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Modelling the compression of perceived time by a cerebral system with nervous excitability deficit and near the perception threshold

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ABSTRACT

A macroscopic model of the nervous tissue is used to explain temporal aspects of perception in human cases with deficitary nervous excitation due to loss of neural cortical mass. In these cases, the cerebral system is less excitable, has a lower reaction time and a slower decay of excitation than in a normal case. The model considers the macroscopic nervous excitation response in the brain, involved in the integrative process, and contains essential ingredients such as permeability to the excitation of the network implicated and its reaction time. Non-local temporal aspects subjected to causality are taken in account. This simple model accounts for the observed shortening of the perceived duration of a stimulus, the perceived reduction of the lapse of time between two events, and the lower discrimination between repeated stimuli, when the network of the cerebral system has a deficit in nervous excitability and the intensity of the stimulus is close to the perception threshold. Temporal summation is involved in these effects. Stimuli can be visual, tactile or auditive, though we restrict the references and data to the visual system.

1. Introduction

Since very early it is known that the capability to discriminate between two consecutive stimuli in time decreases as luminance decreases (logarithmic Ferry-Porter law [1,2]) for a wide range of luminous intensities. Also, for targets of constant size and constant luminous intensity, temporal resolution between repeated stimuli decreases with eccentricity in the visual field in a range of intensities not too low [3]. This finding is confirmed more recently by several authors (e. g., [4–6]) and represents a decrease of "temporal acuity" towards the periphery of the visual field. Temporal acuity is often measured by means of the flicker fusion frequency, defined as the minimum frequency at which a flickering stimulus (repetitively presented stimulus) appears as continuous. It was also pointed out that the perceived speed of moving objects increases at low luminance [7], an assertion that is confirmed by several authors ([8–10] and references therein), or if it is perceived in the periphery of the visual field [11,12]. According to what is exposed in the present work, all these examples would illustrate a lowering of the nervous cerebral excitation for stimuli with low luminance or in the periphery of visual field.

This type of phenomenology was extremely pronounced in human cases with brain damage reported by Gonzalo [7,13] and referred to as *central syndrome* cases. They suffered from a marked loss of cerebral excitation due to the neural mass lost in a unilateral parieto-occipital cortical lesion, in a rather unspecific (or multisensory) zone equidistant from the visual, tactile and auditory primary areas (the middle of area 19, the anterior part of area 18 and the most posterior of area 39, in the Brodmann terminology), the corresponding projection paths being untouched. These cases showed bilateral and symmetric multisensory disorders strongly dependent on the intensity of the stimulus and on crossmodal effects that tend to improve the perception. The fundamental disorder in this syndrome is a functional depression where sensory functions (or qualities) of all sensory systems are loss in a defined order according to their different excitability demands as the intensity of the stimulus diminishes. Higher (or complex) functions are then the first to be lost. A functional gradients model was proposed [13], where the sensory specificity of the cortex is distributed. For a sensory system, the specificity is maximum in the corresponding projection area and decreases in gradation towards other zones, the final decline of the gradients reaching other primary areas. The cortical lesion in central syndrome corresponds to the zone where the different specific sensory gradients would overlap (a rather unspecific multisensory zone). The gradients model explains the multisensory affection, the general functional depression and the enhanced crossmodal effects [7,13–16]. This is in close relation and in agreement with other works and recent results (e. g., [17–28]).

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A key feature of the central syndrome is its similitude with a normal man. The sensory system maintains its organization but at a reduced scale of nervous excitability [7,13,16]. Spatial and temporal acuity, as well as movement perception, requiring high spatial and temporal discrimination, are complex functions that would need high nervous excitation degree (and cerebral integration), and therefore they are among the first functions lost when the excitability of the neural network decreases, as reported in central syndrome cases [7,13]. In them, and under low intensity of the stimulus, the temporal interval between successive stimuli was perceived as shortened. The flicker fusion frequency was then lower than that in normal, which supports the idea that the temporal integration underlying flicker fusion does not occur at the level of the retina (untouched in these cases), but that it takes place later in the visual hierarchy, as also sustained by other authors (e. g., [29]). Movements were perceived at a higher speed and with a much shorter trajectory than they were. Since there is also a compression of space, the overestimated speed of a moving object would be due to a compression of time in a greater degree than the shortening of the trajectory. These disorders were present in visual, tactile, motor and auditory systems, and were accompanied by many other disorders such as tilt or inversion perception of the stimulus, diminution of its size, loss of its shape and color [7,13,14,16,30]. For example, corporal movements, including waking, were felt as shorter in amplitude and with an overestimated speed.

Although little is currently understood about the mechanisms that determine processing time, we explain here some features of the above phenomenology from a model that accounts for the seemingly paradoxical fact that a cerebral system with a deficit in nervous excitability and slow response can perceive events as if they were faster than they actually are. In this model, we focus on macroscopic properties of the nervous tissue such as the permeability to the excitation and the time response, or reaction time. These properties account for the capability of temporal summation, which is magnified in a slow system. In them, there is a long delay between the stimulus and the initiation of perception (long latency), and also a slow decay of the sensation (long persistence) and, if a second stimulus arrives before the excitation of the first one has completely fallen down, there is a summation effect in the cerebral excitation. The same occurs for a train of intermittent stimuli, so that it is possible to reach the excitation threshold to produce a sensorial perception in spite that a single one could be unable to do it.

In this macroscopical model we do not consider the internal structure of the neural network involved. What is crucial in this model is the non-local temporal aspects subjected to causality, that is, the cerebral excitation at a time t is the result of the stimuli produced at that instant and all preceeding instants. This causal feature is analogous to that encountered in material media supporting time-varying electromagnetic fields, and is generally referred to as time-dispersive media. This is the reason why we used the term time-dispersive model in previous works [31,32]. Here we improve the time-dispersive model initially proposed in [31] in order that it can account for the shortening of the duration of a stimulus, the shortening of the time interval between two stimuli, and the temporal discrimination between consecutive stimuli, as well as the dependence with the intensity of the stimulus. The model establishes a relation between these temporal aspects and the macroscopic excitation response of the neural system, and can be applied to stimuli of different nature, as visual, tactile or auditive. However, we refer only to data of the visual system.

2. The model

We consider a simple model in which the cerebral excitation response at an instant of time is determined by the stimulus at that time and all previous instants of time. Thus, for a stimulus $S(t)$ acting on the cerebral system, the excitation $E(t)$ produced at a time t in the cerebral system depends on the stimulus at all times $t' \le t$. Within the simplest assumption of a linear dependence, the most general expression for the excitation is expressed by

$$E(t) = \int_{-\infty}^{t} \chi(t-t')S(t')\,dt' = \int_{-\infty}^{+\infty} \chi(\tau)S(t-\tau)\,d\tau, \tag{1}$$

where $\tau = t - t'$, and χ is related to the capability or permeability of the system to become excited. For a cerebral system that does not change in time, χ depends only on the time difference τ between the stimulus and the excitation. Further, causality imposes that $\chi(\tau) = 0$ for $\tau < 0$. The permeability $\chi(\tau)$ can be interpreted as the cerebral excitation under a delta pulse stimulus, i.e.,

$$E_\delta(t) = \int_{-\infty}^{+\infty} \chi(\tau)\delta(t-\tau)\,d\tau = \chi(t). \tag{2}$$

A simple model for $\chi(\tau)$ that accounts for most of the relevant aspects of the excitation response to different stimuli is

$$\chi(\tau) = \chi_0\, a\tau\, e^{-a\tau}\theta(\tau), \tag{3}$$

where $\theta(\tau)$ is the Heaviside step function ($\theta(\tau) = 1$ for $\tau \ge 0$, and $\theta(\tau) = 0$ for $\tau < 0$). It can be shown that Eq. (1) with $\chi(\tau)$ in Eq. (3) is the solution for the differential equation

$$\frac{d^2E(t)}{dt^2} = a\chi_0 S(t) - a^2E - 2a\frac{dE}{dt}, \tag{4}$$

with initial conditions $E = 0$ and $dE/dt = 0$ at $t = 0$. The term $a\chi_0 S(t)$ represents an external "force" due to the stimulus S acting on the system, the term $-a^2E$ is like a recuperation "force" to the initial equilibrium state of the system and $2a(dE/dt)$ represents an intrinsic "damping" of the excitation.

We apply this model for an overall or gross description of the excitation $E(t)$, which represents a macroscopic measure of the cerebral excitation and involves the propagation of many induced nervous impulses. In Eq. (3), the constant χ_0 measures the excitation permeability, and the factor $a\tau$ is introduced to describe the initial growth of the response, which was ignored in a similar model used previously in [31]. The growth of the excitation is followed by an exponential decay $e^{-a\tau}$. The parameter a is then directly related to the velocity of the system, i.e., with the chronaxia of the nervous tissue (related to the reaction time $1/a$). If a is small, the system is slow and the excitation increases and decays slowly. The value of $1/a$ is the time required for the nervous excitation to reach a maximum, related to the latency period, and $2/a$ is approximately the decay time elapsed from the maximum of the excitation to half the maximum, thus being related to the persistence of the excitation.

To show the phenomena described by this model, we compare the nervous excitation response of two cases conveniently chosen so that one of them represents a normal case (denoted as N) with excitations well-above the threshold of perception, and the other one represents a deficitary case (denoted as D) characterized by lower values of χ_0 and a than N, with excitations close to the threshold perception, and that could represent a central syndrome case [7]. A reasonable assumption is that the *cerebral* excitation threshold E_{th} to obtain minimum sensation is the same for a normal case N and for a deficitary case D. What is different for N and D is the stimulus threshold since their reaction times $1/a$ and permeabilities χ_0 are different. We take for these examples the ratio $a_D/a_N = 0.25$ for the reaction times and the ratio $\chi_{0D}/\chi_{0N} = 0.15$ for the permeabilities to the excitation, in such a way that their responses can be represented in the same graphic at the same scale for the different situations. Taking in account the visual temporal resolution of about 40 ms estimated for characters [33], and that the flicker fusion frequency in direct vision in humans is known to be around 60 Hz (temporal resolution about