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A Model-Based Approach for the Quantification of H Reflex Depression in Humans

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Abstract - A dynamical model is used to describe the H reflex depression in humans in response to trains of stimuli.
Keywords: H reflex, H reflex depression, dynamical model

I. INTRODUCTION

The analysis of the H reflex, a homologue of the stretch reflex, is useful as an aid in understanding certain neurologic disorders as it reflects some aspects of spinal cord neuronal circuitry and dynamics. A single H reflex has a prolonged depressing effect on a second H reflex elicited afterwards. There has been some controversy about the mechanisms behind this depression, presynaptic inhibition and homosynaptic depression being the most cited. H reflex depression has been described for durations longer than 5s and, therefore, it is unlikely that presynaptic inhibition is a relevant mechanism in the latter part of the depression, since the duration of its effect beyond 600 ms has not been demonstrated [1].

A simple model is used here to quantify the depression of H reflexes when a train of stimuli is applied. It describes the interplay of neurotransmitter depletion and replenishment in the presynaptic boutons and has been used to study transmitter turnover in the Ia to motoneuron synapses in the cat [2].

II. METHODS

A. The Model

The model assumptions are: (1) a train of N stimuli at 1/T Hz is applied, (2) each presynaptic action potential is followed by the release of a fraction p ($0 \leq p \leq 1$) of the available storage of neurotransmitter, (3) the replenishment rate is proportional to the amount of depleted neurotransmitter at any instant of time. If τ is the time constant of the replenishment process, the faster this process is the smaller will τ be, and vice-versa. Let $s(iT_-)$ be the amount of stored neurotransmitter available for release immediately before time instant iT, where T is the interval between stimulation pulses in the train. Let $s(iT_+)$ be the amount of stored neurotransmitter immediately after a release. Both $s(iT_-)$ and $s(iT_+)$ are normalized with respect to the initial amount of neurotransmitter. The following equations then follow:

$s(iT_+)=(1-p)\, s(iT_-)$

$s((i+1)T_-)= s(iT_+)+[1- s(iT_+)](1-\exp(-T/\tau))$

for $i=0,1,\ldots,(N-1)$ and with $s(iT_-)=1$ for $i=0$.

Calling r(iT) the amount of released neurotransmitter at time instant iT taken with respect to the amount released at time 0, it is clear that due to the normalizations being used r(iT) is equal to $s(iT_-)$. The equations above may be solved for the steady state and after some additional algebra one gets

$p=[r(T)+r-1-rr(T)]/[2r-rr(T)-1]$

$\tau = T/\log[(1+pr-r)/(1-r)]$

where r is the steady state value of the released amount of neurotransmitter and r(T) is the value of released transmitter for the second pulse in the train.

We noticed that there is an intrinsic limitation in the practical use of the model which is related to the problem of noisy measurements of r and/or r(T). It is straightforward to show that there is a large sensitivity of parameter p when r is large, i.e., near 1 and, hence, for the narrow range of feasible values for r(T) (i.e., $r \leq r(T) \leq 1$) the value of p will go rather abruptly from 0 to 1. Therefore, one should not expect reliable estimates for p whenever there is little depression.

B. Experimental Procedures

H reflexes were recorded by surface electrodes placed over the soleus muscle and the Achilles tendon. The stimuli consisted of 1 Hz trains of N=10 1ms rectangular current pulses delivered to the tibial nerve by means of surface electrodes with the cathode in the popliteal fossa and the anode on the patella. The intensity of stimulation was adjusted to produce a baseline (i.e., undepressed) H reflex with an amplitude of 20% the maximum direct M response. Averages were computed from the results obtained from at least 10 trains delivered at least 1 minute apart. The peak to peak value of each H reflex was measured. From each experimentally obtained average curve of H reflex depression (at a given pulse rate 1/T) the corresponding values of r and r(T) were used to determine the estimates for p and τ.

III. RESULTS

In Fig. 1 we compare the depression obtained in a normal subject (male, age 38) with that predicted by the model. The stars are the mean H. From this data we estimated p=0.40 and τ = 3.21 which were then used in a program that simulates the synaptic dynamics according to the model described above. The result of the simulation agreed quite well with the data (circles in Fig. 1).

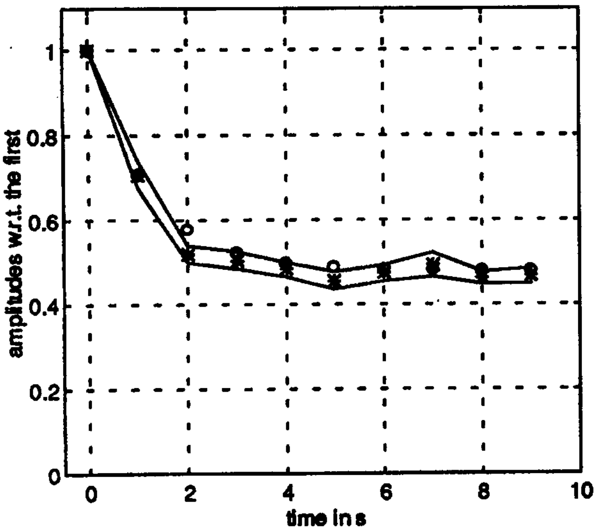

Fig. 1 - Experimental data of H reflex depression for a train of 10 stimuli at 1 Hz (*) and synaptic depression predicted by the model (o). The continuous lines show the standard error of the mean for the experimental data. The ordinates are the amplitudes with respect to (w.r.t.) the first value.

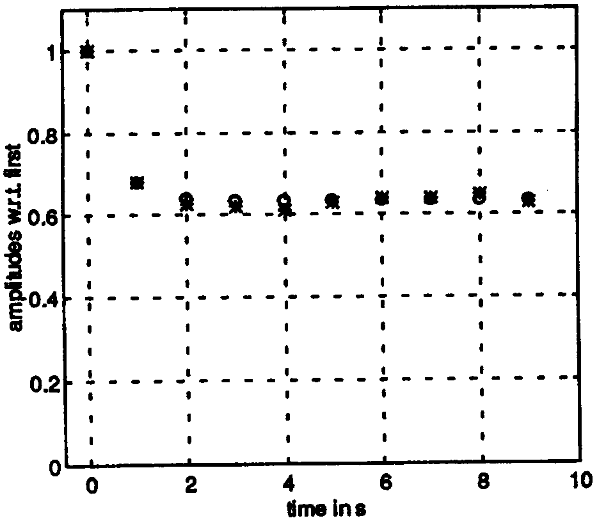

Fig. 2 - Experimental data (*) of H reflex depression compared with model simulation (o), both for a 1Hz train. The ordinates are the amplitudes with respect to (w.r.t.) the first value.

The experimentally obtained H reflex depression curves for the normal subjects that were analyzed showed different relations between r(T) and r, sometimes r(T) was quite above r, as in Fig. 1, while at other times it was somewhat closer to r, as shown for a different normal subject (male, age 26) in Fig. 2 (stars). This "sharper corner" is characterized in the model by a larger value of p, i.e., a larger depletion percentage per transmitter discharge. For the experimental points in Fig. 2 the values for the model parameters were $p=0.70$ and $\tau=1.27$ and the corresponding simulated result is plotted with circles in Fig. 2. The maximum standard error of the mean for the H reflex train was 0.014.

IV. DISCUSSION

Our results indicate that the use of the simple model is helpful in characterizing H reflex depression curves. From the viewpoint of pattern recognition, the two features p and τ are better quantifiers for the curves than the steady state value r which has been in use, e.g. [3], and hence better for discrimination purposes. Therefore, these parameters could be used with advantage in an effort to characterize different neurological dysfunctions based on H reflex depression data. In addition, as the parameters have a proposed physiological interpretation, it may be possible to obtain some insight about internal processes not available for direct measurements in humans. The model results will suggest some hypotheses on mechanisms and these could then be tested by other types of experiments or by doing experiments in animal models. It is clear that there are intervening processes not being considered here between the amount of released neurotransmitter in the Ia to motoneuron synapses and the recorded H waveforms at the muscle. If they have an influence in our study of H reflex decrement it is probably more in terms of static nonlinearities. The data we have analyzed so far were almost always well fitted by the model so we have not yet felt the need to introduce more complicated models. The model indicates that it is possible to describe H reflex depression by means of homosynaptic depression.

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