BioCaen : A Causal Qualitative Network for Cerebral Information Propagation Modeling

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Abstract

Functional brain mapping studies in humans may show contradictory results, as no one to one correspondence can be found between activated cerebral zones and cognitive functions. An explanation could be the networked physical organization of brain zones and the information propagation mechanisms through the network. As we focus on language-related brain subsystems, AI models are the single alternative to animal models. The brain being considered here as a physical, rather ill-defined system, AI qualitative approaches, especially causal methods, fit perfectly our purposes. The major constraint in the approach, i.e. the fact that phenomena related to the system's functioning must be time-ordered, is compatible with our knowledge on brain behavior.

This paper presents a tentative two-level model of brain information propagation mechanisms. At the structural level, the brain anatomical structure is represented as a component network whose nodes are cerebral zones connected by propagating or inhibiting anatomical links (axon bundles). At the functional/behavioral level, each zone is modeled by a causal qualitative network instanciated from a generic model. A component/connection approach derives the global functional model corresponding to a structural network - from the above models. As models must constantly evolve with new hypotheses and findings in brain research, we propose a flexible « hypothesis simulator », BioCaen, for implementing them. BioCaen is an offspring of Ca~En *(Bousson & Travé-Massuyès, 1993, 1994)* that extends its capabilities by : (1) dealing with components, (2) giving more flexibility to time-variation expressions, (3) coding causal network nodes by couples of variables.

Introduction

Since the 19th century, people have tried to identify cerebral locations of cognitive functions such as language or reasoning. The first evidences came from the comparison between cognitive disorders in patients and locations of their brain lesions, observed after death. Coarse relationships were found between structure and function : supposedly « missing » cognitive functions were assumed to be located at the lesions. Today, a better understanding is obtained from activation studies. In those experiments a patient, or a healthy subject, is asked to perform a specific cognitive task while evidences of his/her brain functioning are obtained through imagery techniques (Positron Emission Tomography -PET-, Magnetic Resonance Imagery -MRI- or Quantified Electroencephalography -EEGq-). The anatomical correlates of the task are supposed to be those brain zones whose activation (as seen through cerebral blood flow changes for PET and MRI, event-related potentials -ERP- for EEGq) is significantly different in the performed task when compared to a reference task.

However, clinical observations as well as experimental studies may show that no one to one correspondence exists between brain zones and cognitive functions *(Démonet et al, 1994; Raichle, 1993)*. Those incongruent results may have different explanations such as the existence of a real functional polymorphism in the cerebral areas or a wrong interpretation of experimental results *(Sergent, 1994)*. A third answer *(Damasio, 1989; Friedman & Goldman-Rakic, 1994; Mesulam, 1990)* may be the existence of anatomical links between remote cerebral zones and the possible network organization of cerebral structures involved in a specific task performance. Our assumption is that, besides the possible functional specialization of cerebral zones, the influence of brain network organization (cerebral zones and anatomical links connecting them) and of information propagation mechanisms through the network may explain the links between cognitive tasks processing and patterns of cerebral activation.

Questions arise also on the automatic/controlled, data/goal driven information processing in the brain. Clearly, both stimulus-driven (whether visual *(Fox & Raichle, 1984)* , auditive *(Price et al, 1994)* or motor *(Sabatini et al, 1993)* stimulus), automatic,

bottom-up mechanisms and goal-driven, controlled *(Corbetta et al, 1990 ; Funahashi et al, 1993)*, top-down processes coexist in the brain. Our assumption is that the efficiency and speed of the brain processing come from the major part played by automatic and stimulus-driven mechanisms. Therefore, the brain can be considered as a rather ill-defined dynamic physical system, whose function is information processing and whose behavior is constrained by its physical structure and networked organization of components (the cerebral zones) connected through anatomical links (axon bundles). No direct observation of the behavior exists and the only external evidences are indirect measures of the neuronal activity provided by tomography or ERPs. Comparing these measures, that will not feed our model, with outputs from the model simulation will help validate our assumptions and get a new insight on apparently conflicting cerebral activation patterns.

Our research is twofold : modeling human brain information propagation mechanisms and building a flexible simulator that will allow to quickly implement model changes and assess new hypotheses on brain behavior and function. This paper presents our first tentative model of propagation mechanisms, based on a former language-related experiment *(Démonet et al, 1994)*, model which has been the starting point for the simulator specifications. We will also describe the main features and implementational status of BioCaen , the model simulator.

AI tools and brain modeling

Models of brain behavior belong generally to two major trends in AI, the classical AI symbolic approach, on one hand, and connectionism, on the other hand, or the combination of both, hybrid methods *(Wallace et al, 1993)*.

The **symbolic AI approach** provides purely functional architectures that model high level cognitive processes such as reasoning *(Feigenbaum et al, 1971)* or memory *(Schank & Farrell, 1988; Anderson, 1988)*. This approach, that focuses on mind considered as a set of emerging cognitive functions independent of the biological device that generates them, does not meet our goal, which is the modeling of cognitive functions considered as an offspring of brain activity.

Connectionism is widely used for implementing " pure mind " cognitive models *(McClelland, 1981)*, as well as for modeling cerebral functioning *(Mitchell et al, 1991)*. However, connectionism does not fill our goal, i.e. the explicit modeling of cerebral brain propagation mechanisms, for it provides black box models that give an accurate approximation of a cognitive function output without giving any information on the brain physical mechanisms that generate it. Our work cannot either take advantage of current researches in " biologically plausible formal neurons ", for our modeling level is at the cerebral zones' behavior, which represents the integrated behavior of thousands of neurons.

In our view *(Pastor et al, 1995)*, the brain is a biological device, whose dynamics is largely automatic and constrained by its physical structure. The system's behavior can be quantified through functional imagery data, although not very precisely. All these facts suggest that the **AI qualitative approach** is the best modeling paradigm. **Causal methods**, which describe cause-effect relationships between a system's state parameters, fit perfectly the current knowledge on brain structure and mechanisms *(Travé-Massuyès et al, 1993; MQ&D group, 1996)*. The major constraint in this approach, i.e. the fact that phenomena represented by causal relationships must be time-ordered, is perfectly compatible with our knowledge on brain behavior. The Ca~En formalism and software *(Bousson & Travé-Massuyès, 1994)* are the core of the tools used in this work.

Qualitative modeling of brain information processing

Modeling the brain information processing at the integrated level of zones requires to take into account both a structural representation, the anatomical organization of these zones, and a behavioral and functional representation, the description of the information processing within and between the zones.

Structural modeling

Our goal is to disambiguate the interpretation of cerebral zone activation by modeling and simulating information propagation in the brain. Two major assumptions relative to this propagation *(Pastor et al, 1995)* have been suggested by former experiments conducted in positron emission tomography *(Démonet et al, 1992; Démonet et al, 1994)* and ERPs *(Doyon et al, 1995)*. Their results support the hypothesis that *cerebral information is automatically propagated from one cerebral zone to another through the in-between anatomical links, each zone having an acknowledgment mechanism that lets the information in, for further processing, or stops it. Once the information is processed, it is transmitted.*

A Cerebral Zone (CZ) is defined here as a closed, functionally homogeneous, part of the brain that can be located anatomically; its contours however

may be fuzzy. CZs are connected by anatomical links, either neighborhood relationships, made of a limited number or neuronal steps, or remote links built of white matter. Information can only be transmitted through these links.
The brain can hence be represented as a network of interconnected CZs. Anatomical links constitute oriented, across and inner links in the network. Two links with opposite orientations may exists between two CZs. In the CZ network, propagation is largely automatic and constrained by the network structure.
As shown in figure 1, two kinds of anatomical connections exist. Most links are simple information transmitters. Some are inhibitory links that transmit to the target zone an inhibition order instead of to-be-processed information.

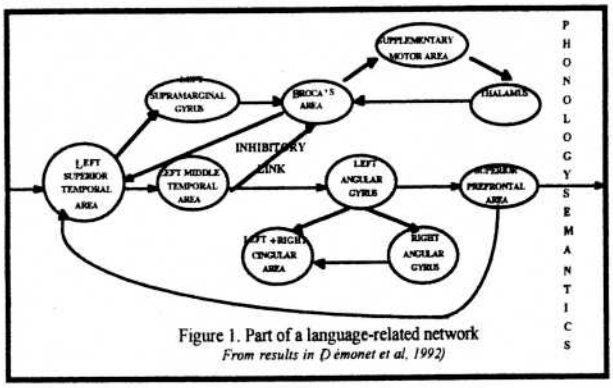

Figure 1. Part of a language-related network
From results in Démonet et al. 1992)

In summary, any subpart of the brain can be represented as an oriented graph whose nodes are cerebral zones and links are physical connections between the zones. Knowledge for building the corresponding structural network will be extracted from our experiments (as in figure 1) and/or from data in the literature. Each CZ can be interpreted as to be a component, associated with a behavioral and functional qualitative causal network.

Functional and behavioral modeling

- ***Hypotheses***

One of our most fundamental hypotheses is *the existence of common operating mechanisms among all the CZs, different CZ behaviors deriving from different instanciations of the mechanisms*. This assumption is supported by knowledge on brain plasticity and cortical reorganization *(Sabatini et al, 1994)*. The basic assumption of brain activation experiments is that CZ's activation variations are good measures of their involvement in the performing of a specific cognitive function. From a computational viewpoint, this means that information acknowledgment or processing in a CZ will raise its activation level. Therefore, in a straightforward manner, *activation can be coded as an energy level*. Moreover, *information propagating through the CZ network can be represented as a couple (energy, type)*. An energy level can be computed from physical parameters of auditory or visual stimuli (i.e. exogenous information). In the brain, information being the result of a neuronal discharge, it makes sense to code it also as energy. However, from our former hypotheses on brain transmission information, any CZ must be able to acknowledge incoming information. As energy level is not discriminant enough, a type has been added in order to characterize a specific piece of information. Each CZ " knows " to what extent it is able to accept and process information signed by a certain type.

- ***Model***

The common operating mechanisms assumed hereabove are implemented by a generic model. Each CZ is considered as an energy processor modeled by a dynamic causal network, the nodes of which represent major mechanism components as information acknowledgment, integration and processing, inhibition, activation... An oriented link between two nodes means an influence and a temporal ordering between the origin and the target. The nodes' states can, from our current knowledge, be given only qualitative values, i.e. real number intervals instead of precise numerical values.
The CZ's Type Preference Table (TPT) supports the acknowledgment mechanism that assesses the type of the received information. This mechanism calculates the Type fuzzy weights for the zone. The more the Type is preferred by the zone, the larger the weight.
The generic model has five nodes (figure 2):
(1) Integration Buffer Node (IBN) : The rationale for this node is that the information sampling period is generally too short for a sample to be meaningful. Meaning can be reached only after an integration time-lag, specific to the zone. When the generic model is instanciated for a specific zone, there are as many integration buffer instances $IBN_i = (IBNE_i, IBNT_i)$ as input CZs (i.e. CZs originating anatomical pathways to the zone). If the current zone is a primary zone (i.e. it has no preceding zone), there are as many IBN_i as stimulus. Each IBN_i is influenced by an Input Information $II_i = (IIE_i, IIT_i)$, which can be either a preceding zone's Broadcast Node (see *(4)*), BN_i, or an information instance directly computed from a stimulus properties. Each $IBNE_i$ instance is the result of the integration of the input energy IIE_i over time. IBN instances have the following properties :

* there is a rapid decrease of $IBNE_i$ when the input Type changes,
* a too quick shift in the input Types allows no acknowledgment,
* input information is considered as long as the inhibition node is not active and the activation energy AE is lower than a saturation threshold.
* $IBNT_i$ is the type of the II_i currently being integrated in IBN_i.

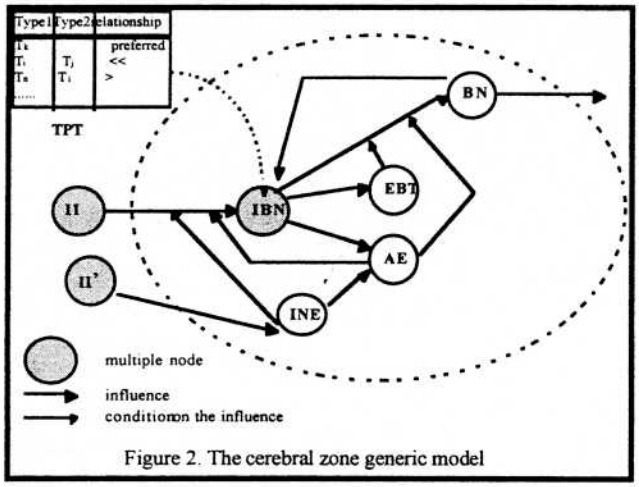

Figure 2. The cerebral zone generic model

(2) Energy Broadcast Threshold (EBT) : As long as the Activation Energy AE is below this threshold, there is no information broadcast. EBT is a function of all the $IBNE_i$ that varies with the heterogeneity of the information pieces arriving simultaneously at the zone and with the information ambiguity. This implements the fact that information processing in CZs is more efficient (i.e. quicker, with a lower energy level) when the zone is not overloaded by the mass of information or puzzled by information ambiguity. The less there are pieces of information and the more they differ, the lowest the threshold.

(3) Activation Energy (AE) : Information broadcast occurs when AE > EBT. The Activation Energy is a function of the Integration Buffer Node Energies, weighted by the corresponding Type weights ; it is also influenced by the Inhibition Node Energy INE. AE varies between a Minimal Threshold (MT) and a Saturation Threshold (ST). MT represents the permanent brain activity while ST allows a regulating mechanism to be implemented (no input to a zone as long as its AE is close to ST). AE variations are influenced by two marginal components: information processing and inhibition. The first component varies only with the weight of the $IBNE_i$ influence (in fact, the Type weights) : the more a Type is preferred, the more its corresponding Energy will contribute to AE. AE is an estimate of the zone activation level; therefore, as a first model validation, its variations will be compared to those of the activation measurements.

(4) Broadcast Node (BN) : In the current state of the research, the simplifying assumption is that there is no real Type processing within the zone and that BN = (BNE, BNT) is such that it corresponds to the Integration Buffer Node that has contributed the more to the variation of AE. BNT is the Type of this Buffer Node and BNE is an amplifying function of its Energy. The Broadcast Node constitutes an Input Information for **all** the CZs downstream the current zone. When information is sent out of the zone, BN influences all IBNs belonging to the zone by provoking an energy discharge.

(5) Inhibition Node Energy (INE) : Inhibition belongs to the class of regulation mechanisms. For example, when many pathways are competent in the processing of a specific information, inhibition can allow deactivation of the less efficient of these pathways. An inhibition link and a normal connection cannot simultaneously exist between two zones. An inhibition link between two zones has, in our model, the following effects :

* the target zone's Activation Energy increases briefly and the zone remains impermeable as long as the Inhibition Node Energy is positive,
* after a peak increase, the INE decreases, if no new inhibition impulse arrives at the zone.

For a given CZ, its model is an instanciation of the generic model. Each parameter of the generic model is given a value, specific to the zone, and calculated with identification methods drawn from Automatic Control techniques.

- ***Global model***

A global model is directly derived from the corresponding structural network and from the CZs' instanciated networks (figure 3). When a CONNECT link exists between two zones, the upstream zone's BN node is connected, for each downstream zone, to one of its IBN nodes. An IBN node is dedicated to a single preceding zone. An INHIB link between two zones, creates a link between the upstream BN and the downstream INE. BN nodes belonging to different zones connected to a specific CZ through INHIB links will be connected to its unique INE node.

Such a model provides a way to simulate a fully automatic, self-regulated, information propagation and brain activation mechanisms. Zone activation is completely data-driven and there is no need of a goal to be expressed for some CZs to be activated. The preferential activation of a given network does not need either any strategy to take place for it can be perfectly explained by the "all

direction broadcast" feature, in addition to the acknowledgment and the inhibition mechanisms. Another interesting feature is the "natural" implementation of the behavior of structural network cycles such as the « articulatory loop » which is supposed to be a fundamental mechanism of working memory in humans.

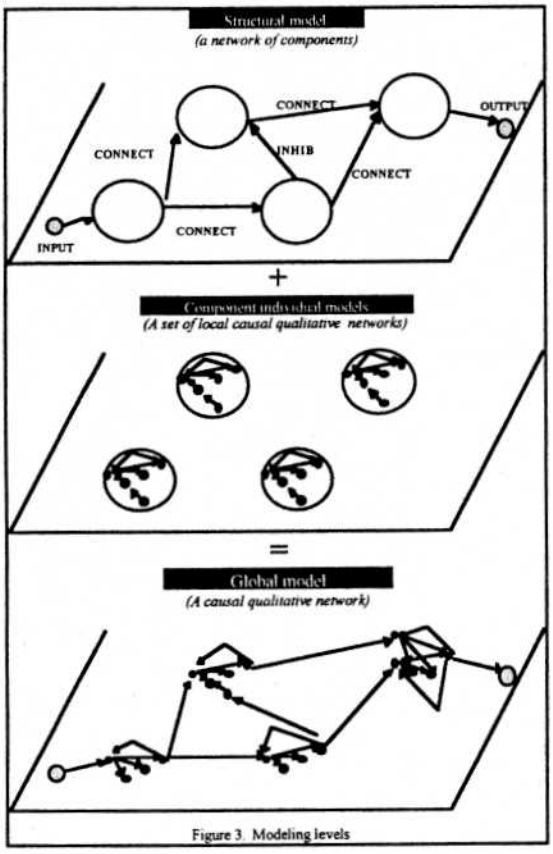

Figure 3. Modeling levels

BioCaen : a flexible causal qualitative network language and simulator

The structural model as well as the generic functional model are written in BioCaen which is an offspring of the Ca~En software *(Bousson-& Travé-Massuyès, 1994)*. We will describe hereafter Ca~En, BioCaen and the specific implementation of the generic functional model.

The causal qualitative simulator Ca~En

- ***The Ca~En knowledge representation formalism***

In Ca~En, time is dealt with explicitly, abstracted to a logical clock-based sampled time. The clock is set accordingly to the swiftness of the process. The process variables may be either numeric (real-valued) or symbolic. Numeric variables' *quality space* is a value set which is assumed to be a closed interval of reals; that of a symbolic variable is a finite set of symbols linearly ordered according to the application context. The value of a numeric variable may be any subinterval of its quality space. Its *variation* is defined as its value change within the unit of time. The variation value may be a real number (if precisely known) or any interval of reals.

The Ca~En formalism is based on a two-level representation scheme for the description of the relationships between the process variables: *a local constraint level* and a *global constraint level.*

The **local constraint level** is in agreement with our perception of a physical process as a net of interacting variables influencing one another. This is represented by a directed graph in which the paths presume the perturbation flow causality. The influences supported by the edges of the graph allow for representing causal dependency type knowledge. The Ca~En formalism allows for two kinds of causal relations : (1) influence-based relations which are concerned with cause-effect interactions among numeric variables; (2) information-based relations which are concerned with direct information about variable states. Relations of the latter kind are not useful in our application and they are not described in this paper.

An influence-based relation between a variable X and a variable Y is assumed to represent a linear first order type relation, which means that the influence corresponds to a linear first order differential equation. These relations are described by means of the following predicates:
$I+(X, Y, c, K, T_d, T_r)$, or $I-(X, Y, c, K, T_d, T_r)$
where :

- X, Y are the influencing and the influenced variable, respectively;
- c is the activation precondition of the causal relation (the influence from X to Y is said to be activated whenever c holds);
- K is a positive interval or real number representing the static gain of the influence;
- T_d is a positive real number or interval representing the delay of the influence, i.e. the time needed by Y to react to a variation of X;
- T_r is a positive real number or interval representing the influence response time, i.e. the time needed by Y to get a new equilibrium state after having been perturbed.

Note that the formalism allows one to cope with *imprecise temporal knowledge* since T_d and T_r can be defined as intervals.

A weight w may eventually be associated with the influence from X to Y to represent combination distortion phenomena. It is expressed by means of its relative order of magnitude with respect to the weights of other influences on Y in a fuzzy counterpart of the O(M) formalism that allows one to compute automatically the fuzzy value of weights as explained below *(Bousson & Travé-Massuyès, 1993)*.

Given two quantities a and b, the O(M) formalism proposes the seven following order of magnitude relations :

$a<<b$ $(a>>b)$	a is much smaller (larger) than b
$a-<b$ $(a>-b)$	a is moderately smaller (larger) than b
$a\sim<b$ $(a>\sim b)$	a is slightly smaller (larger) than b
$a==b$	a is exactly equal to b

If r is an order of magnitude relation between a and b, then $(a\ r\ b)$ if, and only if $(a/b\ r\ 1)$) *(Mavrovouniotis & Stephanopoulos, 1988)*. Hence a relation r can be characterized by the set of numbers which are in relation with 1. In our fuzzy counterpart, the sets corresponding to the order of magnitude relations are not crisp but fuzzy sets, presenting then an overlapping region which makes smoother the transition from one relation to the other.

The automatic generation of numeric weights is based on the marginal influence axiom: *the marginal influence of a variable X on a variable Y is the highest influence that X is able to exert on Y when it is combined with influences coming from other variables.* In addition, it assumes that the weights precedence graph is a high semi-lattice, the maximal weight at the top of the lattice being equal to one (from the above axiom). The algorithm explores the weight lattice from its top towards its leaves, in a width first manner *(Bousson & Travé-Massuyès, 1993)*.

The **global constraint level** is composed of functional numeric constraints associated with interval domains, e.g. constraints arising from physical laws. In other words, a global constraint is any mathematical equation - which may be non linear as well - in which each unknown is assumed to take on interval values; which still allows us to manage imprecise knowledge at this level.

- ***The Ca~En simulation algorithm***

The input data are the causal model - including initial conditions - and the evolution of the measured variables of the causal graph over time. The output of the system is the behavior of each process variable *(Bousson & Travé-Massuyès, 1994)*. The temporal unit of the simulation is that of the logical clock. The following steps are executed :

1. ***At the local constraint level :***

(1.1) Computation of the net variation : The computation of the net variation is given by:

$$\delta y(t) = \sum_{i=1}^{n} w_i \delta y^i(t) \qquad (1)$$

where $\delta y(t)$ is the net variation of Y from instant $t-1$ to instant t and $\delta y^i(t)$ is the variation induced by $\delta x_i(t)$ on Y assuming that only X_i influences Y, that is the *marginal variation* of Y_i on X. X_i , $i=1,...n$, are the active variables influencing on Y, $\delta x_i(t)$ is the net variation of X_i from instant $t-1$ to instant t and w_i is the weight associated to the influence from X_i.

For $I+(X_i, Y, c, K, T_d, T_r)$, the influence-based relation, marginal variations are expressed as:

$$a_1 \delta y(t)/\delta t + a_2 y(t) = b_1 x_1(t - T_d) \qquad (2)$$

where coefficients a_1, a_2 and b_1 are real numbers or intervals.

If T_s is the sampling period of the process behavior, the values of Y and those of X_i at the sampling instants can be directly related by a recurrent equation which is equivalent to (2) in a sampled temporal scale.

$$y(t+1) = a y(t) + b x_1(t-d) \qquad (3)$$

where:

- t stands for the logical time (an integer)
- d is the least integer greater than or equal to T_d / T_s
- $a = e^{-T_s/\tau}$ and $b = K(1 - e^{-T_s/\tau})$ if T_r is finite (τ is the *time-constant* of the influence which corresponds with a good accuracy to $T_r/3$)
- $a=1$ and $b= KT_s$ if T_r is infinite (integration relation)

If we had a negative influence $I-(X_i, Y, c, K, T_d, T_r)$ instead of $I+(X_i, Y, c, K, T_d, T_r)$, then the coefficient a would remain unchanged, but b would undertake a negative sign.

The recurrent equation relating the variation of Y to that of X_i (the marginal variation) at sampling instants is directly obtained from (3):

$$\delta y^i(t+1) = a_i \delta y^i(t) + b_i x_i(t - d_i) \qquad (4)$$

where :

- $t=0,1,2,...,\ i=1, ... ,n$,
- a_i, b_i and d_i are the coefficients related to the influence of X_i on Y and are defined in the same manner as in equation (3). The delays

and response times are automatically taken into account.

The net variation of Y is then obtained by equation (1) which sums up all the marginal variations weighted by the fuzzy weights of every influence w_i, $i=1,...n$. These fuzzy weights are updated according to the set of active influences at hand.

(1.2) Computation of the updated variables' values : For each variable Y the net variation is summed with the value of the variable at the previous clock tick. The result is an interval.

$$y(t) = y(t-1) + \delta y(t) \qquad (5)$$

2. ***At the global constraint level :***

(2.1) Refinement of the updated variables' values : The numeric intervals obtained for the updated values (Eq. 5) are refined with the global constraints by performing a tolerance propagation algorithm *(Hyvönen, 1991)* on the set of variables.

The simulation results produced by the Ca~En prediction module are envelopes that provide the upper and lower bounds of the variable values at each sampled instant.

BioCaen : adapting Ca~En to brain modeling requirements

BioCaen altogether is a restriction of Ca~En capabilities and extends the flexibility and expressiveness of Ca~En remaining features. It works exclusively at the causal level : Ca~En global constraint level cannot be used here, as it is for respresenting analytical knowledge which is not available here. In fact, Ca~En global constraints, when available, are only used to narrow the range of qualitative results obtained from the local constraint propagation. Their use is not compulsory. Therefore, in our domain, a restriction to the local level can only improve the computational efficiency.

BioCaen's three major extensions of Ca~En causal level are required by features of the cerebral information transmission model :

(1) Components : A structural model may be considered as a network of components (the CZs), each component being itself a causal qualitative network. A global model is a causal qualitative network, automatically generated from its structural model and its components' local networks (figure 3).

(2) Node definitions : In the CZ generic model, node variables represent a couple (energy, type). BioCaen needs therefore to extend Ca~En's node definition from a real (number or interval) variable to a couple N=(Y,S) where :

- Y is a real variable whose values are numbers or intervals,
- S is a symbolic variable having a finite value set $\{s_1, s_2, \ldots, s_k\}$.

(3) Propagation functions : In Ca~En, the function used to compute the nodes' temporal variations is unique and the same for every node (cf. equations 4 and 1). In BioCaen, propagation functions are not unique. They are provided in functional libraries that are loaded together with BioCaen. Therefore, anytime a propagation function is called by a model, its use is straightforward, provided the function exists in the libraries. The choice of a model's functions is up to the user that builds the model. He/she can either use a basic library provided with the system or add his/her own library. This extension gives more flexibility to the system behavior modeling. It is made necessary by the fact that brain models evolve very quickly and that we do need an « hypothesis simulator ». However, if implementational problems linked to this extension are currently solved, computational properties still need to be studied. Completeness and soundness concern only the energy component of a BioCaen node (types have symbolic values). BioCaen, like Ca~En, provides complete and non sound results, that are value envelopes. However, new propagation functions may change dramatically the simulator's complexity, which must be studied under different conditions (i.e. different propagation function classes).

Four functions and a partial O(M) relation are defined at each node N=(Y,S), the value set for S being $\{s_1, \ldots, s_k\}$. If $\{M_i=(X_i,C_i),\ i=1\ldots n\}$ is the set of the nodes influencing N, $\{c_1^i, c_2^i, \ldots, c_{m_i}^i\}$ the value set of C_i for $i=1\ldots n$, and δY^i the marginal variation of Y under X_i, we have :

- R, a fuzzy O(M) relation on $\bigcup_{i=1}^{n} \{c_1^i, c_2^i, \ldots, c_{m_i}^i\}$ and $W=\bigcup_{i=1}^{n} \{w_1^i, w_2^i, \ldots, w_{m_i}^i\}$ the set of the corresponding fuzzy weights
- $F : \bigcup_{i=1}^{n} \{c_1^i, c_2^i, \ldots, c_{m_i}^i\} \rightarrow \{s_1, \ldots, s_k\}$ gives the value of the target symbol from the influencing node symbol values; the application of F is submitted to a precondition SC.

- f_m^i, the marginal variation function, associated to the influence of M_i on N, such that

$$\delta y^i(t) = f_m^i(\delta y^i(t-1), S(t-1), \delta x_i(t-T_d), C_i(t-T_d)) \quad (4')$$

The application of f_m^i is submitted to a precondition RC^i.

- f_c, the combination function, such that

$$\delta y(t) = f_c(\delta y^1(t), \delta y^2(t), \ldots, \delta y^n(t)) \quad (1')$$

- f_u, the update function, such that

$$y(t) = f_u(y(t-1), \delta y(t)) \quad (5')$$

BioCaen must be flexible enough to include various local R, F, f_m, f_c and f_u. The implementation strategy has therefore shifted from a global package (Ca~En) to a toolbox where function libraries are provided and can be added. Currently, the function library is restricted to the implementation of the generic model.

Implementation of the generic model

The generic functional model is a simple application of BioCaen possibilities. Let CZ be a specific zone, and CZ^i, i=1... r, such that for all i, $CONNECT(CZ^i, CZ, T_d^i)$ and CZ^j, j=1... p, such that for all j, $INHIB(CZ^j, CZ, T_d^j)$; T_d^i and T_d^j mean that information propagation or inhibition order transmission have delayed effects.

$\bigcup_{i=1}^{n} BNT^i \subset TPT$, where TPT is CZ's type table, i.e. it is possible to give a weight to any incoming type. That does not mean that any type will be recognized, i.e. will be given a high weight. The O(M) relation R, associated to CZ, has been defined for TPT, with resulting weights W.

CZ's nodes may be described as follows :

- **IBN nodes**

There are as many IBN nodes in CZ as preceding zones to CZ. For i=1,...,r, $IBN^i = (IBNE^i, IBNT^i)$, with $IBNE^i$ being the node's energy and $IBNT^i$ being its type.

(1) Outputs from the CZ^is influence the variations of $IBNE^i$ in two different ways : their energy variation is integrated in $IBNE^i$ as long as their type does not change whereas a type change produces a decrease of $IBNE^i$. Quick type shifts in the inputs will therefore prevent $IBNE^i$ to be high enough for the information getting pertinence. The following influences hold :

$$I+(BNE^i, IBNE^i, ((INE=0)and(AE<ST)), 1, T_{d_i}, \infty)$$

$$constant\_influence(-K_Z, IBNE^i, ((INE=0)and(AE<ST)and((prec(IBNT^i,1) \neq prec(BNT^i, T_{d_i}))))$$

where prec(X,T) = X(t-T)

The above influences hold as long as the zone is not inhibited and not saturated. Inhibition and saturation make the cerebral zone « deaf », which will entail a rapid decrease of the activation energy. The first one is a Ca~En influence, while the second one's precondition is an extension of Ca~En's conditions.

(2) It is assumed that after CZ has emitted information, there is a sort of hush, and it becomes less able to receive and interpret new information :

$$I-(BNE, IBNE^i, (prec(AE,1) > prec(EBT,1)), K_b, 0, 0)$$

The precondition being more complex than in Ca~En, this is an extension of a Ca~En influence.

(3) Combination and update are similar to Ca~En's .

(4) The integration buffer type is that of the received information: $IBNT^i(t) = BNT^i(t - T_{d_i})$

- **INE node**

(1) As no zone is supposed to resist an inhibition order, there is no type associated to an incoming inhibition. Therefore the inhibition node has no type.

(2) An inhibition order may have different strengths (energy levels) and it is assumed that CZ's response to such an order is not immediate after CZ receives the order (after the delay T_d^j). The response time to inhibition is supposed to be intrinsic to each CZ. Therefore, $I+(BNE^j, INE, t, 1, T_d^j, T_r^{inhib})$ holds.

(3) When CZ receives two or more inhibition orders, they reinforce each other and their energies combine. Ca~En's combination and update functions are therefore used .

- **EBT node**

(1) This node is an energy threshold : as long as the activation energy is below, there is no information emission. Therefore, it has no type.

(2) It models the fact that CZ is sensitive to the heterogeneity and the number of its incoming pieces of information. Therefore its energy is not dependent on variations of individual (IBNE^i, IBNT^i). Rather, it depends on the global incoming information pattern. This can be expressed by the fact that the energy at time t can only be calculated through an update function. There are no influence or combination functions. Anytime, the following equations hold :

$$EBT = EBT_{min} - K_o \sum_{i=1}^{r} p_i \, Log p_i - K_a \sum_{i=1}^{r} q_i \, Log q_i$$

where $p_i = \dfrac{weight(IBNT^i)}{\sum_{l=1}^{r} weight(IBNT^l)}$,

$$q_i = \frac{weight(IBNT^i) * IBNE^i}{\sum_{l=1}^{r} weight(IBNT^l) * IBNE^l}$$

$0 < K_o << K_a$ to make overload less difficult than ambiguity ;

EBT_{min} is the value of EBT when there is only one active IBN node.

This update function is based on the notion of entropy. EBT will increase with overload, if too many different types enter the zone at the same time. It will increase also with ambiguity.

- **AE node**

(1) The activation energy is influenced by all integration buffers energies according to IBN's type weights. When incoming information to the zone is not « recognized » (low weight), its energy participates only to AE as acknowledgment energy. When it is recognized (high weight), its energy participates as processing energy. In addition, we suppose that processing activity in itself creates energy. This can be expressed, in an influence with a variable gain, which extends a Ca~En influence:

$I+(IBNE^i, AE, t, K_a * weight(IBNT^i), 0, T_a^i)$

(2) Inhibition temporarily increases AE before the rapid decrease due to the lack of incoming information. This is written as $I+(INE, AE, t, K_{inhib}, 0, 0)$.

(3) AE is calculated simply by adding up these influences.

- **BN node**

When different information compete in order to be processed, it is assumed that both the level of recognition of the information and its energy will select the information to be broadcast. Therefore :

$$BNE(t) = K_{out} * max\left\{weight(IBNT^i(t)) * IBNE^i(t), i = 1 \cdots r\right\}$$

$BNT(t) = IBNT^{i_{max}(t)}(t)$ with :

$weight(IBNT^{i_{max}(t)}(t)) * IBNE^{i_{max}(t)}(t) =$

$max(weight(IBNT^i(t) * IBNE^i(t), i = 1 \cdots r)$

Information broadcast only occurs when the activation energy is higher than the broadcast threshold, that is if the condition AE > EBT is met.

Conclusion

This paper, that reports a preliminary work, aims at highlighting possible interactions between QR and human brain mapping. Currently our work is still in a development phase. A restricted version of BioCaen compiler and simulator is already implemented. From Ca~En to BioCaen, the language has been notably augmented at two levels : the structural network description level has been added and the influence descriptions have been enriched (for example, the influence gain which was a constant is now an expression, and the influence condition syntax is more complex). These changes, together with the use of function libraries give BioCaen all the expressiveness needed for brain activation modeling. In fact, building the first generic model aimed principally at getting pertinent specifications for BioCaen language. The only current limitation to this expressiveness is the fact that propagation function libraries accept only Lisp functions. A more « natural » language is going to be developed. In addition, a graphic HMI is being designed so that any neuropsychologist could be able to design and simulate any model. The two following steps of our work will be completing the implementation and studying BioCaen's computational properties. Besides its obvious necessity, this will allow to get more confident in the model simulation and to separate model errors from computational errors. It is also a way to falsify the model if experimental data fit the simulation outputs. Indeed, BioCaen flexibility allows to define node functions that could lead to a non convergent system, which may not be found out if test data are not numerous enough.

The generic model takes into account our first hypotheses on brain information propagation. This model that has few nodes and relationships, has been designed to give a self-regulation capability to the cerebral zones and an autonomous, data-driven behavior to a structural network. Currently it is evolving in order to give additional properties to a zone, such as memory and learning capabili-

ties that are zone-dependent. It has to be implemented and validated in order to make a pertinent assessment of the model errors and to guide the model evolution. The experimental validation is particularly needed to assess the model physiological plausibility. We planned to focus the validation experiments on basic auditory processes in human brain. We will capitalize on studies *(Celsis et al, 1997)* showing that brief acoustic events are coded in the temporal cortex as separate units and that this can be assessed in functional imaging experiments. These experiments can manipulate the temporal structure of stimuli so that brain counterparts of temporal coding can be identified. Mapping ERP with multiple surface electrodes will be the best method to do so, combined with the use of the BESA (Brain Electrical Source Analysis) software *(Scherg, 1990)*. Every experiment will be based on the same principles : the simulation inputs are a brain zone network, the links of which are exclusively established through anatomical and physiological knowledge, neurophysiological and neuropsychological data and stimulus values ; the simulation outputs will be the envelopes of the zones' activation node curves. These envelopes will be compared to the activation values obtained during the experiments on human subjects.

Extensions are already planned. An important software extension concerns the information extraction from stimuli. Currently this is "hand made", that is the stimuli are artificially coded. It would be an important improvement to extract energy and type from the stimuli physical parameters. This would lead to define more refined functions for converting an information type, as it circulates through the network. Another extension is the modeling of brain information propagation when sensory or visual stimuli are implied. It is grounded on our belief that the brain propagation mechanisms are not linked to a specific behavior and that behavior is the outcome of different type recognition capabilities and of connection patterns between zones. One support to this assumption are the well-known studies on hippocampus *(Carpenter & Grossberg, 1993 ; Squire & Zola, 1996)* that show that the non-specific mid-term memory capability of this brain structure could be linked to the great number of its connections with different neocortical zones (*Squire & Zola-Morgan, 1991)*. Although we assume that the temporal evolution of an activation pattern is the evidence of a specific cognitive function, the system is not able to provide this function. Interpretation in terms of cognitive functions is up to the neuropsychologist. This new approach, based on a systemic viewpoint, of brain functioning, has shown, than despite the biological complexity, cerebral modeling, when taken at the right level of interpretation, is not out of the scope of QR methods.

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