

Neuromimetic model of interval timing

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Abstract. Neuromimetic models of time processing mechanisms in the sub-second to minute range are mainly focussed on the mean and variance properties of time estimation (scularity) but offer no appropriate account of attention manipulations: a systematic underestimation of time with decreasing levels of attention. Our model is able to reproduce the scularity and attentional effects, and fits both behavioral and brain imaging data.

1 Introduction

Time processing in the sub-second to minute range is an inter-species ability, suggesting ubiquitous basic mechanisms which enable for anticipatory behaviour (e.g. pavlovian conditioning) and for many human skills (e.g. music playing). The duration of both signals and motor acts can be accurately timed, as shown by two major categories of temporal protocols concerned with either perceptual discrimination or motor production. In discrimination tasks, signal duration or inter-stimulus interval is manipulated, with all other features being steady across trials. Subjects estimate, for example, whether the duration of a signal delivered on a current trial is identical to or different from a previously memorized duration. Motor timing tasks involve accurate regulation of sustained motor responses (e.g. finger-pressing on a key) or inter-response intervals.

Perceptual and motor temporal performances share common fundamental trends. Typically, temporal performance is scalar (cf. Weber's law): the coefficient of variation of temporal estimates is constant across different duration ranges, which implies that the distributions of estimates superimpose when scaled by relative time (Fig 6, right panel). Furthermore, the form of the distributions is quasi-Gaussian. Also remarkable are the systematic biases due to arousal and attention changes. Firstly, target durations are judged longer with increasing arousal levels induced by activating drugs or increases in body temperature, and shorter with opposite manipulations. Secondly, target durations are judged shorter with less attention allocated to timing performance due to interference with a concurrent task; the proportion of attention paid to the elapsing time can even be voluntarily controlled (Fig. 7, right panel).

1.1 State of the art

Basically, models of duration encoding imply either pattern recognition or accumulation mechanisms (review in [1]). In the first category, it is proposed that oscillatory activities of various frequencies could be synchronized when the target duration starts, such that the temporal estimate depends on detection of the subset of neurons which are spiking synchronously when it ends. Hebbian strengthening of the

relevant synapses ensures neural coding of this momentary pattern. The same network is activated at each subsequent target occurrence, thus forming a memorized template of the target duration. Other possibilities are that, rather than firing synchrony, phase relationships between oscillators, or a spectrum of different neuronal events could be coded to define the target limits. In any case, these models are designed to code the limits of a target duration rather than the time elapsing between them. On the other hand, the accumulator concept [2] implies the storing of periodic or stochastic pulses (time bases) from start to end of the target. Temporal estimates are correlated with pulse number, which increases monotonically with the passage of time. Although the accumulator is normally switched on during the entire target duration, it can be transiently switched off if this duration includes gaps (i.e. is divided into segments separated by irrelevant sub-periods).

Most models account for scalar timing but not attentional effects. Mattell and Meck [1] suggested that attention, like arousal, modifies oscillators frequency. Experimental data, however, indicate that attention and arousal manipulations have independent effects on temporal performance [3]. The accumulator framework enables to conceive that arousal impinges on pulse sources (e.g. drug effects) whereas attention shifts provoke transient interruptions in pulse accumulation. Interfering signals that are processed while the target duration elapses can indeed act as gaps that fragment the target duration. The model we present thereafter is grounded on the accumulator concept and is designed to match data issued from both behavioural and brain imaging studies [10].

2 Neuromimetic model of interval timing (Fig. 1)

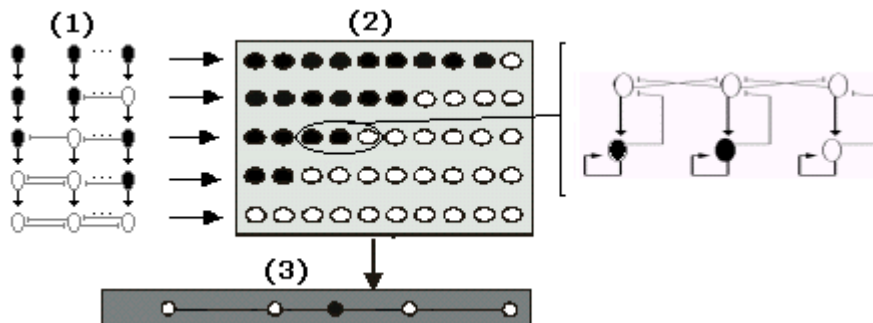


Fig. 1: The 3 modules of our model: (1) a frequency generator which outputs signals of decreasing frequencies, (2) an accumulator that transforms the temporal signals into spatial signals (on right a detail of the accumulator structure), (3) a memory unit that allows for performance improvement over repetitions. In black, the neurons currently spiking. All neurons are spiking neurons.

2.1 Frequency generator module

To be able to measure delays ranging from sub-second to minute with scalar temporal performance (Weber's law), our model implements frequency generators ranging from 80 Hz down to 7 Hz. This is achieved with a series of neurons layer, each layer

connected to the following by 1-to-1 excitatory connections. Intralayer inhibitory connections – which percentage increases from one layer to the following – account for the decreasing of the generated frequency (Fig. 2). In our model, the first layer of neurons exhibits a spontaneous activity at 80 Hz.

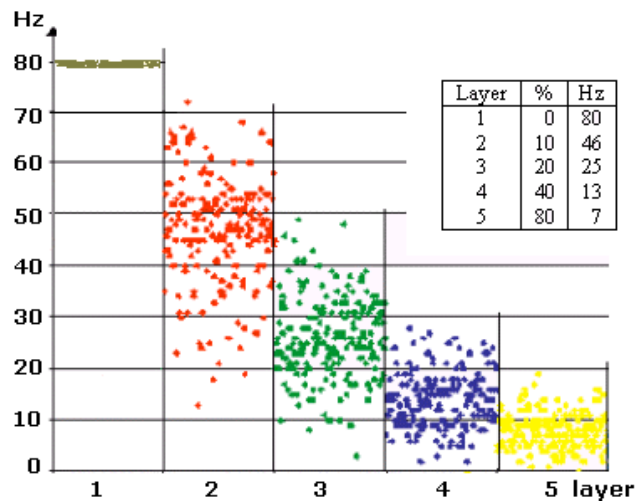


Fig. 2. Left: Distributions of the individual neuron frequencies in each layer. There are 300 neurons per layer, neuron threshold is 100, interlayer connection weight value is 100 and intralayer inhibitory weight value is -3. Top right: Percentage of inhibitory intralayer connections and the corresponding mean frequency in Hz.

2.2 Accumulator module

The accumulator module is composed of several layers (one for each frequency generator layer) of bistable units implemented by pairs of neurons (see (2) in Fig. 1). The upper neuron of the bistable unit counts, whereas the lower neuron memorizes. The accumulator module realizes the temporo-spatial transform. A given delay becomes a specific pattern of neuron activation. Each (upper) neuron receives inputs from the neurons of the corresponding frequency generator layer. The order of activation of the (upper) accumulator neurons remains constant over repetitions because the percentage of connections from the neurons of the frequency generator are all different, ranging from 100% (300 connections) down to 17% (50 connections). In Fig. 1, the upper neurons are ranged in descending order depending on their percentage of connections. After an upper neuron has reached its threshold, it must keep its activation. This is achieved by the lower neuron of the bistable unit which has recurrent excitatory connection that allows for a continuous spiking activity. The inhibitory connection with the upper neuron will forbid it to spike again. Moreover, the activation of the upper neuron also resets all the other upper neurons of its layer. All these features guarantee that the interval of time between two neuron activations is constant (on average). The number of bistable units per layer is 25, the threshold of the upper neurons is 1000, the connection weight to the frequency generator neurons is +1. All lower neurons are connected with inhibitory connections to an external reset signal (not displayed) that allows resetting of the accumulator before any new measure of delay.

2.3 Memory module

Time is one dimension, therefore we thought that a 1-D Kohonen map [4] should be a particularly well-suited implementation for the memory module. As time elapses, the number of activated neurons in the accumulator increases (more rapidly in the upper layers due to the greater frequencies of the frequency generator). Fig. 3 shows that each neuron of the topological map is associated to a prototype pattern of accumulator activation. Due to the neighbourhood property (each neuron is connected to 2 other neurons), similar accumulator patterns are coded by neighbour neurons. Fig. 4 displays the learning of a 2 seconds delay.

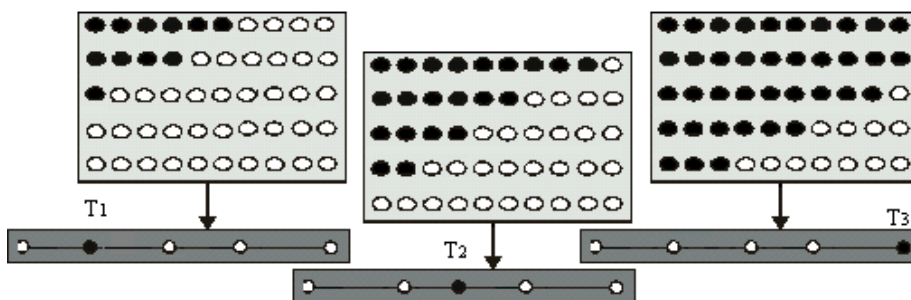


Fig. 3. Three delays (T1, T2 and T3) coded by the accumulator and the corresponding activation of the topological map. Neighbourhood property of the topological map explains why a long (T3), relative to a short (T1) delay, is coded further right on the 1-D map.

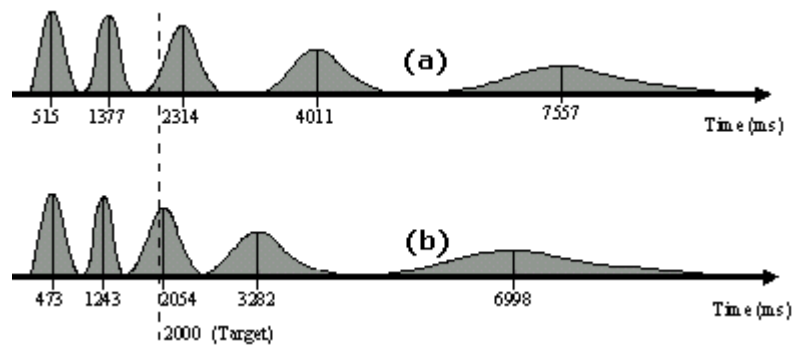


Fig. 4. Each of the 5 neurons of the 1-D topological map codes for a specific delay (represented in abscissa). (a) The initial situation. (b) The same map after learning a 2 seconds delay. Now, the third neuron codes now for a value of 2.054 s. (very close to 2 s) and its two neighbour neurons have converged closer to this neuron. This will account for the learning and generalization property that is observed in experimental situations. In this simulation experiment, the number of neurons is 5, the learning parameters (μ , β) values are 0.2 and 0.02 respectively, the number of learning iterations is 500, whereas the number of samples in the learning set is 1000 (parameters and equations in agreement with the formalism shown in [5], pages 44-47).

3 Simulation results

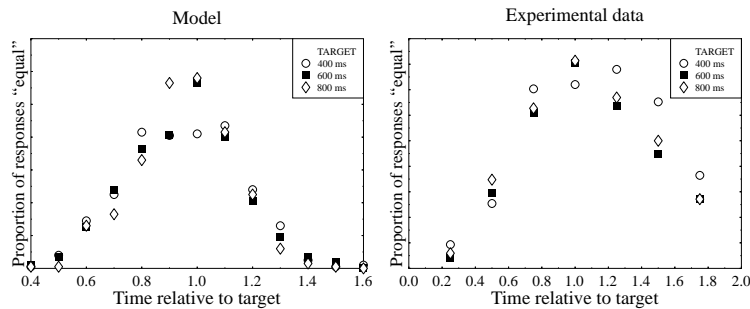


Fig. 5. Scularity of the temporal estimates: distributions superimpose when rescaled relative to the target durations (here targets are 400, 500 and 600 ms, set equal to 1 on the abscissa). Our simulation results (left) are similar to human performance in temporal discrimination: when subjects judge intervals as being equal or not to a pre-memorized target, the proportion of responses “equal” depends on the current interval-to-target difference, whatever the target value (right: adapted from [6]). This is a strong argument for the validity of our model.

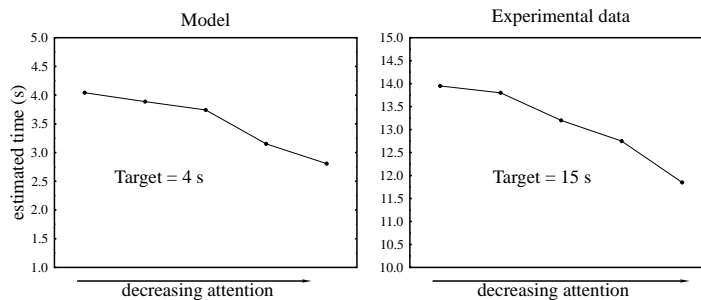


Fig. 6. Attentional effect. The attentional process acts at the level of the link between the frequency generator module and the accumulator module (see Fig. 1). A reduction of attention is simulated by a diminution of the connection weight, which results in lower accumulation rate, and, hence, shorter estimated time. Our simulation results (left) are similar to human performance in a task of temporal reproduction with variable attention levels (right: adapted from [7]).

4 Discussion

One important feature of our model is that it includes few *a priori* constraints: most properties emerge from the functioning of the entire network population rather than from arbitrarily well tuned properties of individual neurons or inter-neuron connections.

As such, the model is able to reproduce well-established properties of temporal performance as well as data issued from brain imaging studies [10]. First, it accounts for the scalarity observed in humans and animals without introducing any Gaussian-like variance. Secondly, it fits with the monotonous relationship found between brain activation and estimated time in regions thought to subservise the temporal accumulator [8]. In our model, this relationship is paralleled by a lengthening of the estimated time as the number of active neurons in the accumulator module increases. Thirdly, the effects of arousal and attention processes are simulated at distinct levels, as required by behavioural data [3]. While arousal effects can impinge on the frequency generator module, attentional changes affect its link with the accumulator module. By decreasing the number of activated neurons in the latter, our simulation accounts for the finding that decreased levels of attention paid to a target duration correspond to its underestimation (review in [9]), and, furthermore, to a decrease in the activation of brain regions that subtend timing performance [10].

Although certain aspects of our model need further specification, we view it as a first step toward integration of major behavioural and neurophysiological data in sub- and supra-second timing.

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