

## THE LEARNING OF INSERTIONS BY THE CEREBELLUM

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### Abstract

A computational model of the cerebellum is developed to learn how to guide the learning of insertions in syntax or motor responses by means of a teacher (assumed in the hippocampus). A short simulation is shown, in which LTP/LTD is used for synapses from the granule cells onto the Purkinje cells, attaining suitable effectiveness.

### Introduction

The cerebellum is well known to be important for learning temporal aspects of control, and its features in developing temporal sensitivity have been explored by numerous experimental studies of conditioned learning [1, 2]. It has also been recognized as important in supporting cognitive processes, both from the effects of cerebellum deficits on language processing as well other cognitive task areas. There are also effects of interaction and combination of the cerebellum and hippocampus which prove of importance, as trace conditioned learning shows [3]. More generally we expect the cerebellum to be the basis of error-based learning driving the learning of action responses (including motor planning and covert language use) from being under attention from parietal and prefrontal control to be housed more securely at an automatic response level in prefrontal circuits crucially involving the basal ganglia.

The purpose of our work is to develop a model of the cerebellum based on the known architecture it possesses in the brain, and then attempt to extend the ability it has been shown to possess computationally [1, 2, 3], to learn to be sensitive in response to timing in conditioned learning to similar sensitivity that we suspect is at the basis of syntactic analysis in language and in planning of motor actions. In particular we will attack the problem of insertions of endings (or also pre-fixes, although that will not be considered specifically but should also be amenable to our treatment). We extend the architecture modeled in [1, 2, 3] by considering feedback from the deep cerebellar nucleus (DCN) to the pons so as to guide the insertion process. However our paradigm needs a teacher throughout, acting as an unconditioned stimulus (US). We assume this arises from the hippocampus or nearby parahippocampal cortex, to be used later to help improve the production, under cerebellar guidance, of the initially incorrect but later corrected responses/stem-ending. Thus our simulation is in two stages:

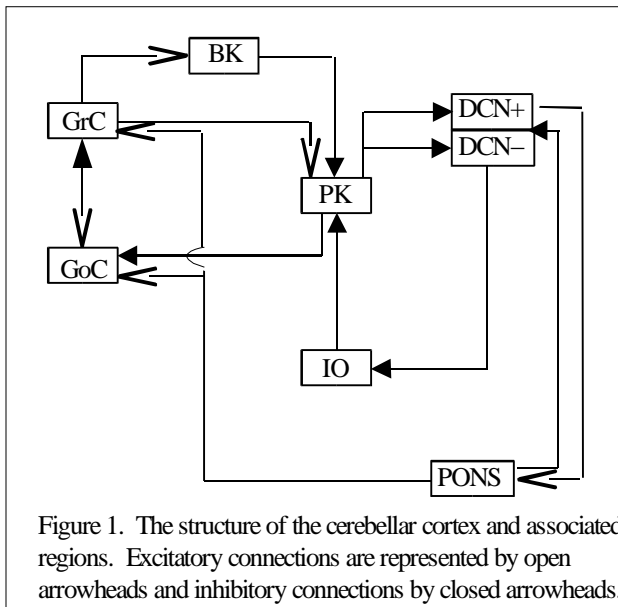
- 1) We consider the learning of a stem representation in the cerebellar cortex;
- 2) We then develop the learning of the stem-ending temporally defined response, assuming that this total sequence is presented as the US in a conditioned-type of learning process.

The paper consists of a description of the model, a description of the simulation results, and a final discussion.

### Description of Cerebellum

The Cerebellar cortex is composed of golgi cells (GoC), granule cells (GrC), basket inhibitory interneurons (BK) and purkinje (PK) cells, as in figure 1. There are 2 routes of input to the cerebellum: the climbing fibres and mossy fibres. The climbing fibres originate in the Inferior Olive (IO) and are excitatory to the PK cells, with a single synapse on each PK node. The mossy fibre input, also excitatory, originates in brain stem nuclei such as the PONS, and contacts the GrC and GoC, and (external to the cerebellar cortex) the deep cerebellar nuclei (DCN). It has been suggested that GoC inhibition on GrC forms time-windows from GrC firing [4]; another effect of the connections between these neurones is the setting up of 2 populations of GrC: an on set and an off set [3] when there is an input via the PONS. PK neurones receive excitatory input from GrC and IO, and inhibitory input from the BK nodes; PK neurones have intrinsic currents which lead to spontaneous firing rates, which when combined with its inputs leads to resting firing rates of 50 – 100Hz. PK cells inhibit 2 populations of DCN neurones: excitatory (DCN+) and inhibitory (DCN-); further inputs to the DCN come from the pons. Reciprocal connections from DCN+ to the pons allows for the reintroduction of excitatory activity into the cerebellar cortex, whereas

the DCN- inhibit the IO. This structure is summarised in fig. 1.



IO neurones have a slow rate of firing rate of complex spikes (1–2Hz) during resting action, and complex spike input to PK cells causes a pause in PK cell activity. The output of the cerebellar cortex is via the PK cells to the DCN. This connection is inhibitory thus keeping DCN activity reduced, so that any release of this inhibition will lead to increased DCN firing from a resting rate of 10 – 25Hz to ~200Hz.

GrC to PK node synapses undergo long-term potentiation (LTP) and long-term depression (LTD); LTP occurs when there is GrC input without IO input, and LTD occurs when a GrC input follows a IO input within ~50 –100ms, though changes have been seen over longer time scales as well. Learning has also been detected at DCN synapses formed from mossy fibre inputs. The crucial factor here is the firing rate of the PK neurones: for high firing

rates (>40Hz, so this includes the normal PK resting spike rates) LTD occurs for these synapses, but when PK rates drop below 40Hz LTP of the synapse takes place.

### Conditioned Learning

A normal conditioned learning task is the eye-puff. During this task the unconditioned stimulus (US) is an eye-puff which typically lasts for ~50ms. The conditioned stimulus (CS) is often a tone active for 100–500ms inputting to the cerebellum via the pons. During conditioned learning the CS and US co-terminate, so the CS has a period of 50–450ms when it is the only external stimulus. This paradigm leads to 2 stages of learning of the GrC to PK synapses. An initial period of LTP is caused by increased GrC activity without a change in IO firing rates due to the input of the CS (figure 2). This is followed by a period of LTD when the US causes firing of the IO by its input there (figure 2). The input of the US has 2 methods of reducing PK cell activity 1) the LTD of the GrC→PK synapse, and 2) the pause in PK activity caused by climbing fibre complex spikes. This combined reduction of PK activity causes disinhibition of the DCN. As training continues the output of DCN- can cause the annihilation of the US input, as we would expect in error-based learning.

### Method

We use a neurone model based on that used in [5] which is a leaky integrate-and-fire spiking neurone. Two excitatory glutamate pathways are modelled: the fast AMPA and the slower acting NMDA, and two GABAergic inhibitory currents: GABA<sub>A</sub> and GABA<sub>B</sub>, and an inactivating after-hyperpolarisation (AHP) current. The neurone equation is:

$$CdV/dT = I_{LEAK} + I_{AHP} + I_{NMDA} + I_{AMPA} + I_{GABA_A} + I_{GABA_B} \quad (1)$$

where  $I_{LEAK}$  is a leak current, negative above the neuron's resting potential,  $I_{AHP}$  is an inactivating after-hyperpolarisation current, and the synaptic currents (NMDA, AMPA and GABA A and B) are modelled with two channel kinetic variables representing fractions of open channels, which give rise to positive (NMDA, AMPA) or negative (GABA<sub>A</sub>, GABA<sub>B</sub>) currents.

The learning of GrC synapses on PK is adapted from [2], and controlled:

$$\Delta w_i = \delta_{LTP} GR_i CF(T) (Ca_{conc} - 0.0175) + \delta_{LTD} GR_i (1 - CF(T) (Ca_{conc} - 0.0175)) \quad (2)$$

where  $\delta_{LTP} = 0.225$ ,  $\delta_{LTD} = 0.465$ ,  $Ca\_conc$  is the calcium concentration in the cell which is thresholded,  $GR_i$  is 1 if the  $i^{th}$  GrC is firing and 0 otherwise,  $CF(T)$  is 1 if a climbing fibre input spike has arrived within the last  $T$  ms and 0 otherwise.

The cerebellum and associated regions are modelled as above (fig. 1) with two changes: the DCN is modelled as a single region with nodes having an excitatory connection to pons and an inhibitory connection to IO; a cortical region is added as a relay of DCN output to the pons; this route is given a delay time of  $\sim 100$  ms and with suitable recurrence in the cortical site its output can last for  $\sim 500$ ms. The numbers of neurones and synapses formed are given in table 1, there is a general topographic nature to the connections formed.

<i>Pre-synaptic cell (number of neurones)</i>	<i>Post-synaptic cell</i>	<i>Number of synapses (formed on post-synaptic cell)</i>	<i>Weight value</i>
GrC (100)	GoC	20	0.025.
	BK	10	0.12.
	PK	50	0.008.
GoC(50)	GrC	20	-0.00015.
BK (20)	PK	10	-0.00002.
PK (60)	DCN	10	-0.0008.
	GoC	10	-0.00004.
DCN (18)	PONS	10	0.0001.
	Cortex excitatory	10	0.03.
	Cortex inhibitory	5	0.03.
	IO	6	-0.00008.
PONS (30)	GoC	10	0.003.
	GrC	10	0.03.
	DCN	10	0.004.
IO (4)	PK	1	0.0001.
Cortex excitatory (30)	PONS	10	0.03.
	Cortex inhibitory	10	0.03.
Cortex inhibitory (10)	Cortex excitatory	10	-0.00015.

Table 1. Summary of connections. All weight values can vary by  $\pm 10\%$ .

The cerebellum is initially trained with a CS1 and US1 both being the word stem. These inputs last for 500ms and 50ms, respectively and co-terminate and so the timings and durations of the CS1 and US1 do not differ from that used in conditioned learning. The conditioned response (CR), that is the output of the cerebellum via the DCN, can re-enter the cerebellum via the cortical route and the direct route via the pons. This CR is then the new CS, termed CS2, for the learning of a stem – ending, with the new US (termed US2) being the associated ending. Since CS2 is not an externally driven signal we do not have fine control over its timing, hence the CS2 and US2 do not necessarily co-terminate. We do not wish the CS2 input to have terminated before the US2 input starts (since this would cause LTD of synapses based on resting rate GrC firing rates), so the US2 input is timed to occur about half way through the CS2 input. This obviously changes the 2 stages of learning described above, and we expect to see an early LTP due to CS2 input, followed by a period of LTD caused by activation of US2 whilst CS2 input is on going, finally a period of LTP when US2 has terminated and CS2 is still on going. We follow [2] in constraining the PONS to produce phasic and long-lasting responses to a CS. To produce the same

effect when the CR1 is re-entrant to the PONS via the cortical site we include both excitatory and inhibitory cells in the cortical module. Each US input only excites one of the four IO cells.

### Results

The two stages of learning of GrC to PK synapses are shown in fig 2: there is early LTP during the presentation of the CS due to the GrC firing rate, followed by LTD when the firing rate of the IO increases because of US input. The weight shown in fig. 2 changes quickly and after 3 presentations of the CS1 US1 pair the weight has reached zero value, where it will remain due to a lower weight cap of zero; this is the most dramatic weight change seen, and can be controlled by smaller values for:  $\delta_{LTP}$  and,  $\delta_{LTD}$ .

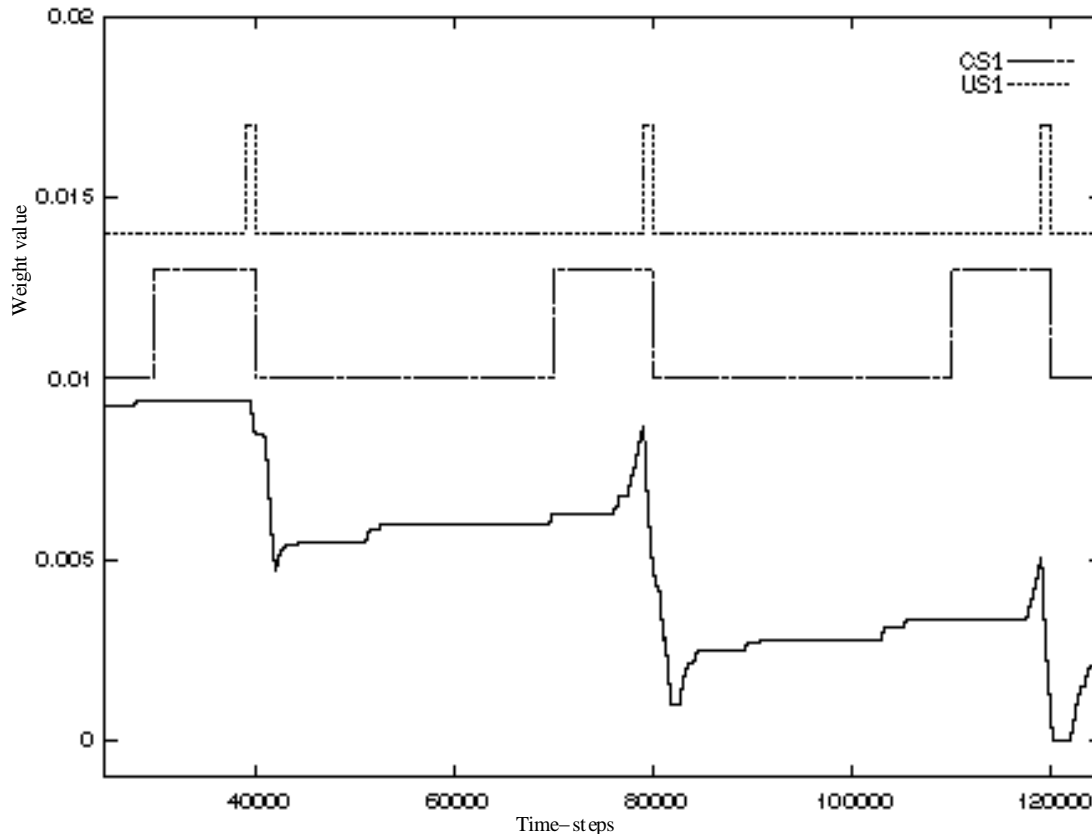


Figure 2. The weight changes of a GrC to PK synapse, and timings of CS1 and US1 input. 1S is 20000 time steps.

As training progresses the response of DCN neurones changes. Initially due to high PK firing rates the DCN firing rates are low, exhibiting a small burst when the US is presented. The US presentation causes an increase of IO complex spikes which leads to a pause in the PK potential, preventing firing and hence releasing the inhibitory action on the DCNs. Figure 3 shows the response of an untrained DCN neurone and the same neurone after training.

These results show the effects of training for the identity case, where both CS1 and US1 are the stem, and the resultant combined DCN output forms the CR1. With suitable weights from DCN to the cortical module for the CR1 and recurrent weights between the cortical excitatory neurones the cortical response is relatively weak until shortly before and during US1 presentation. This prevents the CR1 input to PONS via the cortical pathway overlapping with the ongoing input to the PONS of CS1.

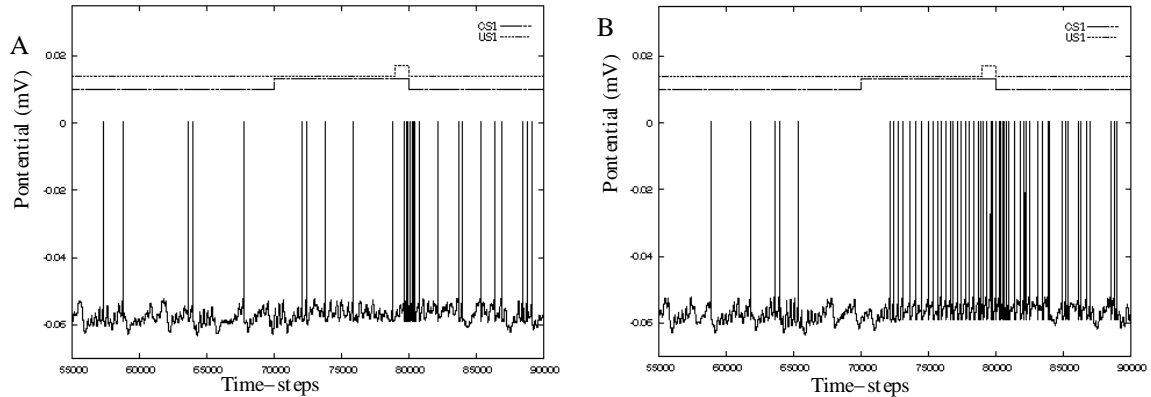


Figure 3. Spike train of a DCN neurone: (A) the untrained case shows a low firing rate comparable with rest during presentation of the CS1 alone, followed by a small burst with presentation of the US1; (B) The same neurone when trained shows an increased firing rate 125ms (2500 time-steps) after CS1 presentation with a growth in firing rate until presentation of the US1.

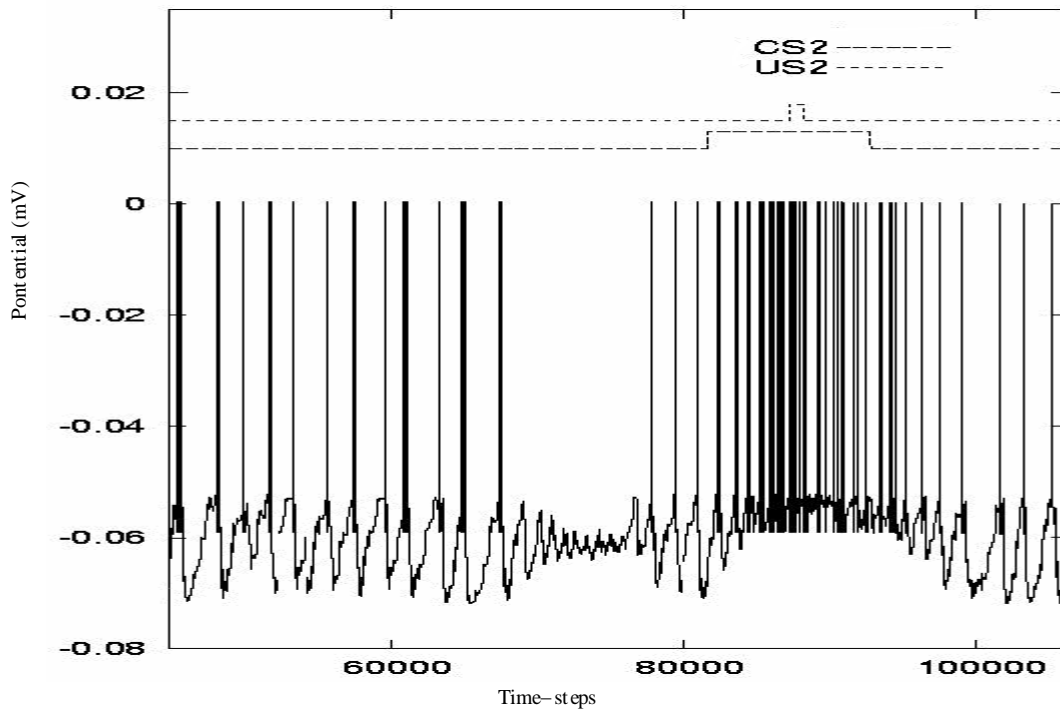


Figure 4. Potential of a GrC which is active during the presentation of CS2. This same neurone is off during the earlier presentation of CS1 (which occurs between 70000 and 80000 time-steps).

A number of different GrC groupings are formed during the complete presentation of CS1, US1, CS2 and US2, they can be on or off during both CS1 and/or CS2. Figure 4 shows a cell which is off during presentation of CS1 but is on during CS2, it also shows that the US2 occurs during CS2 and the 2 stimuli do not co-terminate.

As with the GrC the training process generates different forms of DCN response. Figure 5A indicates the firing rate of a DCN neurone which is only slightly responsive to CS1 but shows a strong response to the input of CS2. A single training trial is shown (figure 5B) indicating the 3 stages of learning that occur during the presentation of the CS2 US2 pair.

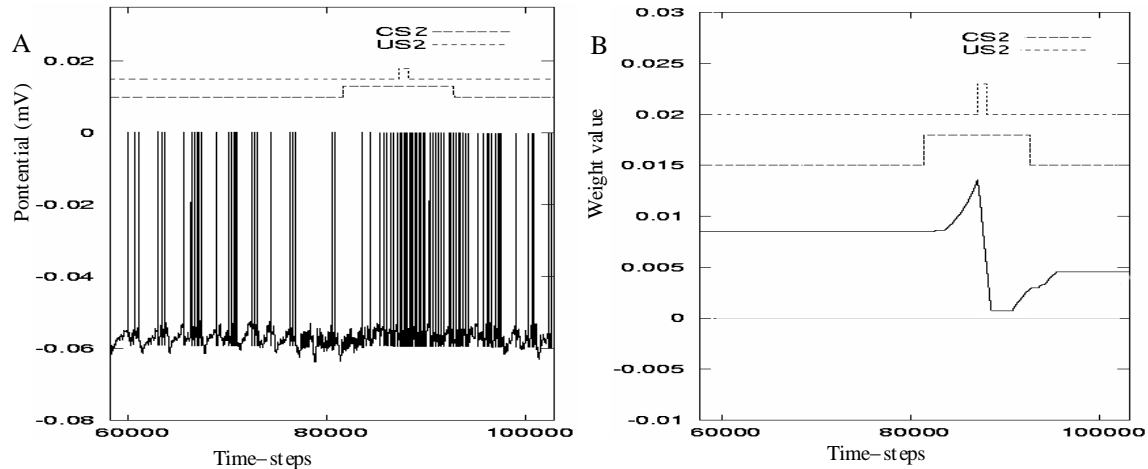


Figure 5. The potential of a DCN neurone (A) during presentation of the full CS1, US1, CS2 and US2 sequence. This neurone shows a slight increase in firing rate during presentation of CS1 and US1 but its largest change is during the presentation of CS2 and US2. (B) The weight change during presentation shows 3 stages of learning.

### Conclusions

We have applied the computational powers of the cerebellum to learn ending insertion in a stem–ending generation process. It was assumed that the correct stem–ending structure was stored elsewhere in the brain (such as in the hippocampus). This 'teacher' system was used as the US in a conditioned learning paradigm when the CS was only the stem, as was the CR. The feedback of the incorrect CR1 output from the DCN to the IO, when compared to the correct US of the stem–ending, led to learning of the ability to produce the full stem–ending sequence by the cerebellum, from the DCN as the CR1 and the CR2. This will be assumed to enable further learning of a prefrontal representation of the stem–ending, so that when primed from a suitable brain site, giving the context 'past' or 'present', there is priming of the corresponding prefrontal/BG/thalamus representation built up by cerebellum drive, but now able to be produced automatically. We propose to extend the system by addition of a hippocampus (already available in the NETSIM system at KCL) and the ACTION net (also developed as part of NETSIM) so as to develop a more complete syntactic analyser.

A question arises from the simulation about the topographic nature of the cerebellum which should be included. If the connectivity described in table I is random then a problem arises during learning. As the rate of LTD exceeds LTP (seen in figures 2 and 5B), during initial trials LTD is dominant and the total connectivity strength from GrC to PK decreases. As learning continues a number of the weight values will reach the minimum value of zero and be held there, but without a fixed upper limit to weight values LTP will continue. This situation occurs with a random weight connectivity in models with more than one IO neurone and leads to PK becoming excited once again during presentation of a CS preventing disinhibition of DCN nodes except when the US is presented. With a topographic arrangement through much of cerebellum this problem can be avoided. But then the question becomes: at which point in the structure is there cross–over in information? We have used a non–topographic connectivity between DCN neurones and the IO, and for DCN nodes to the cortical site; other alternatives need to be explored.

### References

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