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# Bayesian Modeling of Cerebral Information Processing

Vincent LABATUT

Josette PASTOR

INSERM U455

Fédération de Neurologie, CHU Purpan

F31059 TOULOUSE Cedex

France

{Vincent.Labatut, Josette.Pastor}@purpan.inserm.fr

## Abstract

Modeling explicitly the links between cognitive functions and networks of cerebral areas is necessitated both by the understanding of the clinical outcomes of brain lesions and by the interpretation of activation data provided by functional neuroimaging techniques. At this global level of representation, the human brain can be best modeled by a probabilistic functional causal network. Our modeling approach is based on the anatomical connection pattern, the information processing within cerebral areas and the causal influences that connected regions exert on each other. The information processing within a region is implemented by a causal network of functional primitives that are the interpretation of integrated biological properties. This explicit modeling approach allows the formulation and the simulation of functional and physiological assumptions.

## 1 Introduction

In Neurology and Neuropsychology, the understanding and the prediction of the clinical outcomes of focal or degenerative cerebral lesions, as well as the assessment of rehabilitation procedures, necessitate knowing the cerebral substratum of cognitive or sensorimotor functions. Human brain mapping is performed through activation studies, where subjects are asked to perform a specific task while data of their brain functioning are obtained through functional neuroimaging techniques. Such studies, as well as animal experiments, have shown that sensorimotor or cognitive functions are the offspring of the activity of large-scale networks of anatomically connected cerebral regions [Bressler, 1995]. However, a one to one correspondence between activated networks and functions cannot be found in all cases [Démonet *et al.*, 1994]. Understanding such incongruent results is crucial for the care of cerebral lesions.

Neuroimaging techniques and their traditional interpretation methods only address the following topics:

- (1) *Visualization of activated areas* (tomographic techniques) and *times of specific cerebral events* (surface electromagnetic techniques);
- (2) *What areas could participate in the same function* (“functional connectivity” [Herbster *et al.*, 1996]) and *what is the role of anatomical links on the activation* (“effective connectivity” [Büchel and Friston, 1997]).

Clearly, if the “where” and “when” (1), and the “what” and “how” (2) are answered, the “why”, i.e. how the activation of large-scale networks derives from cerebral information processing mechanisms, is missing. Our goal is the understanding of that “why”, which only can explain apparently conflicting activation data. Our research is twofold: providing plausible models, at the level of large-scale networks, of cerebral information processing mechanisms in humans and building a flexible simulator, allowing a quick implementation of the models, for a better interpretation of cerebral functional images.

Connectionist methods are the dominant approach in the modeling of the cerebral functional structure. However, they focus on functions emerging from a networked architecture of *populations of undifferentiated neuronal cells* [Grossberg *et al.*, 1997]. Modeling explicitly the role of *networks of regions* on information processing requires departing from this dominant viewpoint for at least two reasons. On one hand, we aim at modeling the function that emerges from the activity of *networks of differentiated cerebral areas*. On the other hand, the information processed by a cerebral area can be considered as the abstraction of the global signal emitted by the region’s neurons, representing both the pattern of firing neurons as well as their average firing rate, and can therefore hardly be modeled by a single numerical value. Moreover, since the cerebral response to a given stimulus may vary, the brain can be considered as a probabilistic information processor. In the next paragraphs, we will demonstrate that these constraints are in favor of a Bayesian approach, and more especially of causal functional networks.

## 2 Biological constraints

Modeling is constrained both by the necessity of a certain biological plausibility and by the purpose of the model building, that is allowing neurologists to express *explicitly*, as *cause-effect relationships*, their knowledge and hypotheses about the human brain.

### A networked architecture

The nodes of a large-scale cerebral network are functionally homogeneous, anatomically well-defined, *cerebral regions*, connected by *oriented anatomical links* (axon bundles), which are the network's edges [Pastor *et al.*, 2000]. Each region can itself be considered as a functional network of *processors*, such as *information processors* that are specific neuronal populations (e.g. GABA neurons) implementing *functional primitives* (e.g. inhibition).

### Causality and temporality

Every function (primitive or cognitive/sensorimotor function) is, in the brain, the outcome of the activation of an oriented network (called hereafter cerebral network), whose nodes are neurons or neuronal populations, and oriented links are axons or axon bundles. Information propagation results from a *cascade of causal events*, since the signal or information emitted by the firing of a node provokes the activation of its downstream nodes. The brain can therefore be considered as *a causal network*. According to Hume, A is the cause of B if they are contiguous, if A precedes B and if the relationship is regular. Our definition of causality, which extends Hume's one, is based on contiguity, probabilistic regularity and temporal consistency (ie. the beginning of A must precede the beginning of B).

Since anatomical links, which convey information with very short transmission delays, connect physically nodes in a cerebral network, the nodes are spatially and temporally adjacent and the condition of *contiguity* is strictly met.

Either at a large or small scale level, the response of a neuronal population to a given stimulus or information is not deterministic. The relationship between two nodes in a cerebral network has therefore a *probabilistic regularity*.

Physiologically, the temporal consistency is met, that is there is an order of activation for the cerebral areas. But the interpretation of activation data requires *representing explicitly time* in the models, and this representation has to be consistent with both the sampling time of neuroimaging techniques and the cerebral processing time. Depending on the temporal granularity chosen in the model, a cause-node and an effect-node could fire within the same time unit. This could lead to cycles in the network and hence to a loss of the causality. Imposing the model's network not to be cyclic will be necessary to keep the model causal.

### A two-dimensional representation of information

Cerebral information can be considered as the abstraction, at the level of a neuronal population, of the integrated activity of the individual cells. Any piece of information is defined as a couple of an *energy* and a *category*, where category

stands for which neurons react to a specific stimulus, and energy determines how they respond [Pastor *et al.*, 2000].

The cerebral *energy* reflects roughly the number of firing neurons and their firing rates. The energy of a stimulus can be extracted from its physical parameters (e.g. the intensity for a sound). It has a numerical representation.

The *category* of a stimulus summarizes the minimal set of physical properties that characterizes the information (e.g. the frequency for a tone). This "external" category is consistent with the "internal" category, that corresponds to the general pattern of neurons excited by the information. Information categorization is reflected in the "topic" organization of primary cortices and other areas [Alexander *et al.*, 1992]. For example, the auditory cortex can be decomposed in subareas reacting to precise frequency intervals. The category has a symbolic representation.

The pattern and the number of activated fibers of an axon bundle [Leiner and Leiner, 1997], which correspond to the pattern and the number of activated neurons in the emitting cerebral node, represent the category and the energy that is transmitted between two nodes.

### Uncertainty and imprecision

Uncertainty arises from the probabilistic regularity of cerebral events.

Furthermore, in humans, the only external evidences of energy values are provided by neuroimaging techniques and are therefore very imprecise. For example, the metabolic activity (tomographic signal) is an indirect measure of the neuronal activity.

### Conditions and non-linearity

The relationships between cerebral nodes (neurons or areas) are intrinsically non-linear. Moreover, the presence of conditions on information propagation increases the non-linearity of the brain processing. These conditions may go from very simple (firing thresholds) to very complex (role of special areas on the propagation between other regions).

### Habituation and learning

Both are related to the brain's adaptability. Habituation is a transient decrease of the activation that occurs when a neuronal population receives consecutively, several times, the same stimulus and that disappears when a new stimulus is presented. This kind of "energy saving" phenomenon may happen as soon as the second presentation of a stimulus [Miller *et al.*, 1991].

Learning is a permanent change of the brain state that occurs when a neuronal population receives regularly the same information pattern. The population's response becomes more efficient, that is fewer neurons fire and they become specialized in the processing of that information. The population is supposed to create a new information category, which represents the information pattern.

## 3 A new formalism for cerebral modeling

So-called "causal networks" should meet the two first

constraints of §2. However, all do not cope with the other requirements and all do not deserve to be called “causal”. The pros and cons of different causal formalisms are described hereafter, and our arguments in favor of probabilistic functional causal networks are given.

### 3.1 Causal Qualitative Networks

Causal Qualitative Networks (CQNs) have initially been designed to model physical devices and they are largely inspired by process control. CQNs are oriented graphs, whose nodes are qualitative variables, generally state variables, and edges are cause-effect relationships, generally influences between the state variables.

Causality is based here on three requirements [de Kleer, 1979]: locality (the cause acts only on its direct neighbors), precedence and regularity. Locality is weaker than contiguity, the neighborhood being not precisely defined. Precedence and regularity are stronger constraints than temporal consistency and probabilistic regularity. Therefore, CQNs do not meet our definition of causality

Qualitative algebras are at the core of CQNs. They take imprecision into account implicitly, by representing numerical values by some qualitative properties: signs, orders of magnitude or real intervals centered on the values. CQNs do not support uncertainty and, since imprecision is implicitly represented, it is not measurable or controllable.

An interval-based CQN, with an explicit discrete time representation, has been used in a previous tentative modeling of large-scale networks [Lafon *et al.*, 1999; Pastor *et al.*, 2000]. In order to meet biological constraints, the basic formalism was augmented by a limited non-linearity (piecewise linearity) and uncertainty (multivalued logic). However, it suffers drawbacks: a classical flaw of interval calculus [Struss, 1990] makes the range of intervals increase dramatically at each simulation step and uncertainty and imprecision are defined by different formalisms. Moreover, all the causes to a node are processed independently and then combined. This is the opposite of what happens in formal neural networks when the node processes the weighted sum of inputs. Whether the combination precedes the processing or not is still an open question in neuroscience. Those drawbacks restrict considerably the applicability of the system to cerebral modeling and has moved the research effort to Bayesian approaches.

### 3.2 Dynamic bayesian networks

Among the different dynamic Bayesian networks [Dean and Kanawaza, 1989], State Space Models (SSMs) [Ghahramani, 1998], an extension of Hidden Markov Models (HMMs), seem to be the most interesting formalism for our cerebral modeling approach. Relationships are defined by the probabilities of the current response variables conditionally to the current hidden state variables, and the expression of every current hidden state variable as a linear function of the past values of the hidden state variables, plus a random variable.

SSMs meet our definition of causality. The respect of temporal consistency, contiguity and probabilistic regularity is derived from the definition of the oriented, autonomous and stable relationships [Pearl 2000]. Other constraints are respected: the explicit and discrete representation of time, the possible handling of the numerical (energy) and symbolic (category) parts of cerebral information, the expression of conditions in the relationships’ deterministic part, a straightforward measure of uncertainty and learning mechanisms implemented by probability revisions.

However, two major requirements are not satisfied: non-linearity cannot be represented in the deterministic part of hidden state variables and no instantaneous relationship can be defined.

### 3.3 Causal functional networks

Causal Functional Networks (CFNs) [Pearl, 2000] are based on structural equations. Basic structural equations are asymmetric linear relationships, that is the equality symbol in each equation should be replaced by an affectation symbol ( $:=$  or  $\leq$ ) [Druzdzal and Simon 93]. Therefore, they are causal relationships.

However, in most applications of Structural Equation Modeling (SEM), relationships are symmetric and the equations system is identified globally, by fitting the theoretical covariance matrix to the observed one. This non-causal version is used in the “effective connectivity” image interpretation approach [Büchel and Friston, 1997].

CFNs [Pearl 2000] extend *causal* SEM in different aspects: variables can be numerical or symbolic, non-linear functions are used to model relationships and time can have an explicit, discrete representation. In fact, like SSMs, CFNs respect, temporality, uncertainty, conditioning, learning and cerebral information representation constraints and, in addition, they allow non-linearity and instantaneous relationships.

Their main drawback concerns the representation of imprecision: probability theory can directly measure only uncertainty, while imprecision can be only estimated by an average value and a dispersion value. A direct measure of both imprecision and uncertainty could be obtained by the use of the possibility theory [Dubois and Prades, 1994]. However, three points are in favor of the probability calculus: it has a well developed mathematical theory, neuroimaging data are statistical summaries, and overall, brain processing is mostly probabilistic.

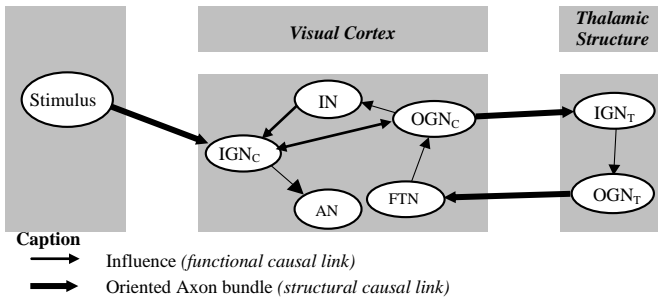
CFNs seem to be the best paradigm for cerebral modeling. Moreover, like all causal Bayesian models, they can answer clinical questions such as: “What happens in area A when area B is activated?” (observation) and “What happens when area A is damaged?” (intervention). In addition, they have the specific ability of answering “What would happen if area A was activated, knowing that it is not activated in reality ?” (counterfactual).

## 4 A Tentative Model of a Cerebral Area Network

CFNs seem to be the most adapted formalism to model cerebral mechanisms. The adaptation, in terms of a CFN, of the model described by Pastor *et al.* [2000], is given. This model aimed at explaining results from Fox and Raichle's experiment [1984] that study focused on the modulation of the activation of the striate cortex by the presentation rate of visual stimuli.

### 4.1 The causal network

The hypothesis is that the experimental results can be explained by the interactions between the striate cortex and the thalamus [Pastor *et al.*, 2000]. The "large-scale" network is a simple anatomical loop, the cortex and the thalamus being connected by opposite oriented axon bundles. The global functional network is the connection of the two functional networks representing the striate cortex and the thalamus (*Figure 1*), plus an additional node standing for the stimulus. Since delays are associated to the links in the network, at a given time, the network is an acyclic oriented graph.



**Figure 1.** The structural and functional network

### 4.2 Modeling Approach

The cerebral information, or part of it, is processed at each node. It is therefore a flowing entity, while nodes are processing entities and links are propagating entities.

#### Information Representation

The flowing entity is characterized by the values of its *Magnitude* (the representation of the information energy) and its *Type* (the representation of the category). Its state is, functionally (at each node of the causal network, *after* it has been processed by the corresponding information processor) and temporally (at each discretized instant  $t$ ), represented by a two-dimension random variable  $X(t) = (X_M(t), X_T(t))$ , attached to the node.  $X_M$ , the magnitude, is a real variable.  $X_T$ , the type, takes a multiple symbolic value  $\{s_1, \dots, s_n\}$ . A probability  $P(s_i/X)$  is associated to each symbol  $s_i$ . A symbol  $s_i$  represents a pure type (something theoretical), and the associated probability stands for the proportion of energy (i.e.  $X_M$ ) emitted by the  $s_i$ -typed neuronal population of the  $X$  node. At the metabolic processor nodes, the

information representation is limited to its magnitude part.

#### Propagation and Processing

A relationship is a couple of two functions dedicated, respectively to the magnitude and the type.  $X(t)$  is updated at each instant  $t$  of the simulation, according to the values of its causes, previously computed.

$$X_c(t) = f_{X_c}(PA_X(t), U_{X_c}), X_M(t) = f_{X_M}(PA_X(t), U_{X_M})$$

In the equations,  $PA_X(t)$  stands for the parents of  $X(t)$ , and includes generally  $X(t-1)$ . The  $U_{X_c}$  are error variables that do not depend on time.

For each region  $R$ , a Type Preference Table (TPT) contains the region's sensitivity to pure types. It is represented by the set of  $P(A/R, s_i)$ , where  $A$  stands for "Activation" and  $P(A/R, s_i)$  represents the chance for  $R$  to be activated, given that the received stimulus' category is of the  $s_i$  type.

The conditions are expressed by logical expressions that are included in the functions. These conditions take probabilistic values. Currently, to simplify the computation, we only calculate an expression according to the most probable value (true or false) of the corresponding condition (i.e. we do not care about the other case).

### 4.3 An example

Two processors exist both in the cortex and the thalamus. The Input Gating Node (IGN) expresses the area's neuronal reactivity to the stimulus. It may be considered as the abstraction, in terms of pattern and average firing rate, of the activation of the area's pyramidal cells' somas.

$$\begin{cases} IGN_{cM}(t) = [OGN_{cM}(t-1) > a] \times [b \times f_{TPT}(STIM_T(t-2) \times IGN_{cPT}(t-1)) \times STIM_M(t-2)] + \\ \frac{c \times IGN_{cM}(t-1) + d \times IN_M(t-1) + u_{IGNc}}{M_2} \\ IGN_{cT}(t) = \frac{M_1}{M_1 + M_2} \times STIM_T(t-2) + \frac{M_2}{M_1 + M_2} \times IGN_{cT}(t-1) \end{cases}$$

The Output Gating Node (OGN) sends information to the downstream areas. It represents, more or less, the integrated activity at the junction between the cells' somas and axons.

$$\begin{cases} OGN_{cM}(t) = [IGN_{cM}(t-1) > FTN_M(t-1) \wedge FTN_M(t-1) > 0] \times IGN_{cM}(t-1) \\ + b \times OGN_{cM}(t-1) + u_{OGNc} \end{cases}$$

Three other processors are specific to the cortex. The Activation Node (AN) reflects the level of the cortex's metabolic activity, linked to the neuronal energy demand. The inhibitory node (IN) represents the integrated behavior of the GABA-neurons. The dynamic Firing Threshold Node (FTN) is modulated by the thalamus that can lower it.

$$FTN_M(t) = c - (b \times (c - FTN_M(t-1)) + a \times OGN_{tM}(t-1)) + u_{FTN}$$

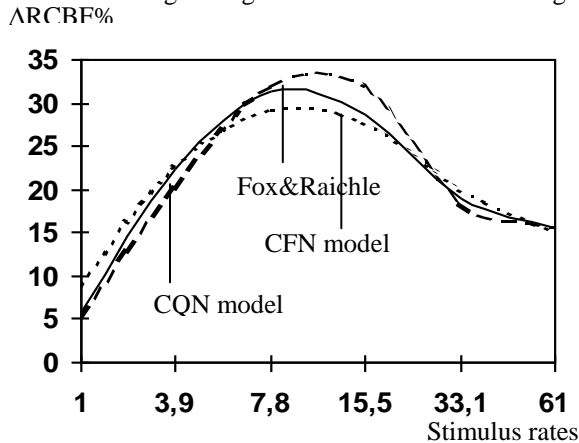
In the visual cortex, as soon as the energy, at IGN, is greater than FTN, OGN transmits information to the thalamus. The two points are illustrated by the definition of the cortical input node  $IGNc$  (*Figure 1*).

#### Simulation results

In the reference experiment [Fox and Raichle, 1984], the stimuli are orange square-waves pulses of constant intensity and duration (5ms) that are presented during 40s scans (PET) at rates of 1, 3.9, 7.8, 15.5, 33.1 and 61 Hz. For the

simulation, we suppose that the stimulus is deterministic with a magnitude of 1 and the type “orange”. The results are measures of the metabolic activation, i.e. measures, for each 40s-scan, of the regional cerebral blood flow variations ( $\Delta rCBF\%$ ) in the visual cortex measure.

In the model, the time unit is 1ms. The summation over 40s of all the AN values is a measure of  $\Delta rCBF\%$ , once the brain’s average activation level is set in the model, at its experimental value. *Figure 2* shows slightly better results for our model than for the CQN model [Pastor *et al.*, 2000], the main advantage being a better control of the divergence.



*Figure 2. Results of the simulations (mean values)*

## 5 Conclusion

Modeling large-scale cerebral networks, so that new evidences can be incorporated in the model and hypotheses can be assessed, is still a challenge. In the paper, we went through two major steps. The most important result is that causal functional networks are the best approach to cerebral modeling, since they fulfill theoretically all the requirements. Moreover, in the brief description of our modeling approach, we showed the flexibility and the adaptability of the formalism. Then, we proved that this formalism is really applicable, describing an example of cerebral model. With this model, we managed to approach experimental data, and furthermore we obtained (slightly) better results than with our previous CQN formalism.

The next steps will be on one hand to deepen the theoretical aspects of our modeling approach, and on another hand to assess the model by comparing simulation results to new experiments, involving more complex large-scale networks and a better temporal definition.

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