Exploiting Memristance for Implementing Spike-Time-Dependent-Plasticity in Neuromorphic Nanotechnology Systems

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Abstract

In this paper we show that STDP can be implemented using a crossbar memristive array combined with neurons that asynchronously generate spikes of a given shape. An attenuated version of such spikes needs to be sent back through the neurons input terminal. The shape of the spikes turns out to be very similar to the neural spikes observed in biology for real neurons. The STDP learning function obtained by combining such neurons with memristors is exactly that of the STDP learning function obtained from neurophysiological experiments on real synapses.

I. INTRODUCTION

As nanotechnology develops, it is becoming apparent that devices and interconnects will suffer much stronger variability and reliability issues than traditional CMOS. An option to overcome this, which is gaining consensus, is to adopt neuromorphic principles mimicking biological neural structures. Biological neural systems are made of unreliable devices (neurons) interconnected through synapses, but they overcome their limitations through learning and adaptation. The original and well established learning mechanism discovered in 1949 by Hebb [12] has been elaborated during the last decade into what has been called Spike-time-Dependent-Plasticity (STDP) [5]-[11]. STDP is a learning rule for spiking neurons where the precise relative timing among individual spikes coming from different neurons determines the evolution of learning. STDP was originally postulated as a computer learning algorithm [5], and is being used by the machine intelligence and computational neuroscience community [8]-[11]. At the same time its biological and physiological foundations have been reasonably well established during the past decade [13]-[20]. If memristance and STDP can be related new neuromorphic-like computers built out of nanotechnology memristive devices could incorporate the biological STDP mechanisms yielding a new generation of self-adaptive ultra-high-dense intelligent machines. We recently showed [27] that by combining memristance models with the electrical wave signals of neural impulses (spikes) converging from pre- and post-synaptic neurons into a synaptic junction, STDP behavior emerges naturally. This result serves to understand how neural and memristance parameters modulate STDP and provides a direct mean to incorporate STDP learning mechanisms into a new generation of nanotechnology computers employing memristors.

II. MEMRISTANCE

Memristance was postulated in 1971 by Chua [4] based on circuit theoretical reasoning's. Fig. 1 shows the basic two-terminal devices relating the fundamental magnitudes *i, v, q,* and φ (with $\dot{q} = i$, $\dot{\varphi} = v$). The memristor relates $d\varphi$ and dq through the device's memristance *M*. If *M* is constant, the memristor degenerates into a linear resistor. In general, a memristor can be current-controlled, in which case it would be described as [4]

$$v_{MR} = R(w, i_{MR}, t) i_{MR} \qquad (1)$$
$$\dot{w} = f(w, i_{MR}, t)$$

or, voltage-controlled, being described as [4]

$$i_{MR} = G(w, v_{MR}, t) v_{MR} \qquad (2)$$
$$\dot{w} = f(w, v_{MR}, t)$$

where *w* represents the state (memory) of the memristor, i_{MR} is the current through the device, and v_{MR} the voltage drop across it.

Memristance has been recently demonstrated in nanoscale two-terminal devices, such as certain titanium-dioxide [1]-[2] and amorphous Silicon [3] cross-point switches. Memristance arises naturally in nanoscale devices because small voltages can yield enormous electric fields that produce the motion of charged atomic or molecular species changing structural properties of a device (such as its conductance) while it operates. In this paper we will restrict our discussion to voltage-controlled memristors of the form

$$i_{MR} = G(w, v_{MR}) v_{MR}$$
$$\dot{w} = f(v_{MR}) \qquad (3)$$

In memristive nanoscale devices, function *f* may describe ionic drift

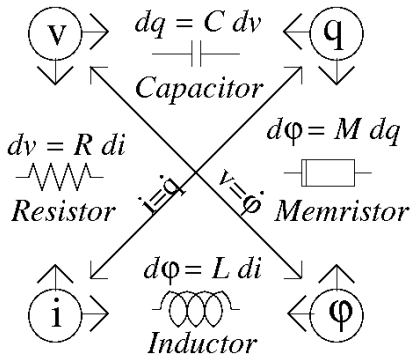

Fig. 1 The four basic two-terminal devices relating the four basic magnitudes

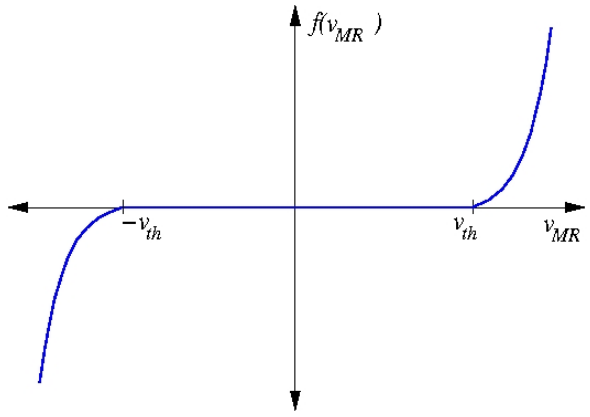

Fig. 2 Memristor characteristic function $f(v_{MR})$, as defined by eq. (4).

under electric fields. Although a linear dependence of f with voltage v_{MR} yields memristive behavior [1], it is clear that in reality such dependence is more likely to grow exponentially and/or include a threshold barrier v_{th}. For our discussions, let's assume the following generic dependence

$$f(v_{MR}) = A\ sign(v_{MR})\left[e^{|v_{MR}|/v_o} - e^{v_{th}/v_o}\right] \quad if \quad |v_{MR}| > v_{th}$$
$$f(v_{MR}) = 0 \qquad\qquad otherwise \qquad (4)$$

where A and v_o are parameters which may or may not depend on w. The shape of f is shown in Fig. 2. Many other mathematical formulations can be used, but the bottom line is to describe a thresholding behavior, an exponential behavior beyond threshold, and a bidirectional behavior (symmetric or not). Note that the memristor, as opposed to the other 3 devices in Fig. 1, is non-symmetric: reversing the polarity of v_{MR} not only reverses the polarity of i_{MR} but also the time derivative of w, thus producing a different evolution for $G(\cdot)$. Therefore, the memristor symbol must indicate its polarity.

III. STDP IN BIOLOGY

Spike-time-dependent plasticity (STDP) is a learning mechanism originally postulated [5] in the context of artificial machine learning algorithms (or computational neuroscience) exploiting spike-based computations (as in brains). It has been shown to improve Hebbian correlation-based plasticity at explaining cortical phenomena [10]-[11], and has been proven successful to learn hidden spiking patterns [8] or to perform competitive spike pattern learning [9]. Astonishingly, experimental evidences of STDP have been reported by several neuroscience groups worldwide during the past decade [13]-[20], so that today we can state that the physiological existence of STDP has been reasonably well established. However, the ultimate molecular and electro-chemical principles behind STDP are still under debate [21]. Before describing STDP mathematically, let us first explain how neurons interchange information and what the synaptic connections are. Fig. 3 illustrates two neurons connected by a synapse. The pre-synaptic neuron is sending a pre-synaptic spike $V_{mem\text{-}pre}(t)$ through one of its axons to the synaptic junction. Neural spikes are membrane voltages from the outside of the cellular membrane V_{pre}^{+} with respect to the inside V_{pre}^{-} (see inset at Fig. 3(b)). Thus $V_{mem\text{-}pre} = V_{pre}^{+} - V_{pre}^{-}$ and $V_{mem\text{-}pos} = V_{pos}^{+} - V_{pos}^{-}$. The "large" membrane voltages during a spike (in the order of hundreds of mV) cause a variety of selective molecular membrane channels to open and close allowing for many ionic and molecular substances to flow or not through the membrane. At the same time, synaptic vesicles inside the pre-synaptic cell containing "packages" of neurotransmitters fuse with the membrane in such a way that these "packages" are released into the synaptic cleft (the inter cellular space between both neurons at the synaptic junction).

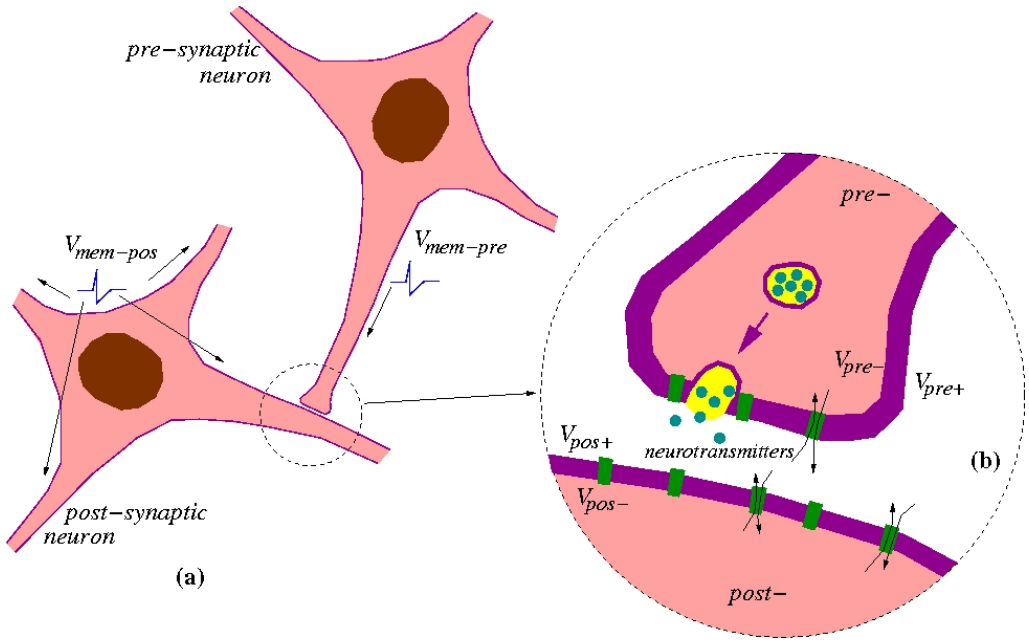

Fig. 3 Illustration of synaptic action. (a) A synapse is where a pre-synaptic neuron "connects" with a post-synaptic neuron. The pre-synaptic neuron sends an action potential $V_{mem\text{-}pre}$ travelling through one of its axons to the synapse. The cumulative effect of many pre-synaptic action potentials, generates a post-synaptic action potential at the membrane of the post-synaptic neuron, which propagates through all neuron's terminations. (b) Detail of synaptic junction. The cell membrane has many membrane channels of varying nature which open and close with changes of the membrane voltage. During a pre-synaptic action potential vesicles containing neurotransmitters are released into the synaptic cleft.

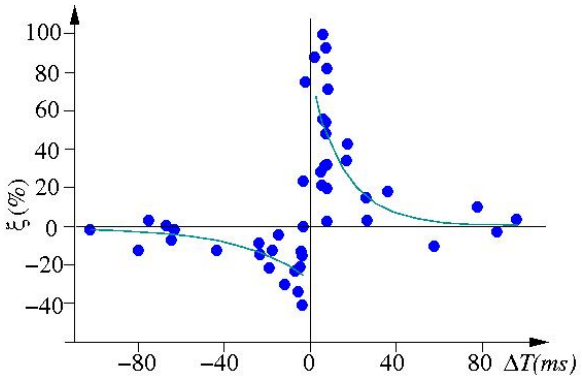

Fig. 4 STDP function ξ(Δ*T*) measured experimentally on biological synapses (data from Bi and Poo [14]-[15]).

Neurotransmitters are collected in part by the post-synaptic membrane contributing to a change in its membrane conductivity. The cumulative effect of pre-synaptic spikes (coming from this or other pre-synaptic neurons) will eventually trigger the generation of a new spike at the post-synaptic neuron. Each synapse is characterized by a "synaptic strength" (or weight) "*w*" which determines the efficacy of a pre-synaptic spike in contributing to this cumulative action at the post-synaptic neuron. This weight *w* could well be interpreted as the size and/or number of neurotransmitter packages released during a pre-synaptic spike. However, for our analyses, we will interpret *w* more generally as some structural parameter of the synapse that controls directly the efficacy of this synapse per spike (for example, the mechanism that controls the amount of neurotransmitter per vesicle). The synaptic weight *w* is considered to be non-volatile and of analog nature, but changes in time as a function of the spiking activity of pre- and post-synaptic neurons. This phenomenon was originally observed and reported by Hebb in 1949, who introduced his hebbian learning postulate [12]:

> "*When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased*".

Traditionally, this has been described by computational neuroscientists and machine learning computer engineers as producing an increment in synaptic weight Δw proportional to the product of the mean firing rates of pre- and post-synaptic neurons. STDP is a refinement of this 1949 rule which takes into account the precise relative timing of individual pre- and post-synaptic spikes, and not their average rates over time. In STDP the change in synaptic weight Δw is expressed as a function ξ of the time difference between the post-synaptic spike at t_{pos} and the pre-synaptic spike at t_{pre}. Specifically, $\Delta w = \xi(\Delta T)$, with $\Delta T = t_{pos} - t_{pre}$. The shape of the STDP function ξ can be interpolated from experimental data from Bi and Poo as shown in Fig. 4 [14]-[15]. For positive ΔT (which means, the pre-synaptic spike has a highly relevant role in producing the post-synaptic spike) there will be a potentiation of synaptic weight $\Delta w > 0$, which will be stronger as $|\Delta T|$ reduces. For negative ΔT (which means, the pre-synaptic spike is highly irrelevant for the generation of the post-synaptic spike), there will be a depression of synaptic weight $\Delta w < 0$, which will be stronger as $|\Delta T|$ reduces.

IV. MEMRISTANCE CAN EXPLAIN BIOLOGICAL STDP

Here we will show how the mathematical function ξ obtained in biology arises naturally when using memristive concepts in biological synapses. The key is to consider carefully the shape of the electric neural spikes. The exact shape of neural spikes, usually called "action potentials" among neuroscientists, is difficult to measure precisely since the experimental setup influences strongly. Furthermore, different action potential shapes have been recorded for different types of neurons, although in general they keep a certain resemblance among them. For our discussion it suffices to assume a generic action potential shape with the following properties (see Fig. 5). During spike on-set, which happens during a time t_{ail}^{+}, membrane voltage increases exponentially until a positive peak amplitude A_{mp}^{+}. After this, it changes quickly to a peak negative amplitude $-A_{mp}^{-}$ and returns smoothly to its resting potential during a time t_{ail}^{-}. A shape of the type shown in Fig. 5 can be expressed mathematically, for example, as

$$spk(t) = \begin{cases} A_{mp}^{+} \dfrac{e^{t/\tau^{+}} - e^{-t_{ail}^{+}/\tau^{+}}}{1 - e^{-t_{ail}^{+}/\tau^{+}}} & if \quad -t_{ail}^{+} < t < 0 \\ -A_{mp}^{-} \dfrac{e^{-t/\tau^{-}} - e^{-t_{ail}^{-}/\tau^{-}}}{1 - e^{-t_{ail}^{-}/\tau^{-}}} & if \quad 0 < t < t_{ail}^{-} \\ 0 & otherwise \end{cases} \tag{5}$$

Parameters τ^{+} and τ^{-} control the curvature of the on-set and off-set sides of the action potential. Consider the case of pre- and post-synaptic neurons in Fig. 3 being of the same type, and thus generating the same action potential shape, *spk*(*t*) of eq. (5), when they fire. Axons and dendrites operate as transmission lines, so it is reasonable to expect some attenuation when the spikes arrive at the respective synapses. Let α_{pre} be the attenuation for the pre-synaptic spike $V_{mem\text{-}pre}(t) = \alpha_{pre}\, spk(t\text{-}t_{pre})$, and α_{pos} for the post-synaptic spike $V_{mem\text{-}pos}(t) = \alpha_{pos}\, spk(t\text{-}t_{pos})$. When both spikes are more or less simultaneously present at the two cell membranes of the synapse, then channels on both membranes are open. Consequently, in principle, it makes sense to assume that during such time there could be a path for substances in the inside of one cell to move directly to the inside of the other cell and vice versa. Furthermore, let us assume now that such motion of substances obeys a memristive law similar to those described by eqs. (3-4). This means, that we would have a two-terminal memristive device between the inside sides of the two cells. More specifically, between V_{pos}^{-} and V_{pre}^{-} in Fig. 3(b). Consequently, the memristor voltage would be $v_{MR} = V_{pre}^{-} - V_{pos}^{-}$. On the other hand, since the outside nodes of both membranes V_{pos}^{+} and V_{pre}^{+} are very close together, both voltages will be approximately equal. Consequently,

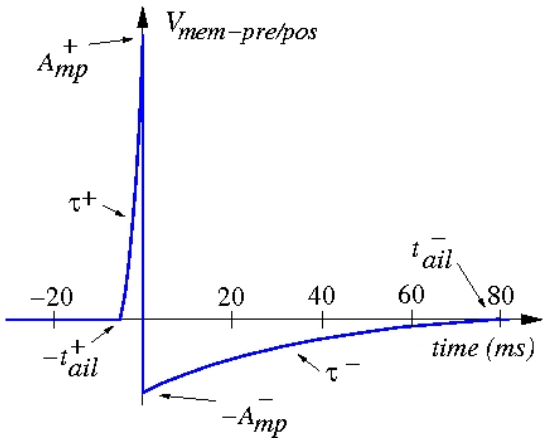

Fig. 5 Details of membrane voltage action potential, as by eq. (5).

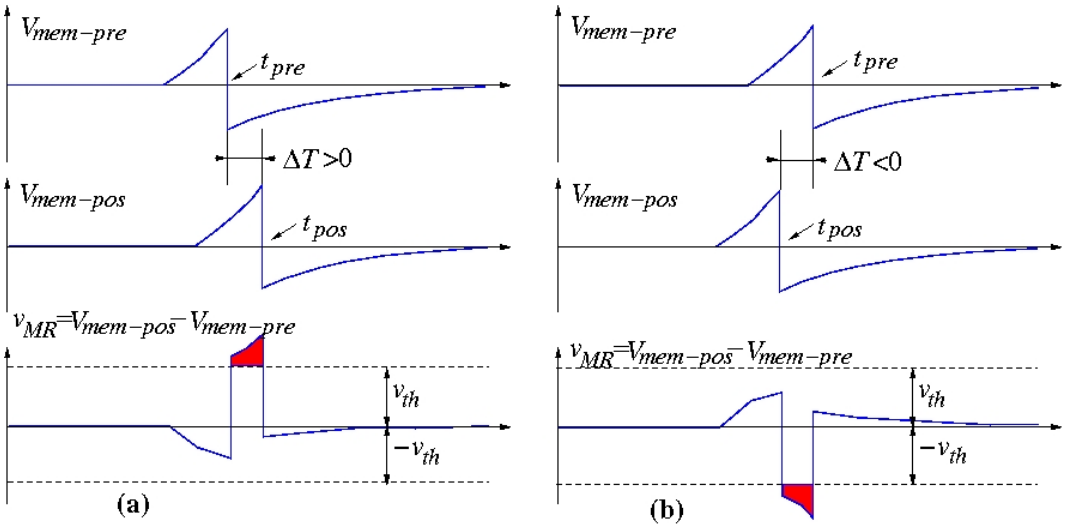

Fig. 6 Membrane and memristor waveforms. Pre- and post-synaptic membrane voltages for the situations of positive ΔT (a) and negative ΔT (b). The voltage at the memristor v_{MR} is the difference between the post-synaptic membrane voltage $V_{mem\text{-}pos}$ and the pre-synaptic membrane voltage $V_{mem\text{-}pre}$. When the signs are opposite, voltages add up, and may exceed the memristor threshold voltage v_{th} of function $f(\ )$. When this happens, the area in red (amplified exponentially) contributes to a change in synaptic efficacy. If ΔT is positive synaptic efficacy is increased, while if it is negative synaptic efficacy will be decreased.

the memristor voltage drop would be given by

$$v_{MR}(t') \approx V_{mem-pos}(t') - V_{mem-pre}(t') = \alpha_{pos} spk(t'-t_{pos}) - \alpha_{pre} spk(t'-t_{pre}) \quad (6)$$

Doing a simple change of variables $t = t' - t_{pos}$ and recalling that $\Delta T = t_{pos} - t_{pre}$, results in

$$v_{MR}(t,\Delta t) = \alpha_{pos} spk(t) - \alpha_{pre} spk(t+\Delta T) \quad (7)$$

This memristor voltage is shown in Fig. 6 for the cases of ΔT being positive or negative. According to eq. (4), memristive action will take place only if v_{MR} exceeds threshold v_{th}, as indicated by the shaded area in Fig. 6. As we postulated before, during this memristive action some amount of synaptic structural substance(s) Δw would be interchanged among both sides of the synapse. This amount of substance Δw will ultimately affect the synaptic strength of this synapse. If this amount of synaptic structural substance interchanged between the two synaptic terminations obeys a memristive law as in eqs. (3-4), then

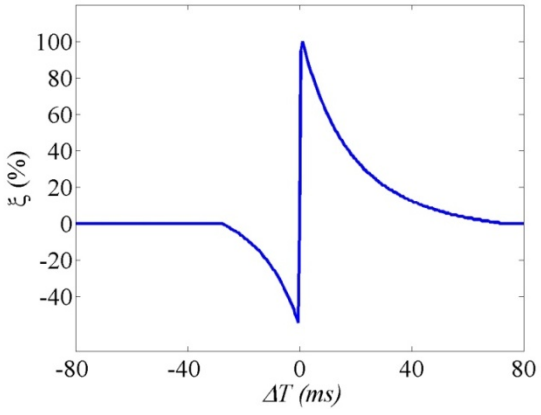

Fig. 7 STDP function $\xi(\Delta T)$ predicted using the memristance-based model and spike shapes from eqs. (1-7).

$$\Delta w(\Delta T) = \int f(v_{MR}(t,\Delta T))dt \quad (8)$$

Which is the area of the shaded regions in Fig. 6, previously amplified exponentially through function $f(\ )$ of (4). Positive areas (above v_{th}, when $\Delta T > 0$) yield increments for w ($\Delta w > 0$), while negative areas (below $-v_{th}$, when $\Delta T < 0$) result in decrements for w ($\Delta w < 0$). As $|\Delta T|$ approaches zero, the peak of the shaded area in v_{MR} is higher. Since this peak is amplified exponentially, the contribution for incrementing/decrementing w will be more pronounced as $|\Delta T|$ is reduced. The resulting function $\Delta w(\Delta T)$, computed using the memristor model through eqs. (3-8) is shown in Fig. 7. It follows indeed the behavior of the STDP function ξ obtained by Bi and Poo from physiological experiments, shown in Fig. 4. For this numerical computation we used the following parameters: $\alpha_{pos} = 1$, $\alpha_{pre} = 0.9$, $v_{th} = A_{mp}^{+} = 1$, $A_{mp}^{-} = 0.25$, $v_o = 1/7$, $t_{ail}^{+} = 5ms$, $t_{ail}^{-} = 75ms$, $\tau^{+} = 40ms$, $\tau^{-} = 3ms$. Making $\alpha_{pos} \neq \alpha_{pre}$ breaks the symmetry of function $\xi(\Delta T)$, and making them very distinct removes one of the branches in $\xi(\Delta T)$.

V. INFLUENCE OF ACTION POTENTIAL SHAPE

The shape of the action potential function $spk(t)$ influences the shape of the resulting STDP function $\xi(\Delta T)$. As an illustration, Fig. 8 shows the situation for several shapes of action potential (left graphs). For example, if the exponential shape degenerates to a triangular type of shape, then the central region of $\xi(\Delta T)$ results in a smoother transition from the negative peak to the positive peak. Note that this would penalize the learning for cases with $|\Delta T|$, which is an undesired effect. Making the positive peak of the spike smaller than the negative one, results in a stronger negative branch for $\xi(\Delta T)$ than the positive one. If the action potential is substituted by a rectangular shape signal, then the central region becomes linear and a saturation effect might occur. If the rectangular spike is made more symmetric, then $\xi(\Delta T)$ degenerates to a triangular type of shape, which is very distinct from the original biological STDP learning function.

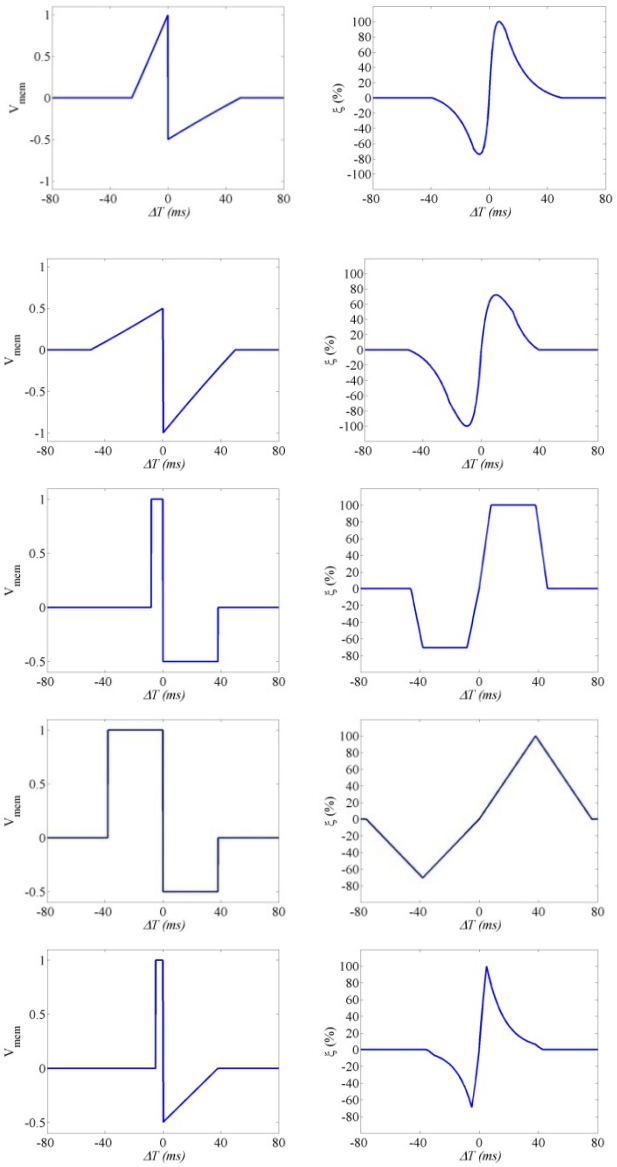

Fig. 8 Influence of action potential shape on the resulting STDP function $\xi(\Delta T)$.

VI. NANOTECHNOLOGY IMPLEMENTATION

This result shows that a memristive type of mechanism could be behind the biological STDP phenomenon. On the other hand, for a neuromorphic engineer, this result provides clear hints on how STDP learning rules could be implemented in nanotechnology based neuro-inspired computers using memristors. For example, one can fabricate very high density memristor crossbar structures [1]-[2] which connect neural layers, as shown in Fig. 10. Memristive crossbars can be fabricated using very dense nanowire fabric, while inter-synaptic neurons can be made using conventional CMOS technology underneath the memristor fabric. In order to equip the system with STDP learning, CMOS neurons could generate fully asynchronous action potentials similar to those shown in Fig. 5 and not only propagate them forward but also backwards with some attenuation.

Although the purpose of this paper is not to provide detailed circuit implementations of the underlying CMOS circuitry, we indicate here some hints on what conditions it should fulfill for proper system level

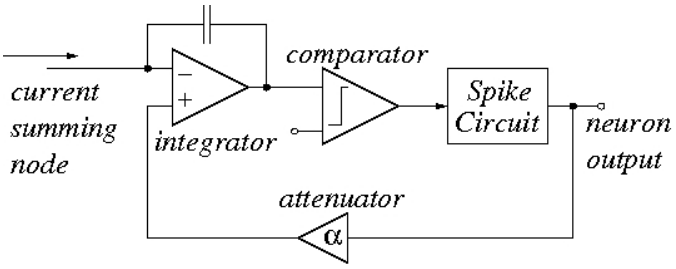

Fig. 9 Possible neuron circuit implementation for memristive compatible STDP learning system.

operations. A possible schematic diagram for the neuron block is shown in Fig. 9. The neurons needs to include a current sinking input terminal so that in absence of spike output it computes the integral of input spike signals. This can be done by using an integrator with a clamped voltage input. During absence of output spikes, its voltage is clamped to analog ground, and the integrator accumulates the contribution of incoming spikes. Note that spikes need to have a non zero overall integral (area). The output of this accumulated integral is compared against a reference. If this reference is reached the comparator output will trigger a spike generation circuit, which provides the output of the neuron. An attenuation version of this voltage is used to clamp the voltage of the neuron input node.

CONCLUSIONS

In this paper we have shown that STDP learning can be implemented using nanotechnology memristive crossbar arrays, by using underlying CMOS spiking neurons that send forward and backward electric spike signals. These spikes need to fulfill certain shape conditions, which makes them similar to the neural spikes observed in biology and neuroscience. We have shown, through numerical computations, that voltage controlled memristors with an internal nonlinear symmetric control function that includes a threshold and an exponential shape above threshold, combined with neurons that generate exponential spikes with a positive onset and a negative relaxing offset, yield naturally the STDP synaptic learning rule observed in biological experiments from neural synapses. We have

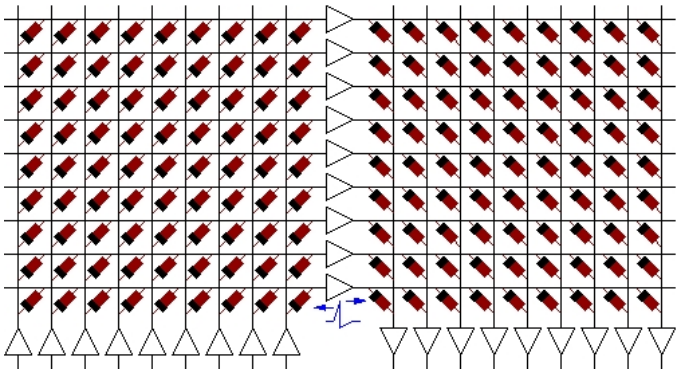

Fig. 10 Neuromorphic memristive computer equipped with STDP. Neuromorphic computing structure with three layers of neurons (made in CMOS technology) and two fully-connected inter-layer meshes of memristors (made with nanowires on top of a CMOS substrate). STDP is implemented by having neurons generate action potentials with shapes similar to that of Fig. 5, and sending them forward without attenuation and backward with a slight attenuation.

also shown how the shape of the electric neural spikes might degrades the STDP learning function. And finally, we have given some hints on how to implement CMOS neurons for use in STDP systems with crossbar memristor arrays.

There exists an extensive knowledge on how to perform signal sensing and processing by means of spiking neuromorphic circuits [22]-[26]. Such body of knowledge could be directly incorporated into nanotechnology memristive adaptive spiking neuromorphic systems.

ACKNOWLEDGEMENTS

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