Dynamic Bayesian modeling of the cerebral activity

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Abstract

Conventional methods used for the interpretation of activation data provided by functional neuroimaging techniques provide useful insights on what the networks of cerebral structures are, and when and how much they activate. However, they do not explain how the activation of these large-scale networks derives from the cerebral information processing mechanisms involved in cognitive functions. At this global level of representation, the human brain can be considered as a dynamic biological system. Dynamic Bayesian networks seem currently the most promising modeling paradigm. Our modeling approach is based on the anatomical connectivity of cerebral regions, the information processing within cerebral areas and the causal influences that connected regions exert on each other. The capabilities of the formalism's current version are illustrated by the modeling of a phonemic categorization process, explaining the different cerebral activations in normal and dyslexic subjects. The simulation data are compared to experimental results [Ruff *et al.*, 2001].

1 Introduction

In Neurology and Neuropsychology, the diagnosis of the neurological causes of cognitive disorders, as well as the understanding and the prediction of the clinical outcomes of focal or degenerative cerebral lesions, necessitate knowing the link between brain and mind, that is what the cerebral substratum of a cognitive or a sensorimotor function is and how the substratum's activity can be interpreted in cognitive terms, i.e. in terms of information processing.

Studies in humans and animals [Bressler, 1995; Démonet *et al.*, 1994] have shown that sensorimotor or cognitive functions are the offspring of the activity of oriented large-scale networks of anatomically connected cerebral regions (Figure 1). In humans, functional neuroimaging techniques provide activation data, which are indirect measures of the brain's electrical or metabolic activity during a task performance. Statistical analyses of the activation data allow determining *where* [Fox and Raichle, 1985], i.e. in which areas, and/or *when* [Giard *et al.*, 1995] during the task performance, the activation reaches local extrema. Through the study of covariation between local activations, they give a sketch of *what* the network of cerebral areas involved in the cognitive function is [Herbster *et al.*, 1996]. A known oriented anatomical link between 2 areas allows determining *why* the activation of one area can affect the other one [Büchel and Friston, 1997]. Above methods allow identifying the substratum of a cognitive function and the activation level and dynamics of the substratum during the function performance. They do not give any clue of *how* the cognitive processes participating in the function are implemented by the substratum and *how* the activation derives from the processing. That is, they do not allow *interpreting* neuroimaging data as the result of information processing at the integrated level of large-scale networks.

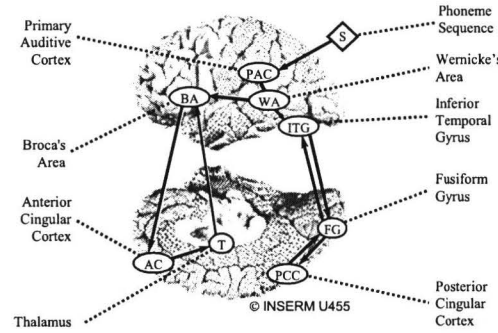

Figure 1: Large-scale network involved in phoneme monitoring, according to results from [Démonet, *et al.*, 1994]

Interpretative models, linking a networked structure activity to the realization of a function, are at the core of Computational Neurosciences. Most existing works in the domain are based on formal neural networks, with varying levels of biological plausibility, from physiology [Wang and Buzsaki, 1996], hardly interpretable in terms of information processing, to more or less biologically

plausible models of how basic cognitive functions emerge from neuronal activation [Grossberg *et al.*, 2002], and to purely functional models [Cohen et al., 1990], not concerned with cerebral plausibility. Although these models answer the *how*, they do not meet two major requirements for an interpretative approach of functional neuroimaging data. The models are not explicit enough to be directly used for clinical purpose, and they cannot evolve quickly and easily with new findings in neuroscience, such as the integration of more detailed knowledge on the substratum, which often necessitates a complete rebuilding of the formal network.

The causal connectivity approach [Pastor *et al.*, 2000] aims at answering the how *and* satisfying the constraints. However, the underlying formalism, causal qualitative networks based on interval calculus, limits severely the biological plausibility of the models, since it cannot represent major cerebral features, such as learning or the non-linearity and the uncertainty of cerebral processes. Dynamic Bayesian networks only meet the three major constraints: temporal evolution, uncertainty and nonlinearity [Labatut and Pastor, 2001]. The utility of graphical probabilistic formalisms for cognitive modeling has also been demonstrated in the representation of visuomotor mechanisms with Bayesian networks [Ghahramani and Wolpert, 1997].

Hereafter, we describe how the interpretation of functional images for a clinical purpose can be tackled. Section 2 presents our viewpoint on large-scale cerebral networks. After a short introduction to dynamic Bayesian networks, section 3 describes the characteristics of our formalism. Section 4 illustrates the formalism's capabilities by an example. We conclude with some perspectives.

2 Representation of Large-Scale Cerebral Networks

2.1 Structural and Functional Nodes

The function implemented by a large-scale network depends on three properties: the network's structure [Goldman-Rakic, 1988], the functional role of each node (E.g. Wernicke's area (Figure 1), which is supposed to realize the early stages of phoneme processing), and the properties of the links (length, role: inhibitory or excitatory, ...). In each network, regions, which are the *structural nodes*, are information processors and connecting oriented fibers are information transmitters [Leiner and Leiner, 1997].

All neurons in a region do not have the same structure or the same role. Similar neurons constitute generally local populations that realize a specific function. For example, the inhibitory role of GABAergic neurons on other neuronal populations may explain the fact that every visual stimulus is not perceived in high frequency stimulation [Pastor, *et al.*, 2000]. Therefore, each region is itself a network of smaller neuronal populations (*functional nodes*), connected through neuronal fibers. These nodes are information processors that implement *functional primitives*, which may all be different.

A large-scale network has therefore neurophysiologically constrained, oriented edges and possibly differentiated nodes. The explicit representation of the nodes' function allows the direct expression of hypotheses on cerebral processing, and their easy modification in order to follow the evolution of knowledge in neurosciences. This cannot be dealt with by formal neural networks' implicit modeling that requires modifying the whole network architecture to implement functional changes. Hereafter, a structural or a functional structure will be indifferently named a *cerebral zone*.

2.2 Information Representation and Processing

The cerebral information processed by a neuronal population can be seen as the abstraction of the number and the pattern of the neurons firing for this information. It can be represented both by an energy level and by a category. Energy is indirectly represented by the imprecise activation data provided by neuroimaging techniques. The category representation is in agreement with the "topical" organization of the brain, which reflects category maps of the input stimuli, and can persist from primary cortices to nonprimary cortices and subcortical structures [Alexander *et al.*, 1992], through transmission fibers [Leiner and Leiner, 1997]. The energy and the category of a stimulus can also be easily extracted from its psychophysical properties.

Modeling cerebral processes necessitates an explicit and discrete representation of time, both for taking into account the dynamics of cerebral mechanisms (transmission delays, response times...), and for complying with sampled functional neuroimaging data.

According to a definition of causality inspired by Hume [Hume, 1740] and consistent with Pearl's probabilistic causality [Pearl, 2001], information processing in a large-scale network can be considered as mediated through *causal* mechanisms. Causality is defined by three properties: spatial and temporal contiguity, temporal consistency, and statistical regularity [Labatut and Pastor, 2001]. In other words, two entities A and B are causally linked if they are contiguous relatively to the system they belong to, if the beginning of A precedes temporally the beginning of B, and if most of the times, A provokes B. In the brain, oriented anatomical links provide spatial and temporal contiguity between cerebral nodes, cerebral events are temporally consistent (a firing zone provokes the activation of downstream zones), and there is a statistical regularity in the response of a specific neuronal population to a given stimulus.

3 Description of the Formalism

3.1 Dynamic Bayesian Networks

In summary, the brain can be viewed as a network whose nodes are differentiated dynamic and adaptive informa-

tion processors and oriented edges convey causality. Moreover, cerebral mechanisms, which are the abstraction, at the level of a neuronal population, of the chemical and electrical mechanisms at the cell levels, are often nonlinear. Causal dynamic Bayesian networks are the paradigm that meets best the constraints derived from these properties [Labatut and Pastor, 2001].

A causal Bayesian network consists of a directed acyclic graph where nodes represent random variables and edges represent causal relationships between the variables [Pearl, 1988]. A conditional probability is associated with each relationship between a node and its parents. If the node is a root, the probability distribution is a prior. When some nodes' values are observed, posterior probabilities for the hidden nodes can be computed thanks to inference algorithms such as the junction tree algorithm [Jensen, 1996].

In a dynamic Bayesian network (DBN), the evolution of random variables through time is considered. Time is seen as a series of intervals called time slices [Dean and Kanazawa, 1988]. For each slice, a submodel represents the state of the modeled system. DBNs are used to model Markovian processes, i.e. processes where a temporally limited knowledge of the past is sufficient to predict the future. The choice of the inference algorithm, generally an extension of the junction tree algorithm [Murphy, 1999], depends on the DBN's structure, the nature of its variables (discrete or continuous), and relationships (linear or nonlinear).

Activation data and/or the subject's responses to the stimuli are the only observable variables we have. Therefore, they must be integrated in our models. One may reasonably consider that the hidden variables, describing the successive states of the cerebral network, constitute a Markov chain, and that observable variables depend only on them. Moreover, the variables are continuous and their relationships may be nonlinear. This is typically the description of a type of DBNs called fully nonlinear state space models. Specific and recent algorithms allowing dealing with nonlinearity exist for this type of structures. Their general principle is to linearize the model in order to apply the classic Kalman filter. These algorithms differ on the used linearization method: first-order Taylor approximations for the extended Kalman filter [Julier and Uhlmann, 1997; Norgaard *et al.*, 2000] or polynomial approximations for the unscented Kalman filter [Julier and Uhlmann, 1997], the divided difference filter (DDF) [Norgaard, *et al.*, 2000], and others [Van Der Merwe and Wan, 2001]. The algorithms based on polynomial approximations seem to give more reliable results [Norgaard, *et al.*, 2000]. Their computational complexity is $O(L^3)$, where L is the state dimension [Van Der Merwe and Wan, 2001]. They offer equivalent qualities, but those of the DDF are more accurate according to its author [Norgaard, *et al.*, 2000].

3.2 Formal definition

Static and Dynamic Networks

A *static network* is the graphical representation of a large-scale network, whose nodes are cerebral zones and edges are the oriented axon bundles connecting zones. Due to anatomical loops, it is often cyclic. The DBN is the acyclic temporal expansion of the static network. Each node of the DBN is the *processing entity* related to a cerebral zone, i.e. the mathematical expression, at a given time slice, of information processing in the zone. Each edge is the *propagation entity*, whose orientation is its corresponding axon bundle's orientation. When deriving the *DBN* from the static network, values are given to the temporal parameters, according to known physiology results (e.g. the transmission speed in some neural fibers). That is, the length of the time slices is fixed, and a delay representing the average propagation time in the bundle's fibers is associated to the propagation entity.

Information Representation

Cerebral information is the *flowing entity* that is computed at each spatial (cerebral zone) and temporal (time slice) step, by a processing entity. It is a two-dimensioned data. The first part, the *magnitude*, stands for the cerebral energy needed to process the information in the zone. It is represented by a real random variable in the DBN. For the second part, the type, which represents the cerebral category the zone attributes to the information, the representation is based on the *symbol* and *categorical field* concepts.

A *symbol* represents a "pure" (i.e. not blurred with noise or another symbol) category of information. For example, when the information represents a linguistic stimulus, a symbol may refer to a non ambiguous phoneme. For cerebral information, the symbol represents, in each zone, the neuronal subpopulation being sensitive to (i.e. that fires for) the corresponding category. It may be, in the primary auditory cortex, the subpopulation sensitive to a specific frequency interval. A *categorical field* is a set of symbols describing stimuli of the same semantic class. The "color" categorical field contains all the color symbols, but it cannot contain phonemes.

A type concerns several symbols, due to the presence of noise or because of some compound information. Let $\mathbb{S}$ be the set of all existing symbols. We assume that a type T is defined for only one categorical field. Let S_T be the subset of $\mathbb{S}$, corresponding to this categorical field. The type T is an application from S_T to $[0,1]$, with the property $\sum_{s \in S_T} T(s) = 1$, i.e. it describes a symbol repartition for a specific categorical field. In a stimulus, this repartition corresponds to the relative importance of each symbol compounding the information carried by the stimulus. Inside the model, $T(s)$ stands for the proportion of s-sensitive neurons in the population that fired for the information whose type is T. Unlike the magnitude, the

type is not represented by a random variable. Indeed, it is not necessary to represent its uncertainty (and hence to make the computational complexity harder) since we cannot compare it to neuroimaging data.

At time t and node X, the information is represented by the type T_X^t and the magnitude M_X^t at the output of X.

Propagation and Processing

For a zone X, both the cerebral propagation mechanisms (i.e. the relationships towards the zone) and the processing (spatial and temporal integration of the inputs, and processing as such) are described by a pair of functions, the type f_{T_X} and the magnitude functions f_{M_X}. In the general case where n zones $Y_1,\ldots,Y_n$ are inputs to X, let $\partial_1,\ldots,\partial_n$ be the corresponding delays of these relationships. In the DBN, the general form of the magnitude functions is:

$$M_X^t = f_{M_X}\left(M_{Y_1}^{t-\partial_1},\ldots,M_{Y_n}^{t-\partial_n},M_X^{t-1},u_X\right) \quad (1)$$

where f_M can be a nonlinear function. The random variable $u_X \sim N(0,\sigma^2)$ models uncertainty in the cerebral processing.

The type function is any combination of the incoming types and of the previous type that respects our type definition. If all types are defined on the same categorical field S, the type function can be the linear combination:

$$T_X^t(s) = c_{Y_1}T_{Y_1}^{t-\partial_1}(s)+\ldots+c_{Y_n}T_{Y_n}^{t-\partial_n}(s)+c_X T_X^{t-1}(s),\ \forall s\in S \quad (2)$$

where S is the categorical field of T_X^t, $T_{Y_1}^{t-\partial_1},\ldots,T_{Y_1}^{t-\partial_n}$, and T_X^{t-1}; and with $\sum c_\bullet = 1$ in order to keep the property $\sum_{s\in S} T_X^t(s) = 1$.

The functions' definition, as well as the setting of the parameters' values (e.g. the value of a firing threshold), utilize mostly results in neuropsychology or in neurophysiology. The existence of *generic models*, that is, non instantiated, *reusable*, models of functional networks, is assumed. For example, primary cortices may implement the same mechanisms, although they are parameterized so that they can process different types of stimuli [Pastor, *et al.*, 2000].

4 Example

The model, presented hereafter, is based on an experimental study [Ruff, *et al.*, 2001] that focused on the differences between normal and dyslexic subjects during a passive phonemic categorization process.

Six patients and six controls were submitted to a passive hearing of stimuli that are mixes of the two phonetically close syllables /pa/ and /ta/. The *pivot* is noted *dev0* and the *deviants* are 4 different mixes of /pa/ and /ta/, noted *dev2M, dev1M, dev1P, dev2P* (Table 1). The measurements were made with fMRI. An experiment is constituted of 5 blocks, corresponding to the pivot and the deviants. Each block contains 6 sequences of 4 sounds, 3 pivots and the block's deviant, in a random order.

We focus on a single region, a part of the right temporal superior gyrus involved in the early processing of auditory stimuli and activated differently in controls and dyslexic subjects. Phylogeny is in favor of the existence of specialized phonemic processors in this area (Figure 2). Since their location is unknown, they cannot constitute separate structural nodes. They are supposed to have the same building functional nodes. According to our genericity hypothesis, the processors' structure and parameters are based on a previously released visual cortex model [Pastor, *et al.*, 2000]. The Input Gating Nodes ($IGN_\bullet$) express the phoneme processors' sensitivity to the stimulus. The Output Gating Nodes ($OGN_\bullet$) send information to the downstream areas. Intra and inter (lateral) inhibitions ($IN_\bullet$ and $LIN_\bullet$) are assumed between the /pa/ and /ta/ processors. $LIN_\bullet$ make the activation of an $IGN_\bullet$ cause an inhibition in the opposite $IGN_\bullet$. Each Firing Threshold Node ($FTN_\bullet$) is modulated by an $OGN_\bullet$ that can lower it. Since only one activation measure is provided by fMRI for the area, it is represented by the sole *AN* node in the static model. *Stim* stands for the stimulus.

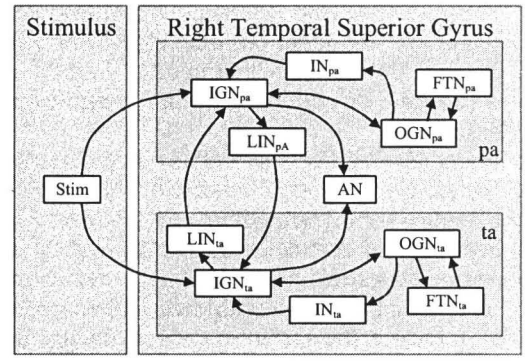

Figure 2: Static network used to model the cerebral phonemic categorization process.

Except for the parameterization of the $IGN_\bullet$ nodes, which reflects the specialization of each phonemic processor to the phoneme category (/pa/ or /ta/), the functions for both the /pa/ and /ta/ parts share exactly the same structure and parameters. Thus, only the *pa* part will be presented. In the following equations, the ith parameter of the function of a node X is noted $a_X^{(i)}$.

The refractory period of the processor's neurons is modeled in IGN_{pa} by a sigmoid function φ_{IGN} that makes the node sensitive to the incoming stimulus only if the magnitude of the output is already close to zero:

$$\begin{aligned} M_{IGN_{pa}}^t = {} & a_{IGN}^{(1)} g_{IGN_{pa}}\left(T_{Stim}^{t-2}\right)\left(1-\varphi_{IGN}\left(M_{OGN_{pa}}^{t-1}\right)\right)M_{Stim_{pa}}^{t-2} \\ & + a_{IGN}^{(2)} M_{IGN_{pa}}^{t-1} - a_{IGN}^{(3)} M_{IN_{pa}}^{t-1} - a_{IGN}^{(4)} M_{LIN_{ta}}^{t-1} + u_{IGN_{pa}}^t \end{aligned} \quad (3)$$

The categorical field contains two symbols (*pa* and *ta*). The type of a stimulus represents the proportions of the two symbols (Table 1).

name	*dev2M*	*dev1M*	*dev0*	*dev1P*	*dev2P*	$sens_{pa}$	$sens_{ta}$
pa value	0.7	0.55	0.4	0.25	0.1	0.8	0.2
ta value	0.3	0.45	0.6	0.75	0.9	0.2	0.8

Table 1: Constants for both phonemic categorization models

The sensitivity of each $IGN_\bullet$ to the received type is defined by a constant type $sens_\bullet$. IGN_{pa} is more sensitive to the symbol *pa*, and IGN_{ta} to *ta*. The function $g_{IGN_{pa}}$ in equation (3) is used with the constant $sens_{pa}$ and the incoming stimulus' type T_{Stim}^{t-2} in order to modulate the magnitude of IGN_{pa}:

$$g_{IGN_{pa}}\left(T_{Stim}^{t-2}\right) = T_{Stim}^{t-2}(pa)sens_{pa}(pa) + T_{Stim}^{t-2}(ta)sens_{pa}(ta)$$

The types are used only for the input gating; they do not intervene in the rest of the model. The sigmoid φ_{OGN} in OGN_{pa}'s magnitude function allows it to fire only if the magnitude coming from the IGN_{pa} is greater than the firing threshold's (FTN_{pa}) one:

$$M_{OGN_{pa}}^{t} = a_{OGN}^{(1)}\,\varphi_{OGN}\left(M_{IGN_{pa}}^{t-1} - M_{FTN_{pa}}^{t-1}\right)M_{IGN_{pa}}^{t-1} + a_{OGN}^{(2)}M_{OGN_{pa}}^{t-1} + u_{OGN_{pa}}^{t} \quad (4)$$

$$M_{IN_{pa}}^{t} = a_{IN}^{(1)}M_{OGN_{pa}}^{t-1} + a_{IN}^{(2)}M_{IN_{pa}}^{t-1} + u_{IN_{pa}}^{t} \quad (5)$$

$$M_{LIN_{pa}}^{t} = a_{LIN}^{(1)}M_{IGN_{pa}}^{t-1} + a_{LIN}^{(2)}M_{LIN_{pa}}^{t-1} + u_{LIN_{pa}}^{t} \quad (6)$$

$$M_{FTN_{pa}}^{t} = a_{FTN}^{(1)} - \left(a_{FTN}^{(2)}\left(a_{FTN}^{(1)} - M_{FTN_{pa}}^{t-1}\right) + a_{FTN}^{(3)}M_{OGN_{pa}}^{t-2}\right) + u_{FGN_{pa}}^{t} \quad (7)$$

AN consists in the sum of the successive *IGN*s' activations during one experimental block:

$$M_{AN}^{t} = M_{IGN_{pa}}^{t-1} + M_{IGN_{ta}}^{t-1} + M_{AN}^{t-1} \quad (8)$$

This is a gross approximation of the fMRI data, which models only the part of the information processing mechanisms in the activation building and neglects metabolic processes at the level of the cerebral blood flow. Since, except the Stim and the AN nodes, all nodes represent neuronal activities, the time unit is set to 1 ms. We used the *DD2* algorithm [Norgaard, *et al.*, 2000] to perform the simulations.

The hypothesis is that the difference of processing between normal and dyslexic subjects is caused by a disorder in the inhibitory mechanisms. Thus, the two models, one for the average patient and the other for the average control, use the same functions and share the same parameters, except for the inhibition nodes ($IN_\bullet$ and $LIN_\bullet$). There are no lateral inhibitions in the dyslexic model. It can be interpreted in cognitive terms as the fact that all the processors compete for each stimulus and that no clear category can be built. Also, the dyslexic model's internal inhibitions are slightly stronger than in the normal one, leading to a slowing in the stimulus perception. These two tentative interpretations are good starting points for new experiments.

The differences in the inhibition parameters are sufficient to obtain very different activation data. For controls, the more distant (from the pivotal stimulus, categorically speaking) the deviant is, the stronger the activation is (Figure 3). This is supposed to be caused by a habituation mechanism that lowers the activation, followed by an activation the force of which depends on the "surprise" caused by the deviant. Dyslexic subjects do not correctly categorize the different phonemes, both the *pa* and the *ta* parts of the gyrus activate for each block. This illustrates how activation data can be explained thanks to the understanding of the cerebral information processing mechanisms expressed in the models.

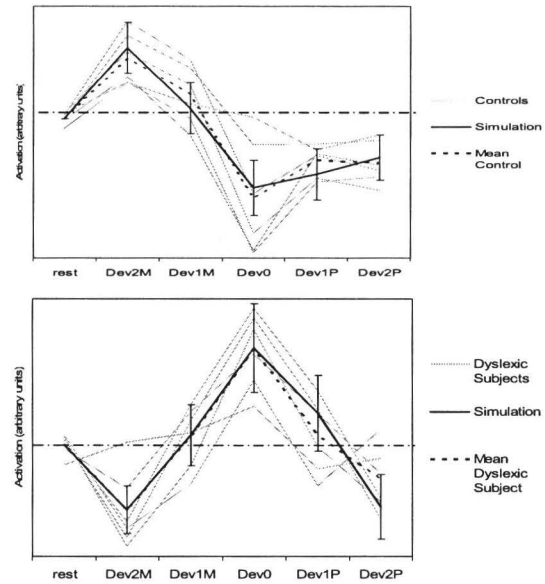

Figure 3: Compared results between simulated data (± 2 standard deviations) and experimental measures.

5 Conclusion

Instead of building a specialized model, designed for a specific function or cerebral network, we have presented a general framework, allowing the interpretation of functional neuroimaging data. This framework has been designed to be open to evolutions of the knowledge in neuropsychology and neurophysiology. Using DBNs allows modeling the brain as a dynamic causal probabilistic network with nonlinear relationships. We have illustrated this with an example concerning a language-related process. Currently, our framework is adapted to automatic processing, which is dominant in cerebral functioning. In function of the stimulus type, nodes can react differently and different networks may be activated, thus implementing different functions. Our future work will focus on the integration of more biological plausibility in the framework. The representation of complex relationships between and inside the zones will allow the representation of controlled processes and contextual modulation of the cerebral activity. The combination of types from different categorical domains and the search for regularities in the combinations will allow the implementation of learning mechanisms. Another essential topic is to make our mod-

els independent of the used data acquisition technique, thanks to *interface* models, able to translate cerebral information processing variables into neuroimaging results. Our long-term goal is to progressively include in our framework various validated models and to build a consistent and general brain theory based on large-scale networks.

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