an arterio-venous malformation in the territory of the anterior cerebral artery. The malformation was surgically resected but the operation resulted in destruction of middle and posterior third of the corpus callosum, including the splenium. He was seen two years after the operation, at the age of 36. MRI showed destruction of truncus and splenium coroporis callosi with visible preservation of some fibres in the central and parietal portion (Fig. 4.4). On the left side the lesion extended into the posterior thalamus affecting the pulvinar and the nucleus ventrolateralis posterior. However, callosal transfer of information was not completely interrupted, as evidenced by above chance performance on the imitation of meaningless gestures test, as evidenced by above chance performance on the imitation of meaningless gestures test (see Fig. 4.2B). For a detailed description of the patient's performance on various neuropsychological tests refer to Goldenberg et al. (2001).

4.2.2 Procedure

The subject was seated in front of a computer monitor. At the start of each trial a central fixation point was shown for 1000 ms. Then, the stimulus for imitation appeared either in the left- or the right-visual field. Stimuli subtended a visual angle of 8°x8° and their medial edge was 2° lateral from fixation. Duration of presentation was 150 ms for controls and 180 ms for PU who in preliminary trials with 150 ms ascribed his difficulties to the shortness of presentation and asked for a longer duration of the stimuli. Fixation was controlled by an experimenter standing behind the monitor. Within each block of trials the same hand was examined, while the laterality of stimulus presentation varied randomly.

4.2.3 Stimuli

The stimuli used for the study depict meaningless postures of the hand relative to the head with line drawings (Fig. 4.1). A left hand was shown for right-handed imitation and vice versa. Eleven different gestures were presented at least once and at most three times, in both the right and left visual fields.

4.2.4 Data Analysis

For fine-grained error analysis we broke down the visual stimuli into five objective geometric variables, which taken in combination entirely define the stimulus (as in Chapter 4). We used these geometric variables as criteria for scoring the patient's performance. The variables can be classified into two separate measures of how well the stimulus was reproduced (see Fig. 4.5):

1) Geometric variables related to the position of contact between the face and hand (PC), which include:

a) PC-F, position of contact on the face. A score of 0 was given whenever the patient was not touching the correct part of the face (e.g. the patient is touching the nose instead of the ear), where the face was subdivided into the following parts: nose, mouth, chin, cheek, forehead and ear. A score of 1 was given whenever the patient was touching the correct part of the face but not at the correct location (e.g. the tip of the nose was touched instead of its base). A score of 2 was given when the correct part of the face was touched at the correct location. The PC-F variable relates to visually identifying the part of the face involved in the contact, finding its tactile counterpart representation and determining its current spatial position vector, which then guides the reaching component of the imitative movement.

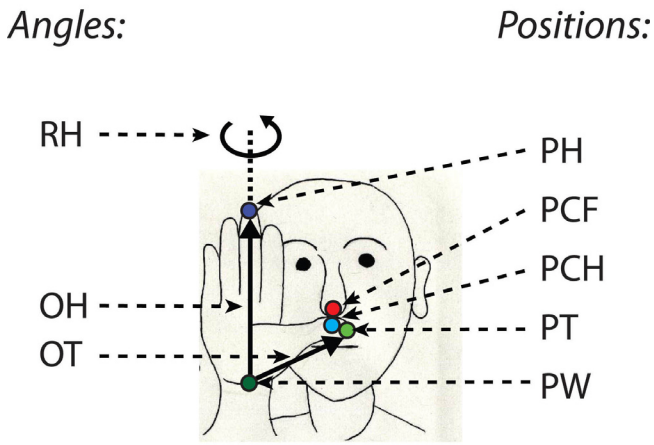

Figure 4.5: Geometric variables used for scoring the patient's performance. Two variables are related to the position of contact between face and hand (PC): PC-F stands for position of contact on the face and PC-H for position of contact on the hand. Three variables are related to the posture of the hand (HP) and are expressed in angles: HP-OH is the orientation of the hand (relates the position of the tip of the hand HP-PH and the position of the wrist HP-PW), HP-OR is the angle of rotation of the hand and finally HP-OT is the orientation of the thumb (relates the position of the tip of the thumb PT and the position of the wrist HP-PW).

b) PC-H, position of contact on the hand. A score of 0 was given whenever the patient was touching the face with an incorrect part of the hand (e.g. with a fingertip instead of the thumb), where the hand was subdivided into the following parts: thumb, 4 fingers, palm and back of the hand. A score of 1 was given when the hand-part was correct but the location was wrong (e.g. the patient touched the face with the tip of the thumb instead of the side of the thumb). Finally, a score of 2 was given when the face was touched with the correct part of the hand and at the correct location within this part. The PC-H variable relates to visually identifying the part of the hand involved in the contact, finding its tactile counterpart representation and determining its current spatial position vector, which then guides the reaching component of the imitative movement.

2) Geometric variables related to the hand posture *per se* (HP), which include:

c) HP-OH, orientation of the hand in extrinsic space, defined as the vector between the position of the tip of the middle finger (HP-PH) and the position of the wrist (HP-PW). For example, a vertical orientation of the hand would mean that the HP-PH and HP-PW vectors differ only in their vertical components in Cartesian space. We gave a score of 0 when the angle between the imitated and correct orientations of the hand exceeded 45 degrees (e.g. the hand was horizontal instead of vertical), a score of 1 when it was between 30

and 45 degrees, and a score of 2 when the discrepancy was less than 30 degrees. The variable HP-OH relates to visually identifying the orientation of the hand (i.e. relationship between the tip of the middle finger and the wrist) and effects the hand flexion/extension and abduction/adduction degrees of freedom.

d) HP-RH, angle of rotation of the hand about the axe of the forearm. A score of 0 was given whenever the hand of the patient was rotated more than 45 degrees away from the correct rotation angle (e.g. the back of the hand is visible instead of the front or the side), a score of 1 when the angle of hand rotation was between 30 and 45 degrees, and a score of 2 when the angle of hand rotation was less than 30 degrees of the correct hand rotation angle. The variable HP-RH relates to visually identifying the rotation of the palm and sometimes involves fine or indirect visual analysis, such as the presence or absence of finger nails and orientation of the thumb. This rotation angle is then used as a control signal for rotating the hand around the axe of the forearm.

e) HP-OT, orientation of the thumb in the extrinsic space, defined as the vector relating the position of the thumb (HP-PH) and the position of the wrist (HP-PW). This variable HP-OT was scored as in d). Note that for the first half of the stimuli the thumb orientation was considered irrelevant (see Fig. 4.1). The variable HP-OT relates to visually identifying the orientation of the thumb (i.e. relationship between the tip of the thumb and the wrist) and controls the thumb flexion/extension and abduction/adduction degrees of freedom.

4.2.5 Statistical Analysis

A three-factor analysis of variance (ANOVA) was performed on the patient's score with the following within-subject factors: "condition" (4 levels: LL, LR, RL and RR), "geometric variable" (5 levels: PC-F, PC-H, HP-OH, HP-RH and HP-OT) and "stimulus" (11 levels). Two-factor analyses of variance (ANOVA) were introduced to explain interactions. In subsequent steps we considered each condition separately and compared the patient's behavior with normal behavior along the factors "geometric variable" and "stimulus" using t-tests. We also performed a new set of similar analysis by grouping the levels of the factor "geometric variable" into a new factor "goal" (2 levels, position of contact between the hand and the face PC and the hand posture HP) and the levels of the factor "stimulus" into a new factor "thumb" (2 levels, stimuli 1-6 where the thumb is not shown vs stimuli 7-11 where the thumb is relevant).

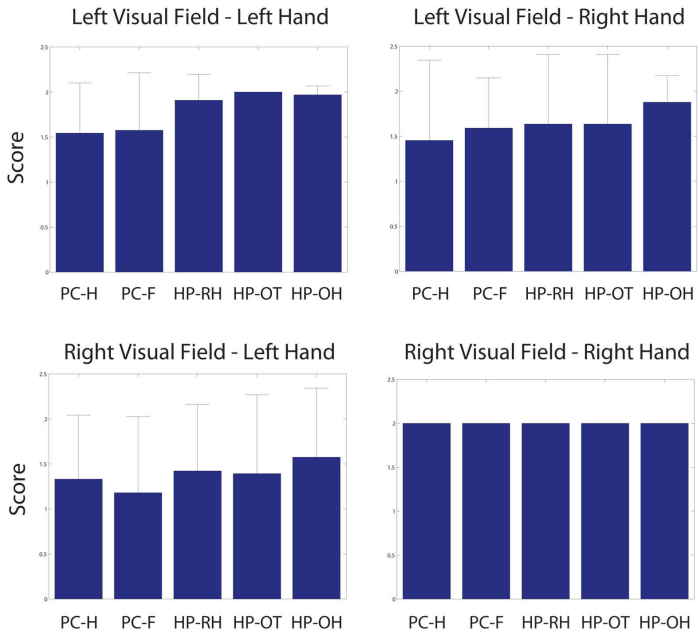

Figure 4.6: Scores per measured variable given for each condition, averaged over trials and stimuli. PC-H stands for position of contact on the hand, PC-F for position of contact on the face, RH for rotation of the hand, HP-OT and HP-OH stand for orientation of the thumb and hand respectively.

4.3 Results

4.3.1 Quantitative Analysis of the Errors

The three-way analysis of variance on the patient's performance showed that all three factors had significant effects. A main effect of "condition" indicated that the crossed conditions LR and RL were significantly more impaired than the ipsilateral conditions LL and RR ($F_{3,120} = 26.27$, $P < 0.001$). Note that PU's score in the RR condition was perfect. A main effect of "geometric variable" indicated that the position of contact located on the hand PC-H was the most affected geometrical variable, followed by the position of contact located on the face PC-F ($F_{4,120} = 5.94$, $P < 0.001$). A main effect of "stimulus" indicated that not all of the imitation gestures were similarly affected ($F_{10,120} = 12.8$, $P < 0.001$). We found one significant interaction effect between the factors "condition" and "stimulus" ($F_{30,120} = 6.89$, $P < 0.001$) indicating that the gestures were not equally impaired across conditions.

Effect of the geometric variable

Figure 4.6 shows the scores per geometric variable measured for each condition. For the following steps of statistical analysis we removed three stimuli

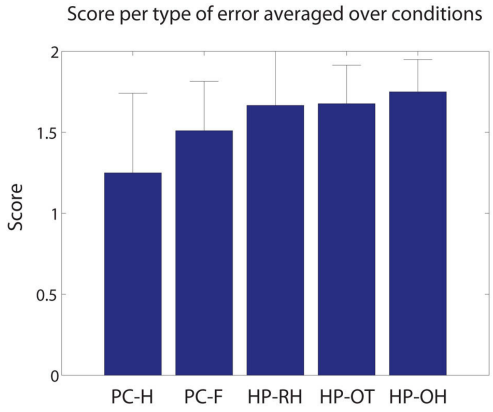

Figure 4.7: Gradation of the score per measured variable, averaged over all the trials and three impaired conditions. PC-H stands for position of contact on the hand, PC-F for position of contact on the face, HP-RH for rotation of the hand, HP-OT and HP-OH stand for orientation of the thumb and hand respectively. PC-H and PC-F are related to the position of contact PC between the face and the hand, while HP-RH, HP-OT and HP-OH are related to posture of the hand HP. Stimuli 2, 4 and 6 and condition RR that did not yield any errors were removed as they were considered uninformative.

(n° 2, 4 and 6) as these stimuli had perfect scores and were thus considered uninformative. We also discarded the data from condition RR where PU scored at ceiling.

Each of the geometric variables measured was impaired and we observed a gradation in their respective scores (see Fig. 4.7). We first compared the patient's behavior with normal behavior (behavior from condition RR) using paired t-tests for each "condition" and "geometric variable". The results indicate that the variables PC-H and PC-F, related to the position of contact between the face and hand, were significantly affected in all conditions. The variables HP-RH, HP-OT and HP-OH related to the posture of the hand were only affected in condition RL.

Based on these results, we grouped the geometric variables into a new factor "goal" with 2 levels: position of contact (PC) and hand posture (HP). For each condition we performed a 2-way ANOVA with this new factor and the factor "stimulus". We observed a main effect of the factor "goal" in conditions LL ($F_{1,24} = 19.52$, $P < 0.0005$) and LR ($F_{1,24} = 12.54$, $P < 0.002$) implying that in the left visual field conditions, the imitation of the position of contact was significantly more impaired than the imitation of the hand posture. There was also a main effect of "stimulus" in the crossed conditions LR ($F_{7,24} = 19.86$, $P < 0.0001$) and RL ($F_{1,24} = 7.09$, $P = 0.0001$) that interacted significantly with the factor "goal" in LR ($F_{7,24} = 3.81$, $P < 0.01$).

Finally, we conducted paired t-tests for comparison of each "goal" to the performance in RR. The results show that the position of contact was significantly impaired in all of the conditions: LL ($T_30 = -3.38$, $P = 0.002$), RL ($T_30 = -4.4$, $P < 0.0001$) and LR ($T_30 = -3.6$, $P < 0.0001$). The hand posture was significantly impaired only in the crossed conditions RL ($T_46 = -4.3$, $P < 0.0001$) and LR

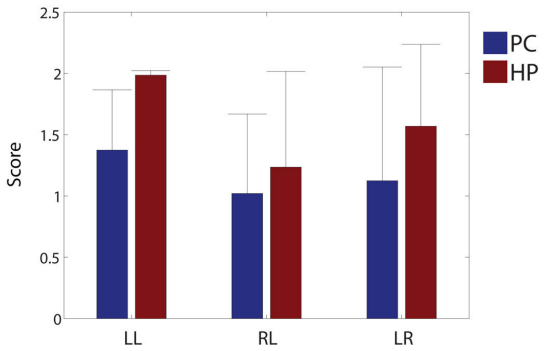

Figure 4.8: Scores per goal given for each condition, averaged over trials and stimuli. The goal PC related to the position of contact between the face and the hand regroups the geometric variables PC-H (position of contact on the hand) and PC-F (position of contact on the face). The goal HP related to the posture of the hand regroups the geometric variables HP-RH (rotation of the hand), HP-OT (orientation of the thumb) and HP-OH (orientation of the hand). The PC scores were significantly impaired in all conditions, whereas the HP score was high in condition LL, leading to a dissociation in performance in this condition. Stimuli 2, 4 and 6 and condition RR that did not yield any errors were removed as they were considered uninformative.

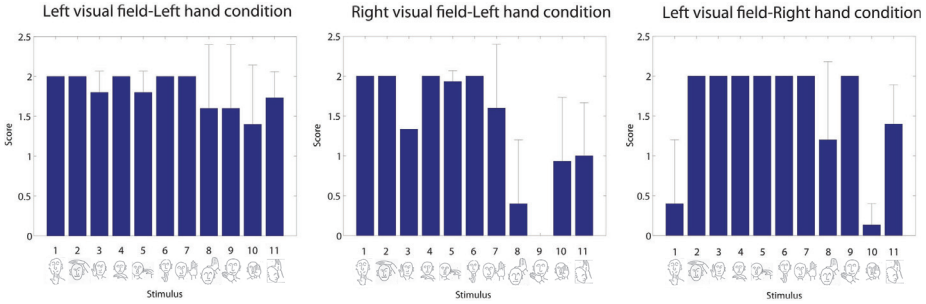

Figure 4.9: The score per stimulus given for each condition, averaged over the geometric variable, showed that not all stimuli are equal in the face of apraxia.

(T_46= -2.67, $P < 0.02$), but not in the LL condition ($P > 0.1$) giving rise to a dissociation (see also Fig. 4.8).

Effect of the stimulus

To explore the significant interaction between the factors "stimulus" and "condition" we performed one-way ANOVAs for the "stimulus" factor for all of the conditions except RR. The results showed a significant effect of the factor "stimulus" in condition RL ($F_{10,44} = 9.43$, $P < 0.0001$) where stimuli 8 and 9 were imitated significantly worse than the other stimuli, and in condition LR ($F_{11,26} = 9.43$, $P < 0.0001$) where stimuli 1 and 10 were imitated significantly worse than the other stimuli (see Fig. 4.9).

We then grouped the stimuli into two groups according to whether the orientation of the thumb was visible (stimuli 7-11) or not (stimuli 1-6). We compared these two groups with a paired t-test and found significant differences in the left hand conditions (LL: T_53= 2.1, $P < 0.05$, RL: T_53= 6.23, $P < 0.0001$ and

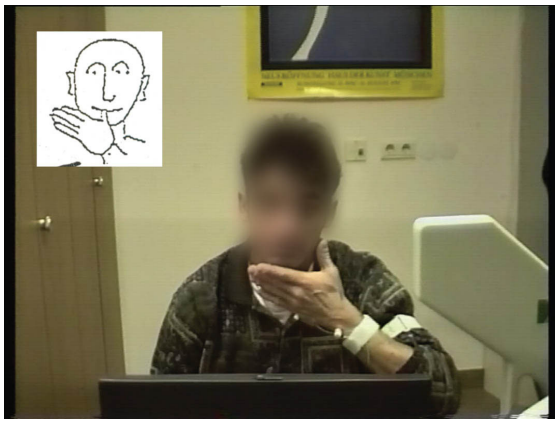

Figure 4.10: Example of error where the hand posture is imitated correctly, but not the position of contact between the face and the hand.

LR: $P > 0.5$). The performance was much worse in gestures that involved the thumb. Finally, stimuli 2, 4 and 6 were perfectly imitated in all conditions (see Fig. 4.9).

4.3.2 Discussion of the quantitative analysis results

Discussion of the geometric variable effect

The effect of the factor "geometric variable" in the left visual field-left hand condition uncovered a dissociation between the preserved imitation of hand postures and impaired imitation of positions of contact between the face and hand. We give an example of this type of error in Figure 4.10. This dissociation has important theoretical implications.

Theories of action imitation vary in their emphasis on either sensori-motor or cognitive connections between the model and its imitation (Rumiati & Bekkering, 2003). The direct matching approach suggests that the perception of an action is directly mapped onto the motor system (Butterworth, 1990; Gray, Neisser, Shapiro, & Kouns, 1991; Brass & Heyes, 2005). Proponents of this theory find support in the experimental observation of so-called mirror neurons that are activated both when a monkey perceives and performs a specific action (Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992; Gallese et al., 1996). By contrast, the "active intermodal matching" (Meltzoff & Moore, 1997) or "body part coding" (Goldenberg et al., 2001) models propose that perceived gestures are transferred into a supramodal code based on a classification of body parts, and that motor imitation uses this interpolated common code rather than direct input from visual perception. Finally, the theory of "goal-directed imitation" postulates that the imitator decomposes the perceived action into a set of goals that are reproduced according to a hierarchy of relative importance

(Bekkering, Wohlschläger, & Gattis, 2000; Bekkering & Wohlschläger, 2002; Wohlschläger, Gattis, & Bekkering, 2003). Our observation of a clear dissociation between preserved imitation of the hand posture and impaired imitation of the position of contact between the hand and face speaks against the direct mapping approach but is compatible with the two other theories. If the visual stimulus is mapped directly onto the motor system as a whole, all of the geometric variables measured would have been similarly affected.

Discussion of the stimulus effect

The patient's performance varied as a function of the stimulus to imitate. As a general rule, the stimuli where the position of contact with the face was on the palm were easier to imitate than those where the contact position was on the thumb. This significant effect of the factor "stimulus" is very important for the assessment of visuo-imitative apraxia. If only movements of the first type were used, the patient would not have been diagnosed as imitation-apraxic. The dependence of performance on the stimuli could be attributed to several aspects of the movement: 1) the number of constraints that define the gesture, i.e. in some stimuli the orientation of the thumb is added as a constraint (in addition to the posture of the hand and position of contact between the hand and face) making the stimulus more difficult to imitate, 2) the degree of "naturalness" of the gesture, e.g. a gesture that one would execute spontaneously in everyday life, and 3) the number of constraints imposed by the biomechanics of the arm (e.g. the number of possible postures of the hand at the top of the head and other workspace limits is lower than in front of the body). In Section 6.6 we will provide an additional and less obvious explanation that is supported by the qualitative analysis of the errors we present next. To summarize, the results indicate that not all stimuli are equal in the face of apraxia.

4.3.3 Qualitative Analysis of the Errors

The above quantitative analysis of errors uncovered *which* of the geometric variables to imitate were impaired. In order to understand how these variables are affected by apraxia, we now turn to a detailed qualitative analysis, where we categorize the nature of observed errors within each condition. This qualitative description of the errors was useful for the characterization of a possible cause underlying the deficit in apraxic imitation.

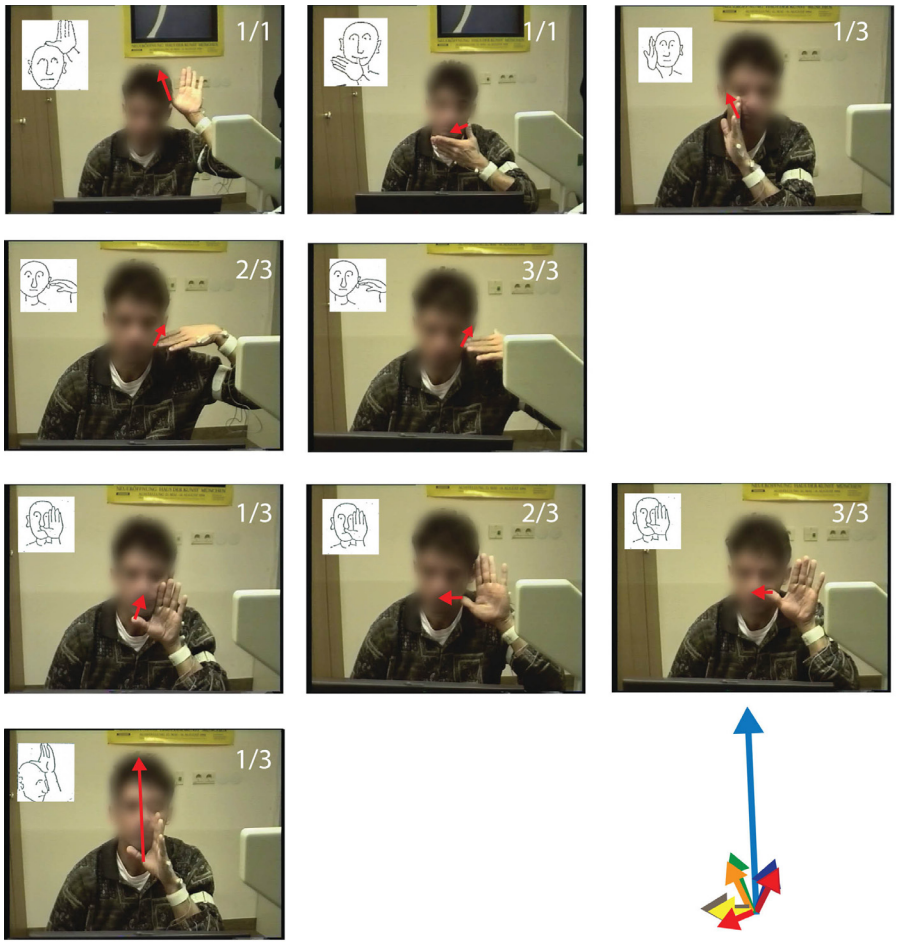

Figure 4.11: All of the errors in the *left visual field-left hand condition*. Note that only the position of contact between the face and hand is affected, whereas the posture of the hand is correctly reproduced. The numbers on the pictures indicate the trial versus the total number of trials for the specific stimulus. The vector deviations from the correct contact position are superimposed in the last quadrant and show a bias that roughly covers an angle of 135° as well as a tendency for under-reaching (the hand is stopped before it gets to the target position).

Left visual field - left hand condition

In Figure 4.11 we show all of the errors that the patient made in the left visual field-left hand condition (LL); that is, errors related to the position of contact between the hand and face, using a correct posture of the hand. According to our model, these errors speak in favor of a specific role of the left hemisphere for the determination of the position of contact (see Fig. 4.3A). We analyzed the deviations of the executed positions of contact and found that the directions and amplitudes of the error vectors are not uniformly distributed around the desired position, but cover a limited sector of 135° on the right part of the frontal plane, and also show a tendency for under-reaching (see the last quadrant of Fig 4.11).

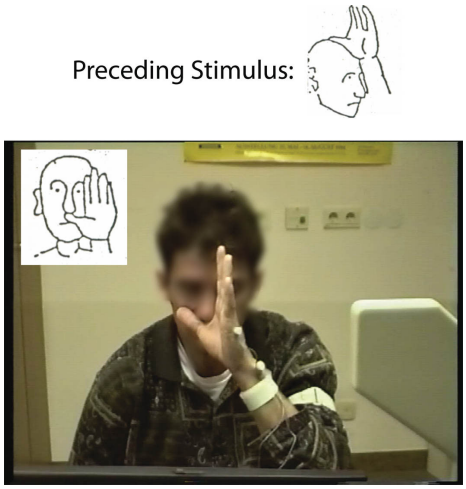

Figure 4.12: A partial perseveration error in the right visual field-left hand condition, where the patient reproduces the posture relative to the previous left visual field-left hand stimulus but at an updated and correct face contact position. Note that within one block of trials the hand was fixed and the visual field was randomly varied, such that the left visual field-left hand and right visual field-left hand stimuli were randomly intermingled within one block of trials (see Section 4.2.2).

Right visual field - left hand condition

In the right visual field-left hand condition (RL) we observed two errors similar to those in condition LL (e.g. incorrect position of contact with a correct hand posture). However, the most prominent errors were perseveration errors, where the previous LL stimulus was either totally repeated (4 cases out of 6) or only the previous LL hand posture was combined with an updated position of contact between the hand and face. In Figure 4.12 we give an example of this particular variant of perseveration errors. Note that the hand was fixed and the visual field was randomly varied within one block of trials, such that LL and RL stimuli were randomly intermingled (see Section 4.2.2). There were also two "no-idea" errors when imitating stimuli n° 3 and 9, where the patient expressed total ignorance about the stimulus to imitate and did not take any action.

Left visual field - right hand condition

In the left visual field-right hand condition (LR) we identified mainly perseveration errors in which the complete previous RR stimulus was repeated (3 times) or the previous RR hand posture was combined with an updated position of contact (1 instance). There were also errors characterized by extensive searching. They consisted of a sequence of up to six distinct simple postures that could not be consistently related to any of the presented stimuli. 3 out of 4 of these errors were observed when the patient imitated stimulus n° 10.

4.3.4 Discussion of the qualitative analysis results

Discussion of the errors in the left visual field - left hand condition

The most informative errors in this study are the errors made in the left visual field-left hand condition (LL). In this condition, both visual processing of the stimulus and motor control of the hand are located in the right hemisphere (see Fig. 4.3A). Thus, the errors in this condition are not influenced by the insufficiency of ipsilateral motor control, but reflect the extent and limits of the right hemisphere competence. When contrasted to the perfect performance in the symmetrical right visual field-right hand condition (RR), these LL errors reveal which contributions depend exclusively on the left hemisphere.

Solely imitation of the position of contact between the hand and face, but not of the hand posture, was affected in the LL condition (see Fig. 4.11). This observation suggests that only the left hemisphere is able to imitate the position of contact between the hand and face, whereas both the right and left hemispheres are capable of controlling the posture of the hand. Note that the position of contact between the face and hand can be defined as a spatial relationship between two body parts. Thus, the selective vulnerability of the position of contact to withdrawal of the left hemisphere contribution would fit well with the hypothesis of a left hemisphere dominance for body part coding (Goldenberg et al., 2001).

Alternatively, incorrect coordination of the reproductions between multiple imitation goals can explain the observed goal-dissociation and qualitative errors shown in Figure 4.11. For example, in order to imitate the stimuli used in this study, the subject needs to reach to a particular point on the face and at the same time put the hand in a particular orientation. Note that these two goals act upon the same end-effector, the hand. If these two goals are satisfied independently, i.e. the movement due to one goal does not take into account the movement initiated to satisfy the other goal, one obtains errors similar to those observed in the LL condition (illustrated in Fig. 4.13). Only the position of contact between the face and hand is really affected by this decoupling or incorrect coordination, as slightly shifting the hand in space has only little influence on the hand posture. Note that the stimuli that present consistent goals, such as stimuli n° 2 and 4 in which touching the face occurs naturally in the hand posture specified by the stimulus, are perfectly reproduced. In addition, incorrect coordination may or may not lead to visible errors depending on the order of execution of the goals (see Fig. 4.13), which would explain why not all of the trials related to an incorrectly imitated stimulus are affected (see Fig. 4.11).

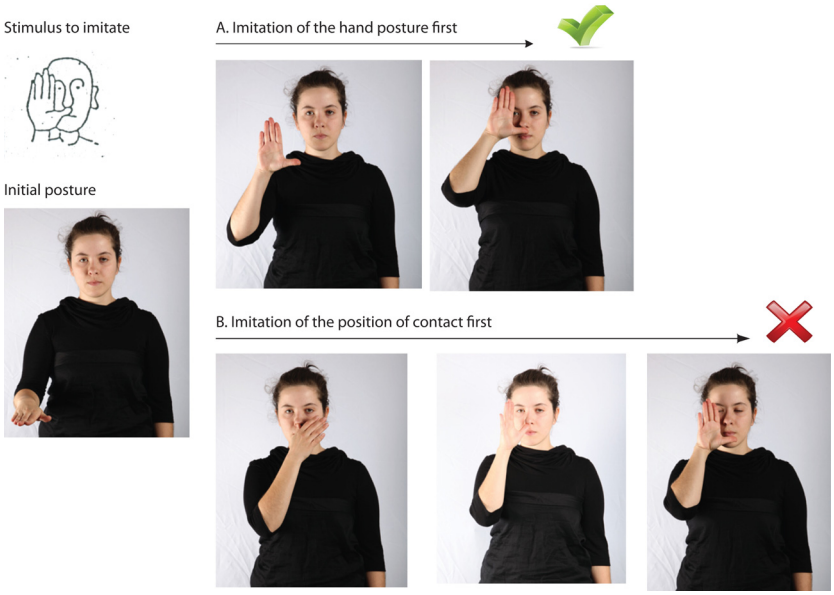

Figure 4.13: An illustration of the errors caused by incorrect coordination between the reproductions of two imitation goals (healthy subject). First row, the subject first imitates the hand posture and then translates the hand to the correct face position leading to a non-observable error. Second row, the subject first brings the hand to the correct position of contact on the face and then imitates the hand posture, this time giving rise to a visible error at the level of the contact position only (the imitation of the hand posture was preserved). Thus incorrect coordination may explain the patient's errors in the left visual field-left hand condition. Interestingly, incorrect coordination leads to observable errors only when the goals are imitated in a particular order, which would explain the differential performance of the patient across trials (see Fig. 4.11).

Discussion of the errors in the crossed conditions

The errors observed in the two other conditions (RL and LR) were expected, as in these conditions the visual information is processed in a different hemisphere than the motor command, which requires transfer of information through the impaired corpus callosum (see Fig. 4.3B and C). Interestingly, the majority of errors in these conditions were total or partial perseveration errors. These perseveration errors and the two "no-idea" errors suggest that the hemisphere controlling the hand did not have access to one or several features of the stimulus.

Whenever the immediately preceding stimulus was still present at the level of the motor command, it was executed instead of the actual stimulus. Occasionally, the previous hand posture was imitated at the correct position of contact, giving rise to partial perseveration errors. This goal-decomposition of the gesture resulted into some very surprising imitations such as the one shown in Figure 4.12, and speaks in favor of goal-directed imitation (see Section 4.3.2).

These partial perseveration errors also suggest that the position of contact was probably processed more quickly or more reliably than the hand posture. As for the extensive searching behavior and hesitations observed in the left visual field-right hand condition, we still do not know from where they arise.

Importantly, errors that could be attributed to incorrect coordination between multiple imitation goals were only observed in the conditions where the patient imitated with his left hand, i.e. conditions in which the motor control area was right-lateralized and did not have reliable access to the information processed in the left hemisphere, possibly in the intraparietal sulcus area (see Fig. 4.3A and C).

4.4 General Discussion

In this paper we re-analyzed the imitation errors of a patient with disconnected brain hemispheres presented in Goldenberg et al. (2001), with respect to *objective geometric criteria* and relative to the *specific stimulus to imitate*. We chose the geometric variables such that when taken in combination they completely define the visual stimulus. Each of these variables measures how well a specific geometric aspect of the stimulus was reproduced. This new analysis of the patient's errors provides additional evidence in favor of the hypothesis of goal-directed imitation. The results also help to better understand the differential performance of imitation relative to specific stimuli, and the contribution of the left hemisphere during imitation of meaningless gestures (see model in Fig. 4.3).

We found that the right hemisphere is capable of reproducing the posture of the hand *per se* but not the position of contact between the face and hand. The errors observed in the left visual field-left hemisphere could be attributed to the incorrect coordination between multiple imitation goals (illustrated in Fig. 4.13). This hypothesis of incorrect coordination not only explains all of the errors observed in condition LL, but also provides an explanation for the differential performance across trials (see Fig. 4.11). Depending on the order in which the two decoupled goals are imitated, one may or may not observe errors. The hypothesis predicts that the patient will not make errors when asked to imitate singles goals: solely a hand posture irrespective of the position of contact with the face, or solely a contact position between the face and hand irrespective of the orientation of the touching hand. These conditions have not been assessed in Goldenberg et al. (2001). However, errors in touching parts of the body including the face (irrespective of the orientation of the touching hand) are a core symptom of autotopagnosia. Autotopagnosia is invariably caused by left parietal lesions and associated with defective imitation of meaningless gestures (e. g. De Renzi and Scotti (1970); Buxbaum and Coslett (2001)). This parallel makes it likely that deficient localization of the contact point between the hand and face can occur also without the additional task demand of coordinating two imitation goals.

Brain imaging and lesion studies have demonstrated the importance of the left inferior parietal cortex (BA40, near the intraparietal sulcus) for processing

meaningless gestures (Decety et al., 1997; Hermsdörfer et al., 2001; Peigneux et al., 2004; Mühlau et al., 2005; Tessari et al., 2007). Following our hypothesis of incorrect cordination between multiple imitation goals, we speculate that this brain area may be responsible for the combination of simultaneous movements, each initiated to satisfy a particular imitation goal (e.g. a posture of the hand, a position of contact). In order to satisfy several imitation goals that act upon the same end-effector, the brain needs to integrate the underlying movements into a single movement (which would require knowledge about the structure of the body and current positions of the body parts). Consistently with this hypothesis, the left intraparietal sulcus has been identified as the neural substrate for both the "body schema" that codes for the dynamic positions of body parts in space (Bonda, Petrides, Frey, & Evans, 1995; Parsons et al., 1995) and "body structural description" that codes for the spatial relationships among body parts (Le Clec'H et al., 2000; Felician et al., 2004; Corradi-Dell'Acqua, Hesse, Rumiati, & Fink, 2008).

Our results do not support the direct matching hypothesis where the perception of the stimulus as a whole activates a corresponding motor representation (Butterworth, 1990; Gray et al., 1991; Brass & Heyes, 2005). The results are, however, compatible with both the proposal of goal-directed imitation (Bekkering et al., 2000; Bekkering & Wohlschläger, 2002; Wohlschläger et al., 2003) and the proposal of body part coding (Goldenberg et al., 2001). Indeed these hypotheses are not mutually exclusive. When humans imitate meaningless gestures their goals are arranged in a hierarchy possibly guided by visual salience. Our results suggest that the orientation of the hand is usually higher in this hierarchy than the position of contact between the face and hand (see Fig. 4.7). Specifically, imitation of the position of contact requires knowledge that provides a classification of the body parts and specifies the boundaries that define them (Goldenberg, 1996, 1999; Goldenberg & Hagmann, 1997; Meltzoff & Moore, 1997; Sirigu, Grafman, Bressler, & Sunderland, 1991). For example, stimulus n° 1 shown on Fig. 4.1 can be summarized as follows: the position of the tip of the hand (a) coincides with the position of the mouth and (b) is above the position of the wrist. Application of knowledge about the structure of the human body helps to reduce the visual features of the demonstrated gesture to a limited number of simple relationships between the demonstrator's body parts. As such, it has the advantage to accommodate novel and meaningless gestures into combinations of familiar elements. The body part coding hypothesis thus bridges the differences in perspective and the differences between the imitatee and imitator body size (Goldenberg, 1996, 1999; Goldenberg et al., 2001), known as the "correspondence problem" (Nehaniv & Dautenhahn, 2001; Alissandrakis et al., 2002; Goldenberg & Karnath, 2006).

Finally, we observed that the errors varied as a function of the stimulus, such that not all stimuli seem equal in the face of apraxia. Our analysis sheds some light on why the patient can imitate only some of the gestures and not

others. We believe that only stimuli that present consistent goals never lead to errors: where touching the face at a certain position is naturally performed with the same posture of the hand that is specified by the stimulus (see Fig. 4.9). Stimuli used during the clinical examination of imitation in apraxia thus need to be chosen carefully.

Chapter 5

Variability and Position Errors in Apraxic Imitation: a Short Report

The question of doubt and uncertainty is what is necessary to begin; for if you already know the answer there is no need to gather any evidence about it. Well, being uncertain, the next thing is to look for evidence [...] and to try to enforce a logical consistency among the various things that you know.

The pleasure of finding things out. Richard P. Feynman.

In the previous chapter we revisited data from Goldenberg's callosal patient. This analysis revealed that the patient imitates using a goal-directed strategy. Furthermore, when the task was processed by the right hemisphere, solely the patient's ability to imitate the position of contact between the hand and face was impaired. This chapter investigates from where these errors may arise, by duplicating Goldenberg's study with three apraxic patients. We find that errors in the position of contact arise only when the patient needs to imitate a specific posture of the hand, indicating that both the spatial representation of the face and motor control of the hand are preserved. In Chapter 3 we hypothesized that brain lesion impairs imitation probabilistically. This chapter tests the validity of that hypothesis by looking at the error variability across the patient trials. We find that our neurocomputational model of imitation needs to be revised, as the observed errors were highly reproducible across trials.

Abstract

This chapter presents the results of two extended neuropsychological experiments of imitation in apraxia that we conducted in collaboration with the Vaud University Hospital Centre. These experiments investigate the nature of apraxic errors in imitation, by assessing the *error variability*. Specifically, we tested three left parietal patients for the repeated imitation of meaningless gestures. We used Goldenberg's visual stimuli, namely hand postures relative to the head. Moreover, we tested the imitation of *partial* versions of Goldenberg's stimuli that represent only a position of contact between the face and hand.

First we show that apraxic errors in imitation are not random but reproducible. Second we find that in the partial imitation condition, the patients perfectly imitate positions of contact. Therefore, position errors in the normal imitation of Goldenberg's stimuli are not due to basic sensory, motor or body representation deficits, but to the complexity of the visual stimuli used. The results are interpreted with respect to the neurocomputational model of apraxia presented in Chapter 3. Specifically, we find that two of the hypotheses underlying our model (i.e. probabilistic lesion and left-lateralized body-schema) need to be revised.

5.1 Introduction

Functions related to voluntary movement are frequently altered following brain lesion, giving rise to *apraxia* - a complex pattern of impairments that has proven difficult to assess or interpret (Petreska et al., 2007). Apraxia is generally defined as "a disorder of skilled movement not caused by weakness, akinesia, deafferentation, abnormal tone or posture, movement disorders such as tremor or chorea, intellectual deterioration, poor comprehension, or uncooperativeness" (Heilman & Rothi, 1993). Apraxia is thus defined in terms of the absence rather than presence of specific deficits, which is due to the poor understanding of this phenomenon. Note that apraxia is a high-level disorder of movement, not caused by elementary sensory and/or motor deficits, such that it specifically effects sensori-motor transformations. *Imitation* is one of the sensori-motor transformations affected by apraxia.

This study investigates two questions. The first question is whether there is a specific imitation deficit in apraxia, or if to the contrary imitation is randomly disturbed. It is essential to answer to this question. In the case that there is a specific imitation deficit, understanding this deficit would help to target the impaired mechanism and affected family of movements. To provide elements of response, we assess the error variability during several trials and several weeks, and across patients. The results are encouraging, since there was a high coherence across the errors within patients.

The second question asked is whether the frequent errors in the position of contact between the face and hand arise from (1) an impaired body schema, i.e. spatial representation of the body in the brain (Pick, 1922; Holmes & Spence, 2004) or (2) an impaired motor command. We thus investigate whether the patients make errors in the position of contact because he or she does not know where the features of their face are located or does not control their hand appropriately. Specifically, we test a baseline condition for the imitation of what we term *partial Goldenberg stimuli*, where solely the position of contact between face and hand needs to be imitated, without the need to reproduce a particular posture of the hand. The results show that neither an impaired body schema

nor impaired motor control can explain the position errors in apraxic imitation, since the patients precisely reach towards their face with both the index finger and thumb.

5.2 Methods

In order to provide more data on the imitation in apraxia, we have conducted extended versions of Goldenberg's experimental studies of the imitation of meaningless gestures (Goldenberg & Hagmann, 1997; Goldenberg et al., 2001; Goldenberg, 2001).

5.2.1 Patients

We test three stroke patients (CM, IB and AL) with large lesions that encompassed the left parietal cortex. All three patients suffered a cerebrovascular accident in the territory of the left internal carotid artery and in particular its sylvian branch.

5.2.2 Experiments

We tested the patients in two different conditions. In the first *normal imitation* condition, the subject imitates Goldenberg's visual stimuli, i.e. hand postures relative to the head. In the second *partial imitation* condition, the subject imitates solely positions of contact between the face and hand, i.e. with no specific hand posture. In both conditions, the subject was seated in front of a computer monitor and asked to imitate the displayed visual stimulus with the left non-paretic hand.

Experiment A: normal imitation condition

In the normal imitation condition, we presented 10 visual stimuli of head postures relative to the head (shown in Fig. 5.1). Each stimulus was presented three times with randomized repetitions. The subject was given unlimited time to imitate and stopped the imitation whenever he or she felt satisfied with their posture. This is a significant extension of the original experiment, since we allowed for corrections. The idea behind unlimited time was to determine the lower bound on the patient's capacity to imitate, regardless of short-term memory issues and stress. When possible, we re-tested the patient two weeks later (patients CM and IB).

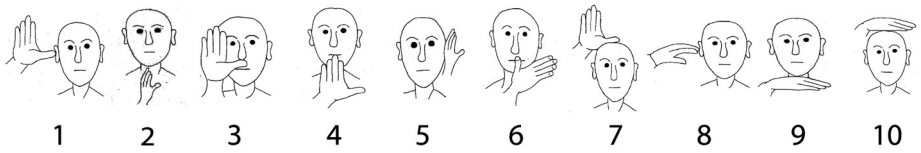

Figure 5.1: Goldenberg's visual stimuli used in the neuropsychological experiments of imitation of meaningless gestures, i.e. hand postures relative to the face. We used these stimuli in the *normal imitation* condition.

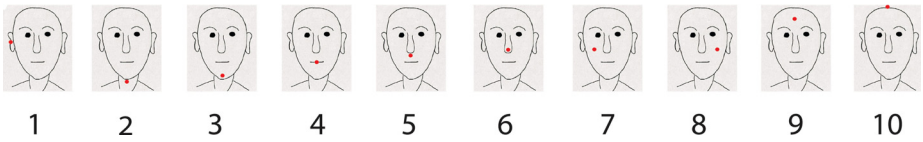

Figure 5.2: Partial Goldenberg stimuli, i.e. the patient imitates the position of contact between the face and hand, where no posture for the hand is specified. We used these stimuli in the baseline *partial imitation* condition.

Experiment B: partial imitation condition

In the partial imitation condition, we introduced a supplementary baseline condition, where the subject was asked to imitate solely the position of contact between hand and face, using the left non-paretic hand. The stimuli presented a face with a red dot that specified the position of contact (see Fig. 5.2). The subject was instructed to precisely reach to the corresponding position of the red dot on his or her face. We did not provide a specific posture for the hand, and thus the subject could reach with a natural and comfortable hand posture. The stimuli were presented three times in a randomized fashion. The patient was instructed to reach either with the index finger (CM and AL) or with the thumb (IB).

5.2.3 Data Analysis

For fine-grained error analysis we broke down the visual stimuli into five objective geometric variables, which taken in combination entirely define the stimulus (as in Chapter 4). We used these geometric variables as criteria for scoring the patient's performance. The variables can be classified into two separate measures of how well the stimulus was reproduced (see Fig. 5.3):

1) Geometric variables related to the position of contact between the face and hand (PC), which include:

a) PC-F, position of contact on the face. A score of 0 was given whenever the patient was not touching the correct part of the face (e.g. the patient is touching the nose instead of the ear), where the face was subdivided into the following parts: nose, mouth, chin, cheek, forehead and ear. A score of 1 was given whenever the patient was touching the correct part of the face but not at

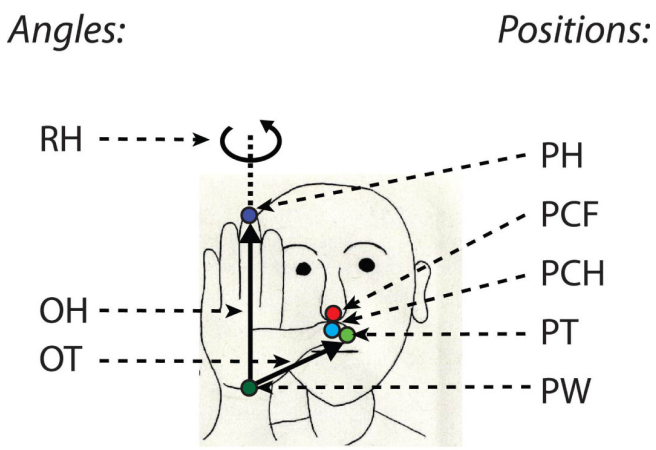

Figure 5.3: Geometric variables used for scoring the patient's performance. Two variables are related to the position of contact between face and hand (PC): PC-F stands for position of contact on the face and PC-H for position of contact on the hand. Three variables are related to the posture of the hand (HP) and are expressed in angles: HP-OH is the orientation of the hand (relates the position of the tip of the hand HP-PH and the position of the wrist HP-PW), HP-OR is the angle of rotation of the hand and finally HP-OT is the orientation of the thumb (relates the position of the tip of the thumb PT and the position of the wrist HP-PW).

the correct location (e.g. the tip of the nose was touched instead of its base). A score of 2 was given when the correct part of the face was touched at the correct location. The PC-F variable relates to visually identifying the part of the face involved in the contact, finding its tactile counterpart representation and determining its current spatial position vector, which then guides the reaching component of the imitative movement.

b) PC-H, position of contact on the hand. A score of 0 was given whenever the patient was touching the face with an incorrect part of the hand (e.g. with a fingertip instead of the thumb), where the hand was subdivided into the following parts: thumb, 4 fingers, palm and back of the hand. A score of 1 was given when the hand-part was correct but the location was wrong (e.g. the patient touched the face with the tip of the thumb instead of the side of the thumb). Finally, a score of 2 was given when the face was touched with the correct part of the hand and at the correct location within this part. The PC-H variable relates to visually identifying the part of the hand involved in the contact, finding its tactile counterpart representation and determining its current spatial position vector, which then guides the reaching component of the imitative movement.

2) Geometric variables related to the hand posture *per se* (HP), which include:

c) HP-OH, orientation of the hand in extrinsic space, defined as the

vector between the position of the tip of the middle finger (HP-PH) and the position of the wrist (HP-PW). For example, a vertical orientation of the hand would mean that the HP-PH and HP-PW vectors differ only in their vertical components in Cartesian space. We gave a score of 0 when the angle between the imitated and correct orientations of the hand exceeded 45 degrees (e.g. the hand was horizontal instead of vertical), a score of 1 when it was between 30 and 45 degrees, and a score of 2 when the discrepancy was less than 30 degrees. The variable HP-OH relates to visually identifying the orientation of the hand (i.e. relationship between the tip of the middle finger and the wrist) and effects the hand flexion/extension and abduction/adduction degrees of freedom.

d) HP-RH, angle of rotation of the hand about the axe of the forearm. A score of 0 was given whenever the hand of the patient was rotated more than 45 degrees away from the correct rotation angle (e.g. the back of the hand is visible instead of the front or the side), a score of 1 when the angle of hand rotation was between 30 and 45 degrees, and a score of 2 when the angle of hand rotation was less than 30 degrees of the correct hand rotation angle. The variable HP-RH relates to visually identifying the rotation of the palm and sometimes involves fine or indirect visual analysis, such as the presence or absence of finger nails and orientation of the thumb. This rotation angle is then used as a control signal for rotating the hand around the axe of the forearm.

e) HP-OT, orientation of the thumb in the extrinsic space, defined as the vector relating the position of the thumb (HP-PH) and the position of the wrist (HP-PW). This variable was not used in this study, as the thumb was present in less than half of the stimuli.

5.2.4 Data Collection

In order to distinguish between our two hypotheses of the underlying cause for the contact position errors - faulty body schema and faulty motor control - we measured the kinematic properties of the imitation gesture[1]. Data was recorded using 3D inertial measurement unit/motion sensors (Xsens Technologies B.V., The Netherlands). The sensors were attached to three arm segments (the upper arm, the forearm and the hand) and were calibrated in the upright position with the arm vertical. The orientation of the three arm segments during the execution of the movements was recorded at a frequency of 50 Hz.

5.2.5 Statistical Analysis

A four-factor analysis of variance (ANOVA) was performed on the patient's scores, with the following within-subject factors: "patient" (3 levels: CM, IB, and AL), "geometric variable" (4 levels: PC-F, PC-H, HP-OH and HP-RH),

[1] Similar to Hermsdörfer et al. (1996).

"stimulus" (10 levels) and "trial" (3 levels). One-way analyses of variance (ANOVA) were introduced to explain interactions. We also performed a t-test to compare the scores relative to the position of contact, i.e. variables PC-F and PC-H, in the normal (Goldenberg's stimuli) and partial (only position of contact) imitation conditions.

5.3 Results

Even though the patients were given unlimited time to imitate, they made a substantial number of errors. Here we provide a systematic quantitative analysis of these errors.

5.3.1 Experiment A: Error Variability

The four-way analysis of variance on the patients' performance showed that the factor "trial" does not have a significant effect ($P > 0.05$) and does not interact significantly with the other factors. The factor "geometric variable" also fails to reach significance ($P > 0.05$), meaning that all of the quantities measured (see Section 5.2.3) are similarly impaired.

A main effect of the factor "patient" indicates that patient CM scored significantly less well than patient IB ($F_{2,504} = 12.27$, $P < 0.001$). A main effect of the factor "stimulus" indicates that some of the stimuli are more difficult to imitate than others ($F_{9,504} = 13.14$, $P < 0.001$). We observed two significant interaction effects. The interaction effect between factors "patient" and "stimulus" indicates that the patients have difficulties with imitating different stimuli ($F_{18,504} = 8.44$, $P < 0.001$). Similarly, the interaction effect between factors "geometric variable" and "stimulus" indicates that the reproduction of specific geometric aspects varies as a function of the stimulus to imitate ($F_{27,504} = 3.62$, $P < 0.001$).

Interaction Effects of the Factor "Stimulus"

We further investigated the interaction effect between the factors "patient" and "stimulus" with a one-way ANOVA. Each patient presented significantly impaired imitation for certain stimuli: patient CM of stimuli 2, 4, 7 and 9; patient AL of stimuli 2 and 5; and patient IB of stimulus 8 (see Fig. 5.4).

The interaction effect between the factors "geometric variable" and "stimulus" was also investigated using a one-way ANOVA. Imitation of the position of contact on the face PC-F was significantly impaired in stimulus 2; the position of contact on the hand PC-H in stimuli 2, 4, 5, 8 and 9; the orientation of the hand in stimulus 4 and finally the rotation of the hand in stimuli 2, 5 and 9

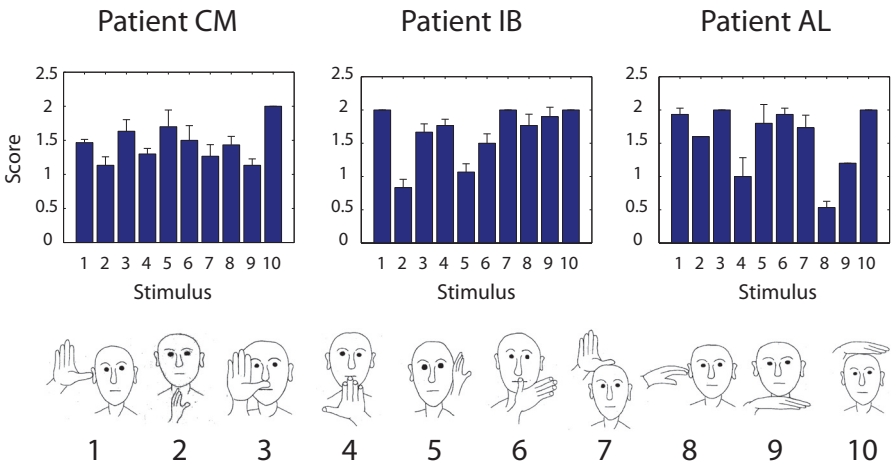

Figure 5.4: Interaction effect between the factors "patient" and "stimulus". Each patient displayed significantly impaired imitation for certain stimuli: patient CM of stimuli 2, 4, 7 and 9; patient AL of stimuli 2 and 5; and patient IB of stimulus 8. Thus a visual stimulus could be "easy" to imitate for one patient, but "difficult" to imitate for another patient.

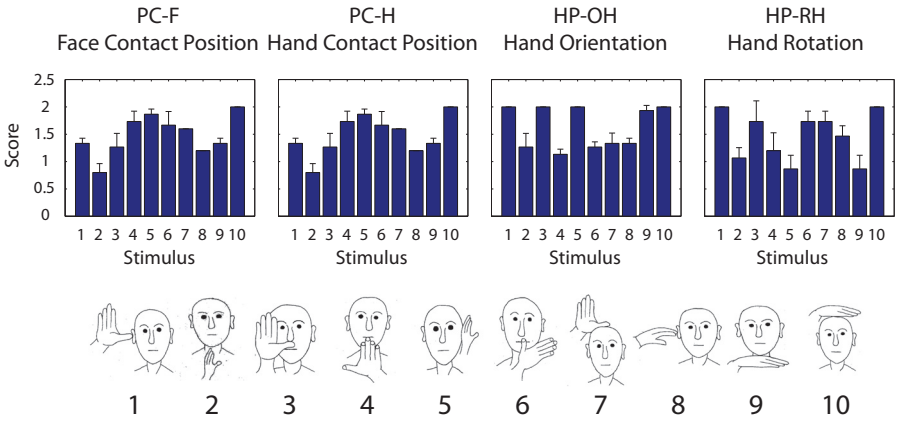

Figure 5.5: Interaction effect between the factors "geometric variable" and "stimulus". Whether a geometric aspect of the stimulus is correctly reproduced depends on the imitated stimulus.

(see Fig. 5.5). Furthermore, within the geometric variable PC-H, the imitation was significantly more impaired for stimuli that touched the face with the back of the hand and index finger, when compared to stimuli that touched the face with the thumb or palm ($F_{3,146} = 14.06$, $P < 0.001$). We did not find significant effects in the other geometric variables (e.g. differential imitation of a vertical versus horizontal orientation of the hand).

5.3.2 Experiment B: Imitation of Contact Positions

None of the patients displayed any difficulty in imitating solely the position of contact between the hand and face, both with the index finger and thumb, when no posture of the hand was specified. The difference in performance of the contact position variables PC-F and PC-H in the normal and partial

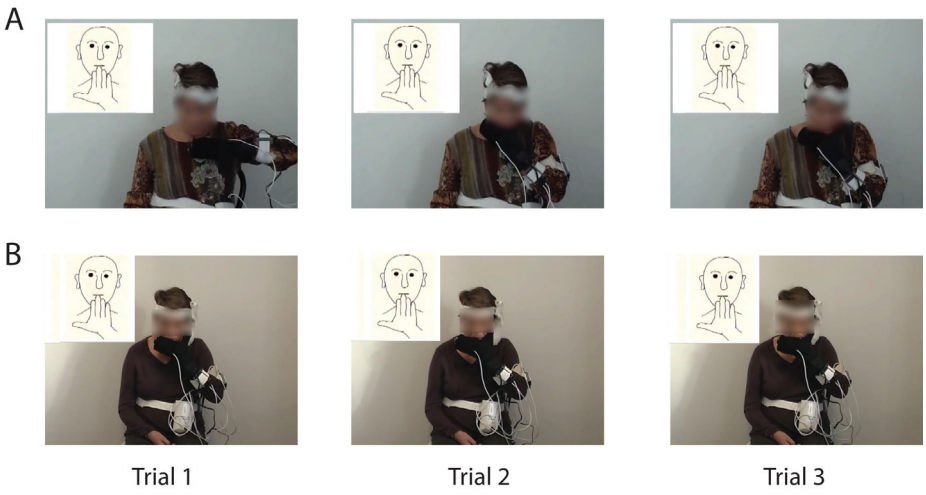

Figure 5.6: The same imitation errors consistently persisted during several trials and also over several weeks. **A** First examination of patient CM. **B** Second examination two weeks later.

imitation conditions was highly significant (T_{118} = -10.17, $P < 0.0001$). The patients made errors in the position of contact only in the normal condition (i.e. imitation of Goldenberg's stimuli, see Fig. 5.1), but not in the partial condition (i.e. imitation of solely the position of contact between the hand and face; see Fig. 5.2).

5.4 Discussion

With unlimited time to imitate a given body posture, the patients were relieved from stress and memory-load. Nevertheless, their imitation was still significantly impaired. Corrective movements using compensation strategies highly improved the patients' performance, but were not sufficient to reach normal behavior. The patients were often aware that something was wrong with their gesture, but expressed ignorance regarding the impaired aspects or appropriate solution.

The "trial" effect was not significant in all three left parietal apraxic patients: the patients did not learn or get habituated to the stimuli, and the same error persisted during repetitive trials. Many errors were qualitatively reproduced even after several weeks (see Fig. 5.6) and some errors were similar across patients (see example in Fig. 5.7). This consistency in the **error structure** indicates that the deficit underlying imitation in apraxia *is characterizable*. This body of neuropsychological evidence argues in favor of a specific neural mechanism impaired in apraxic imitation.

Another important result is that imitation of solely the position of contact between the hand and face is not impaired. We asked the patients to imitate reaching to the face, where the position of contact was presented visually using

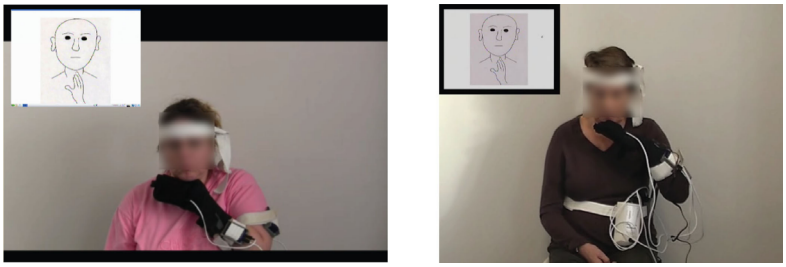

Figure 5.7: Different patients sometimes executed the same imitation errors.

a red dot. We did not constrain the hand posture, such that patients could use natural and comfortable hand gestures. The results show that all of the patients correctly reached to the target part of the face, and with the correct part of the hand (thumb and index finger). Therefore, the **position errors** during the imitation of Goldenberg's stimuli can not be attributed to: (1) an impaired spatial representation of the face, or (2) an incorrect motor control of the hand. The deficit that creates position errors is thus high-level and probably related to the complexity of Goldenberg's imitation stimuli. We suggest that position errors are caused by multiple interdependent constraints that need to be simultaneously resolved (e.g. constraints on both the position and orientation of the hand).

Relation to our neurocomputational model of imitation. These results have important repercussions on our neurocomputational model of imitation, and on how we simulate the callosal lesion. Our assumption of a probabilistic lesion that impairs the transfer of information between the two brain hemispheres is invalidated by the observed structure of apraxic errors. Thus impaired access to the neural processes of the left parietal cortex does not necessarily lead to uncertain mental representations of the target position as suggested by Hermsdörfer et al. (1996), which would provoke random rather than systematic errors.

The observation that the right hemisphere is capable on its own to precisely reach to targets located on the face argues against our hypothesis of a left-lateralized body schema. Thus the unique tactile representation of the body in our neurocomputational model should be distributed and localized in both hemispheres. According to this new evidence, the left hemisphere is more concerned with the coordination between the reproductions of different aspects of the imitated body posture.

Effect of the "stimulus" factor. We did not observe a clear pattern of errors or systematic degradation of the performance *with respect to the geometric variables* measured. Even though the errors themselves were systematic across trials, we could not identify any geometric variables that were more impaired than others. Furthermore, the pattern of errors varied as a function of the stimulus. As shown on Fig. 5.4, the stimuli that were difficult to imitate varied

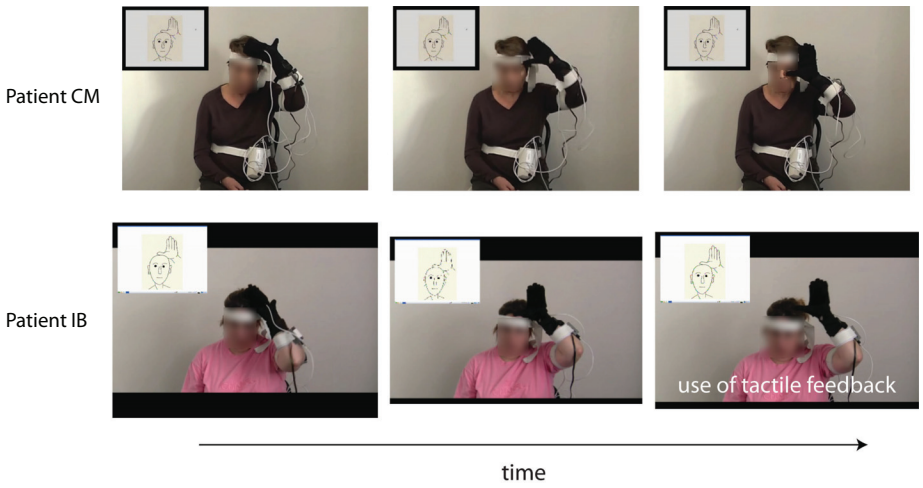

Figure 5.8: Use of compensation strategies. Both patients satisfy the two imitative goals sequentially, i.e. position of contact and posture of the hand. Patient IB also uses tactile feedback to precisely reposition her hand on the top of the head.

across patients. In addition, the stimuli had an influence on which geometric aspects were incorrectly reproduced (see Fig. 5.5). It is very interesting to note that stimuli that presented the same posture of the hand at different positions in space (such as stimuli 1-3-7, 8-9-10 and 2-5) had very different error profiles. This neuropsychological observation suggests that the processes that compute the position and orientation of the hand are tightly linked, and may share the same neural substrates. The specification of a hand position effects the hand posture and vice-versa. In addition, uniquely stimulus 10 was correctly reproduced by all patients (see Fig. 5.1). We believe that stimulus 10 is an easily reproducible "natural stimulus" in the following sense: the instruction to simply touch the top of head would result in a horizontal posture of the hand, which is exactly the hand posture shown in the visual stimulus. These results suggest that the stimuli for the clinical assessment of imitation in apraxia need to be chosen carefully. Specifically, natural and familiar movements should be avoided as they might not be helpful at uncovering the imitation deficit.

Compensation Strategies. Corrective movements were allowed in order to see whether the patients were aware of their errors and to what extent. All of the patients showed the following compensation strategies: 1) sequential satisfaction of the imitation goals (i.e. spatial relations between parts of the body), 2) the use of tactile feedback to refine the position of contact and 3) the occasional use of visual feedback to check the posture of the hand. Whereas a normal subject is able to simultaneously satisfy all of the constraints imposed by the visual stimulus to imitate, the patients showed a tendency to satisfy these imitation constraints *sequentially*. For example, a patient would start by taking the hand to the position of contact on the face (using a natural posture of the hand), and only then focus on the reproduction of the desired hand posture (see Fig. 5.8). Correction of the hand posture usually did not take into account

the position of contact on the face, such that this position was displaced (e.g. patient CM in Fig. 5.8). Finally, the patients frequently used tactile feedback to bring the hand back to the original position of contact, while keeping the desired hand posture (e.g. patient IB in Fig. 5.8). The transitions between states that partially fulfill the imitation goals should be investigated in more detail, as the associated corrective movements may well correspond to movement primitives, i.e. simple movements that the brain may use to create complex movements by the principle of superposition.

To summarize, we find that the imitation errors in apraxia are highly reproducible across trials and time, such that the underlying deficit is not probabilistic in nature. In addition, the errors in the imitation of the position of contact between the face and hand are not due to an impaired spatial representation of the face or incorrect motor control of the hand. Interestingly, these contact position errors arise solely when a specific posture of the hand also needs to be imitated.

Chapter 6

Movement Curvature Planning through Force Field Internal Models

This work was previously published in:

Biljana Petreska and Aude Billard. Movement curvature planning through force field internal models. *Biological Cybernetics*, 100:331–350, 2009.

In the previous chapter we collected kinematic data during the imitation of meaningless gestures, in order to investigate whether motor control is impaired in apraxia. We showed that the patients executed normal reaching movements, suggesting that motor control was also normal. In this chapter we develop a mathematical model of a motor control system that accounts for the kinematics of the movements observed in Chapters 4 and 5. We considered it important to develop a biologically plausible model that could reproduce natural reaching movements, as reaching is an integral part of the imitation task. The reaching model presented here takes as input a desired target position for the hand, which is computed according to the neurocomputational model presented in Chapter 3. The model introduced in this chapter thus accounts for the final execution stage of imitation.

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ORIGINAL PAPER

Biological Cybernetics

Movement curvature planning through force field internal models

Biljana Petreska · Aude Billard

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Abstract Human motion studies have focused primarily on modeling straight point-to-point reaching movements. However, many goal-directed reaching movements, such as movements directed towards oneself, are not straight but rather follow highly curved trajectories. These movements are particularly interesting to study since they are essential in our everyday life, appear early in development and are routinely used to assess movement deficits following brain lesions. We argue that curved and straight-line reaching movements are generated by a unique neural controller and that the observed curvature of the movement is the result of an active control strategy that follows the geometry of one's body, for instance to avoid trajectories that would hit the body or yield postures close to the joint limits. We present a mathematical model that accounts for such an active control strategy and show that the model reproduces with high accuracy the kinematic features of human data during unconstrained reaching movements directed toward the head. The model consists of a nonlinear dynamical system with a single stable attractor at the target. Embodiment-related task constraints are expressed as a force field that acts on the dynamical system. Finally, we discuss the biological plausibility and neural correlates of the model's parameters and suggest that embodiment should be considered as a main cause for movement trajectory curvature.

Keywords Motor control · Neural control of movement · Dynamical systems · Computational model · Goal-directed reaching movements

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1 Introduction

The vast majority of motor control studies have focused on highly constrained reaching movements, limiting the movements to a two-dimensional plane, and in particular to the frontal plane. These constraints are meant to ensure the reproducibility and controllability of the task. They have led to the observation of so-called "quasi-straight" reaching movements with a stereotyped single-peaked, bell-shaped velocity profile (Morasso 1981; Flash and Hogan 1985). The gentle curvature responsible for the term "quasi" has proved hard to explain. Some have suggested that it is due to distortions in the visual perception of the target (Wolpert et al. 1994, 1995), which could however not explain the fact that these are also observed in congenitally blind subjects (de Graaf et al. 1994). Others have attributed the curvature of the movement to the dynamics of the arm's biomechanics, i.e., inertial and viscoelastic resistive forces (Flash 1987; Bullock and Grossberg 1988). This again could not explain the fact that the curvature persists in isometric tasks, which indicates rather that the curvature is encoded directly in the activation patterns of the muscles (Pellegrini and Flanders 1996). Another possible explanation for the curvature of arm movements is Listing's law, as the arm rotation movements were shown to roughly lie in a 2D curved surface (Liebermann et al. 2006). Importantly, when participants are instructed to generate straight paths, they produce movements much straighter than those generated spontaneously (de Graaf et al. 1994; Desmurget et al. 1997; Osu et al. 1997), which argues against the hypothesis of imperfect control (Flash and Hogan 1985). In addition, the curvature depends on the location of the target (Soechting and Lacquaniti 1981) and is systematic within trials and across subjects (Soechting and Lacquaniti 1981; Pellegrini and Flanders 1996). Curved trajectories are also more frequently observed during unconstrained movements

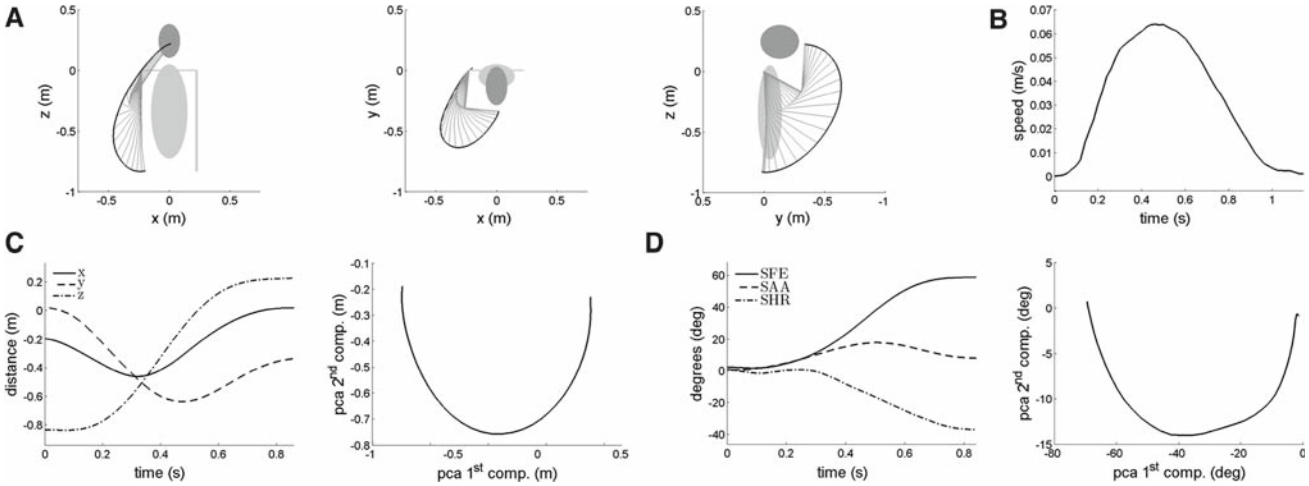

Fig. 1 An example of the curvature of an unconstrained self-oriented movement (the subject was asked to touch his nose). **a** Projections of the movement in the *xy*-, *xz*-, and *yz*-planes. **b** The velocity profile is bell-shaped and single-peaked, similarly to the velocity profiles of straight point-to-point movements. **c** The movement is curved in the extrinsic hand Cartesian space (*left*), which is best visible when projected on the first two principal components following a principal component analysis (PCA) (*right*). **d** The movement is curved also in the intrinsic joint angles space (*left*) and its two principal components (*right*). The joint angles represented here correspond to the three degrees of freedom of the shoulder: shoulder flexion–extension (*SFE*), shoulder abduction–adduction (*SAA*) and shoulder humeral rotation (*SHR*)

(Soechting and Lacquaniti 1981; Lacquaniti et al. 1986; Miall and Haggard 1995; Desmurget et al. 1997; Osu et al. 1997). Overall, the above evidence indicates that the curvature underlying human motion might be a "natural" feature of the movement, and the observed straightness an artifact of the restricted workspace.

We show in this paper that these non-linearities are particularly important when considering reaching movements directed to ourselves (see Fig. 1). Self-oriented movements are part of our daily repertoire (e.g., to eat). They are among the first to emerge in life and are likely the result of evolutionary old neural structures. Their study may thus reveal basic neural processes of motor control. For instance, electrical stimulation of the precentral and motor cortices evoked natural multijoint movements that reached to different points in space, such as for example characteristic hand-to-mouth movements (Graziano et al. 2002, 2005). These movements are also routinely used in neurological examinations to test and diagnose various movement deficits following brain lesion (De Renzi and Lucchelli 1988; Goldenberg and Hagmann 1997; Petreska et al. 2007), which directly inspired the stimuli used in our study. All in all, the study of reaching movements toward oneself is particularly interesting from both a behavioral and a neurological perspective.

We will argue that movement curvature is planned by the central nervous system (CNS) and takes into account the geometry of the body. The idea that embodiment can be encapsulated in the control system itself is in line with our earlier observation that differences in the kinematic features of reaching movements in macaques and humans could be related to the biomechanical properties of the macaques' and humans' shoulder joints (Christel and Billard 2002). Importantly, the model proposed here is not limited to self-oriented movements and can be applied to any point-to-point reaching movement such as for example reaching to targets in the extrapersonal frontal workspace.

2 Computational approach

Modeling studies are particularly useful for distinguishing among all of the plausible mechanisms to encode movements, as long as their predictions are tested and validated against empirical behavioral or neurophysiological data.

However, existing models are unsuccessful at reproducing the curvature of natural human movements (Admiraal et al. 2004), up to several exceptions (Torres and Zipser 2002; Biess et al. 2007; Guigon et al. 2007). For instance, while the so-called 2/3 power law (Lacquaniti et al. 1983) could account well for the curvature observed during handwriting and drawing motions, it was unsuccessful at explaining the curvature of reaching movements in the 3-dimensional space (Schaal and Sternad 2001), including the movements we consider in this paper as shown on Fig. 2. Furthermore, the minimum work model (Soechting et al. 1995) successfully reproduces the final joint postures of pointing movements starting from different initial joint postures, but does not explain the time dependency across joint trajectories. A kinematic model that intrinsically constrains the arm joints according to Listing's law (i.e., such that the arm rotation vectors lie in a 2-dimensional surface) was partially successful at describing the experimental data (Liebermann et al.

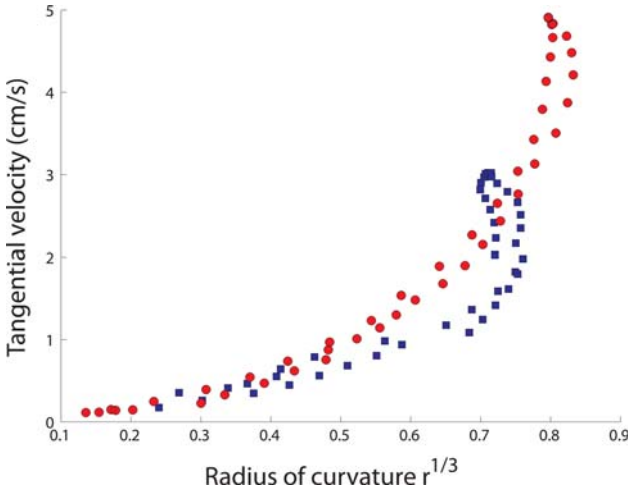

Fig. 2 Two examples of unconstrained self-oriented movements where the 2/3 power law was degraded. The tangential velocity versus radius of curvature to the power 1/3 is shown. The subject was asked to touch his nose (*circles*) or to touch his left ear (*squares*)

2006). The minimum hand jerk[1] model (Flash and Hogan 1985) maximizes the smoothness of the hand trajectory in the extrinsic space. The result is a straight-line trajectory, whereas curved trajectories are obtained by specifying via-points (e.g. for avoiding obstacles). However, it predicts a bimodal velocity profile which is at odds with the experimental data (Atkeson and Hollerbach 1985). Later it was suggested that the hand trajectory is the result of a compromise between planning a straight line in the task space and planning a straight line in the joint space (Cruse and Brüwer 1987; Okadome and Honda 1999; Hersch and Billard 2007). Such hybrid computations offer numerous advantages for controlling 3-dimensional reaching movements, such as avoiding singularities and avoiding hitting the joint limits (Hersch and Billard 2007). Unfortunately there is currently no direct neurophysiological evidence in support of such a control strategy. It has also been proposed that arm movements are controlled by minimizing the derivative of joint torques (Uno et al. 1989; Nakano et al. 1999; Wada et al. 2001). However, this model overestimates the magnitude of curvature of pointing movements (Biess et al. 2007). In Torres and Zipser (2002), the hand path is computed in the intrinsic joint angles space by minimizing an energy-like quantity, giving realistic predictions for curved paths. However, this model assumes a separate processing for the spatial and temporal dynamics of motion and displays some imprecisions for movements similar to those addressed here. The model by Biess et al. (2007) computes a geometrical joint angles geodesic path with respect to a kinetic energy metric in the Riemannian configuration space and subsequently minimizes the squared jerk along this path. This model also treats the spatial and temporal dimensions separately and predicts identical path trajectories for different speeds. We find it difficult to evaluate how well this model would predict highly curved reaching movements as the pointing movements addressed in the study were quasi-straight, but we could observe that the model has difficulties with reproducing mixed curvatures (i.e., movements that deviate first to one side and then to the other side of the idealized straight trajectory). Another class of reaching models are stochastic models that take into account the noise inherent to the motor system. It has been consistently observed that the standard deviation of neuromotor commands increases with its mean (Sutton and Sykes 1967; Schmidt et al. 1979; Clamman 1969; Matthews 1996; St-Amant et al. 1998; Clancy and Hogan 1999; Osu et al. 2004). In line with this evidence, it was suggested that the brain minimizes the variance of the final arm position in the presence of such signal-dependent motor noise (Harris and Wolpert 1998; Hamilton et al. 2002). Even though this model succeeds at reproducing the curvature of 2-dimensional reaching movements, it does not specify which control laws generate these movements. In Todorov and Jordan (2002), an optimal feedback theory of motor control is proposed, in which the variability of the movement is distributed optimally among different degrees of freedom that do not interfere with the task goal. This qualitative model is appropriate for explaining the variability observed in reaching movements, it is however imprecise in its prediction of the curvature of movements. This is partly due to the determination of the appropriate cost function to optimize. This performance criterion is chosen arbitrarily and varies with the task. Another model based on the optimal feedback control theory was successful at reproducing the joint and hand trajectories of 3-dimensional movements (Guigon et al. 2007), but the authors admit that the movements reproduced are rather stereotyped. For example the model does not account for nonsymmetric velocity profiles or avoidance of extreme joint limits.

While it has been suggested that two different control strategies underlie straight and curved reaching movements (Desmurget et al. 1997; Moran and Schwartz 1999), we argue that these two types of movements are generated by a unique adaptive control mechanism. While none of the existing models offers a satisfactory solution for modeling the highly variable curvature of human movements, here we propose a dynamical model that accounts for both gently and highly curved hand trajectories, consistent with recent neurophysiological findings. First, unlike many of the models above, our model is closed-loop. Closed-loop control takes into account the uncertainty of the "real-world" and allows intelligent online corrections as well as robust responses to perturbations, rather than "playing a prerecorded tape" (Todorov 2004). Such an approach is in agreement with the observa-

[1] The jerk corresponds to the derivative of the acceleration and is a measure of the smoothness of the trajectory.

tion that the CNS is able to estimate and anticipate the state of the limb. This is achieved by integrating delayed sensory input and motor output through afferent and efferent internal feedback loops (Desmurget and Grafton 2000). The state information is used to continuously update the motor commands, which is likely to occur in the posterior parietal cortex and cerebellum.

Our model also takes advantage of the signal-dependant neuromotor noise mentioned earlier, which may be responsible for the speed-accuracy trade-off known as Fitts' law (Fitts 1954) and trail-to-trial variability (Todorov 2004). Finally, our model hypothesizes that the curvature of the hand trajectories is not an undesirable noise on otherwise perfect straight-line reaching movements. Rather, it is necessary and planned as such by the CNS in order to, for example, avoid impossible trajectories that go through the body and uncomfortable joint limit postures.

3 Model description

Our work was driven by the assumption that (a) a unique controller underlies both straight and curved reaching movements, and (b) that this controller is such that all the variables can be accounted for by known neurophysiological processes. Thus, to start with, we considered the vector integration to endpoint (VITE) model for point-to-point reaching (Bullock and Grossberg 1988) that accounts for typical kinematic features of human reaching movements such as bell-shaped velocity profiles and speed-accuracy trade-off. The model has been used to explain control in both hand extrinsic and joints intrinsic spaces (Ajemian et al. 2001; Hersch and Billard 2007). Most importantly, the dynamics of the VITE model's response displays a profile of activity similar to that of populations of neurons in the primate's brain. In particular, the model could account for these neurons' sensitivity to change in the velocity of the movement and for the latency of activity at the movement onset (Bullock et al. 1998). The VITE model, however, suffers from a major restriction: it can generate only *straight* movements.[2] Next, we describe the VITE model and give a formal definition of our extension that accounts for curved reaching movements.

[2] An extension of the VITE model has been proposed to account for highly curved handwriting movements (Bullock et al. 1993; Paine et al. 2004), where three coupled VITE models control the displacement of the hand in a 2-dimensional plane and the rotation of the wrist. The curvature results from the coupling between the three models and the fact that each model is initiated with a slight delay at onset. This approach is not optimal for modeling simple point-to-point reaching movements as it necessitates the characterization of a sequence of multiple arbitrary targets, one for each change in the curvature.

3.1 The original VITE model

The original VITE model is a dynamic controller that at each point in time reduces the distance between the estimated and desired states of the controlled variable. First, it computes the desired movement acceleration based on the difference between the present and endpoint vectors. Second, this acceleration is integrated and primed with a faster-than-linear time-dependent "go signal" to specify the desired speed, which is the control signal sent to the muscle motoneurons. This priming signal is essential for the obtention of a bell-shaped velocity profile.

In its complete form the VITE model succeeds for example at: maintaining accurate proprioception while controlling voluntary reaches to spatial targets, maintaining postures despite perturbations, complying with an imposed movement, exerting force against obstacles, compensating for static and inertial loads and reproducing muscle vibration effects (Cisek et al. 1998). For simplicity, we only use the concise form of the model presented in Bullock and Grossberg (1988). For a description of the original VITE model please see the Appendix.

3.2 Modification of the original VITE model

Our modified VITE system is governed by a non-linear and noisy spring-damper system given by:

$$\ddot{\mathbf{x}}(t) = \overbrace{-\alpha\dot{\mathbf{x}}(t)}^{\text{damping factor}} + \overbrace{\beta\mathbf{g}(\mathbf{x}^*(t) - \mathbf{x}(t) + \boldsymbol{\eta})}^{\text{noisy endpoint attractor}}\mathbf{u}(t) \quad (1)$$

The first term is a damping factor proportional to the speed $\dot{\mathbf{x}}(t)$ of the end-effector that prevents the system from oscillating too importantly. The second term corresponds to an elastic force that drives the end-effector from its actual position $\mathbf{x}(t)$ toward the desired target position $\mathbf{x}^*(t)$. Note that the desired position is written as a function of time in order to emphasize the ability of the system to track the target in real time without any additional computation (as a result the system is robust to perturbations of the target position). α is a time constant set to 50. $\beta \in \mathbb{R}^+$ determines the amplitude of the speed at which the system moves globally (increasing β would result in a higher velocity peak and shorter movement duration, see Fig. 3a). $\mathbf{g}$ is a nonlinear function that modulates the dynamics of the system so that it presents a typical bell-shaped velocity profile (refer to the Appendix for the exact form of $\mathbf{g}$). Finally $\boldsymbol{\eta}$ is a multiplicative gaussian noise with zero mean and standard deviation proportional (by a factor of 0.005) to the distance between the actual and desired end-effector positions, namely $|\mathbf{x}^*(t) - \mathbf{x}(t)|$. This noise factor is necessary to initiate the movement and to account for the trial-to-trial variability at the onset of movement (see the Appendix).

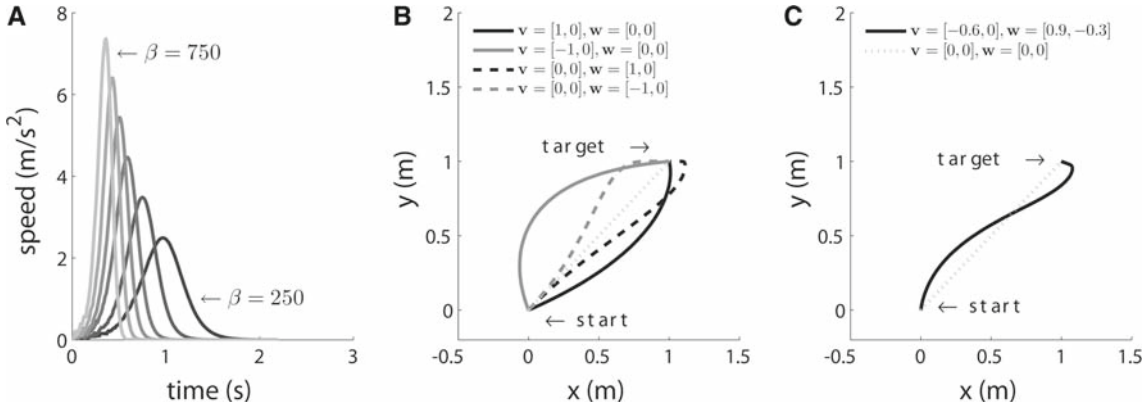

Fig. 3 **a** Effect of gradually increasing the parameter β of the modified VITE model (see Eq. 1) on the velocity profile of the movement. Higher β values increase the velocity peak and shorten the movement duration. **b** Behavior of the extended F2REACH model (see Eq. 2) under different repulsive forces **v** and **w**, for illustrative purpose the forces shown are applied only on the horizontal dimension. The forces are modulated such that **v** affects mostly the beginning of the movement and **w** mostly the end of the movement. Note that the direction of the deviation from the straight trajectory is determined by the sign of the force. **c** By combining two forces **v** and **w** of different signs one can obtain very interesting deviations that change their direction during the execution of the movement. Reference values: $\alpha = 50$, $\beta = 500$, noise was set to 0.005. Only the speed parameter β is varied throughout the simulations, the two other parameters (time constant and noise) are fixed to the given values

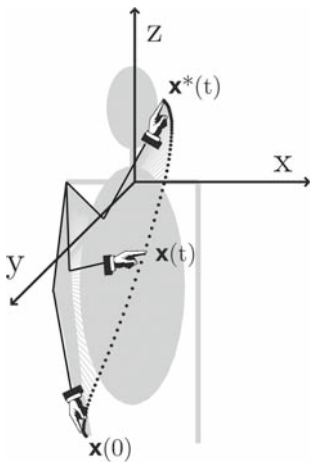

Fig. 4 Description of the task space. The hand position $\mathbf{x}(t)$ is represented in a 3-dimensional space centered on the chest, at the level of the shoulders. The input to the model consists of the initial hand position $\mathbf{x}(0)$ and final target position $\mathbf{x}^*(t)$

The above formulation makes two strong assumptions from a motor control point of view: (a) it takes as control signal the acceleration of the end-effector $\ddot{\mathbf{x}}$, expressed in an extrinsic 3-dimensional Euclidean space centered on the chest (see Fig. 4), and (b) it accounts only for a "high-level" control mechanism, in that it generates the desired end-effector kinematics, and does not account for the subsequent transformation required to control muscle activations.

Expressing the system in terms of desired acceleration is not constraining, since it is conceivable to assume that a neural population coding for the acceleration can be neurally integrated out to obtain a velocity control signal, which can in turn be integrated out to have a position control signal, see Sauser and Billard (2006). Moreover, evidence that muscle activity may be governed by a kinematic signal, such as the acceleration, velocity or position, or any combination of these, has been found in the motor cortex (Wang et al. 2007). Note that we do not address the problem of redundancy mapping between desired hand kinematics and actual muscle activations in this paper. These assumptions will be further developed in Sect. 6.1.

The above system differs from the original VITE model in two ways (see the Appendix for the original VITE formulation). First, the dynamics of the system is now governed by a *single* second order differential equation and is thus expressed in terms of the end-effector acceleration.[3] Second, we replaced the explicit time dependency of the original VITE system by introducing a bounded nonlinearity in the function **g**. In the original VITE system, this explicit dependency in time through the priming signal let the velocity of the system grow exponentially in time, which created instabilities in the case of a long lasting perturbation, and was thus biologically implausible (your arm does not start accelerating if someone holds it).

3.3 Extension of the original model: F2REACH model

To account for the movement curvature, we next introduce a new functional $\mathbf{F}(\mathbf{x}(t))$ that corresponds to a *virtual force*

[3] The original VITE system was driven by two coupled first-order differential equations. We reformulated this by writing the whole system as a second order differential equation. This allows us to relate explicitly the acceleration of the system to the force-field which we introduce in the following section.

field, which encapsulates a geometrical representation of the task constraints. This force field is modulated by the dynamics of the control signal in order to preserve the bell-shaped velocity profile:

$$\ddot{\mathbf{x}}(t) = -\alpha\dot{\mathbf{x}}(t) + \beta\mathbf{g}(\mathbf{x}^*(t) - \mathbf{x}(t) + \boldsymbol{\eta}) + \overbrace{\beta|\mathbf{g}(\mathbf{x}^*(t) - \mathbf{x}(t) + \boldsymbol{\eta})|}^{\text{modulation factor}}\overbrace{\mathbf{F}(\mathbf{x}(t))}^{\text{force field}} \quad (2)$$

The force field $\mathbf{F}(\mathbf{x}(t))$ assigns a vector gradient to each position in space that expresses constraints related to: (a) objects in the environment that one needs to avoid (including the subject's body), (b) dynamic properties of the human body such as inertial properties of the limb, (c) extreme joint angles limits. The contribution of each of these constraints is simply summed to result in the virtual force field. The gradient of the force field at each point in space pushes the hand away from the undesired locations.

This force field framework reconciles the dynamic and kinematic aspects as well as intrinsic and extrinsic approaches to motor planning in a very convenient way. Instead of finding a compromise across systems that would operate simultaneously in conflicting coordinates (e.g., hand position and joint angles, see Sect. 2), our system provides both dynamic (acceleration) and kinematic (speed or position) control signals, taking into account (a) a target for the motion expressed in extrinsic kinematic coordinates and (b) intrinsic dynamic motion constraints. This reconciles the observation that objects in the environment such as a table may influence the kinematic planning of the movement[4] (Brenner and Smeets 1995) and that knowledge of the arm dynamics is necessary for the kinematic planning of complex movements (Uno et al. 1989; Nakano et al. 1999; Sabes and Jordan 1997).

As the particular form taken by the force field is task and context dependent, we chose a very generic expression given by:

$$\mathbf{F}(\mathbf{x}(t)) = h(\mathbf{x}(t))\mathbf{v} + (1 - h(\mathbf{x}(t)))\mathbf{w} \quad (3)$$

where $\mathbf{v}$ and $\mathbf{w}$ are constant force vectors that push the trajectory away from the straight-line generated by the rest of the system. $\mathbf{v}$ affects primarily the *beginning* of the movement, whereas $\mathbf{w}$ affects the *end of movement* (as illustrated in Fig. 3b, c). The modulation function h that associates these two forces to different parts of the movement is given in the Appendix.

In our framework, a 3-dimensional reaching movement needs the specification of seven parameters in total: β that controls the amplitude of the velocity's peak and two 3-dimensional repulsive forces $\mathbf{v}$ and $\mathbf{w}$, where the time constant α and noise can be fixed to 50 and 0.005 respectively. We will show next that the latter two forces give a crude representation of the volume and geometry of the body around which the hand must navigate.

To conclude the description, control policies of the form of autonomous differential equations such as the one proposed here are particularly interesting, as they allow online modifications of the input variables. Thus a very nice property of our model is its robustness to external perturbations, where the model shows smooth adaptation to changes such as blocking or displacing the arm and displacing the target (simulation results not shown here).

4 Experiments

4.1 Subjects

Ten healthy subjects, five female and five male of mean age 33 ± 11 years volunteered for the study. All the participants except for two were right-handed according to the Edinburgh handedness test (Oldfield 1971). All the subjects were naive as to the purpose of the study and had no history of neurological or musculoskeletal deficits.

4.2 Procedure

The subjects were asked to perform natural reaching movements toward targets situated on their head. In order to obtain entirely natural and fully unconstrained movements, the target positions were specified verbally (for example we gave instructions such as "on the go signal touch your nose"). The subjects were left free to determine the location of the reaching target (e.g., at the tip of the nose or just above it), but they were instructed to reach to exactly the same location across one block of repetitions of the same movement. There were six target positions, shown in Fig. 5a, indexed as follows: (1) nose, (2) right ear, (3) left ear, (4) top of the head, (5) under the chin and (6) back of the head. Given that the subjects had different arm lengths and given that the targets were defined with respect to the subject's head, the length of the hand path varied importantly across subjects and movements. This was done on purpose to test the ability of the model to reproduce the generic characteristics of the movements and to account for such body variabilities, which we consider task-independent. The subjects were standing in order to limit undesirable movements of the upper body. There were no external constraints that would confine the movement range. The movements were performed with the right hand independently of the handedness of the subject, since handedness was shown not to affect spontaneous self-oriented movements (Dalby et al. 1980; Lavergne and Kimura 1987). In order to verify the gener-

[4] This type of computation is natural (and especially useful) if the movement is considered in a constantly varying environment full of external objects, instead of isolated in an artificial experimental setup.

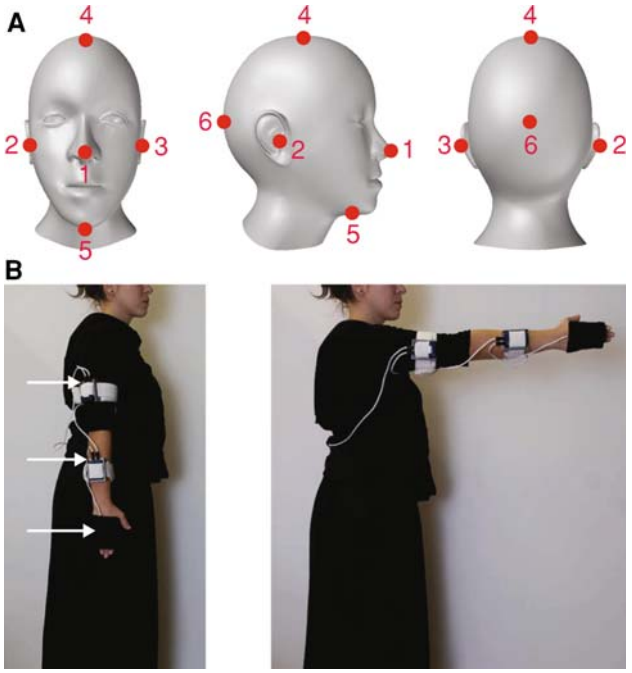

Fig. 5 **a** Target positions on the head used in our experiment. **b** Two initial conditions that yield both highly and gently curved movements. The three motion sensors are indicated with *arrows*

alization of our model over movements with different curvature levels, movements were initiated from two different locations, shown in Fig. 5b: (1) upright position with the arm extended along the body that yielded highly curved movements and (2) upright position with the arm extended in front of the body that yielded gently curved movements. Prior to each experiment, the subjects were asked to assume the same starting position, which was verified by the experimenter. The subjects had at least one trial of practice per movement to ensure that they had understood the instructions. Each movement was repeated five times in order to have a measure of its inherent variability and consequently a measure of the precision of the model's reproduction.

4.3 Data acquisition

Data was recorded using 3D inertial measurement units/motion sensors (Xsens Technologies B.V., The Netherlands). The sensors were attached on three arm segments (the upper arm, the forearm and the hand) and were calibrated in the upright position with the arm vertical (see Fig. 5b, left). The orientation of the three arm segments during the execution of the movements was recorded at a frequency of 50 Hz.

4.4 Data analysis

All analyses were performed with custom software written in Matlab (Mathworks, Natick, MA, USA). The trajectories of each arm segment were reconstructed using the orientation matrices recorded by the inertial measurement motion sensors. We used only *unfiltered raw* values. The movements of interest were extracted using criteria such as percentage of velocity change. The samples were aligned in time so that the inter-trial Euclidean distance per movement and subject (five samples) is minimal. The movement mean and standard deviation (SD) of each trajectory for each movement type and for each subject was computed with respect to the aligned signals. We then solved numerically the original VITE and extended F2REACH models for each of the mean movements, with a time step of 20 ms. The models' parameters were fixed using 3^3 and 3^7 factorial experimental designs respectively, coupled with a local search procedure (Neter et al. 1996; Hoos and Stützle 2004).

To evaluate the predictions of the two models we measured the following Euclidean distances and deviation indices:[5] (1) *mean deviation* (MD) of the predicted hand trajectory compared to the measured hand trajectory at each point in time, (2) *mean squared error* (MSE), (3) *hand trajectory deviation index* (HTDI) defined as the ratio between the maximal distance across the modeled $\mathbf{x}^m(t)$ and real $\mathbf{x}^r(t)$ mean trajectories over the total length of the real path,

$$\mathrm{HTDI} = \frac{\max_{i=1,\ldots,N} |\mathbf{x}^m(i) - \mathbf{x}^r(i)|}{\sum_{i=1}^{N-1} |\mathbf{x}^r(i+1) - \mathbf{x}^r(i)|}$$

where N is the number of points sampled (see Fig. 6a), (4) *speed deviation index* (SDI) and finally (5) *total acceleration deviation index* (ADI), both defined in Fig. 6b. We also considered the standard deviation trajectory (SD) as a possible limit prediction (see Fig. 6c for a definition). We further assessed the *curvature index* of recorded and modeled movements, defined as the ratio between the total arc length of the hand path and the Euclidean distance that separates the initial and final positions. For example a curvature index of 1 indicates a perfectly straight path and a curvature of $\pi/2$ corresponds to a semicircular path. Finally, the *speed asymmetry index* was defined as the ratio $(S^a - S^d)/(S^a + S^d)$ where S^a is the distance traveled up to the time at which the velocity is maximal (referred to as the acceleration phase) and S^d the distance traveled from the time at which the velocity is maximal until the end of the movement (deceleration phase). An additional measure of the precision of the original VITE and extended F2REACH models is the percentage of trajectory points predicted by the models that are comprised within the volumes defined by 1 and 2 SD away from the recorded mean trajectory (per subject and movement type, established over five repetitions of the movement, see Fig. 6c). This measure accounts for the variability inherent to goal-directed reaching

[5] The deviation indices are adapted from Nakano et al. (1999) and Biess et al. (2007).

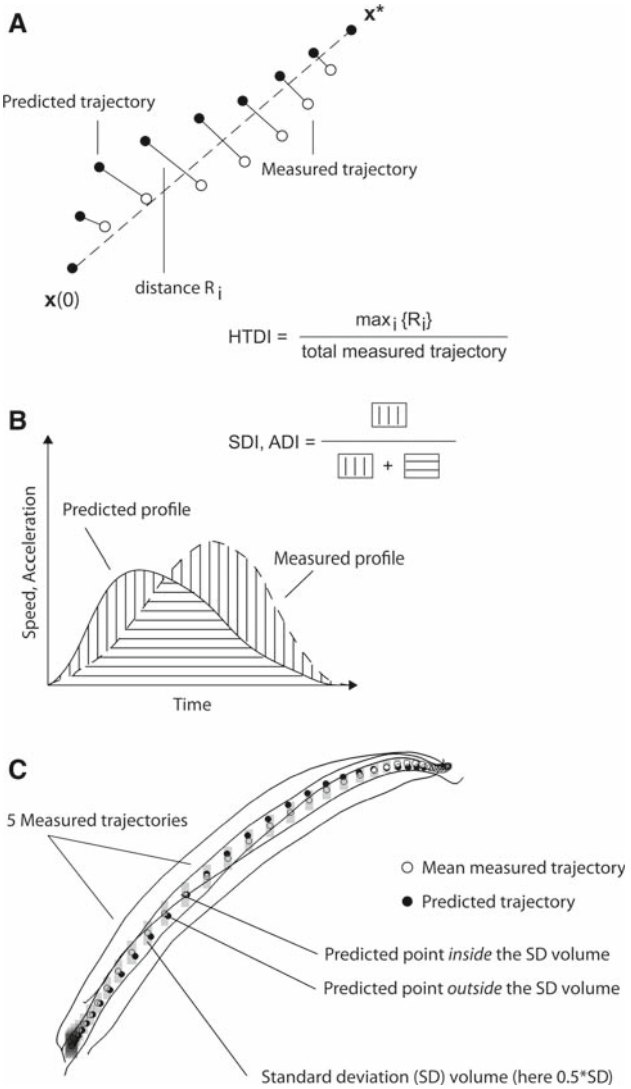

Fig. 6 Definitions of error measures. **a** The hand trajectory deviation index (*HDTI*) of measured and predicted hand trajectories is the ratio of the maximum distance, $R = \max_{i=1,\ldots,N} R_i$, between the two trajectories matched in time over the total length of the measured path. **b** The speed deviation index (*SDI*) and total acceleration deviation index (*ADI*) are defined as the ratio of the noncommon area enclosed by the measured and predicted speed/acceleration profiles and the total enclosed area. **c** Standard deviation volumes (SD), comprised within a multiple of the standard deviation distance (computed from the mean trajectory of five movement trials per subject and movement type) at every point of the movement trajectory. A SD trajectory would follow the corresponding corners of these volumes. We consider that a point was well predicted if it is contained inside the SD volume of its measured counterpart, thus enforcing a higher precision at points with very low variability

movements (Harris and Wolpert 1998; Todorov and Jordan 2002) and penalizes imprecision in parts where the variance of the movement is minimal. For example, the subjects were more consistent in the vicinity of the initial and target positions.

5 Results

In this section we report on a systematic assessment of how well the original VITE and our extended F2REACH models account for the kinematics of the recorded human movements. We also discuss the biological plausibility of our model's parameters. Finally, we conduct a stability analysis of the F2REACH model and define conditions under which the target is a stable attractor of the model and therefore guaranteed to be reached.

5.1 Observed data statistics

We first assessed the general characteristics of the recorded movements (summarized in Table 1). The movements addressed had large spatial extent (mean path length of 1.23 m) with significantly longer path lengths in the first experimental condition (see Fig. 5b) when compared to the second experimental condition (mean path lengths of 1.7 and 0.95 m respectively). Movements in the first condition lasted longer with mean durations of 1.3 and 1 s, respectively. Most importantly, the movements in the first condition were significantly more curved with a mean curvature index of 1.59 compared to 1.21 in the second condition. In addition, the curvature indices of the recorded movements were distributed homogeneously between quasi-straight (<1.1) and highly curved (>2).

We expected to see substantial trial-to-trial fluctuations due to noise of the motor system (Todorov and Jordan 2002), which motivated us to model the mean trajectory of the movement rather than the separate trials. We believe that the mean movement captures the intrinsic nature of the movement, which is task-relevant and free of noise. An example of the inherent variability across trials per subject and movement type is shown in Fig. 7a. Figure 7b shows that the inter-subject variability (attributed to the difference in embodiment of the subjects) is much more important.

5.2 Comparison between the observed and modeled data

Here we assess how well the original and extended models reproduce the human data. The mean movement trajectories were simulated with both the original VITE and our extended F2REACH models. Typical examples of measured and predicted hand path trajectories are given in Fig. 8. The first row in each example shows the five hand trajectories of the movement projected in the *xy*-, *xz*- and *yz*-planes relative to a schematized humanoid. The second row shows the projections of the mean recorded trajectory and generated model trajectories. The subject's trials are represented with light grey lines and show the inherent variability of the movement. The third row shows the *x*-, *y*- and *z*-components of the hand trajectories with respect to time in order to show the quality of the model predictions at the temporal level.

Table 1 Path length, duration and curvature index of the movements in the two experimental conditions (see Fig. 5b)

	Condition 1	Condition 2	2 Conditions	10 Subjects
			P value	*P* value
Path length (m)	1.70 ± 0.32	0.95 ± 0.18	<0.001	NS
Duration (s)	1.28 ± 0.26	0.97 ± 0.20	<0.001	<0.001
Curvature index	1.59 ± 0.22	1.21 ± 0.11	<0.001	NS

We also give one-way ANOVA results for the initial condition and subject effects on these variables. The movements in condition 1 were significantly longer in time and space and significantly more curved when compared to the movements in condition 2. The recorded movements differed significantly across subjects only in their duration

Table 2 Mean deviation (MD), mean squared error (MSE) and mean deviation indices (see Fig. 6) for the trajectory (HTDI), speed (SDI) and acceleration (ADI) (± standard deviation) of the hand as predicted by the extended F2REACH and original VITE models

	F2REACH model	SD	VITE model
MD (mm)	18.85 ± 8.10	35.67 ± 11.63	132 ± 71
MSE (cm^2)	5.62 ± 5.34	15.93 ± 10.61	431 ± 413
HTDI	0.031 ± 0.010	0.04 ± 0.02	0.25 ± 0.06
SDI	0.11 ± 0.03	0.50 ± 0.11	0.29 ± 0.12
ADI	0.38 ± 0.07	0.60 ± 0.08	0.51 ± 0.13

We also consider the trajectory comprised within one standard deviation (SD) from the mean trajectory (per subject and movement type, computed as described in Fig. 6c) as an indication for the limit prediction that would be acceptable for a model. This SD trajectory represents the inherent variability of the movement. One-way ANOVAs performed on the error measures of the extended F2REACH model show that the effect of the subject performing the movement was not significant and that the movements in the second initial condition, i.e., movements with lower curvature, tended to be slightly better predicted (MD and MSE only)

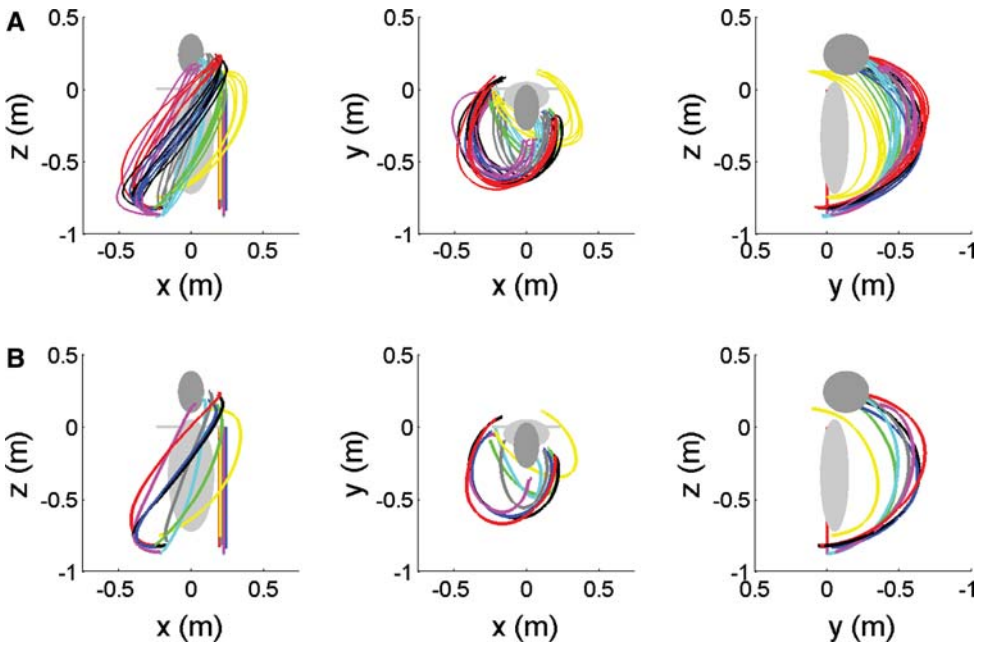

Fig. 7 Trajectories of the hand for ten subjects performing five repetitions of the same movement, reaching to the left ear (movement 3) with the right arm in condition 1 (see Fig. 5). The hand trajectories are shown relative to a schematized humanoid and the color refers to the same subject. **a** All the movement trajectories are shown in order to emphasize the movement's inherent variability. Note that this intra-subject variability is lower than the inter-subject variability, i.e., the hand trajectories of one subject are consistent when compared to those of the other subjects. **b** Only the mean movements are shown. The inter-subject variability can be partially attributed to differences in the subjects' arm lengths and shoulder positions (see *color-coded arms*)

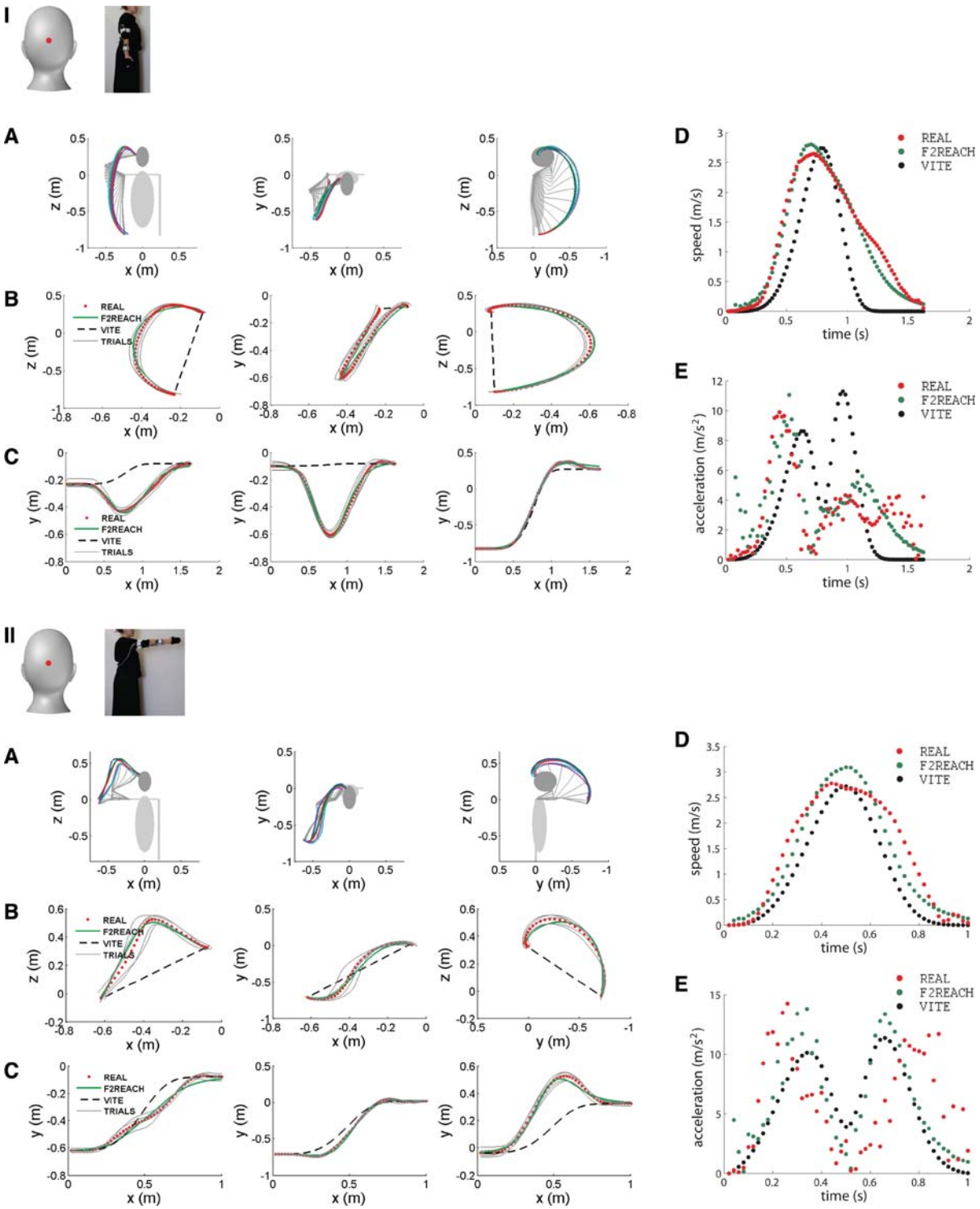

Fig. 8 Two examples of typical movements. The recorded human data is shown with points that respect the sampling rate, the original VITE model is shown with a dashed line and our extended F2REACH model with a plain line. *I*, The subject reaches for the back of the head (movement 6) with as initial condition the right arm extended along the body (condition 1). *II* The subject reaches for the back of the head (movement 6) with as initial condition the right arm extended in front of the body (condition 2). **a** The five recorded hand trajectories of the movement projected in the *xy*-, *xz*- and *yz*-planes and shown relative to a schematized humanoid. **b** The measured and predicted mean movement trajectories projected in the *xy*-, *xz*- and *yz*-planes. The light grey trajectories are the five trials and reflect the intra-subject variability per movement type. **c** The *x*-, *y*- and *z*-components of the measured and predicted mean movement trajectories shown with respect to time. **d** The measured and predicted speed profiles of the movement. **e** The measured and predicted total acceleration profiles of the movement

Table 3 Measured (M) velocity peak amplitude and peak time, asymmetry and curvature indices (± standard deviation) compared to those predicted by the extended (F2REACH) and original (VITE) models

	Measured (M)	F2REACH model	M versus F2REACH P value	VITE model	M versus VITE P value
Velocity peak amplitude (m/s)	2.26 ± 0.68	2.37 ± 0.69	<0.05	8.22 ± 59.31	NS
Velocity peak time (s)	0.50 ± 0.14	0.53 ± 0.13	NS	0.59 ± 0.19	<0.001
Asymmetry index	−0.08 ± 0.15	−0.10 ± 0.11	NS	0.03 ± 0.06	<0.001
Curvature index	1.40 ± 0.26	1.36 ± 0.23	NS	1.03 ± 0.22	<0.001

There were no significant differences between the measured and extended model variables (with the exception of a small difference in the velocity peak amplitude), whereas significant differences were found between the measured and original model for three of the four variables addressed (all except for the velocity peak amplitude)

Finally, on the right we show the measured and predicted speed and acceleration profiles. One can see that, unlike the original VITE model, the F2REACH model is generally in very good agreement with the human data.

We systematically evaluated the predictions of the original VITE and extended F2REACH with several Euclidian distances and deviation indices defined in Sect. 4.4. The results are summarized in Table 2 and show that our model is highly precise at reproducing the kinematics of the recorded movements. The deviation indices are much smaller, generally on a different order of magnitude than those from the SD trajectory and always smaller than the original VITE model. The mean deviation was less than 2 cm for movements of average path length superior to 1 m.

We performed one-way ANOVAs for the extended model using, as dependent data, the different error measures defined in the preceding paragraph. The results show that, regardless of the error measures used, we did not find an effect of the subject executing the movements ($P > 0.05$, with the exception of two subjects for the HTDI and ADI deviation indices). This indicates that our model performed equally well across the ten subjects. A significant effect ($P < 0.001$) was observed for the two experimental conditions (see Fig. 5) for the mean deviation (MD), mean square error (MSE) and speed deviation index (SDI) suggesting that the model is better at predicting low rather than high curvatures. This result is not very surprising since the force field in our model is parameterized with two constant forces, thus approximating the real force field underlying the movement. The more a movement is curved, the more imprecisions related to this parametrization affect the model's performance. Finally, the original and extended models differed significantly in their predictions for all the error measures ($P < 0.001$).

We have further investigated whether our model captures the major temporal characteristics of the movement. We compared the VITE and F2REACH models' predictions to the real data for the peak amplitude, time at peak amplitude and speed asymmetry index, see Table 3. One way ANOVAs confirmed a very good match between our model's prediction and the data for all the above quantities (except for the velocity peak which was slightly lower, $P < 0.05$), whereas the predictions of the original VITE model differed significantly from the data ($P < 0.001$) except for the velocity peak amplitude. To illustrate the quality of the extended and original VITE models' predictions for the time-dependency of the signals, in Fig. 9 we compare instances of measured and predicted speed profiles (normalized in time). Finally we looked at the percentages of trajectory points comprised within the volumes defined by one and two standard deviations (SD) in order to evaluate the performance of the models at portions where the movement is very precise and systematic over trials (see Sect. 4.4 for details). The results show that 81% of the hand trajectory points predicted by our model were within two SDs of the mean trajectory against 40% of the points predicted by the original VITE model (Table 4 shows also the result for 1D).

One should emphasize that the F2REACH model generates these 3-dimensional movements using few parameters: β that controls the amplitude of the velocity and the two repulsive force vectors $\mathbf{v}$ and $\mathbf{w}$ (see Fig. 3b) that parameterize the force field surrounding the subject. The other two parameters α (time constant) and noise were fixed to 50 and 0.005 in all the simulations. The high accuracy with which the model manages to replicate the movements confirms that the model encapsulates the important features underlying free reaching movements. The force field is a key variable of the model. Next we show that the force field can be interpreted in relation to the bio-mechanical constraints of the subject's body.

5.3 Understanding the force field

Figure 10 shows the components of the virtual repulsive forces $\mathbf{v}$ and $\mathbf{w}$ parameterizing the force field of the F2REACH model (Eq. 2). We observe that the values of the components are clustered in two groups depending on the starting location of the movement. They are, thus, consistent

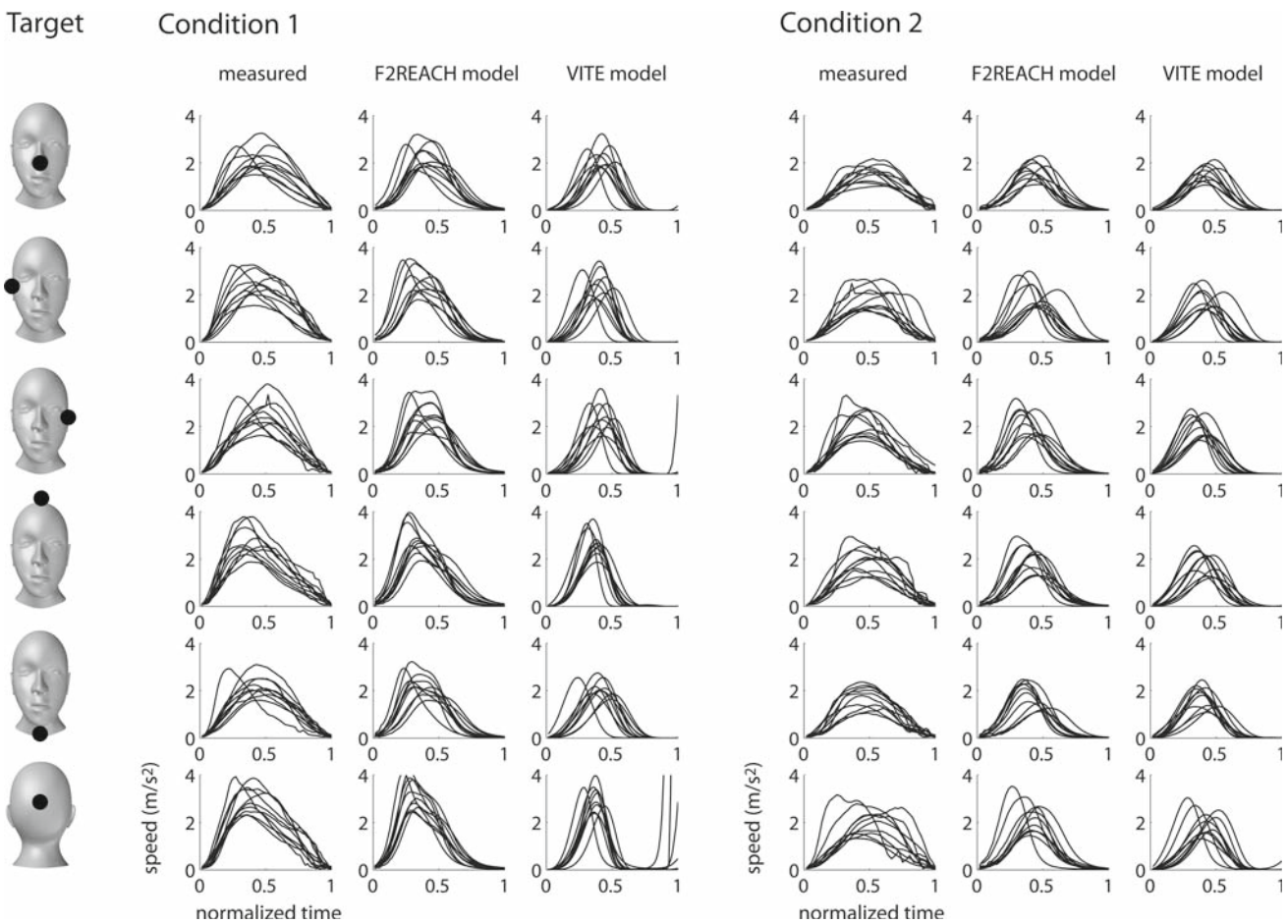

Fig. 9 Normalized time speed profiles of the measured human data and as predicted by the extended F2REACH and original VITE models, for the six target positions and the two initial conditions (see Fig. 5)

within the same condition (see Fig. 10a). The fact that movements to different targets are also clustered (forces underlying similar movements have similar components) suggests a certain regularity in the force field (see Fig. 10b). Finally, the trials related to one movement are clustered according to the subject executing the movement, which shows once more that the parameter values found for the repulsive force fields are not arbitrary (see Fig. 10c). Recall that the sign of the force vector governs the direction of the deviation and that, according to the expression of the modulating function h, the resulting force $\mathbf{F}(\mathbf{x}(t))$ coincides with $\mathbf{v}$ at the beginning of the movement and with $\mathbf{w}$ at the end of the movement, $\mathbf{F}(t = 0) = \mathbf{v}$ and $\mathbf{F}(\mathbf{x} = \mathbf{x}^*) = \mathbf{w}$.

Closer analysis of the clusters shows that the force $\mathbf{v}$, dominating the beginning of the movement, is highly dependent on the starting location in the x and y coordinates (see Fig. 4), whereas the force $\mathbf{w}$, dominating the end of the movement, varies according to the z direction. An intuitive explanation for this result is shown in Fig. 11 where we show the direction and amplitude of the repulsive forces $\mathbf{v}$, $\mathbf{w}$ and their modulated sum $\mathbf{F}(\mathbf{x}(t))$ (Eq. 3) for different types of movement. We see that, in the second starting position (arm extended in front of the body) the subject pushes his or her hand in the direction of the target (see Fig. 11a), whereas, in the first starting position (arm extended along the body) the subject must first push the hand to the right in order to avoid the body, and then bring the hand downwards in order to avoid reaching the limit of the shoulder joints (see Fig. 11b).

Table 4 Percentages of predicted trajectory points comprised within one and two standard deviation volumes (1 SD and 2 SD), see Fig. 6c, for the extended F2REACH and original VITE models

	F2REACH	VITE
1SD	54.64 ± 19.17	31.11 ± 8.44
2SD	80.89 ± 14.82	39.76 ± 9.18

This error measure is highly restrictive as it penalizes the model predictions at points where the five trials per subject and movement type are very consistent

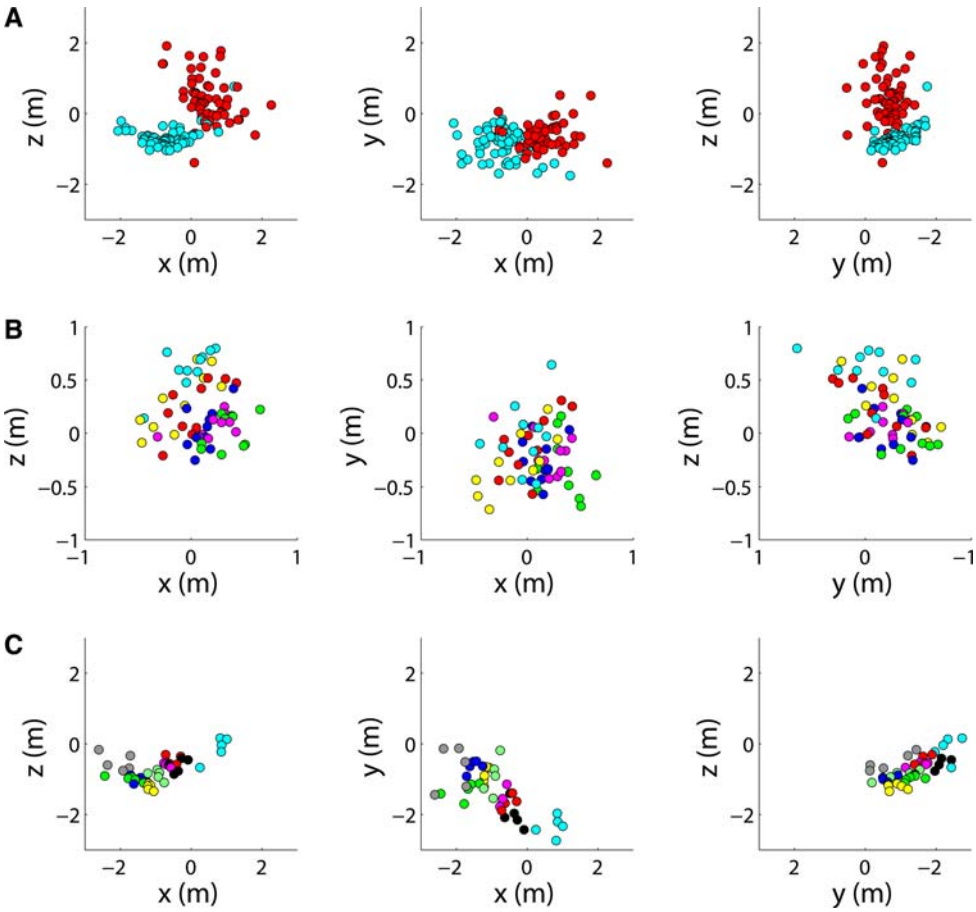

Fig. 10 Components of the repulsive forces **v** and **w**. **a** We show the components of the first repulsive force **v** for the two conditions: arm extended along the body (*red*) and arm extended in front of the body (*blue*). Two practically non-overlapping clusters can be observed showing a consistency of the parameter values within one condition. **b** We show the components of the second repulsive force **w** in the first condition for the six targets (different scale). Again the parameter values are clustered such that movements oriented toward one target are close together, showing a regularity in the repulsive force field. **c** We show the components of the first repulsive force **v** in the first condition and target right ear for the ten subjects. Clusters corresponding to the subjects can be identified for the five trials representing the movement

To better understand the effect of the forces when starting from the same initial condition, we compared the values found for the force components when reaching to two different targets (Fig. 11b, c). Unsurprisingly, the repulsive vector **v** is coherent across conditions irrespective of the target position, whereas the repulsive vector **w** depends on the target position and moves along the normal to the head surface at the target's position.

We also considered whether the magnitude of the repulsive force is related to the geometry of the subject's body, such as the length of the forearm for example. We observed a linear correlation between these two quantities (shown in Fig. 12): the shorter the arm, the more the hand must be pushed away to circumvent the head. Finally, we observed that the vectors of repulsive forces were coherent across subjects. These results are in agreement with the driving hypothesis of our model, namely that the curvature of reaching movements is the result of an explicit encapsulation of the task constraints in a control system which would, in the absence of constraints, produce straight-line motions. However, the opposite is not true, as we find non-null forces for quasi-straight movements, which are parallel to the motion. In the movements we have considered here, the task constraints comprise geometrical constraints related to the body.

5.4 Stability analysis of the model

The dynamical system described in Eq. 1 is globally asymptotically stable around a unique equilibrium point, the target position $\mathbf{x}^*$. We have omitted the analytical proof but the interested reader can convince themselves by computing the determinant of the Jacobian of the dynamical system around the fixed point and observe the latter to be always negative. Next we define the conditions under which the F2REACH model including the repulsive force field (see Eq. 2) is guaranteed to converge to the target. Let there be a perturbation that drifts the hand far away from the initial and target positions, such that $|\mathbf{x}(t)-\mathbf{x}(0)|-|\mathbf{x}(t)-\mathbf{x}^*(t)| < \varepsilon$, with $\varepsilon \in \mathbb{R}$ very small and $h(\mathbf{x}(t))$ and $1-h(\mathbf{x}(t))$ approaching 1/2. The system converges to a stable state iff $|1/2(\mathbf{v}+\mathbf{w})| < 1$ such that the amplitude of the repulsive force field is smaller than the normalized attracting vector, i.e., the distance separating the target from the present position only gets smaller through time.[6] Note that all the forces' components found in our study satisfied the above condition.

[6] This result is, however, not valid in the vicinity of the initial position, which acts a second unstable attractor. Since this affects only a transient part of the motion (onset of the movement), which is unlikely to undergo perturbations, this could be ignored for the present study.

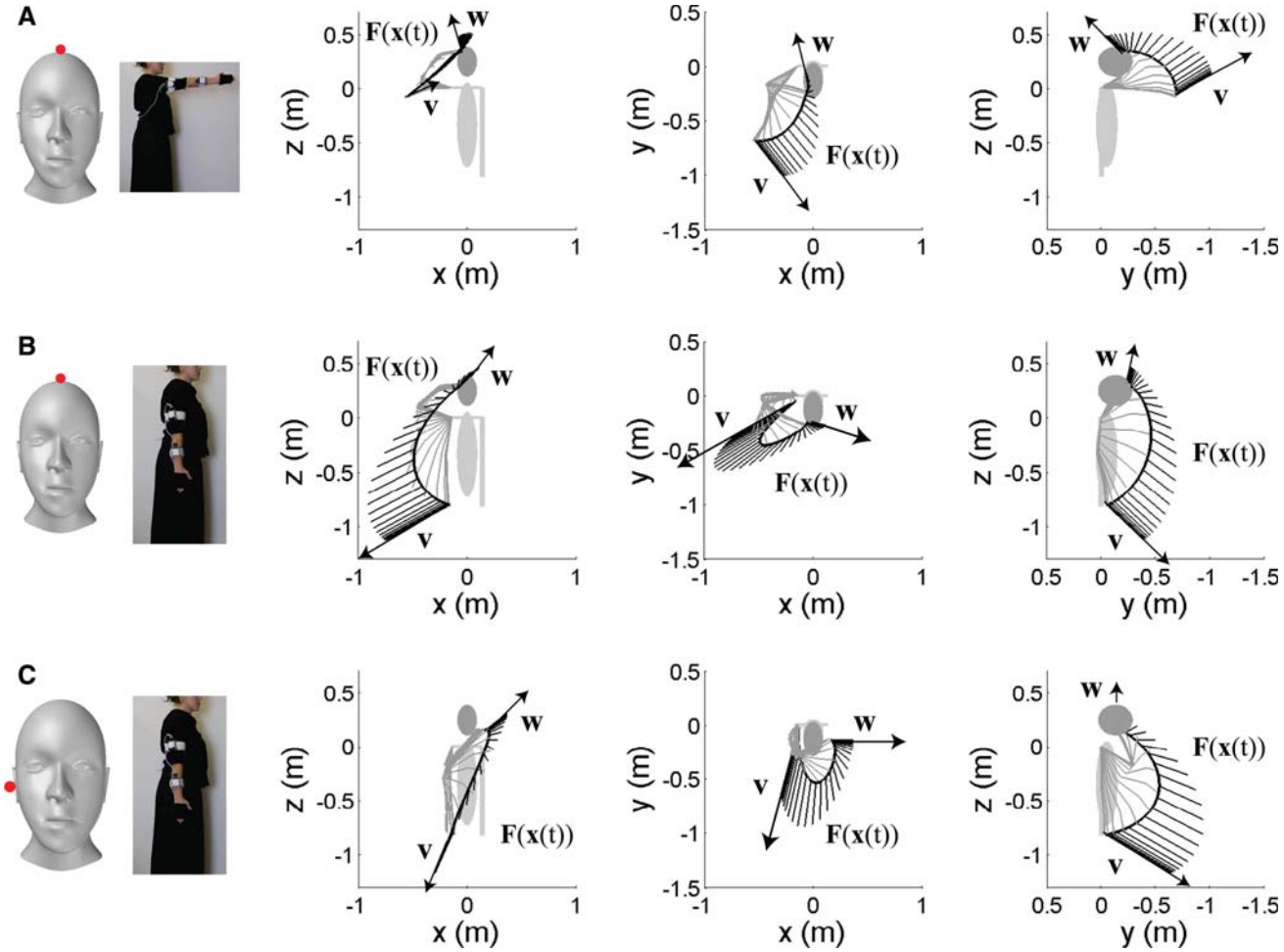

Fig. 11 Physical interpretation of the directions and amplitudes found for the repulsive forces **v**, **w** and their modulated sum $\mathbf{F}(\mathbf{x}(t))$ in our extended F2REACH model (Eqs. 2 and 3) relative to a schematized humanoid. Three movements of the same subject are shown. **a** The subject reaches for the top of the head (movement 4) with the arm extended in front of the body (condition 2). **b** Same position target as in **a** with the arm extended along the body (condition 1). **c** Same condition as in **b**, but the subject reaches to the left ear (movement 3). Due to the nature of the modulating function $h(\mathbf{x}(t))$, i.e., $h(\mathbf{x}(0)) = 1$ and $\lim_{t\to\infty} h(\mathbf{x}(t)) = 0$ (see the Appendix), the resulting force $\mathbf{F}(\mathbf{x}(t))$ coincides with **v** at the beginning of the movement and with **w** at the end of the movement, i.e., $\mathbf{F}(t = 0) = \mathbf{v}$ and $\mathbf{F}(\mathbf{x} = \mathbf{x}^*) = \mathbf{w}$ (Eq. 3). From **a** and **b** one can see that the initial condition affects the repulsive forces **v** and **w**. For example, in the second condition (**a**), **v** is in the direction of the target, whereas in the first condition (**b** and **c**) it is deviated to the right in order to avoid the body and downwards such that the arm does not reach the shoulder extension limit. In addition, **v** is coherent within the same condition (see **b** and **c**). The target position particularly affects the repulsive force **w** (predominant at the end of the movement) that is similar to the normal of the head surface approached. $\mathbf{F}(\mathbf{x}(t))$ was scaled for illustrative reasons

6 Discussion

We have hypothesized that the curvature of unconstrained reaching movements is due to an explicit encapsulation of the task constraints by the CNS in a virtual force field (F2). Movements thus unfold in time according to a dynamical system that attracts the hand to the target position while repelling it from undesirable locations in space (such as objects in the environment, the subject's body and joint limits) and while compensating for unexpected perturbations of the arm. Furthermore, we have argued that the curvature observed in natural movements is not a by-effect of the inherent dynamics of the body but a necessary and voluntarily controlled feature.

In order to probe our hypothesis, we have conducted motion studies in which healthy adult subjects produced natural reaching motions directed to various locations on their head. To highlight the effect that body constraints may have on the curvature of the movement, we asked the subjects to initiate the movement from two locations: one that required the subject to move alongside the body, the other which allowed the subject to move quasi freely. We showed that our mathematical model, the F2REACH model, could predict the major kinematic features of the movements, such as the bell-shaped velocity profile. Most importantly, it could account for both the weak and strong curvatures of the movements.

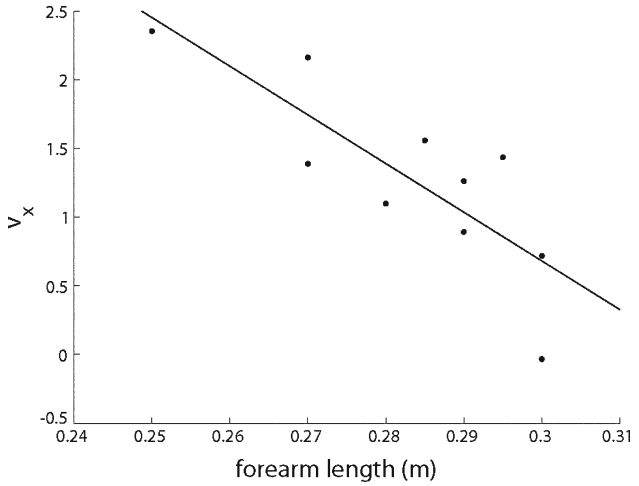

Fig. 12 The amplitude of the repulsive force **w** (Eqs. 2 and 3) is linearly correlated to the length of the subjects' forearms for the movement reaching to the left ear with the right arm (condition 1), showing that the repulsive forces in our model are affected by geometrical features of the body. Intuitively, with a shorter forearm, the hand needs to be pushed stronger away to circumvent the head

This led us to argue that a single controller underlies both straight and curved movements. The controller adapts the trajectory according to multiple constraints the subject has consciously or not decided to take into account. Although we have only shown that it can precisely reproduce the kinematics of self-oriented movements, the model is general and can generate natural movements to any target in both intrapersonal and extrapersonal spaces, e.g., in another study that investigates imitation of unnatural postures we successfully use this model to simulate reaching to objects on a table.

6.1 Assumptions of the model

The first assumption we have followed is that of a *functionally hierarchical motor control system* proposed by Bernstein (1947) and translated partly in Bernstein (1996). The hierarchy consists of four levels: complex actions with abstract goals, dealing with 3-dimensional space, muscle synergies, posture and muscle tone. In our study, we considered the first and second levels, in that we addressed 3-dimensional goal-directed reaching movements, characterized by a single target position. By leaving out the question of how such high-level control is then translated into muscle synergies and the control of posture and muscle tone, we follow the observation that: electrical stimulation of the brain motor area elicits reaching movements in primates (Graziano et al. 2002, 2005) and leg movements in frogs (Bizzi et al. 1982). Interestingly, all of these movements converge to the same position in extrinsic space independently from the initial posture. Thus, the control of these movements seems to use solely the definition of the desired final position, and not a description of low-level muscle activations (in a way functionally similar to muscle synergies when compared to activating individual muscles, see d'Avella et al. (2003)). In addition these studies indicate that reaching movements are extensively represented in the motor cortex.

Another argument in favor of a "high-level" extrinsic 3-dimensional representation of movements come from evidence of the many to one mappings between: (1) muscles and joint configurations, (2) muscles and end-effector positions or (3) joint configurations and end-effector positions. Controlling the hand in a 3-dimensional extrinsic space over an intrinsic joint space is advantageous in that it allows to easily encapsulate task constraints, such as avoiding surrounding objects, and plan movements accordingly (these task constraints would have an infinite number of possible representations in the joint and muscle spaces). Also note that we have assumed that movements were computed in a Cartesian frame of reference located on the body. It would however be conceivable to compute the same movement according to a polar coordinate system without affecting the prediction of the model.

The fact that we do not address the above two lower-levels of motor control, is a limitation of the model. As stated by Bernstein, the problem of translating a kinematic signal encoded in a 3-dimensional extrinsic frame of reference into muscle activations (so-called degrees of freedom problem) is complex because of the redundancy of the muscular system. An infinity of different muscle activations leads to the same kinematic motion of the end-effector. Although this problem is of the highest importance for a complete motor control theory, we do not address this problem here [see d'Avella et al. (2003), Todorov and Jordan (2002) and Guigon et al. (2007) for possible solutions].

Another important assumption we make is that the CNS can represent forces internally. Our model is based upon a force field that encapsulates the constraints of the task, which implies the knowledge of a mapping between different locations in the subject's peripersonal space and virtual repulsive forces. It thus requires the existence of an internal model of the environment in terms of attractive or repulsive force fields in the brain. The above hypotheses are not at odds with the literature. There is substantial evidence that the brain is capable of learning an internal representation of external forces in order to adapt its control of the motion of the hand (Shadmehr and Mussa-Ivaldi 1994; Conditt et al. 1997; Shadmehr and Brashers-Krug 1997; Thoroughman and Shadmehr 2000; Gandolfo et al. 1996), when subjected to these for a long enough period of time. Another force that is centrally represented and integrated in the internal dynamic control models for reaching is the gravitational force (Shadmehr and Mussa-Ivaldi 1994; Papaxanthis et al. 1998). In our model the geometry of the body and external objects, among other factors, contribute to the force field.

Accordingly, McIntyre et al. (1995) have shown that the brain may integrate an external constraint such as a curved surface through an a priori internal model of the surface geometry.

6.2 Properties of the model

Interesting properties of the F2REACH model for motor control are: (i) the system is *asymptotically and globally stable*; (ii) it exploits a biologically plausible *signal-dependant noise* and (iii) planning of the movement is done through closed-loop control. This enables on-the-go re-computation of the motion in the face of perturbation or imprecision in the sensory-motor loop. Closed-loop control through afferent and efferent internal feedback loops (Desmurget and Grafton 2000) allows to take into account the uncertainty of the "real-world" instead of just "playing a prerecorded tape" (Todorov 2004). We suggest that only an online mechanism that tightly couples movement planning with movement execution could explain the irregular curvatures observed in some of the trials; the latter were likely due to an on-the-go correction of the trajectory.

Most importantly, we have proposed a force field framework as a powerful mechanism for integrating various constraints related to, e.g., the dynamics and geometry of the arm, external objects and the person's own embodiment, into a unique and generic controller. Whereas the goal of the controller is encoded according to kinematic variables (a position to reach), the constraints are encoded in dynamic variables, the force field, and may as well be expressed in an intrinsic (limit joint angles) or extrinsic (surrounding objects) frame of reference. This framework could reconcile findings that argue for both dynamic and kinematic planning (Vetter et al. 2002; Admiraal et al. 2004), in providing a computational account for how the dynamics of the arm can be taken into account in kinematic planning (Sabes and Jordan 1997). It also explains how external objects might influence the trajectory of the hand (Brenner and Smeets 1995).

Furthermore, the representation of this environmental force field generalizes to performing the motion faster or slower (Harris and Wolpert 1998). This is equivalent to learning to vary the value of the factor β in our model (see Eq. 1). Finally the representation of the force field, although local, extends to nearby locations (smoothly decaying away from the position of the perturbation). Similarly, our expression of the force field is spatially continuous.

The extent to which the model's predictions can be generalized to any reaching movement remains to be shown, since we only demonstrated a good agreement of the model with data from reaching movements directed to the head. The movements we have addressed are nevertheless quite generic in that they were entirely unconstrained. For example, we did not observe a reduction of the degrees of freedom as in Klein Breteler et al. (1998) where the subjects had a tendency to produce movements in 2D rather than in 3D (see example in Fig. 1). In addition many of the velocity profiles recorded, exhibited asymmetric velocity profiles similar to those observed (Gielen et al. 1985; Brown and Cooke 1990). These characteristics are present in all reaching movements and we are thus confident that the model is generic in its representation of the class of reaching movements.[7]

The force field in our model is parameterized by two constant forces and is thus only an approximation of the real underlying force field. This approximation may lead to imprecisions in the model's predictions, especially in places where the field changes importantly locally.

While our model proposes a way in which the brain may encapsulate all types of motion-related constraints (e.g., body and joint-limits avoidance, inertia of the arm) within a general controller of reaching movements, we do not provide a general method for expressing these constraints in the form of a force field. Our future efforts will concentrate on segmenting the contributions of different constraints and on a mechanism that would allow to learn these through experience.

6.3 Predictions of the model

Our model is consistent with several experimental observations and provides a theoretical basis for their interpretation.

For example, in different pointing and reaching studies, systematic misdirections of the fingertip trajectory were observed (de Graaf et al. 1991, 1994; Brenner and Smeets 1995). The misdirections were clockwise and anticlockwise when pointing to targets on the right and on the left frontal space, respectively. To explain their results the authors hypothesized a distorted and contracted internal representation of space (de Graaf et al. 1991, 1994) or speculated that the subjects anticipate the purpose of the target (Brenner and Smeets 1995). Within the repulsive force field framework we propose in this paper, these misdirections are created by the geometrical relationship between the target and the subject's body.[8] Our model predicts that if one was to repeat the experiment in a different part of the workspace where the misdirections are mainly due to body avoidance, the misdirections would be anticlockwise and clockwise when the target is respectively

[7] Current work of ours has applied the model to account for reaching movements oriented to targets on a table in natural and unnatural postures where an artificial constraint is introduced. Preliminary results show that the model again encapsulates with high accuracy all the features of the movements (unpublished data).

[8] These two similar studies, de Graaf et al. (1991) and Brenner and Smeets (1995), puzzlingly reported different results. We suggest that the differences observed can be attributed to the distance chosen from the subject to the initial position of the hand [25 cm in Brenner and Smeets (1995) and 40 cm in de Graaf et al. (1991)], as the repulsive forces responsible for avoiding the body would fade away as this distance increases.

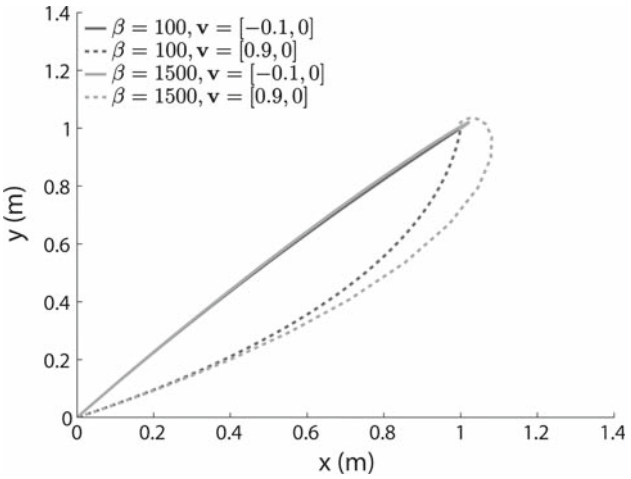

Fig. 13 Prediction of the F2REACH model: the curvature increases with higher speed, here equivalent to higher β values. The effect is not visible in quasi-straight movements

right and left from the closest virtual line connecting the subject's trunk with the hand's initial position.

Furthermore, our model predicts that faster movements may be more curved, as shown on Fig. 13. Even though this prediction has been empirically observed (Klein Breteler et al. 1998), it contradicts several experimental and theoretical studies that have shown curvature-speed invariance (Nishikawa et al. 1999; Sha et al. 2006; Liebermann et al. 2008) and suggest that speed and path are planned independently (Todorov and Jordan 2002; Torres and Zipser 2002; Biess et al. 2007). In our model speed modulates the curvature of the path by construction. However, the deviation is also proportional to the magnitude of the repulsive force field such that this effect is particularly important for highly curved movements (see Fig. 13). This might explain why curvature-speed invariance is more consistently observed, as highly curved movements are rarely studied. Otherwise, an additional compensatory mechanism should be added to the model that modulates the force field as a function of desired speed.

Finally, the model suggests that the asymmetry of the velocity profile is due to the difference in directions between the repulsive force field and attracting vector. Finally, even though the curvature of a movement is highly systematic and reproducible (Soechting and Lacquaniti 1981; Pellegrini and Flanders 1996; Admiraal et al. 2004), our model would predict that if you alter the geometry of the subject's body, such as adding a false belly for example, then reaching movements will be displaced away from the artificial object even if this object does not interfere with the original trajectory. Our model also predicts that the shape of the object would matter.

6.4 Neural correlates of the model

Most importantly, the F2REACH model we propose is compatible with neurophysiological studies. Primate brain areas have been identified as the loci of the computations involved in the original VITE model (Bullock et al. 1998). Specifically, it was shown that the model's variables display the same dynamics of activation (e.g., response profiles and latency of activity onset) as that of populations of neurons: the hand velocity might be represented in area 4, whereas the hand acceleration and position in area 5. Note that the extended F2REACH model solicits only quantities that would be easily accessible to the CNS such as distances from the target and initial positions.

A novel feature of the model is the repulsive force field that shapes the landscape of the workspace, meaning that not each position is equally likely to be visited. In other words, the model assumes the existence of neural populations coding for forces related to the body and surrounding objects. Area 5 is a putative region for the computation of the force field, as it receives abundant somatosensory and visual inputs that are necessary for the encapsulation of the geometrical properties of the body and surrounding objects in an internal model (Scott et al. 1997; Graziano et al. 2000). We thus predict the existence of a population of neurons in area 5, whose activity would be close to baseline during straight movements and would rotate in curved movements. In addition, the activation of these neurons would be modulated by the introduction of new objects in the workspace.

6.5 Conclusion

We showed that not only the spatial, but also the temporal features of unconstrained and naturally curved reaching movements could be modeled through a dynamical system modulated by a virtual force-field. We found that the model was in very good agreement with kinematic data from human motions, during unconstrained reaching movements directed to the head. We showed that the natural curvature of these movements could be attributed to the interplay between a target attractor and virtual repulsive forces that encapsulate a representation of the geometry of the subject's body. Such a representation is a simple and powerful way to generate kinematically-driven trajectories that comply with the underlying dynamic constraints.

7 Appendix

7.1 Original VITE system

The original VITE model's dynamics as given by Bullock and Grossberg (1988):

$$\begin{aligned} \dot{\mathbf{y}}(t) &= \alpha(-\mathbf{y}(t) + \mathbf{x}^*(t) - \mathbf{x}(t)) \\ \dot{\mathbf{x}}(t) &= \beta t^{\nu}\mathbf{y}(t) \end{aligned} \tag{4}$$

where $\mathbf{x}(t)$ corresponds to the current position of the hand in a three-dimensional extrinsic frame of reference and $\mathbf{x}^*(t)$ is the location of the target (see Fig. 4). $\mathbf{y}$ is a secondary variable related to the hand velocity. α and β are real positive time constants and ν is a real positive exponent parameter. The model recomputes at each time step the hand position $\mathbf{x}(t)$, so as to generate an overall straight trajectory to the target that follows a bell-shaped velocity profile. The first term of the equation ensures that the unprimed acceleration vector $\dot{\mathbf{y}}(\mathbf{t})$ is always directed toward the target, i.e., $\mathbf{x}^*(\mathbf{t}) - \mathbf{x}(t)$, so that the target's position $\mathbf{x}^*$ forms a unique attractor of the system. The amplitude of the acceleration $\dot{\mathbf{y}}(\mathbf{t})$ is proportional to the distance separating the hand and the target. $\mathbf{y}(t)$ grows quickly at the beginning of the movement and slows down exponentially towards the end of the movement. To compensate for this asymmetric velocity profile, $\mathbf{y}(t)$ is scaled down in the second equation by a time-dependent variable βt^{ν}. $\dot{\mathbf{x}}(\mathbf{t})$ is the hand's velocity and can be viewed as the output activity of a corresponding neural population that would control agonist muscle motoneurons (Bullock and Grossberg 1988).

7.2 Nonlinear functions used in the F2REACH model

The form of the nonlinear function $\mathbf{g}$ in Eq. 1 is the following:

$$\mathbf{g}(\mathbf{u}) = |\mathbf{d} - \mathbf{u}|\mathbf{u} \tag{5}$$

where the control vector $\mathbf{u}(\mathbf{t}) = \mathbf{x}^*(t) - \mathbf{x}(t) + \boldsymbol{\eta}$ is the vector separating the actual hand position $\mathbf{x}(t)$ from the desired hand position $\mathbf{x}^*(t)$ (does not need to be stationary) with signal dependant noise $\boldsymbol{\eta}$. The operator $|\ |$ stands for the norm of the vector and $\mathbf{d}$ is defined as:

$$\mathbf{d}(t) = \mathbf{x}^*(t) - \mathbf{x}(0) \tag{6}$$

the vector between the target $\mathbf{x}^*(t)$ and initial position $\mathbf{x}(0)$, such that the term $|\mathbf{d} - \mathbf{u}|$ is equivalent to the distance separating the actual position of the end-effector from its initial position. t is set to 0 each time a new movement is initiated. In the absence of noise in the control signal $\mathbf{u}$, the multiplicative factor $|\mathbf{d} - \mathbf{u}|$ would be 0 at $t = 0$ and no movement would be initiated.

The function h that modulates the force field in Eq. 3 is defined by:

$$h(\mathbf{u}) = \frac{|\mathbf{u}|}{|\mathbf{u}| + |\mathbf{d} - \mathbf{u}|} \tag{7}$$

and normalizes the amplitude of the control signal $\mathbf{u}$.

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Chapter 7

Discussion

Be driven by a question and not by a technique.

Eric Kandel. As cited by John W. Krakauer.

7.1 Neural Imitation

In the introduction we raised several questions relative to *neural imitation*. Here we review how this thesis has addressed, and partially answered these questions. We also highlight the neural mechanisms that remain to be explained and provide suggestions for how to tackle these computationally.

7.1.1 Main contributions of the thesis

What information subserves the human ability to imitate? To actually implement a neurocomputational model of imitation in Chapter 3 has allowed the identification of the variables and types of information required. Specifically, the imitation of Goldenberg's stimuli, i.e. hand postures relative to the head, could be achieved by combining: (1) an unsupervised learning mechanism that learns somatotopic representations of the necessary information and (2) a supervised learning mechanism that learns the associations between these somatotopic representations. We have identified the following sensory information as necessary: visual, proprioceptive, tactile, spatial and orientation. The use of visual information is immediate as the stimulus to imitate is visually processed. Similarly, proprioceptive information is necessary, as the brain must know the actual posture (or state) of the body in order to reach the imitated posture. In addition, our model has highlighted the use of tactile information, due to the position of contact between the face and the hand present in the stimuli considered. Finally, as the imitation process reproduced the spatial relations between parts of the body, access to a spatial representation of the body also

was necessary. Note that in the literature this spatial representation is referred to as the body schema (Pick, 1922; Holmes & Spence, 2004).

How is the information underlying imitation represented and processed in the brain? The most interesting contributions of this thesis are related to how the brain processes and transforms information in an imitation task.

In Chapters 4 and 5 we provided **neuropsychological evidence** that meaningless gestures, i.e. gestures that have no apparent goal, are also imitated using a goal-directed strategy. This means that the imitator's body posture is not imitated as a whole, but first decomposed into imitative goals. We define an imitation goal as a geometric constraint on the relative spatial position between two parts of the body. For example, to imitate a person that touches the nose with the thumb would translate into the following imitation goal: the nose and thumb need to have the same spatial position. To imitate a vertical orientation of the hand would correspond to a similar imitation goal: the position of the middle finger needs to be one hand-length above the position of the wrist. Importantly, these imitation goals are interpreted with respect to the observer's body, and reconstructed within their physical embodiment. As such, a goal-oriented strategy (Bekkering et al., 2000; Bekkering & Wohlschläger, 2002; Wohlschläger et al., 2003) solves to some extent the **"correspondence problem"** (Goldenberg et al., 2001), i.e. difficulties related to the differences in size, shape and perspective between the demonstrator and imitator (Nehaniv & Dautenhahn, 2001; Alissandrakis et al., 2002). An alternative hypothesis, where the perception of a stimulus directly matches the corresponding motor action (Butterworth, 1990; Gray et al., 1991), implies that a motor representation of the observed body posture already exists in the brain, which would be a rather inefficient encoding approach due to the infinite number of possible body postures. Instead, we believe that imitation is a generic mechanism that allows one to learn complex and unfamiliar body postures, through the reconstruction of geometric aspects specific to the observed body posture onto one's own body.

After training, the neurons in our neurocomputational model started functioning as **"mirror neurons"**, i.e. neurons that fire both during the execution and observation of a specific action. These neurons, originally discovered in the monkey premotor cortex (Pellegrino et al., 1992), selectively fire during goal-directed actions, such as different types of grasping (Gallese et al., 1996, 1996; Rizzolatti, Fadiga, Gallese, & Fogassi, 1996). Similar mirror neurons were reported in the human brain by a body of neuroimaging studies. We say similar since the mirror neurons reported code not only for goal-directed actions (Gazzola & Keysers, 2009; Kilner, Neal, Weiskopf, Friston, & Frith, 2009), but were also observed in meaningful intransitive actions such as pantomime (Dinstein, Hasson, Rubin, & Heeger, 2007; Chong, Cunnington, Williams, Kanwisher, & Mattingley, 2008; Dinstein, Gardner, Jazayeri, & Heeger, 2008) and

even meaningless actions (Lingnau, Gesierich, & Caramazza, 2009). Moreover, the human brain areas found to contain mirror neurons are more numerous and extensive than in the monkey cortex, such that one speaks of a *mirror-neuron system* located in the inferior frontal cortex, as well as inferior and superior parietal cortices. Parts of these brain regions are thus active when a subject performs an action and also when the subject sees another individual performing an action. However, several studies have failed to observe *selective activation* of these motor areas, i.e. visual and motor brain activations that correlate only when the same action is seen or observed (Dinstein et al., 2007; Chong et al., 2008; Dinstein et al., 2008; Lingnau et al., 2009). Only one study has provided evidence for a common neural code between the observation and execution of motor actions (Lingnau et al., 2009), whereas another study has provided evidence in favor of overlapping, but distinct networks (Dinstein et al., 2008).

In our neurocomputational model of imitation presented in Chapter 3, neurons that initially fired within exclusively one modality - while seeing, feeling or executing a specific body posture - after training also fired during the other modalities in a correlated fashion. This is due to the Hebbian supervised mechanism that we used during learning. More specifically, we associated the visual, tactile and spatial modalities related to a specific body posture, and subsequently used visual information to retrieve the corresponding tactile sensation and motor act. Our model suggests that the mapping between the visual and motor sensation of a body posture needs not to be direct, but can also be performed at the level of extracted features that represent the body posture (e.g. position of contact between two parts of the body). Furthermore, the model suggests that "mirror neurons" can also relate the tactile modality with the motor modality, as well as with the visual modality, if their patterns of neural activation are correlated. This prediction is biologically validated by Etzel, Gazzola, and Keysers (2008), who report the existence of human mirror neurons that fire when a subject executes an action and hears a sound that is provoked by someone else executing the action. Note that a given body posture can be viewed as the outcome of an action, whereas an action is a time-series of body postures. We will use these terms interchangeably, as claims related to body postures can be generalized to actions.

These considerations raise the following essential question: have **"multimodal neurons"** frequently been mistaken for "mirror neurons"? In their initial definition, mirror neurons were restricted to goal-directed movements that mediate action understanding (i.e. a semantic representation of the action). However, what does action understanding reduce to in the context of meaningless gestures? We suggest that understanding a meaningless action reduces to a set of extracted relations between parts of the body that include positions and possibly orientations.

Where in the brain is the information underlying imitation rep-

resented and processed? We have identified three stages in the process of imitation of meaningless gestures:

1) *Visual extraction of imitation goals.* Chapter 4 provided neuropsychological evidence that in order to imitate, the brain first extracts representative features of the observed body posture. These visual representative features may be contact or relative positions between parts of the observed body, as well as orientations of body segments. The extracted features then become goals that will guide the imitation.

2) *Dynamic localization of body parts.* The imitation goals extracted in Stage 1 need to be interpreted *with respect to the subject's embodiment.* For example, an imitation goal may correspond to bringing *one's own body part* to a location relative to another body part. In order to execute an imitation goal, the brain first needs to compute the current positions of the body parts involved. This process of localization of body parts is equivalent to the body schema, and requires a dynamical computation of the current state of the body based on muscles activations (partially modeled in Chapter 3).

3) *Motor execution of the imitation goal.* A visual stimulus to imitate provides only the *final* body posture that the subject desires to reach, as computed in Stage 2. For this reason, we argue that motion attractors (see Chapter 6) are particularly appropriate for guiding the motor execution stage of imitation. The motion of a body part towards the desired final position then unfolds in time without the need to specify a complete trajectory. The main advantage of such a simple control mechanism is robustness to external perturbations, such as changes of the desired final position or enforced displacement of the end-effector.

When there is more than one imitation goal acting on the same end-effector (e.g. constraints on both the position and orientation of the hand), there is the need to coordinate these imitation goals. The *goal-coordination* might be performed either before or after the Stage 3. In the preceding stages each goal is processed independently, which means that several instances of neural activation associated to different goals may coexist in the brain. If goal-coordination is performed before stage 3, the brain directly computes a motor program that resolves a multiple constraints problem, where each goal is treated as an additional constraint.[1] If goal-coordination is performed after stage 3, the brain first computes motor programs that solve each goal separately and subsequently coordinates these motor programs.

Interestingly, four brain areas were consistently activated in PET and fMRI imaging studies of the imitation of meaningless gestures (Decety et al., 1997; Hermsdörfer et al., 2001; Peigneux et al., 2004; Chaminade et al., 2005; Mühlau et al., 2005). We speculate that each of these brain areas is the neural correlate of one of the four imitation stages identified above. The brain pathway of imitation described below is summarized in Figure 7.1.

In particular, the first *visual extraction* stage might be processed in the **"Ex-**

[1] Note that the terms "goal" and "constraint" will be used interchangeably in this chapter.

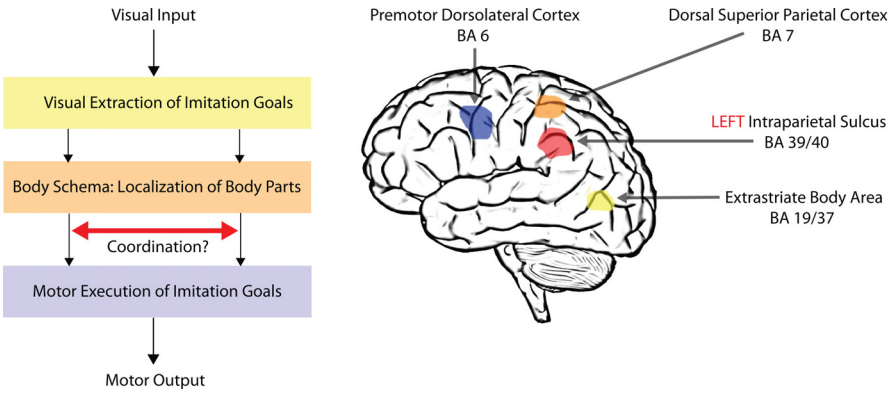

Figure 7.1: Stage decomposition (left) and corresponding brain pathway (right) of imitation of meaningless gestures.

trastriate Body Area" at the level of the occipito-temporal junction in Brodmann Area (BA) 19/37, a brain area that visually processes stimuli that represent body postures (Peigneux et al., 2000; Downing et al., 2001; Astafiev et al., 2004).

The second stage of *dynamic body parts localization* may be performed by the **dorsal superior parietal cortex (BA 7)**, since this brain area is consistently activated when human subjects are asked to localize their own body parts (Felician et al., 2004). Accordingly, a lesion in this brain area leads to a deficit in pointing to one's own body parts, but preserved ability to point to the body parts of other persons (Felician, Ceccaldi, Didic, Thinus-Blanc, & Poncet, 2003; Felician & Romaiguère, 2008). In primates, the superior parietal cortex integrates inputs from the somatosensory cortex, so as to monitor the position of the limbs (Sakata, Takaoka, Kawarasaki, & Shibutani, 1973; Mountcastle, Lynch, Georgopoulos, Sakata, & Acuna, 1975; Graziano et al., 2000). These kinematic aspects of limb movement seem to be processed in a body-centered coordinate system (Kalaska, Cohen, Prud'Homme, & Hyde, 1990; Caminiti, Johnson, Galli, Ferraina, & Burnod, 1991; Lacquaniti et al., 1995). In addition, the brain area BA7 is part of the "where" visual stream that processes the spatial and dynamic (e.g. motion) aspects of visual stimuli (Ungerleider, 1985), which makes it a good candidate for processing the body schema. Indeed, visuospatial signals that arise from the body need to be integrated in order to build a coherent representation of the body posture. A different role of the superior parietal cortex may be to direct attention to specific parts of the visual stimulus, by modulating the activity in the visual cortex (Kastner & Ungerleider, 2000).

The third *motor execution* stage may be processed by the brain region with the highest motor gradient, which is the **dorsolateral premotor cortex (BA 6) in the inferior frontal gyrus**. This brain area has been related for example to sensory-guided movements (Toni, Schluter, Josephs, Friston, & Passingham, 1999), movement selection (Deiber et al., 1991) and motor learning (Jueptner et al., 1997).

Finally, the best candidate for processing the *goal coordination* stage is the **left intraparietal sulcus (IPS) within the inferior parietal lobule (BA 40)**, since lesions in this area systematically lead to apraxic imitation (Haaland et al., 2000; Goldenberg & Karnath, 2006; Tessari et al., 2007). Indeed, the types of apraxic errors analyzed in Chapters 4 and 5 are best explained by an impairment that affects the goal-coordination stage (see Section 7.2.1 for more details). However, the role of the left intraparietal sulcus may not be limited to goal-coordination and could also be involved in processing the body schema, as this brain area is consistently activated when subjects are asked to mentally rotate their body parts (Parsons et al., 1995; Bonda et al., 1995; Parsons & Fox, 1998) or to access topographical knowledge about their body (Corradi-Dell'Acqua et al., 2008; Le Clec'H et al., 2000)[2]. A lesion in the left IPS leads to the inability to point to the body parts of other persons, but not one's own body parts (Felician et al., 2003; Felician & Romaiguère, 2008). Furthermore, this brain area is truly a visuomotor area, activated in action observation tasks (Grèzes, Costes, & Decety, 1998) and disrupting the execution of grasp movements under TMS (Tunik, Frey, & Grafton, 2005; Rice, Tunik, & Grafton, 2006).

7.1.2 Limitations and Future Work

In this section we review several limitations of our neurocomputational and neuroanatomical models. In addition, we propose research directions that may help to overcome these limitations.

Data dimensionality and frames of reference. Our neurocomputational model of imitation uses data with varying dimensionality: very large for the tactile, but small for the spatial and visual information. In addition, appropriate frames of reference for representing this information were were chosen according to some specific neurophysiological data. However, due to lack of clear neurophysiological evidence, it is possible that the dimensionality and frames of reference used in our model do not reflect the reality of the brain.

Estimation of the spatial information. This thesis proposed a connectionist neural model of imitation that learns a compact spatial map of the body. Whereas the body surface contains tactile and proprioceptive sensors, there are no position sensors. Therefore, the spatial information would need to be estimated from correlations between modalities that are directly available to the CNS such as visual, tactile and proprioceptive information.

[2]To assess the topographical knowledge about body parts in neuroimaging studies, subjects are asked to determine the distance between two parts of their body or to judge whether a given body part is higher than the shoulder.

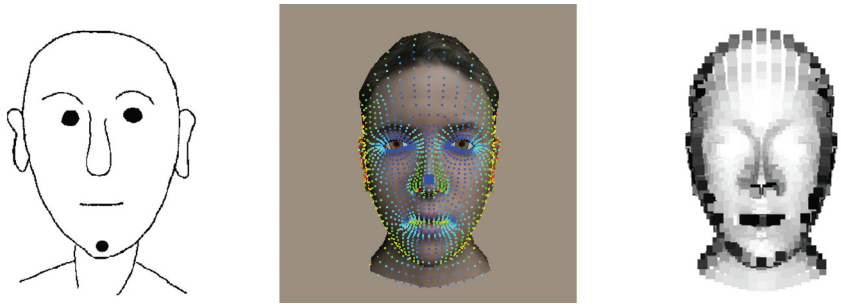

Figure 7.2: Basis for Bayesian imitation. The distribution of pixels in the visual stimulus (left) is very similar to the distribution of body surface gradients at the level of tactile sensors (middle), in particular when these gradients are projected onto the stimulus plane (right).

Bayesian imitation. The neuro-computational model developed in this thesis hypothesizes that imitation is acquired through an associative process (e.g. by learning the visual appearance of an executed body posture while looking at oneself in the mirror). However, imitation existed well before mirrors became common place. We suggest that imitation may also be achieved by looking at the similarity between the distribution of points in the visual stimulus to imitate (e.g. pixels) and orientation information of the body (e.g. gradients of the body surface at the level of the tactile sensors, projected onto the stimulus plane); see Fig. 7.2. The extraction of imitation goals (not directly addressed by this thesis) thus may be achieved through a Bayesian process that infers the underlying posture (e.g. by maximizing the likelihood) given the distributions of visual and orientation information.

Brain pathways of imitation. This thesis proposes which brain areas are crucial for the imitation of meaningless gestures, but does not provide the detailed information flow across brain areas. In particular, we can not distinguish between the information flows shown on Fig. 7.3, which correspond to resolving the multiple-constraints problem of imitation at the level of: (A) motor programs, (B) spatial goals or (C) a mixture of both. Note that all of these brain areas are extensively interconnected, and probably in a bidirectional fashion, such that the flows shown in Fig. 7.3 by no means give the complete picture.

Formally address the coordination between multiple imitation goals. This thesis does not formally address the extremely interesting research issue of how reproductions of multiple goals may be neurally coordinated. We envision two different means to resolve the related multiple-constraints problem. One possibility is that the brain *explicitly* finds an optimal body posture, or motor program, that best satisfies the imitation goal. For example Gribovskaya and Billard (2009) propose a dynamical system that guides the hand to a target position, constrained by a specific orientation. Another possibility is that the problem is *implicitly* resolved at the level of coordinated motor programs,

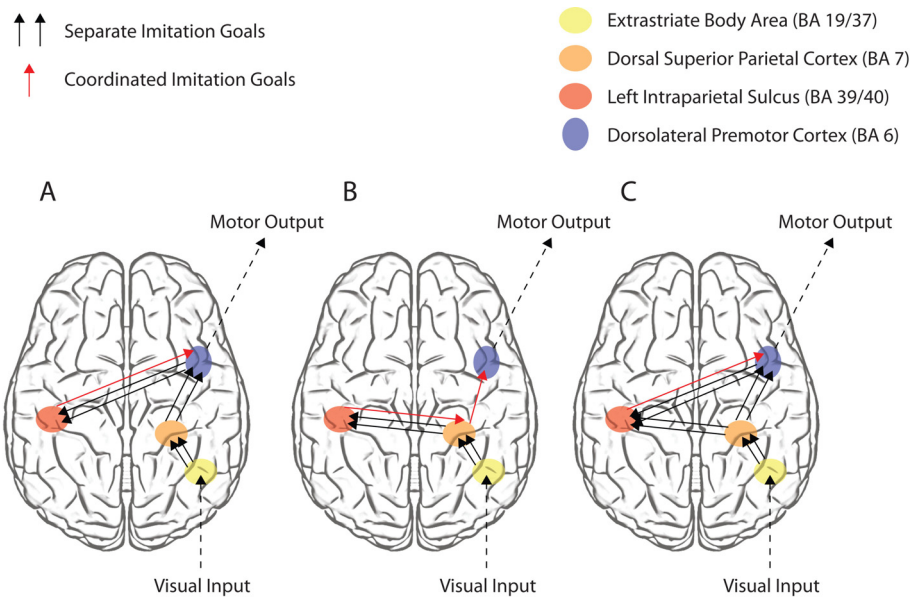

Figure 7.3: Our model does not distinguish between different types of information flow, shown for the most complex *left visual field - left hand* condition in Goldenberg's tachistoscopic experiment. In this condition only the right hemisphere "sees" the visual stimulus and also controls the imitating hand, such that information needs to be transferred to and from the left hemisphere (see Chapter 3). According to our "goal-coordination" hypothesis, the left hemisphere is solicited to coordinate multiple imitation goals or to resolve the imitation multiple-constraints problem. The manner in which the left hemisphere is solicited is not specified by our model, and could be at the level of: (A) motor programs, (B) spatial goals directly or (C) a mixture of both. Note that not all the brain pathways are shown, such the complete picture is probably more complex.

where each goal drives a different dynamical system. In this framework, the goals are coordinated by coupling the concurrent motor programs. A consistent experimental finding are functional units in the frog spinal cord that generate a specific pattern of muscle activation, which can be characterized as a force field (Bizzi et al., 1991). Interestingly, the force fields of different units seem to be combined independently with a linear sum (d'Avella & Bizzi, 1998).

In addition, the brain may optimize the desired body posture in a specific planning stage *prior to* the generation of the imitation movement. We advocate however that the problem is resolved on the fly during movement generation, as in Gribovskaya and Billard (2009). Future work can focus on modeling the potential processes for goal-coordination, and contrast the model predictions to experimental data.

7.2 Apraxia

In the introduction we also asked two questions related to *apraxia*. We here discuss the findings that this thesis has provided, their limitations and several promising research directions.

7.2.1 Main contributions of the thesis

Is the imitation deficit in apraxia characterizable? The first question we asked is whether the imitation deficit in apraxia is characterizable; that is, the deficit creates reproducible and systematic errors, rather than random and noisy errors with no interpretable structure. If the observed imitation deficit is characterizable, then it makes sense to search for a specific neural mechanism whose impairment might produce these errors, by thoroughly investigating the error structure. Neuropsychological experimental studies of imitation, conducted in collaboration with the Vaud University Hospital Centre (CHUV), indicate that the *imitation deficit in apraxia is characterizable* (see Chapter 5). Specifically, we extended regular clinical tests of apraxia to encompass multiple trials and a second examination several weeks later. The errors observed were highly systematic across trials and time, and sometimes similar across patients, encouraging us to look for a specific neural mechanism whose impairment could lead to the errors observed. We describe this neural mechanism next.

Can we characterize the imitation deficit in apraxia? To gain a better understanding of the impaired neural mechanism underlying apraxic imitation, we decomposed the errors observed in Goldenberg's callosal patient. We looked at five geometric variables that taken together define the visual stimulus to imitate (see Chapter 4). This decomposition allowed us to observe a very interesting goal dissociation. When the imitation process could not properly access the left hemisphere, Goldenberg's patient made only errors in the position of contact between the hand and face, but correctly imitated the hand posture (an example is given in Fig. 7.4).

Following this observation, we tested two hypotheses for the mechanism causing these errors in the position of contact: (1) faulty representation of the body in the brain, i.e. an impaired body schema meaning that the patient does not have access to the precise position of contact on the face or (2) faulty control of the hand, meaning that the patient knows where to bring the hand but sends an incorrect motor command. To test these two hypotheses we extended Goldenberg's experiment with a baseline condition where the patient imitated only the position of contact between hand and face (see Chapter 5). Neither of these two hypotheses was validated, as the three left-parietal patients precisely reached to the correct position of contact, with both the thumb and index

Figure 7.4: Example of a typical right-hemisphere apraxic error in Goldenberg's patient with callosal lesions. The posture of the hand is correctly imitated but not the position of contact between face and hand.

finger.[3]

These experimental findings suggest that errors in the position of contact occur only when the stimulus is sufficiently complex and composed of multiple goals to imitate. An imitation goal can be either the relative spatial position between two parts of the body or the orientation of a body segment. We propose that the specific deficit underlying imitation in apraxia is the *incorrect coordination between multiple imitation goals*. Specifically, the errors seem to arise from the partial satisfaction and/or incorrect synchronization of the imitation goals. For example, patients frequently decomposed the imitation task into two phases: they would first imitate the hand's position of contact and then the hand's posture, or vice-versa. There was thus no clear hierarchical organization between the goals as suggested by Bekkering et al. (2000); Gleissner, Meltzoff, and Bekkering (2000).

We advocate that the absence of hierarchical organization is due to the random process that allocates visual attention. Therefore, which goal is performed first varies from case to case, and is a combination of the goal's visual salience and gaze exploration process. Importantly, it seems that when the goals were satisfied sequentially, the first goal (e.g. position of contact) was ignored while satisfying the second (e.g. posture of the hand); see Fig. 7.5. Even though we occasionally observed sequential satisfaction of the goals, most of the time the goals were satisfied simultaneously. Moreover, the imitation could be a mix of both a sequential and simultaneous approach, such that the goals were not necessarily combined together from the start of the imitation process, but following a variable delay. Therefore, we believe that this goal-combination can be explained by gaze exploration and attentional and short-term memory processes, such that the goals are combined whenever they are identified and remembered.

The hypothesis that the left-hemisphere contributes to "goal-coordination" is very powerful. In the case of Goldenberg's patient, this hypothesis explains why the patient made errors only in some of the trials. Although we advocated before

[3]We tested these patients at the CHUV.

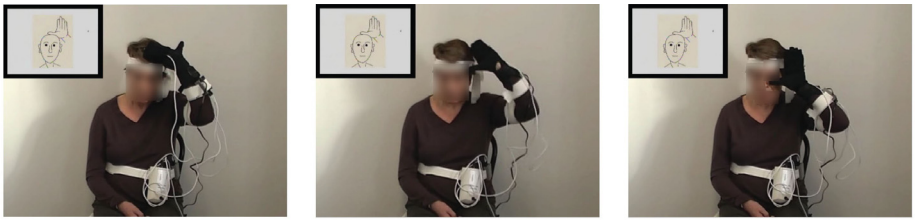

Figure 7.5: Sequential satisfaction of the two imitation goals. Ignoring the first goal (i.e. hand position of contact) while satisfying the second goal (i.e. hand posture) leads to incorrect imitation.

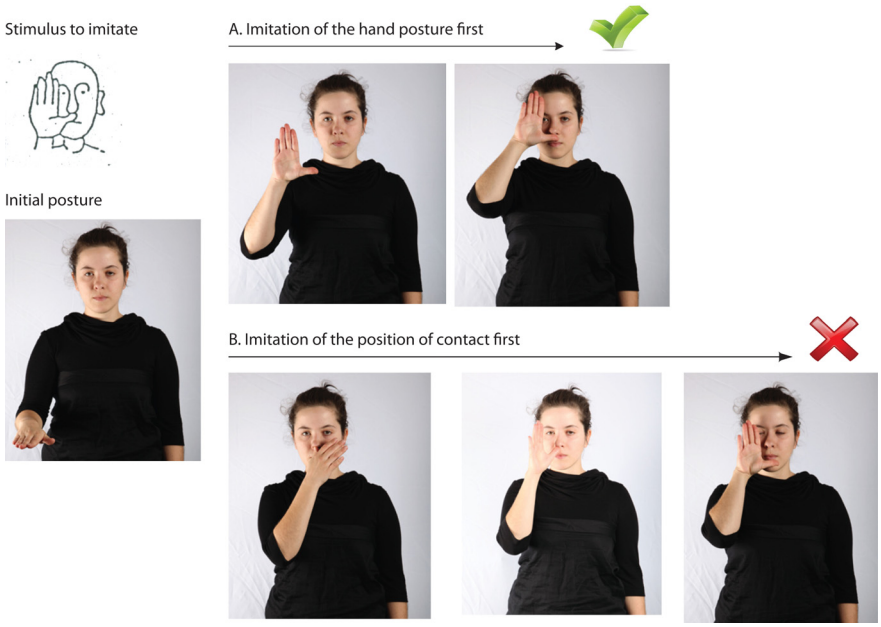

Figure 7.6: The order of execution of the imitation goals influences the patient's performance. Imitation is correct if the patient imitates the posture of the hand first (upper row), but impaired if the patient imitates the position of contact first - since updating the hand posture will displace the contact position (lower row).

that the apraxic errors were consistent across trials, whether the patient made an error or imitated correctly was not consistent. This puzzling phenomenon could be explained by the *order of execution* between imitation goals that are decoupled and solved sequentially. This is the case when the goals are satisfied one after the other and the first constraint is *ignored* while satisfying the second. For example, imitation will be correct if the subject first imitates the posture of the hand and then the hand's position of contact with the face (see Fig 7.6). However, if the subject first touches the correct position on the face, but then updates the posture of the hand to match the one shown in the visual stimulus, the position of contact will be displaced, thus leading to an incorrect imitation.

Furthermore the hypothesis of "goal-coordination" also explains why only some of the imitation stimuli led to incorrect imitation, and not others. Note that when the patient imitates only a position of contact between the hand and face, he or she uses a natural and comfortable posture of the hand, that we

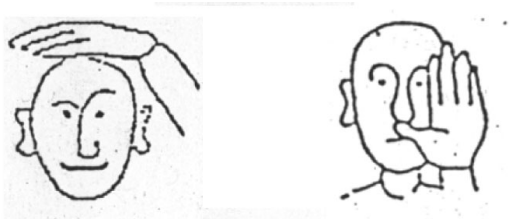

Figure 7.7: Examples of a stimulus with non-consistent (left) and consistent (right) goals. The stimulus with non-consistent goals on the left never led to apraxic errors, since the posture used to touch the top of the head is natural. This type of natural stimuli might be too easy to imitate and should be avoided in the clinical assessment of apraxia.

will refer to as the "default hand posture". We suggest that a visual stimulus might be incorrectly imitated only when this default posture is different from the hand posture specified in the stimulus. On the contrary, a "goal-consistent" stimulus was always correctly imitated, by all of the patients; see Fig. 7.7 (left). Indeed, a subject asked to touch the top of the head would naturally adopt a horizontal orientation of the hand, which corresponds to the hand posture in the stimulus. This observation that natural gestures are not impaired has far reaching consequences. By natural we mean a gesture that may be frequently executed in everyday life, that does not strain the muscles such as when reaching extreme joint angles and that touches the body with preferred parts of the hand (e.g. parts with a good tactile resolution). It is thus crucial to carefully choose the stimuli used for the clinical assessment of apraxia: if only stimuli with consistent goals are used, the patient might not have been diagnosed as imitation-apraxic.

7.2.2 Limitations and Future Work

We here review the neurocomputational model of imitation presented in Chapter 3 in light of the neuropsychological evidence collected and analyzed in Chapters 4 and 5. We also suggest a feasible way to test and further exploit our hypothesis of incorrect coordination between multiple goals.

Revision of the neurocomputational model of imitation. The neurocomputational model of imitation of meaningless gestures in Chapter 3 was based on several hypotheses, two of which were not verified by the experimental data. Next we revise these two hypotheses.

We first hypothesized that the callosal lesion in Goldenberg's patient corresponds to a probabilistic impairment of the information transferred between the two brain hemispheres. In Chapter 4, we reanalyzed the experimental data and observed systematic apraxic errors that do not confirm our hypothesis of random or noisy errors. Nevertheless, given a frame of reference centered on the hand, our neurocomputational model correctly predicts that the target on

the face would be *under-reached* (e.g. the hand is brought in the vicinity of the target, but stops before the target is reached), as the lesion slows down the integration of the desired position of the hand. The errors observed in the right visual field-right hand condition are indeed under-reaching errors (see Fig. 4.11), but present several inconsistencies.

We also assumed that only the left hemisphere contains a *unified body schema* that mediates spatial information relative to the body surface (Pick, 1922; Holmes & Spence, 2004). In our model, the body schema was grounded within the tactile modality. Neuropsychological data presented in Chapter 5 suggests that both hemispheres, and not only the left one, have access to spatial information relative to the body. Indeed, three apraxic patients that made contact position errors when imitating Goldenberg's stimuli, had no difficulty at reproducing the contact position between the face and hand when no posture of the hand was specified. Instead of a unified body schema, we suggest that the left hemisphere may process the relative distance between two parts of the body (Corradi-Dell'Acqua et al., 2008), or coordinate movements that simultaneously satisfy different imitation goals (this computation would be equivalent to solving a multiple constraints problem where each imitation goal is treated an additional constraint).

Validate the hypothesis of incorrect coordination between multiple imitation goals. This thesis proposes a simple, yet powerful hypothesis for interpreting the errors observed in apraxic imitation. We suggest that the underlying deficit is incorrect coordination between the reproductions of multiple goals. However, the predictions of this hypothesis still need to be validated against a larger set of lesion data.

One important prediction that our hypothesis makes is that the patient will not make errors when asked to imitate a single goal. Thus the patient will imitate correctly when asked to (1) touch the face at a visually specified position or (2) reproduce a hand posture not represented with respect to the body. We have verified the hypothesis' prediction in the first condition, but the second condition still needs to be tested (see Chapter 5). In addition, our results contradict the **"body part coding"** hypothesis proposed by Goldenberg et al. (2001), which predicts that the patient would make errors when there is a need to imitate a relationship between two parts of the body (in the first, but not in the second condition).

Devise efficient rehabilitation strategies. The validation of our hypothesis in full might reveal the specific imitation deficit in apraxia, which could then be used to devise efficient and adapted rehabilitation strategies. These rehabilitation strategies could directly target the impaired mechanism or affected movements. Another way would be to provide the patient with a strategy that bypasses the apraxic problem. For example, the patient's imitation ability would improve if he or she is instructed to first imitate the posture of the hand and

then the position of contact between face and hand. Of course, the usefulness of these new rehabilitation strategies would subsequently need to be assessed and contrasted with that of standard rehabilitation methods.

7.3 Computational Motor Control of Reaching

This section summarizes how this thesis answered to the two questions related to *human motor control*, as a part of the execution stage of imitation. Furthermore, we discuss the model's limitations and provide several promising research directions.

7.3.1 Main contributions of the thesis

Can we devise a biologically-plausible controller that accounts for the kinematics of natural three-dimensional reaching movements? While attempting to model the kinematic motion data collected from apraxic patients, we encountered a difficulty. Current models of human reaching primarily focus on straight, point-to-point movements that lie within a plane, but goal-directed movements are not straight in general. In particular the self-oriented movements involved in the imitation of Goldenberg's stimuli follow highly curved trajectories (see Fig 7.8). Some models have addressed the spatiotemporal features of realistic three-dimensional movements, but none of these models are satisfactory, for the following reasons. Some of these models preplan the trajectory of the hand (Biess et al., 2007), such that the movement is not robust to external perturbations (e.g. displacement of the target or appearance of an obstacle). Other models only address the spatial, but not temporal, aspects of motor control (Torres & Zipser, 2002). Models that use the framework of optimal stochastic feedback control require the definition of a cost function to optimize in task space (Todorov & Jordan, 2002; Guigon et al., 2007), which influences model precision.

In Chapter 6, we present a 2^{nd}-order time-independent nonlinear dynamical model for the control of reaching that overcomes all of these difficulties. Given the initial and desired positions of an end-effector, the F2REACH model generates a motion trajectory by creating a single stable attractor at the desired target position. Environment- and embodiment-related task constraints are expressed as a virtual force field that acts on the dynamical system and shapes the movement trajectory. This biologically-plausible model was validated against a large set of unconstrained reaching movements directed towards the head and starting from two initial positions. The model reproduces both the spatial and temporal kinematic features of the movements with high accuracy, as the mean

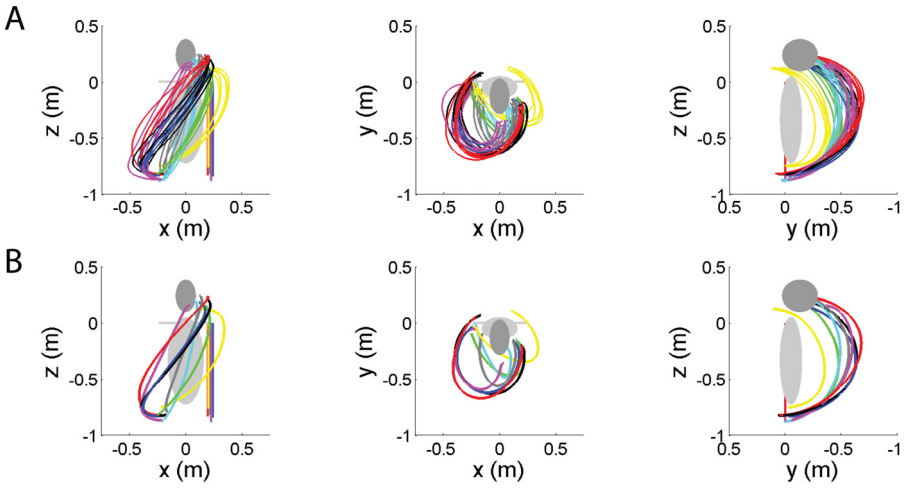

Figure 7.8: Self-oriented movements such as those involved in the imitation of Goldenberg's stimuli follow *highly curved* trajectories. **A** Ten subjects (color-coded) reached to their left ear with the right hand in five trials. **B** The mean movements are shown.

deviation observed was less than 2cm for movements with an average path length greater than 1m. Furthermore, under realistic conditions, the model is globally and asymptotically stable.

Our model suggests that both curved and straight-line reaching movements may be generated by a unique neural controller, where embodiment (e.g. the geometric properties of the human body) should be considered as the main cause for movement curvature. Specifically, curved trajectories may result from an active strategy that avoids the limits of the joints and the subject's body. We believe that the existing models for the control of reaching have for the most part underestimated the effects of bio-mechanical properties of the limb on the kinematics of human movements.

Relation between the thesis models. The neurocomputational model of imitation of meaningless gestures developed in Chapter 3 and nonlinear dynamical model of reaching developed in Chapter 6 are activated sequentially in the imitation process. Specifically, the neurocomputational model of imitation takes as input a visual stimulus to imitate and produces as output a desired target position of the hand that is consistent with the visual stimulus). The dynamical system model for reaching takes as an input the output of this neurocomputational model of imitation, i.e. a desired position of the hand, and generates the hand's kinematic trajectory. In other words, the first model transforms a visual stimulus into a desired outcome of a motor action, whereas the second model executes the corresponding motor action.

At what level does the Central Nervous System (CNS) control hand movements? The low-dimensional parameter space of our reaching model suggests that the CNS may control movement at the extrinsic level, i.e.

by directly controlling the trajectory of the hand. Our model encapsulates the regularity of the hand trajectory of unconstrained natural movements with only one parameter that controls speed and six parameters that approximate the force field. One major advantage of having a high-level neural movement controller is that interaction with the environment is greatly facilitated. For example, the position of an object that needs to be avoided has a unique representation in the extrinsic space, but corresponds to an infinite number of different configurations of the joint angles or of muscle activations (i.e. intrinsic levels). Furthermore, our model couples the spatial and temporal levels of control, such that these levels are intrinsically linked.

Our model is thus consistent with Bernstein's theory of a four-leveled hierarchy in the control of movements: from complex actions with abstract goals dealing with three-dimensional space, to muscle synergies, posture and finally muscle tone (Bernstein, 1947)[4]. Going from a higher to a lower level of motor control could be performed through muscle synergies (d'Avella et al., 2003) or "an internal inverse model", which would implement the transformation from desired consequences to actions (Wolpert & Ghahramani, 2000). Even though there is evidence for each type of control, it is still unclear how the different levels of control are coordinated in the simple task of reaching. As each of these control levels presents complementary advantages, several hybrid models of motor control have been proposed that find a compromise between planning a straight line in task space and planning a straight line in joint space (Cruse & Brüwer, 1987; Okadome & Honda, 1999; Hersch & Billard, 2008). Unfortunately these models mix the intrinsic and extrinsic controls arbitrarily, without providing any insight on how the CNS might resolve this problem. A different approach, proposed by Just et al. (2009), couples two neural controllers associated to the end-effector and elbow joint, suggesting that each body part may be controlled individually.

7.3.2 Limitations and Future Work

We see two main limitations that reduce somewhat the explanatory power and applicability of the F2REACH model.

Dissociation between the extrinsic and intrinsic levels of motor control. The first limitation of our model is that it controls the trajectory of the end-effector in a three-dimensional Cartesian space, but does not specify the trajectories of other joints of the limb such as the elbow. To be more specific, the model does not address the inverse kinematics problem, i.e. the problem of how a desired end-effector trajectory (extrinsic level) is translated into joint angles or muscles activations (intrinsic levels), while taking into account the

[4]Translated in part in Bernstein (1996).

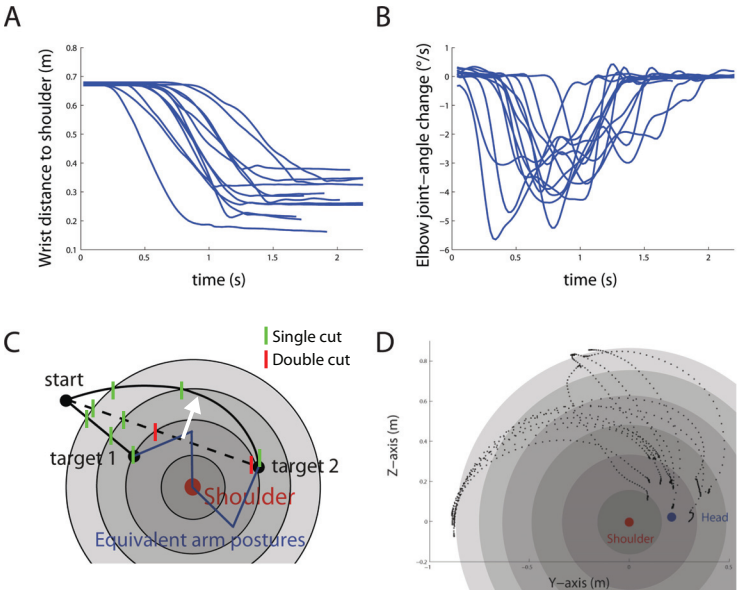

Figure 7.9: Movement curvature invariance reflects bio-mechanical properties. **A** Wrist-to-shoulder distance decreases monotonically in the reaching part of all the movements considered, shown for one subject. **B** Change in the elbow joint-angle is negative in the reaching part of an unconstrained movement (occasional exceptions are observed at the very start or end of the movement). **C** Wrist equidistant spheres centered at the shoulder (frontal plane, axial view), with equal elbow joint angles. A curved trajectory is preferred over a straight trajectory if the path cuts an equidistant sphere twice. **D** Sagittal view of the set of unconstrained hand movements addressed in this study (given for one subject). The movement trajectories are highly curved, as suggested by the hypothesis illustrated under C.

dynamics of the limb.

Although our model controls movement at the extrinsic end-effector trajectory level, it can be influenced by intrinsic control parameters through the so-called virtual embodiment force field. For example, we observed that in the self-oriented movements we have recorded, the motion of the elbow joint angle is *monotonically* decreased (with some exceptions at the very start or in the vicinity of the target, shown in Fig. 7.9A and B). As the elbow joint is a hinge joint, inversion of the direction of movement in the middle of the reaching phase may be energetically costly and inefficient (e.g. because of inertial resistance), when compared to the spheroidal shoulder joint. This means that in addition to maximizing the smoothness of the hand trajectory (or minimizing the jerk), the CNS may also maximize the smoothness of the elbow-joint trajectory.

This experimental observation is very interesting as it allows the prediction of where in the workspace one would do straight-line motion trajectories (that still fulfill the monotonicity of the elbow joint motion). Note that the elbow angle determines univocally the distance between the wrist and shoulder, such that we can define spheres around the shoulder joint that correspond to equidistant surfaces of wrist positions, i.e. surfaces of equal elbow joint-angles. We propose that the kinematic trajectory of an unconstrained movement will be straight in the extrinsic space only when this straight movement cuts a partic-

ular equidistant surface only once (see Fig. 7.9C). If an equidistant surface is cut twice, the elbow angle is first increased and then decreased (or vice-versa), such that the movement trajectory will be curved in order to prefer a monotonic function of the elbow joint-angle (see Fig. 7.9D).

The extrinsic and intrinsic levels of a movement are thus highly interdependent. A promising research direction in order to understand how these two levels of control interact is to investigate the control of movements with a different number of fixed degrees of freedom. For instance, one could compare a baseline movement to movements with a fixed orientation of the hand, or equivalently to movements that reach with a different part of the hand. Differences in the control strategies could be extrapolated from the differences in the movements' kinematics and variability.

Understand the virtual force field. A second limitation of our model is the parametrization of the embodiment force field that shapes the movement trajectory: two three-dimensional forces that are estimated from human motion data. Ideally, we would like to infer these forces from the shape and geometry of the subject's body.

A promising approach for the investigation of the embodiment force field is to observe how it is modulated by different external perturbation force fields. This method has already proven extremely helpful for disentangling the neural processes underlying motor learning (Shadmehr and Mussa-Ivaldi, 1994; Wagner and Smith, 2008). The observation of how a learned perturbation force field is represented by our model can help uncover the different constraints that contribute to the movement curvature: a) avoidance of the joint limits, b) smoothness of the joint trajectories, c) geometry of the body, d) objects in the environment, and e) dynamic properties of the limb such as inertia or viscosity. An extension that characterizes this virtual force field would increase the applicability and explanatory power of the model .

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RESEARCH INTERESTS

Computational neuroscience, neural information processing, machine learning and statistical learning theories, human motor control, nonlinear dynamical systems, neuropsychology, neurophysiology and neurorehabilitation.

EDUCATION

Ph.D. Candidate in Computational Neuroscience 2005 – 2009
Ecole Polytechnique Fédérale de Lausanne (EPFL), defended on December 1st 2009
Advisor: Prof. Aude Billard
Thesis: Modeling the Neural Correlates of Imitation from a Neuropsychological Perspective.

This Ph.D. thesis proposes a neurocomputational model of visuo-motor imitation and a dynamical model for the control of reaching movements. The models are grounded in the fields of machine learning and nonlinear dynamical systems, and take into account evidence from multiple neuroscience fields such as neuroimaging and neurophysiology. To validate these models, I conduct neuropsychological experiments of imitation in patients with apraxia, i.e. a high-level deficit of voluntary movement following a brain lesion.

Doctoral courses: Machine Learning (final grade within the top three), Cellular and Molecular Neurobiology, Functional Systems in the Mammalian Nervous System.

M.Sc. in Computer Science (grade: 5.7 / 6) 2002 – 2004
Ecole Polytechnique Fédérale de Lausanne (EPFL)
Advisor: Prof. Auke Jan Ijspeert
Thesis: Neural Visuomotor Controller for a Simulated Salamander Robot.

This master's thesis investigates the neural mechanisms underlying visually-guided behavior in salamanders. Simple biologically-motivated models of the salamander brain coupled to spinal locomotor circuits can account for typical orientation, gaze stabilization, approaching and snapping behaviors specific to feeding, and reproduce the walking pattern of a monocularized salamander.

B.Sc. in Computer Science 1999 – 2002
Ecole Polytechnique Fédérale de Lausanne (EPFL)

SUMMER SCHOOLS

Machine Learning Summer School 1-15 Sept 2008
Isle de Re, France

Complex Systems Summer School (< 30% of acceptance) 3-19 June 2007
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Clinical Collaborations

SISSA, Trieste, Italy from February 2010
Laboratory of Cognitive Neuroscience of Actions, with Prof. Raffaella Rumiati.
Intend to conduct a large-scale neuropsychological study, in order to validate our hypothesis of incorrect coordination between multiple imitation goals as the main imitation deficit underlying apraxia.

Klinikum Bogenhausen, Munich, Germany 2007 – present
Entwicklungsgruppe Klinische Neuropsychologie, Professors Georg Goldenberg and Joachim Hermsdoerfer.
Re-analyzed their experimental study of callosal apraxia and discovered a pattern of goal dissociation.

Vaud University Hospital Centre (CHUV), Lausanne, Switzerland 2007 – present
Neuropsychology and Neurorehabilitation Dept., under the responsibility of Prof. Stéphanie Clarke.
Conducted several neuropsychological experiments of the imitation of meaningless gestures in apraxic patients.

Geneva University Hospital (HUG), Geneva, Switzerland 2006 – 2007
Department of Neurology, supervised by Prof. Olaf Blanke.
Developed integrated software to record kinematic data during neuropsychological experiments of imitation.

Professional Experience

Web Software Engineer, WEBSPARK, Geneva 2005, 4 months
Developed methods for an automatic and easy creation of complex dynamical web pages (PHP, SQL, Apache).
Participated in the development of a database for contextual web advertising, now Ads-click.

Teaching assistant, Logic Systems Laboratory EPFL, Lausanne 2002 – 2004
Supervised the exercise sessions and evaluated the work of students for the course Computer Architecture.

Personnel, Service for Research in Education, Dept. of Public Education, Geneva Summer 2002 – 2003
Coding and evaluation of the international OECD Programme for International Student Assessment (PISA) in Switzerland. On my initiative I devised an archive for most outstanding or surprising results.

Manager of the Stamping Atelier, ROLEX, Geneva Summer 1999 – 2000
Organized, planned and supervised the production flow and work of 20 employees. I implemented techniques for an automatic predictive production management and was responsible for the precious metals accounts.

Skills

- Theoretical neuroscience, neural information processing and information theory
- Knowledge in multiple neuroscience fields: neuroimaging, neurophysiology, experience in neuropsychology
- Theoretical knowledge and deep interest in machine learning and statistical learning theory
- Nonlinear dynamical systems theory
- Programming (C/ C++, Matlab, PHP, OpenGL, SQL, Java, VHDL, Lisp, Ada, Smalltalk, HTML, Prolog)
- Computer graphics and web design, databases and numerical systems

Languages

- French: mother tongue
- English: fluent, **Cambridge Certificate of Proficiency in English (CPE)**, grade B, 2003
- German: average
- Italian: average

AWARDS AND HONORS

Swiss National Science Foundation (SNSF) fellowship for prospective researchers, 18 months ~ $85 000
To join as a postdoctoral fellow the Gatsby Computational Neuroscience Unit at University College London and Neuromotor Control Lab at Harvard University, 2009.

Best Student Paper Award
IEEE International Joint Conference on Neural Networks (IJCNN), Hong Kong, 2008.

SNSF doctoral fellowship, 12 months ~ $45 000
To enroll as a doctoral student in the EPFL Manufacturing Systems and Robotics Doctoral Program, 2005.

Annaheim Foundation Award
Rewards a high-level M.Sc. thesis devoted to the connection between computer science and life sciences, 2004.

Travel Grants

- €1250, *Pascal 2* to attend WiML, 2008
- $800, *IEEE Computational Intelligence Society (IEEE CIS)* to attend IJCNN, 2008
- £350, *Microsoft Research for students (MSR)* to attend WiML, 2006
- €400, *European Neural Network Society (ENNS)* to attend ICANN, 2006
- €610, *European Science Foundation (ESF)* to attend the ESF Research Conference on Brain Development and Cognition in Human Infants, 2005.

MEDIA COVERAGE

Interview in the *IEEE Computational Intelligence Magazine***.** February 2009 issue, pages 7-8.

INVITED AND CONFERENCE TALKS

Modelling the Neural Correlates Underlying Human Imitation September 2009
Gatsby Computational Neuroscience Unit, University College London (UCL).

A Neural Model of Demyelination of the Mouse Spinal Cord June 2008
International Joint Conference on Neural Networks (IJCNN 2008), Hong Kong.

A Neurocomputational Model of Imitative Apraxia March 2007
Entwicklungsgruppe Klinische Neuropsychologie, Klinikum Bogenhausen, Munich.

Intelligent Robots: Perspectives (in french) November 2006
Centre Dürrenmatt, Neuchâtel, Switzerland.

Neurocomputational Modelling of Incorrect Imitation following Brain Lesion October 2006
Brain Simulation Laboratory, University of Southern California (USC), Los Angeles.

A Neurocomputational Model of an Imitation Deficit following Brain Lesion September 2006
International Conference on Artificial Neural Networks (ICANN 2006), Athens.

A Reconfigurable Hardware Membrane System June 2003
International Workshop on Membrane Computing (WMC 2003), Tarragona, Spain.

PUBLICATIONS

JOURNALS

Petreska B and Billard A (2009) Movement curvature invariance reflects bio-mechanical properties of the limbs (in preparation).

Petreska B, Billard A, Hermsdörfer J and Goldenberg G (2009) Revisiting callosal apraxia: the right hemisphere can imitate the orientation but not the position of the hand. **Neuropsychologia** (accepted).

Petreska B and Billard A (2009) Movement curvature planning through force field internal models. **Biological Cybernetics**, 100:331-350.

Just A, Petreska B, Billard A, Craighero L, D'Ausilio A, Oleynik A and Fadiga L (2009) Point-to-point gestures: modeling wrist and elbow trajectories. **Human Movement Science** (submitted).

Petreska B, Adriani M, Blanke O and Billard A (2007) Apraxia: a review. In C. von Hofsten (Ed.) From action to cognition, **Progress in Brain Research**, Elsevier, Amsterdam, 164:61-83.

Brooks A, van der Zwan R, Billard A, Petreska B, Clarke S and Blanke O (2007) Auditory motion affects visual biological motion processing. **Neuropsychologia**, 45(3):523-530.

REFEREED CONFERENCES

Petreska B and Yossi Y (2008) A neural model of demyelination of the mouse spinal cord. In Proceedings of the IEEE International Joint Conference on Neural Networks (IJCNN 08), pp. 2538-44. **Best student paper.**

Petreska B and Billard A (2006) A neurocomputational model of an imitation deficit following a brain lesion. In Proceedings of the International Conference on Artificial Neural Networks (ICANN 06). Lecture Notes in Computer Science (LNCS), 4131:770-779.

Petreska B and Teuscher C (2004) A reconfigurable hardware membrane system. In C Martin-Vide, G Mauri, G Paun, G Rozenberg and A Salomaa, (eds). Membrane Computing, LNCS, 2933:269-285.

ABSTRACTS

Petreska B, Billard A, Hermsdörfer J and Goldenberg G (2009) What apraxia tells us about the brain hemispheres' contributions to human imitation. Society for Neuroscience Meeting (SfN 2009), Chicago.

Petreska B and Billard A (2008) Neurocomputational modeling of neural deficits. Computational Neuroscience Meeting (CNS 08), Portland, Oregon, USA. BMC Neuroscience 2008, 9(Suppl 1):P76.

Petreska B and Billard A (2008) Neurocomputational modeling of imitation through apraxia errors. Workshop for Women in Machine Learning (WiML 08), collocated with NIPS, Vancouver, Canada.

Petreska B and Billard A (2008) Modeling unconstrained self-oriented movements. Neural Control of Movement Society Meeting (NCM 08), Naples, Florida, USA.

Petreska B and Billard A (2006) A neurocomputational model of impaired imitation. Workshop for Women in Machine Learning (WiML 06), San Diego, California, USA.

Petreska B, Adriani M, Blanke O and Billard A (2006) Neural modeling of imitation deficits. LATSIS Symposium. In Dynamical principles for neuroscience and intelligent biomimetic devices; A Ijspeert, J Buchli, A Selverston, M Rabinovich, M Hasler, W Gerstner, H Markram, D Floreano (eds), Lausanne.

Petreska B, Adriani M, Blanke O and Billard A (2005) Modeling of imitation deficits in apraxic patients. ESF Conference on Brain Development and Cognition in Human Infants, Maratea, Italy.

Professional Activities

Journal Reviewer: Neurocomputing, International Journal of Unconventional Computation.
Society Member: Society for Neuroscience, IEEE Computational Intelligence, Neural Control of Movement.

Personal Interests

Passionate about the Arts: classical literature, author cinema, contemporary art. I enjoy painting, photography, inventive cooking & wine degustation, Pilates. Subscribed to Wired, XXI and The Economist.