VOR Adaptation on a Humanoid iCub Robot Using a Spiking Cerebellar Model

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Abstract—We embed a spiking cerebellar model within an adaptive real-time (RT) control loop that is able to operate a real robotic body (iCub) when performing different vestibulo-ocular reflex (VOR) tasks. The spiking neural network computation, including event- and time-driven neural dynamics, neural activity, and spike-timing dependent plasticity (STDP) mechanisms, leads to a nondeterministic computation time caused by the neural activity volleys encountered during cerebellar simulation. This nondeterministic computation time motivates the integration of an RT supervisor module that is able to ensure a well-orchestrated neural computation time and robot operation. Actually, our neurorobotic experimental setup (VOR) benefits from the biological sensory motor delay between the cerebellum and the body to buffer the computational overloads as well as providing flexibility in adjusting the neural computation time and RT operation. The RT supervisor module provides for incrementally countermeasures that dynamically slow down or speed up the cerebellar simulation by either halting the simulation or disabling certain neural computation features (i.e., STDP mechanisms, spike propagation, and neural updates) to cope with the RT constraints imposed by the real robot operation. This neurorobotic experimental setup is applied to different horizontal and vertical VOR adaptive tasks that are widely used by the neuroscience community to address cerebellar functioning. We aim to elucidate the manner in which the combination of the cerebellar neural substrate and the distributed plasticity shapes the cerebellar neural activity to mediate motor adaptation. This paper underlies the need for a two-stage learning process to facilitate VOR acquisition.

Index Terms—Cerebellar adaptation, neurorobotics, real-time (RT) control, spiking neural network, vestibulo-ocular reflex (VOR).

I. INTRODUCTION

DESCARTES’ famous “cogito ergo sum” back in the 17th century laid the foundations for creating the dualist body-mind humanistic understanding of human beings. This humanistic conception has resulted in a fruitful dualist tradition in which psychology and neuroscience are two essential partners alternating roles as allies or nemeses. Hence, it is common practice to draw a distinction between the body and the mind when it comes to cognition. Conversely, this mind-centered traditional conception of cognition is now matched by the concept of embodied cognition [1], which emphasizes the coexistence of cognition and body-function as a whole beyond a content-container relationship.

According to this embodied cognition concept, the main aim of the central nervous system (CNS) becomes now to solve and facilitate the interaction of the body with the environment. The cerebellum, a neural region underneath the brain whose primary function is to facilitate motor control, naturally fits into embodied neural simulations within a control loop.

The cerebellum is pivotal in contributing to coordination, precision, and accurate timing in voluntary and involuntary movements during body–environment interaction. It is traditionally tested in a behavioral task such as the vestibulo-ocular reflex (VOR) [2] due to the direct correlation between cerebellum and the reflex itself. The VOR is a reflexive eye movement controlled by the cerebellum that stabilizes the images on the retina during head rotations by producing opposite eye movements that maintain the image centered in the visual field (Fig. 1). The Purkinje cells (PCs), the sole output of the cerebellar, are connected via vestibular nuclei (VN) to the oculo-motor neurons that ultimately drive the eye muscles (Fig. 2). Consequently, most cerebellar disorders are echoed in the motion of the eyes and vice-versa. Cerebellar disorders directly affect the VOR response which makes VOR tests a powerful tool for cerebellar study and diagnosis [3]. Nevertheless, the observations of how all the elements in the cerebellum (neurons, synapses, plasticity mechanisms, etc.) play their roles and complement each other in these behavioral in vivo experiments are usually not direct and are difficult to perform, thus making the hypothesis-experimentation cycle extremely difficult. Computational modeling can partially overcome this limitation by bringing together different sciences, such as computational neuroscience, automation, or neurorobotics, and by providing easy access to all the elements modeled (neurons, synapses, plasticity mechanisms, etc.).

To that aim, here we have replicated a well-known embodied cognition setup largely studied in cerebellar neuroscience.
Fig. 1. \textit{r}-VOR experiment. VOR stabilizes the images on the fovea during horizontal and vertical head rotation tests by producing opposite eye movements that compensate the movement.

(a) Schematic of the cerebellar control loop [color encoded functional blocks are corresponding to elements in (b)]. (b) Cerebellar connectome diagram. Connections from semicircular canals in vestibular organ to oculomotor nucleus (OMN) via the flocculus in the cerebellum and the vestibular nuclei (VN), forming the three-neuron reflex arc (MF: mossy filters, GC: granular cells, PF: parallel fibers, PC: Purkinje cells, CF: climbing fibers, IO: inferior olive, GJ: gap-junction, AOS: accessory optic system, Exc: excitatory connections, and Inh: inhibitory connections).

(a synthetic VOR setup) that can help us describe and explain what the cerebellum does and does not do using this holistic view. This setup requires three key elements.

1) The VOR behavioral experiment setup such as our behavioral/cognitive task.
2) A cerebellar spiking model such as our neural structure responsible for facilitating the body interaction.
3) The humanoid iCub robot [4] such as the front-end human-like body.

Modeling and interconnecting each of these three elements leads us to face state-of-the-art challenges rarely addressed as a whole. The next points outline these challenges and how they have been addressed.

1) Our VOR behavioral protocol, a well-known standardized task amongst the neuroscientific community, helps us frame the dialogue between the cerebellar neural model and the humanoid front-end body in a well-defined experimental setup. Adopting this protocol facilitates the cross-validation between biological and humanoid VOR data.
2) Our cerebellar model operates as a feed-forward controller which integrates a variety of neuron models, neural characteristics, and a certain neural system topology that allow us to link bottom-up/top-down observations of the neural cues involved in the body–environment interaction.
3) Our front-end body mimics certain features of the human body (such as eye movements that can be compensated through a vestibular system during head movements). It is important to remark that the challenge here is to operate this robot using the cerebellar structure in real time (RT). This is a highly demanding task in terms of computational efficiency for medium scale neural systems.

Here, we present one of the most comprehensive embodied cognition setups. At the core of this experimental setup, we embed an RT spiking cerebellar model in a feed-forward control loop able to orchestrate the vestibulo-ocular adaptation of the humanoid eye movements mimicking a human being (or standard animal experimental setup).

II. MATERIAL AND METHODS

A. Behavioral Task: VOR

The VOR depends on the vestibular system, which detects both rotational and translational head movements through the stimulation of semicircular canals and otolithic organs [5]. The VOR nature is purely feed-forward since it induces prompt compensatory eye movements as a consequence of head movements (Fig. 2). The existing mismatches between the head movements (signaled by the vestibular organ) and the eye movements (driven by the cerebellar output) represent the sensory errors, which are called retinal slips. The forward adaptive control mediated by the cerebellum aims at minimizing these retinal slips [6].

B. Cerebellar Neural Network

The cerebellar neural network proposed in this paper consists of five neural subpopulations: 1) mossy fibers (MFs); 2) granule cells (GCs); 3) inferior olive (IO); 4) PCs; and 5) VN [see Fig. 2(b)]. This cerebellar model has been implemented in EDLUT [7]–[9], an open source event- and time-driven spiking neural simulator mainly oriented to RT embodiment experiments. The theoretical concepts underpinning our cerebellar model were developed in the previous study. Please see [9] and [10] for a review on spike-analog interfaces; [11]–[13] for a review on cerebellar learning; [14]–[16] for a review on cerebellar granular layer; and [17], [18] for a review on cerebellar control loops and neurorobotics.

The MFs convey the input sensory-motor signals from the eyes and vestibular organ to the cerebellar network. MFs project excitatory afferents onto GCs and VN. The IO cells sense the retinal slip (difference between head and eye velocity
movements) and propagate the teaching information through the climbing fibers (CFs) (i.e., IO cell’s axons). PCs receive the somatosensory activity from the parallel fibers (PFs) (i.e., GCs’ axons) and the teaching signal from the CFs (retinal slip). Finally, the VN close the cerebellar loop with excitatory synapses coming from MFs and IO cells, together with inhibitory synapses coming from PCs. The VN generate the cerebellar output activity, which arrives to the oculomotor neurons (OMNs) responsible for ultimately driving the eye movements.

1) MFs: Hundred MFs are modeled as input neurons able to propagate the sensory-motor information toward GCs and VN. The MF activity is generated by activations of sets of MF neurons following the sinusoidal shapes to encode head velocity movements [6], [19]–[21], consistently with the functional principles of VOR in cerebellar-control [21]. The total number of activated MFs during an r-VOR trial depends on the head velocity to be encoded, which also depends on the head rotation frequency and amplitude (each MF is sensitive to a small range of velocities).

2) GCs: Two thousand GCs are modeled as a state generator [22]–[25]. This layer transforms the sensorimotor neural activity coming from the MFs into somatosensory neural activity by generating spatio-temporal patterns that are repeatedly activated during each learning trial.

3) PCs: Two hundred PCs are modeled as a single compartment Hodgkin–Huxley (HH) model with five ionic currents (two groups of 100 cells each corresponding to agonist/antagonist muscles). This model is able to replicate the trimodal spike modes (tonic, silence, and bursting [26]) observed in PCs.

4) IO Cells and CFs: Two hundred IO cells modeled as leaky integrate and fire (LIF) neurons with electrical coupling (two groups of 100 cells each corresponding to agonist/antagonist muscles) conform the olivary system. Each IO cell, through its corresponding CF, makes contact with one PC and one VN cell. Additionally, IO cells are also electrically interconnected via gap-junctions (GJs). The external input activity of IO cells is generated with a probabilistic Poisson process. Given the normalized error signal \( \epsilon(t) \) and a random number \( \eta(t) \) between 0 and 1, an IO cell receives an input spike if \( \epsilon(t) > \eta(t) \) [17], [27]. These input stimuli together with the electrical coupling amongst IO cells generate the olivary system activity. Each single CF spike encodes well-timed information regarding the instantaneous error. The probabilistic spike sampling of the error ensures a proper representation of the whole error region over trials, whilst maintaining the CF activity between 1 and 10 Hz per fiber (similar to electrophysiological data [28]). The error evolution can be sampled accurately even at such a low frequency [17], [29].

5) VN Cells: Two hundred VN cells are modeled as LIF neurons (two groups of 100 cells each corresponding for agonist/antagonist muscles). Each VN cell is innervated by an inhibitory afferent from a PC and an excitatory afferent from the CF which simultaneously innervates the same PC. Each VN cell also receives excitatory projections from all MFs (which maintain the baseline VN activity). The spike activity of both VN agonist/antagonist groups is translated into an analog output signal (eye velocity) according to the following equation:

\[
\text{VN}_i(t) = \int_t^{t+T_{\text{step}}} \delta_{\text{VN}_{i}(t)} \cdot dt \tag{1}
\]

\[
\text{VN}_{\text{output}}(t) = \alpha \left( \sum_{i=1}^{N=100} \text{VN}_{\text{ag}}(t) - \sum_{j=1}^{N=100} \text{VN}_{\text{ant}}(t) \right) \tag{2}
\]

where \( \alpha \) is the kernel amplitude that normalizes the contribution of each VN cell spike to the cerebellar output correction (VN_{ag} output controls the agonist muscle whilst the VN_{ant} output controls the antagonist muscle). This neural topology is summarized in Table I.

### C. Spiking Neuron Models

Spiking neural networks, also referred as third generation of neural networks, are capable of communicating via spikes (i.e., discrete events occurring at certain instants in time) [30]. Spikes are the biological consequence of the interplay amongst several biological processes (ion channels activation and/or deactivation) whose dynamics are typically modeled through a set of differential equations. The dynamics of the neuron membrane potential is the biological process that ultimately governs the spike generation and propagation. More importantly, the final spike trains obtained allow for the processing of spatio-temporal data in the same event-driven manner than sensory data is acquired.

The LIF neuron model [31] constitutes the most prominent model accounting for spike generation and processing at minimal computational cost. Concretely, an LIF neuron elicits a spike once its membrane potential reaches a certain threshold and, immediately after, the LIF neuron potential is reset. When the membrane potential dynamics of the neuron model is driven by more than a single differential equation, other neuron models come into play (i.e., HH [32], Izhikevich [33],

### Table I

| Neurons | Synapses |
|---------|----------|
| Pre-synaptic cells | Post-synaptic cells | Number of synapses | Type | Initial weight (nS) | Weight range (nS) |
| 2000 GC | 200 PC | 400000 | AMPA | 4 | [0, 10] |
| 200 IO | 200 PC | 200 | AMPA | 40 | . |
| 100 MF | 200 VN | 20000 | AMPA | 0 | [0, 1] |
| 200 PC | 200 VN | 200 | GABA | 1.5 | . |
| 200 IO | 200 VN | 200 | AMPA | 1 | . |
| 10 to IO: 5x5 IO neuron squares connected radially from one corner of each 5x5 square to the other three corners | GJ | 3 | . |
generalized-LIF, etc.). Increasing numbers of differential equations entail increasing levels of computational cost. Hence, RT requires a tradeoff between computational cost and biological plausibility.

1) LIF Model (VN): It is implemented according to (3)–(9). Its neural dynamics is defined by its membrane potential and its excitatory and inhibitory conductances. It is equipped with excitatory (AMPA and NMDA) and inhibitory (GABA) chemical synapses

\[
\frac{dV}{dt} = I_{\text{internal}} + I_{\text{external}}
\]

(3)

\[I_{\text{internal}} = -gL \cdot (V + E_L)
\]

(4)

\[I_{\text{external}} = -(g_{\text{AMPA}}(t) + g_{\text{NMDA}}(t)) \cdot (V - E_{\text{AMPA}}) - g_{\text{GABA}}(t) \cdot (V - E_{\text{GABA}})
\]

(5)

\[g_{\text{AMPA}}(t) = g_{\text{AMPA}}(t_0) \cdot \exp\left(\frac{(t - t_0)}{\tau_{\text{AMPA}}}\right)
\]

(6)

\[g_{\text{NMDA}}(t) = g_{\text{NMDA}}(t_0) \cdot \exp\left(\frac{(t - t_0)}{\tau_{\text{NMDA}}}\right)
\]

(7)

\[g_{\text{GABA}}(t) = g_{\text{GABA}}(t_0) \cdot \exp\left(\frac{(t - t_0)}{\tau_{\text{GABA}}}\right)
\]

(8)

\[g_{\text{NMDA-INF}} = \frac{1}{1 + \exp(-62 \cdot V) \cdot 1.2^{\frac{Vg}{2}}}
\]

(9)

where \(C_m\) denotes the membrane capacitance, \(V\) denotes the membrane potential, \(I_{\text{internal}}\) denotes the internal currents, and \(I_{\text{external}}\) denotes the external currents. \(E_L\) is the resting potential and \(g_L\) is the conductance responsible for the passive decay term toward the resting potential. Conductances \(g_{\text{AMPA}}\), \(g_{\text{NMDA}}\), and \(g_{\text{GABA}}\) integrate all the contributions received by each receptor type (AMPA, NMDA, and GABA) through individual synapses. These conductances are defined as decaying exponential functions [7], [31] where the values are directly incremented proportionally to the synaptic weights \(w_i\) upon each presynaptic spike arrival (Dirac delta functions). \(g_{\text{NMDA-INF}}\) stands for the NMDA activation channel. Finally, when the membrane potential reaches a threshold \((V_{\text{thr}})\), it is then reset to \(E_L\) during the refractory period \((T_{\text{ref}})\). All the parameters are shown in Table II.

2) LIF Model Incorporating Electrical Coupling (IO): This is implemented as the previous LIF model without the NMDA chemical synapse, but accounting for the electrical synapse as indicated by the following equation:

\[I_{\text{external}} = -g_{\text{AMPA}}(t) \cdot (V - E_{\text{AMPA}}) - g_{\text{GABA}}(t) \cdot (V - E_{\text{GABA}}) - I_G\]

(10)

3) HH Single-Compartment Model (PC): This model is based on [36] and [37] and consists of a single compartment with five ionic currents and two excitatory (AMPA) and inhibitory (GABA) chemical synapses

\[
\frac{dV}{dt} = I_{\text{internal}} + I_{\text{external}}
\]

(12)

\[I_{\text{internal}} = -g_L \cdot n^4 \cdot (V + 95) - g_{Na} \cdot m_0 [V]^3 \cdot h \cdot (V + 50) - g_{Ca} \cdot c^2 \cdot (V - 125) - g_L \cdot (V + 70) - g_M \cdot M \cdot (V + 95)
\]

(13)

\[I_{\text{external}} = -g_{\text{AMPA}}(t) \cdot (V - E_{\text{AMPA}}) - g_{\text{GABA}}(t) \cdot (V - E_{\text{GABA}})
\]

(14)

\[g_{\text{AMPA}}(t) = g_{\text{AMPA}}(t_0) \cdot \exp\left(\frac{(t - t_0)}{\tau_{\text{AMPA}}}\right) + \sum_{i=1}^{N} \delta_{\text{AMPA}}(t_i) \cdot w_i
\]

(15)

\[g_{\text{GABA}}(t) = g_{\text{GABA}}(t_0) \cdot \exp\left(\frac{(t - t_0)}{\tau_{\text{GABA}}}\right) + \sum_{i=1}^{N} \delta_{\text{GABA}}(t_i) \cdot w_i
\]

(16)

where \(V\) denotes the membrane potential, \(I_{\text{internal}}\) denotes the internal currents, and \(I_{\text{external}}\) denotes the external currents. \(C_m\) is the membrane capacitance. Conductances \(g_{\text{AMPA}}\) and \(g_{\text{GABA}}\) integrate all the contributions received by each chemical receptor type (AMPA and GABA) through individual synapses. These conductances are defined as decaying exponential functions [7], [31] where the values are directly incremented proportionally to the synaptic weights \(w_i\) upon each presynaptic spike arrival (Dirac delta functions). \(g_{\text{NMDA-INF}}\) stands for the NMDA activation channel. Finally, when the membrane potential reaches a threshold \((V_{\text{thr}})\), it is then reset to \(E_L\) during the refractory period \((T_{\text{ref}})\). All the parameters are shown in Table II.

### TABLE II

**Neurons Model Parameters**

| Parameters | VN | IO | PC |
|------------|----|----|----|
| \(C_m\) (pF) | 2 | 10 | 7.16 |
| \(g_L\) (nS) | 0.2 | 0.15 | 0.15 |
| \(E_L\) (mV) | -70 | -70 | -70 |
| \(E_{\text{AMPA}}\) (mV) | 0 | 0 | 0 |
| \(E_{\text{GABA}}\) (mV) | -80 | -80 | -80 |
| \(\tau_{\text{AMPA}}\) (ms) | 0.5 | 1 | 1 |
| \(\tau_{\text{NMDA}}\) (ms) | 14 | 14 | 14 |
| \(\tau_{\text{GABA}}\) (ms) | 10 | 2 | 2 |
| \(V_{\text{th}}\) (mV) | -40 | -50 | -35 |
| \(V_{\text{peak}}\) (mV) | 1 | 1.35 | 1.35 |
| \(g_{\text{Na}}\) (mS) | 31 | 31 | 31 |
| \(g_{\text{K}}\) (mS) | 0.0075 | 5.65 | 5.65 |

**\(I_G\)**

\[I_G = \sum_{i=1}^{N} w_i \cdot (V - V_i) \cdot \left(0.6 \cdot \exp\left(-\frac{(V - V_i)^2}{50^2}\right) + 0.4\right)
\]

(11)

where \(I_G\) represents the total current injected through the GJ [34], \(w_i\) denotes the synaptic weight between the neuron \(i\) and the target neuron, \(V_i\) is the target neuron membrane potential, \(V_i\) is the \(i\) neuron membrane potential, and \(N\) is the total number of input GJ. For a correct operation of the electrical coupling, this model emulates the depolarization and hyperpolarization phases of an action potential after reaching the membrane potential threshold \((V_{\text{thr}})\) by enabling the generation of a triangular voltage function (with a maximum value \(V_{\text{peak}}\) and a minimum value \(E_L\)) during the refractory period \((T_{\text{ref}})\) [35]. All the parameters are shown in Table II.
incremented proportionally to the synaptic weights \((w_i)\) upon each presynaptic spike arrival (Dirac delta functions) Finally, \(g_k\) is a delayed rectifier potassium current, \(g_{Na}\) is a transient inactivating sodium current, \(g_{Ca}\) is a high-threshold non-inactivating calcium current, \(g_l\) is a leak current, and \(g_m\) is a muscarinic receptor suppressed potassium current.

The dynamics evolution of each gating variable \((n, h, c, \text{and } M)\) can be computed using the following differential equation:

\[
\dot{x} = \frac{x_0[V] - x}{\tau_i[V]} \tag{17}
\]

where \(x\) indicates the variables \(n, h, c, \text{and } M\). The implemented equilibrium function is determined by the term \(x_0[V]\) and time constant \(\tau_i[V]\) (Table III).

The sodium activation variable has been replaced and approximated by its equilibrium function \(m_0[V]\). The \(M\)-current presents a temporal evolution significantly slower than the rest of variables that allows the PC trimodal spike modes named burst, silence, and tonic. For the sake of computational efficiency, \(I_K\) \((-g_k \cdot n^4 \cdot (V + 95))\) and \(I_{Na}\) \((-g_{Na} \cdot m_0[V]^3 \cdot h \cdot (V - 50))\) currents, which control the spike shape, can be substituted by a simple threshold process \((V_{th})\) that triggers the generation of a triangular voltage function (with a maximum value \(V_{peak}\) and a minimum value \(E_L\)) each time the neuron fires [35]. This triangular voltage depolarization drives the state of ion channels similarly to the original voltage depolarization during the spike generation. The final internal current is

\[
I_{\text{internal}} = -g_{Ca} \cdot c^2 \cdot (V - 125) \tag{18}
- g_l \cdot (V + 70) - g_m \cdot M \cdot (V + 95).
\]

All the parameters are shown in Table II.

D. Synaptic Plasticity

The overall input–output function of the cerebellar network model is made adaptive through spike-timing dependent plasticity (STDP) mechanisms at different sites. These STDP mechanisms balance long-term potentiation (LTP) and long-term depression (LTD) (see [13] for an in-depth review of the implemented synaptic mechanisms).

1) \(PF–PC\) Synaptic Plasticity: The LTD/LTP balance at \(PF–PC\) synapses is based on

\[
\text{LTD} \Delta w_{PF_j–PC_i}(t) = \alpha \cdot \frac{I_{CF}^{\text{spike}}}{\tau_{\text{LTD}}} \cdot k \left( \frac{t - t_{CF}^{\text{spike}}}{\tau_{\text{LTD}}} \right) \cdot \delta_{PF_{\text{spike}}}(t) \tag{19}
\]

\[
\text{LTP} \Delta w_{PF_j–PC_i}(t) = \beta \cdot \delta_{PF_{\text{spike}}}(t) \tag{20}
\]

where \(\Delta w_{PF_j–PC_i}(t)\) denotes the weight change between the \(j\)th PF and the target \(i\)th PC; \(\tau_{\text{LTD}} = 100\) ms is the time constant that compensates the sensorimotor delay; \(\delta_{PF}\) is the Dirac delta function corresponding to an afferent spike from a PF; \(\alpha = -0.0304\) nS is the synaptic efficacy decrement; \(\beta = 0.0184\) nS is the synaptic efficacy increment; and the kernel function \(k(x)\) [13] is defined as

\[
k(x) = \exp(-x) \cdot \sin(x)^{10}. \tag{21}
\]

The STDP rule [13] defined by (19) produces a synaptic efficacy decrement (LTD) in PFs when a spike from the CF reaches the target PC neuron. The amount of synaptic decrement depends on the activity arrived through the PFs. This activity is convolved with the integrative kernel defined in (21) and multiplied by the synaptic decrement \(\alpha\). The effect on the presynaptic spikes arriving through PFs is maximal over the 100 ms time window before the postsynaptic CF spike arrival, thus accounting for the sensorimotor pathway delay [16]–[18], [38]. The amount of LTP at PF–PC synapses is fixed (20), with an increase in synaptic efficacy equals to \(\beta\) each time a spike arrives through a PF to the targeted PC.

This STDP mechanism correlates the activity patterns coming through the PFs to PCs with the teaching signals coming from CFs to PCs (producing LTD in the activated PF–PC synapses). The correlation process at PC level identifies certain PF activity patterns and consequently reduces the PC output activity. A diminution in PC activations causes a subsequent reduction on the PC inhibitory action over the target VN. Since the VN are driven by an almost constant MF activation, a lack of PC inhibitory action causes increasing levels of VN activation, which ultimately result in an error reduction during the following iterations.

2) \(MF–VN\) Synaptic Plasticity: The LTD/LTP dynamics at \(MF–VN\) synapses is based on

\[
\text{LTD} \Delta w_{MF_j–VN_i}(t) = \alpha \cdot \frac{I_{CF}^{\text{spike}}}{\tau_{\text{LTD}}} \cdot k \left( \frac{t - t_{CF}^{\text{spike}}}{\sigma_{MF–VN}} \right) \cdot \delta_{MF_{\text{spike}}}(t) \tag{22}
\]

\[
\text{LTP} \Delta w_{MF_j–VN_i}(t) = \beta \cdot \delta_{MF_{\text{spike}}}(t) \tag{23}
\]

with \(\Delta w_{MF_j–VN_i}(t)\) denoting the weight change between the \(j\)th MF and the target \(i\)th VN; \(\sigma_{MF–VN} = 5\) ms standing for the time width of the kernel; \(\delta_{MF}\) representing the Dirac delta function that defines an MF spike; \(\alpha = -0.002048\) nS

| Cond. type | Steady-state Activation/Inactivation | Time constant (ms) |
|------------|------------------------------------|--------------------|
| \(g_k\)   | \(x_0[V] = \frac{1}{1 + \exp((-V + 25) \cdot 0.1)}\) | \(\tau_i[V] = 0.25 + 4.35 \cdot \exp\left(\frac{100}{100}\right); \text{if } V \leq -10\) |
| \(g_{Na}\) | \(x_0[V] = \frac{1}{1 + \exp((-V + 25) \cdot 0.1)}\) | \(\tau_i[V] = 1.15 + \frac{1}{1 + \exp((35) \cdot 0.1)}\) |
| \(m_0[V]\) | \(m_0[V] = \frac{1}{1 + \exp(-\frac{V - 10}{2})}\) |                        |

| Forward Rate Function (\(\alpha\)) | Backward Rate Function (\(\beta\)) |
|------------------------------------|-----------------------------------|
| \(g_{Ca}\) | \(\alpha = 1.6\) | \(\beta = \frac{0.02(V + 8.9)}{\exp\left(\frac{V_{peak} - 5}{5}\right)} - 1\) |
| \(g_m\) | \(\alpha = 0.3\) | \(\beta = 0.001 \cdot \exp\left(\frac{-V - 70}{18}\right)\) |

\begin{table}[h]
\centering
\caption{IONIC CONDUCTANCE KINETIC PARAMETERS OF THE HH MODEL}
\begin{tabular}{|c|c|c|}
\hline
\multicolumn{1}{|c|}{Cond. type} & \multicolumn{1}{|c|}{Steady-state Activation/Inactivation} & \multicolumn{1}{|c|}{Time constant (ms)} \\
\hline
\(g_k\) & \(x_0[V] = \frac{1}{1 + \exp((-V + 25) \cdot 0.1)}\) & \(\tau_i[V] = 0.25 + 4.35 \cdot \exp\left(\frac{100}{100}\right); \text{if } V \leq -10\) \\
\(g_{Na}\) & \(x_0[V] = \frac{1}{1 + \exp((-V + 25) \cdot 0.1)}\) & \(\tau_i[V] = 1.15 + \frac{1}{1 + \exp((35) \cdot 0.1)}\) \\
\(m_0[V]\) & \(m_0[V] = \frac{1}{1 + \exp(-\frac{V - 10}{2})}\) & \\
\hline
\end{tabular}
\end{table}
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is the synaptic efficacy decrement; \( \beta = 0.000792 \) nS is the synaptic efficacy increment; and the integrative kernel function \( k(x) \) [13] is defined as

\[
k(x) = \exp(-|x|) \cdot \cos(x)^2. \tag{24}
\]

The STDP rule defined by (22) produces a synaptic efficacy decrease (LTD) when a spike from the PC reaches the targeted VN neuron. The amount of synaptic decrement depends on the activity arrived through the MFs. This activity is convolved with the integrative kernel defined in (24) and multiplied by the synaptic decrement \( \alpha \). This LTD mechanism considers those presynaptic/postsynaptic MF spikes that arrive before/after the postsynaptic/presynaptic PC spike arrival within the time window defined by the kernel (\( \sigma_{MF-VN} = 5 \) ms). The amount of LTD at MF–VN synapses is fixed, with an increase in synaptic efficacy equals to \( \beta \) each time a spike arrives through an MF to the targeted VN.

This complementary STDP mechanism correlates the activity patterns coming through the MFs to the VN with the teaching signals coming from the PCs to the VN (producing LTD in the activated MF–VN synapses). The correlation process at VN level identifies certain MF activity patterns and consequently increases the output activity, which ultimately increases the cerebellar response. This second STDP mechanism accounts for the learning consolidation in the deepest structures of the cerebellar (VN) by using the PC output activity, which is shaped by the first STDP mechanism, as a teaching signal.

E. VOR Plant (Modeling the Mechanical Circuitry)

The cerebellum operates as a biological feed-forward controller within a control loop. The cerebellar output is meant to drive adaptation from the VN through a set of motor neurons, nerve fibers, and muscles finally to the eye. This VOR mechanical pathway is modeled (within EDLUT) as a VOR mechanical circuitry which is identified as a continuous-time mathematical model with two poles

\[
e(kT), E(s) : \text{eye motion (output)} \tag{25}
\]

\[
h(kT), H(s) : \text{head motion (input)} \tag{26}
\]

\[
\frac{E(s)}{H(s)} = \frac{K TC1 s}{(TC1 s + 1) \cdot (TC2 s + 1)} \cdot \exp(-st delay). \tag{27}
\]

There are four parameters in the model: \( Q = [K, TC1, TC2, t_{delay}] \). The delay parameter \( t_{delay} \) captures the delay that exists in communicating the signals from the inner ear to the brain and eyes. Based on the number of synapses involved in the VOR, this delay is estimated to be around 5 ms [39], [40]. The gain parameter \( K \) models the fact that the eyes do not perfectly cope with the head movement. This parameter is assumed to be between 0.6 and 1 [39], [40]. The \( TC1 \) parameter represents the dynamics associated with the semicircular canals as well as some additional neural processing. The canals are high-pass filters, because after a subject has been put into rotational motion, the neural active membranes in the canals slowly relax back to resting position, so the canals stop sensing motion. Based on the mechanical characteristics of the canals, combined with additional neural processing which prolongs this time constant to improve the accuracy of the VOR, the \( TC1 \) parameter is assumed to be between 10 and 30 s [39], [40]. Finally, the \( TC2 \) parameter captures the oculomotor plant dynamics, that is, the eye, muscles, and tissues attached to it. The \( TC2 \) parameter is assumed to be between 0.005 and 0.05 s.

The temporal response for the VOR transfer function requires calculating the inverse Laplace transform, thus obtaining (28) and (29) (note that the delay is modeled and inserted within the control loop)

\[
\begin{bmatrix}
x_1 \\
x_2
\end{bmatrix} =
\begin{bmatrix}
0 & 1 \\
-a_0 & -a_1
\end{bmatrix}
\begin{bmatrix}
x_1 \\
x_2
\end{bmatrix} +
\begin{bmatrix}
0 \\
h(t)
\end{bmatrix} \tag{28}
\]

\[
y = [b_0 \ b_1] \cdot
\begin{bmatrix}
x_1 \\
x_2
\end{bmatrix} \tag{29}
\]

where

\[
a_0 = \frac{1}{TC1 \cdot TC2}; a_1 = \frac{(TC1 \cdot TC2)}{TC1 \cdot TC2}; b_0 = 0; b_1 = \frac{K \cdot TC1}{TC1 \cdot TC2}. \tag{30}
\]

The VOR plant model parameters are adjusted using a genetic algorithm to fit experimental and clinical observations [39]–[41]. The resulting parameters values are \( k = 1.0, TC1 = 15, \) and \( TC2 = 0.05 \).

F. iCub Robot

The humanoid iCub robot, used as front-end body, can sense its own body position (proprioception) and movement (using accelerometers and gyroscopes) [4]. We control the simulated (in Gazebo [42]) and actual iCub robot using YARP: yet another robot platform [43]. This API enables the sending of motor commands and the receiving of sensory information from the robot.

The VOR protocols implemented in this paper only require moving the iCub head (controlled by the neck) and the eyes. Both neck and eyes consist of a serial chain of rotations with 3 degrees of freedom. Additionally, the eyes incorporate a camera that can be used to check the image motion on the “retinas.”

Prior to the real iCub robot implementation, we have validated and calibrated the VOR protocols using the virtual iCub version.

G. Control Loop

The sensory-motor information needs to flow between the humanoid robot and the cerebellar neural network causing effects on one another. Fig. 3 shows how this interaction is managed by two key elements: 1) the inner/outer control loop (including the cerebellar model and the VOR plant implemented in EDLUT) and 2) the robot interface (using YARP to connect with the robot). Both elements have been independently designed and interconnected via TCP/IP. This modular structure presents two main advantages.

1) The control loop may operate any humanoid robot as long as the robot interface program is adjusted.
The RT supervisor manages this temporal difference to ensure that the neural computational load fluctuations encountered during simulation due to volleys of neural activity can be coped with an RT execution.

H. Real-Time Supervisor

The EDLUT simulator incorporates a hybrid event- and time-driven simulation scheme [7], [8], [45]. EDLUT takes full advantage of parallel processing (in CPU and GPU) for neural layers with high levels of neural activity adopting a time-driven simulation scheme (i.e., the PC layer). EDLUT also takes full advantage of parallel processing (in CPU) for neural layers with sparse neural activity adopting an event-driven simulation scheme (i.e., the GC layer). This hybrid simulation scheme significantly optimizes the simulation speed although it remains inherently nondeterministic due to the volleys of neural activity encountered. This nondeterministic behavior demands the development and integration of an RT supervisor.

The RT supervisor, developed here for EDLUT, ensures that the ST of the neural activity ($ST_{net}$) copes with the iCub RT internal clock and does not surpass the temporal difference between the biological and artificial pathway delays of 85 ms (33). The RT supervisor deploys a set of gradual countermeasures depending on the time distance between $ST_{net}$ and RT, thus affecting the neural computation to a lesser or greater degree (Table IV). The smaller the time distance between $ST_{net}$ and RT, the higher the contingency level and the more drastic the countermeasures are.

During VOR tests, the RT countermeasures taken are usually minimal (0-1 contingency levels). However, under occasional large neural computational loads, the countermeasures taken may range from 2 to 4. Under these conditions, EDLUT disengages some neural computation elements, thus causing a slight degradation over the final cerebellar outcome. A permanent contingency level value between 2 and 4 means that EDLUT does not meet the neural dynamic computation requirements. The outcome, therefore, would drastically differ from what was expected. We monitor these countermeasure levels during all the simulations to validate the obtained results.
TABLE IV

| Cont. level | Contingency tasks |
|------------|-------------------|
| 0          | The time distance between ST<sub>net</sub> and RT is too close to 85 ms. The neural simulation has to be halted. |
| 1          | Standard simulation. No countermeasures are needed. |
| 2          | The time distance between ST<sub>net</sub> and RT is near zero. Learning rules are disengaged to speed-up the neural simulation. |
| 3          | The time distance between ST<sub>net</sub> and RT is even closer to zero. Spikes propagation and neuron model updates are also disengaged to further speed-up the neural simulation. |
| 4          | The time distance between ST<sub>net</sub> and RT is too close to zero. All the non-vital neural dynamic computation is disengaged (i.e., internal spike generation, periodic weight saving operation, etc.). |

I. Experimental Protocol for Cerebellar Validation

We adopt the rotational VOR (r-VOR) test as the experimental protocol used in validating our cerebellar model. r-VOR protocol requires the subject under study (monkey, rabbit, rat, etc.) to be placed on a rotatory chair or table which is able to oscillate at different frequencies and velocities. The subject’s head is restrained, so chair/table and head velocities are regarded as equals. During normal VOR adaptation, the eyes are to follow a stationary visual target thus minimizing the retinal slip. Eye velocity responses are then compared to head velocity movements using VOR gain and phase as markers. The lag between head and eye velocity is called the VOR phase (in degrees), whereas the amplitude ratio of eye and head velocities is called the VOR gain (nondimensional). VOR gain is close to 1 and VOR phase is close to 180° for natural head rotation frequencies [46], [47].

r-VOR test is emulated in the robotic platform by driving a direct robot head rotation whilst the cerebellum, via robot eye movements, compensates for the head rotation. Our cerebellar model is tested in an r-VOR test with incremental rotational frequencies (from 0.5 to 5 Hz) to cross-validate VOR gain-and-phase iCub-data against VOR gain-and-phase monkey-data [46]. Fig. 4 depicts ideal [47], experimental [46], and computational VOR data superimposed. By comparing the three sets of data, it is clearly verifiable that the computational model is able to cope with experimental results keeping the overall VOR performance close to the ideal (i.e., VOR gain = 1 and phase = 180°).

III. Results

Once the cerebellar model is validated (see Fig. 4), the r-VOR behavioral tasks here proposed as a test-bed consists of a robotic r-VOR at 1-Hz frequency and 150 deg/s of velocity amplitude in the horizontal plane. Later on, this r-VOR test at 1 Hz is extended in velocity amplitude ([30, 90 150] and [30, 60, 90] deg/s for horizontal and vertical planes, respectively). This test requires at least 300 trials, 1 s per trial in RT, to converge and compensate the head movement.

A. Cerebellar Adaptation Process

The STDP mechanisms located at PF–PC and MF–VN afferents (see Section II) modulate the cerebellar output response during r-VOR adaptation. Fig. 5 depicts how the cerebellar neural activity evolves during synaptic adaptation (Fig. 6). The cerebellar input signals from the vestibular organ [Fig. 5(left column)] remains unchanged, as the horizontal head rotation movement during the adaptation process. This sensory input activity propagated by the MFs is transformed...
into a sparse neural coding at GCs, which represents univocally the passage of the time by a set of spatio-temporal neural patterns repeatedly activated during each learning trial [Fig. 5(left column)]. The PF–PC STDP mechanism correlates the GC neural activity (propagated by the PFs) with the teaching signal (error signal) sensed by the CFs by modifying the synaptic weights at this site [Fig. 6(a) and (b)]. Once the VOR adaptation is accomplished at PF–PC synapses, it is then transferred (in counter phase due to the inhibitory nature of PC axons) and consolidated in deepest cerebellar structures (MF–VN) [Fig. 6(c) and (d)], consistent with the two learning stages hypothesis proposed by Masuda and Amari [48] (see [13] for an in-depth review).

At the beginning of the learning process, the cerebellum starts with a blank sheet and adapts PF–PC (4 nS) and MF–VN (0 nS) synaptic weights from scratch [Fig. 6(a) and (c)]. CF activations are maximal (10 Hz corresponding to the maximal error sensed) [Fig. 5(a), central column] and the cerebellar output is negligible since the adaptation process is not yet deployed [Fig. 5(a), right column]; the eyes are moving conjointly with the head.

At the end of the learning process, the cerebellar output [Fig. 5(b), right column] fully compensates for the head movement thanks to the adaptation process deployed [Fig. 6(b) and (d)]. The resulting CF activations are minimal (1–2 Hz corresponding to the CF baseline activation in the absence of error sensed) [Fig. 5(b), central column]. The zenith view of the synaptic weight distribution at PF–PC and MF–VN synapses depict the footprints that the STDPs generate; footprints that are the photograph negative of one another [Fig. 6(b) and (d)]. Fig. 6(b) and (d) also shows two differentiated areas representing the final agonist/antagonist microzone balance.

Finally, Fig. 7 shows, in the left-hand column, the mean absolute error (MAE) evolution between movements of the head and eyes during the \( r \)-VOR test with three different amplitudes per plane. This measure helps us to evaluate \( r \)-VOR accuracy during the 300 trials (300 s) needed for fully deploying cerebellar learning. MAE is always maximal at the beginning of the learning process (during the first trials), when no cerebellar adaptation is deployed yet.
The MAE progressively decreases as the cerebellar adaptation takes over. Once the synaptic weight distributions at PFs–PCs and MFs–VN are settled and stabilized, the MAE converges to its minimal; the larger the head velocity amplitude to compensate, the greater the cerebellar compensatory output and the time to obtain the optimal synaptic weight distribution are (the MAE requires more time to converge).

Fig. 7 also shows, in the right-hand column, the velocity curves obtained for the head and eyes during the last trial of each r-VOR task. Both head and eyes curves are similar in amplitude (VOR gain close to 1) but in counter-phase (VOR phase close to 180°). These results are consistent with empirical observations [46].

### B. Meeting RT Requirements and RT Supervisor Process

The RT supervisor implemented within the inner/outer control loop is able to control the ST speed of the cerebellar neural activity to meet the RT bounds (see Section II), thus enabling the sensory-motor information to flow in both ways (cerebellar model to iCub robot and vice versa). Fig. 8(a) depicts the temporal evolution of the cerebellar ST speed (ratio between execution time and ST) corresponding to two horizontal r-VOR tasks (150 deg/s) with and without the RT supervisor engaged. This comparison has been performed using the simulated iCub robot.

The cerebellar simulation time (ST\textsubscript{net}) without the RT supervisor engaged (no RT version) overpasses the RT bounds of (33), making a coherent sensory-motor information propagation in the real robot impossible [Fig. 8(a) and (b)]. In contrast, the cerebellar ST with the RT supervisor engaged (RT version) is slowed down or speeded-up to cope with the RT bounds of (33), making possible a coherent sensory-motor information propagation in the real robot [Fig. 8(a) and (c)]. The RT supervisor, using the RT contingency levels described in Table IV, ensures that the time distance between the ST of the cerebellar neural activity (ST\textsubscript{net}) and RT is always between the bounds required for the robot communication. When this time distance is close to the upper RT bound [Fig. 8(c)], the contingency level switch to 0 [Fig. 8(d)], halting the cerebellar simulation. On the contrary, when the distance is close to the lower RT bound [Fig. 8(c)], the contingency level takes values of between 2 and 4 [Fig. 8(d)], progressively disengaging different neural elements to speed-up the cerebellar simulation.

Table V shows the time spent at each contingency level in our RT simulation. The time spent at contingency levels 0 and 1 is 98.69% of the total ST (standard simulation without degradation). The time spent at contingency level 2 is 1.2693%. Level 2 involves a slight degradation in the neural computation caused by the disengagement of the STDP mechanisms (the learning process is delayed). Finally, the time spent at contingency levels 3 and 4 is 0.0407%. Levels 3 and 4 involve a larger degradation in the neural computation due to the disengagement of critical neural elements, that is, spike generation and propagation.

To measure the neural degradation impact on the RT simulation, we calculate the mean and standard deviation of the difference between the synaptic weight distributions at PF–PC and MF–VN obtained for the RT and no RT simulation. These values are $-0.0059 \pm 0.0523$ nS for PF–PC synapses and $0.00003 \pm 0.0776$ nS for MF–VN synapses. We also calculate the mean and standard deviation of the difference between MAE evolutions of the cerebellar output response for the RT and no RT simulation, thus obtaining $0.1165 \pm 0.3293$ deg/s. These deviations are negligible, which makes the RT supervisor impact in the neural outcome minimal.

### C. Robotic r-VOR Cerebellar Adaptation (Proof of Concept)

Two movies are included to visually verify the entire adaptation of the reflex as supplementary material. Movie S1 shows the evolution of cerebellar adaptation process in a simulated iCub robot whereas movie S2 shows the real iCub robot. Each movie includes the six r-VOR tasks proposed (three in the horizontal plane and three in the vertical plane) and compares the cerebellar initial learning stages with the final stages. Fig. 9 shows a snapshot of both movies for the 150 deg/s
Fig. 9. Snapshot of the movies filming the r-VOR in the (a) simulated and (b) real iCub robot. The left-hand windows represent the initial learning stage whereas the right-hand windows represent the final learning stage. The upper windows in (a) and (b) show the head and eyes movements whereas the lower windows show the images filmed by the eye cameras. The optical flow is computed over the camera images (superimposed in green arrows) indicating quantitatively the level of stabilization of the filmed image on the retina.

IV. DISCUSSION

Different artificial intelligence VOR solutions in robotic platforms have been proposed during the last decade to try to give a better insight into the computational primitives underneath our CNS. These solutions are organized into two families according to their biological plausibility: 1) machine learning and 2) the cerebellar-based family.

A. Machine Learning Family

The embodied cognition approach (VOR) is solved without devoting attention to the biological restrictions imposed by the neural structures within the nervous system. The algorithms mediating the cerebellar role operation are claimed to be inspired in either the cerebellar architecture or the cerebellar functionality or both. However, the parallelisms to be drawn between the cerebellar operation/architecture and the algorithms proposed are generally constrained to a general overview of the cerebellar adaptive mechanisms (they are biologically inspired but not biologically plausible). Thus, biology is only taken into consideration at a very high level of abstraction. The solutions provided are usually purely speculative and difficult to refute/validate from a cellular/neural network point of view. These solutions aim at obtaining performance in the robotic VOR task itself rather than understanding the biological involvements. The most prominent examples found in this machine learning family are as follows.

1) Learning systems derived from the biologically inspired principle of feedback-error learning (FEL) [49] combined with nonparametric statistical learning networks [50]. FEL approximately maps the sensory error into motor error. The motor error is subsequently used to train a neural network through supervised learning by means of a recursive least squares (RLSs) algorithm based on a Newton-like method. RLS facilitates a very fast convergence and robustness without the need for costly parameter adjustments. This system is able to acquire a high performance visual stabilization reflex in a humanoid robot but the biological plausibility is lacking.

2) Learning systems based on adaptive linear filters as cerebellar controllers. The Marr–Albus theory commonly assumes the teaching signal (from CFs) as the motor error. This assumption demands complex neural structures that are able to estimate nonobservable motor errors from their observable sensory consequences. To that aim, a recurrent control architecture with a controller that decorrelates the sensory error from the motor error is used [51]. These learning systems assume the cerebellum operating like a bank of adaptive linear filters supervised by the CF activity [52].

3) Learning systems based on local weight projection regressions (LWPRs) [53] as cerebellar controllers. LWPR is a nonlinear function approximator that operates in high-dimensional spaces. This algorithm is able to cope with redundant dimensions and irrelevant inputs. These learning systems use a cerebellar model in which the granular and molecular layers (also including the interneurons [54]) are modeled using this LWPR algorithm [55], [56]. The input to the PCs is the output of the LWPR algorithm. This cerebellar model has been used to create a gaze stabilization system in [57].
B. Cerebellar-Based Family

The cerebellar-based family solves the embodied cognition approach (VOR) by taking the biological restrictions imposed by the cerebellar neural structures as granted. The cerebellar algorithm performance is a consequence of the built-in biologically plausible integrated characteristics, not the main target. The cerebellar algorithms are biologically constrained and they share a family resemblance with the cerebellar anatomy (they aim to be biologically inspired and biologically plausible). The solutions provided give us a closer and clearer view of the cerebellar computation primitives. The main aim here is to draw humanoid-human analogies that may drive basic cerebellar computation primitives. The main aim here is to aim to be biologically inspired and biologically plausible). The biologically plausible integrated characteristics, not the main target. The algorithm performance is a consequence of the built-in biological plausibility (assuming rate coding at cell-level representation). They are usually easier to implement and more computationally efficient at the expense of being less biologically plausible. These kinds of cerebellar models have been used to recreate an eye blink classic conditioning [58] and a VOR experiment [59]. RT requirements here are easy to cope with due to the simplicity and efficiency of the analog cerebellar model.

1) Analog Cerebellar Models: These models usually present higher abstraction levels than spiking models (assuming rate coding at cell-level representation). They are usually easier to implement and more computationally efficient at the expense of being less biologically plausible. These kinds of cerebellar models have been used to recreate an eye blink classic conditioning [58] and a VOR experiment [59]. RT requirements here are easy to cope with due to the simplicity and efficiency of the analog cerebellar model.

2) Spiking Cerebellar Models: These models are more akin to biology. They try to mimic the cerebellar neural communication by using spikes (thus, even spatio-temporal spiking representations and STDP mechanisms can be studied). Spikes are propagated within cerebellar subcircuits that attempt to mimic the cerebellar architecture. Interestingly, the emerging behavior from the dialogue between the neural code and the different cerebellar subcircuits is intended to cope with the behaviors observed in biology. These spiking models can be designed using the results obtained in experimental neuroscience to increase their biological plausibility. These complex models can then be used conjointly with experimental neuroscience to easily refute/validate new hypotheses that could hardly be studied just by experimental neuroscience due to its inherent technical limitations. Nevertheless, conciliating realistic spiking cerebellar models with behavioral outcomes (i.e., VOR) remains an open issue. Computational models that partly address this problem exist (i.e., modeling and interconnecting certain subcircuits [60] or certain spiking features [61]). Nevertheless, reconstructing the path from cellular to behavior level remains elusive. To the best of our knowledge, the solution proposed in this paper is one of the first initiatives that is able to combine this level of neural detail with several neural adaptive mechanisms all working together to operate a humanoid performing a VOR experiment in RT. In this case, the RT requirements are harder to cope with due to the higher complexity of the spiking cerebellar model.

V. Conclusion

In this paper, we present one of the first cerebellar embodiment case-of-studies able to effectively reproduce r-VOR tasks with a real humanoid robot in RT. The spiking cerebellar model/controller effectively adapts the reflex for a real iCub robot thanks to the two STDP mechanisms located at the PF–PC and MF–VN synapses. Both STDP mechanisms operate conjointly to shape the cerebellar neural activity that ultimately generates the eye motor commands that compensate for the head movement in the iCub robot.

This case-of-study incorporates two key elements, for the first time in cerebellar embodiment, which are pivotal in establishing a coherent communication between the cerebellar controller/model and the front-end body (iCub) in RT: 1) an inner/outer control loop and 2) an RT supervisor. These two elements solve the body-mind dialog technical problem in RT thus ensuring a proper timing between the spiking cerebellar commands generated and their corresponding motor actions/sensory responses.

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