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9:15 - 11:30, October 21, 2017. Location: Multiple-function Hall, 5th Floor, Training Building, Guoyuan Langyi Hotel. Session Chairs:
Prof. Sangmin Han(Chonbuk National University, Korea)
Dr. Qing Zhao (Tianjin University of Science and Technology, China)

ID.7. Evolution Analysis for HA Gene of Human Influenza A H1N1 Virus (2009 - 2017)
Lingyun Liu, Zhaohui Qi and Jinlong Ma
Shijiazhuang Tiedao University

ID.15. Single Layer Negative Dispersion Retarder by Self-Organization of Smectic Reactive Mesogen Molecules
Jinyoung Jeong, Hyoungsuk Kim, Seok-Hwan Park and Ji-Hoon Lee
Chonbuk National University

ID.48. A Frequency Analysis Based Sound Detector for Deaf
Ahhyeon Jo, Wonjun Lee, Yongmin Jo, Sangtae Park, Hyejung Seon and Sooyoung Kim
Chonbuk National University

ID.49. Voltage-dependent Action Potential Generator Circuit for Biological Neuron
Zubaer Ibna Mannan, Yongchae Jeong, Seok-Hwan Park, Jihoon Lee and Hyongsuk Kim
Chonbuk National University

ID.58. Abdominal Obesity Measurement Using Kinect Sensor
Dasom Seo, Yeongtae Han, Min-Gul Kim, Kanghan Oh and Il-Seock Oh
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ID.61. Understanding Convolutional Neural Network Architectures for Small Objects Identification
A CNN-Based System for Whiteflies Identification in Tomato Plants
Alvaro Fuentes[1], *Sook Yoon*[2], *Dong Sun Park*[1]
[1]*Chonbuk National University, Jeonju, South Korea*
[2]*Mokpo National University Mokpo, South Korea*

ID.98. The Study of Seafarer's Brain Functional Connectivity Before and After Sailling Using fMRI
Yuhu Shi and Weiming Zeng
Shanghai Maritime University

ID.102. An Approach to Evaluate Electromagnetic Interference with a Wearable ECG at Low Frequencies
Wei Liao
Shanghai University of Engineering Science, Shanghai, China

Voltage-dependent Action Potential Generator Circuit for Biological Neuron

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***Abstract*—In physiology, action potential is regarded as an important short lasting event through which cell to cell communication is performed. In this paper, we propose and design a voltage-dependent artificial action potential generator circuit. The distinctive properties of the biological action potential are verified through the SPICE simulations of the proposed circuit. The artificial architecture presented in this paper can be used in academia to emulate the response of the biological neuron on the basis of diverse stimulating signals.**

Keywords—Action potential (AP), bioelectronics device, neurotransmitter, Schmitt trigger, summation amplifier, synapse, voltage-controlled oscillator (VCO).

I. INTRODUCTION

In neuromorphology, action potential (AP) plays an important role as the neurons are communicating with each other through this electro-chemical signal. Not only the extracellular communication, such as neuron to neuron or neuron to muscle communication, but also the intercellular, like as axonal propagation, depend on the action potentials [1]. Physiologically, in intercellular case, action potential is defined as the short-lasting event through which the cell membrane potential rapidly rises and falls electrically, following a consistent trajectory [2].

Neurophysiology contains several mathematical modeling of action potential based on experimental observations. Among those models Hodgkin-Huxley model (HH) [3] is considered as the propitious one as they describe the generating phenomenon of action potential as well as the axonal propagation of the action potential. However, the complexity of the HH model compels the researchers to emerge a new simplified model and FitzHugh-Nagumo model (FHN) [4] is developed as the simplified model of HH model. However, a circuit based action potential generator is proposed in [5]. Although the generative techniques presented in [5] is completely different from [3] and [4] but also the qualitatively behavior is similar.

In this paper, we propose a voltage-dependent artificial action potential circuit which is capable of generating the action potential based on biological distinctiveness such as strong stimulation and synaptic plasticity as both cases the frequency of the action potential is higher than the typical scenario. To do so, we use a comparator circuit to compare the membrane potential with artificial action potential (AP) threshold. When the membrane potential is higher than the artificial AP threshold then the follow through switch feedforwarded the membrane potential to succeeding voltage control oscillator (VCO) circuit. The VCO circuit synthesizes the membrane potential to a frequency dependent pulse output and feedforwarded to a pulse shaping circuit where the pulse output of the VCO is converted to artificial action potentials. To match the artificial APs with biological action potentials we use a summation amplifier circuit to summate the artificial APs with resting membrane potential (typically -70mV).

Section II reviews the concepts of biological action potential whereas the proposed artificial circuit is described in section III and the results and simulations are illustrated in section IV. Section V contains a comparative conclusion.

II. EXPLANATION OF BIOLOGICAL ACTION POTENTIAL

In neurophysiology, action potential generates due to the intercellular and extracellular ions movement where the *Ligand-gated ion channels* and the *Voltage-gated ion channels* work as a bridge way for the ions movement. In both *Ligand* and *Voltage gated ion channels*, action potential contains three specific states such as Depolarization, Repolarization and Hyperpolarization [2] as shown in Fig. 1.

The generation of action potential through Ligand-gated channel initiates when the released presynaptic neurotransmitters of chemical synaptic transmission pass through the synaptic cleft and attached with the postsynaptic Ligand-gated receptors [7]. Binding of neurotransmitters in the receptors cause the specific ionic channels to open, and change the ability of ions flow (in or out) of the membrane [2]. After the attachment of Acetylcholine (ACh) neurotransmitters in postsynaptic receptor, the sodium (Na^+) gated channels open and the Na^+ ions rush into the postsynaptic membrane and depolarize the membrane by increasing its membrane potential from the resting membrane potential (typically -70 mV) [6] [7]. If the depolarized membrane potential reaches the threshold, typically -55mV, then it generates an all-or-none phenomenon based electrical impulse (action potential) and reaches the peak voltage +40mV. As the membrane potential reaches to +40 mV [8] [9] the sodium channels begin to close and potassium channels begin to open and K^+ ions move out of the cell due to

potential and concentration gradient, known as repolarization [10]. Due to the permeability of potassium channels the postsynaptic membrane continues to repolarize more than the resting membrane potential and gives an undershoot which is known as hyperpolarization [2]. Afterwards, membrane potential recovers the undershoot and stabilizes to resting membrane potential.

In contrast to the Ligand-gated channel, the generation of action potential through voltage-gated channel initiates when the electrical membrane potential alters the conformation of the channel proteins, regulating their opening and closing [2]. The opening and closing of the channels are triggered by changing ion concentration, and hence charge gradient, between the intercellular and extracellular sides of the cell membrane [11]. When an axonal membrane exhibits potential difference due to charge gradient, the associated electric field induces a conformational change in the ion channels. Before an action potential occurs, the axonal membrane is at its normal resting potential [2]. In response to an electric current, the sodium ion (Na^+) activation gates open, allowing positively charged Na^+ ions to flow into the cell membrane through the Na^+ ion channels, and depolarize the membrane by increasing its potential from resting membrane potential. When the membrane potential is higher than the threshold (-55 mV) then it generates the action potential and when it reaches the peak (+40 mV) the Na^+ channels inactive themselves and the membrane electric field induced conformational change by opening the potassium (K^+) ion channels [7]. Due to the opening of potassium ion channels, K^+ ions move out of the cell and repolarize the membrane. Because of the permeability of potassium channels, membrane potential exhibits an undershoot which recovers afterwards [2].

The frequency of the action potential depends on the strength of the stimulating signal as well as the synaptic plasticity of the synapse [2] [7]. For a weak stimulating signal, the neuron either generates a low frequency action potential or might be exhibited no action potential whereas for strong stimulating signal, the neuron exhibits higher frequency action potential in accordance to the strength of the stimulation [2].

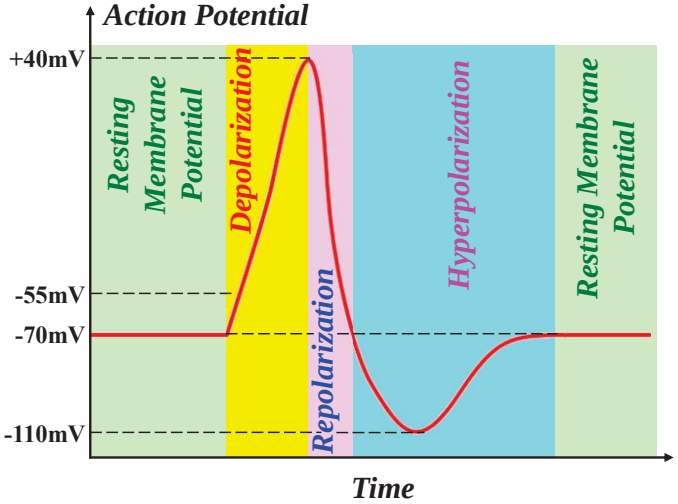

Fig. 1. Typical action potential waveform of biological neuron.

III. ARTIFICIAL ACTION POTENTIAL GENERATOR CIRCUIT

The proposed artificial voltage-controlled action potential generator circuit contains four different segments as shown in Fig. 2. First part of the proposed bioelectronics device contains a comparator circuit along with a follow through switch (M_{con}) which compare the input V_{in} (in this design, V_{in} is imitated the role of membrane potential) with the artificial threshold of the action potential $V_{thr} = 0.1V$. If $V_{in} > V_{thr}$ then the positive pulse of the op-amp (U1) output turn ON the M_{con} switch and V_{in} is feedforwarded to the succeeding voltage controlled oscillator (VCO) circuit [12]. The VCO circuit consists of an integrating amplifier and a Schmitt trigger circuit. The non-inverting terminal of the integrator drops half of V_{in} and the inverting terminal maintains the same voltage as the non-inverting terminal. In contrast, the opposite terminal of R_{Osc} maintains the same voltage as V_{in}. When the switch (M_{dis}) is *ON*, the current through the 49.9k resistor (connected in between the inverting terminal of the integrator and the discharge switch M_{dis}) is the summation of currents generated by the potential difference of R_{Osc} (I_{Rosc}) and the charging capacitor (C_{Osc}) current (I_{Cosc}). In ON state of M_{dis}, the integrating amplifier maintains the steadily rising output voltage V_{Tri}. However, for *OFF* state of M_{dis}, the I_{Rosc} current passes through the capacitor and discharges it for which the output voltage V_{Tri} of the integrating amplifier is steadily decreased. The output voltage V_{Tri} is connected to the inverting terminal of the Schmitt trigger whereas the non-inverting terminal of Schmitt trigger is connected to a reference voltage source (V_{ref}) as well as to the output terminal (V_{Rec}) through a resistive network. When the threshold of Schmitt trigger, $V_{th} > V_{Tri}$ the output of the Schmitt trigger exhibits high voltage ($V_{Rec} = 3.5V$) and for $V_{th} < V_{Tri}$ output voltage of the Schmitt trigger exhibits low voltage ($V_{Rec} = -3.5V$). Moreover the frequency of the output pulse of the VCO circuit can be controlled by the following equation[1]

$$f_{VCO} = V_{VCO} / 8V_{th}R_{Osc}C_{Osc} , \quad (1)$$

where the frequency f_{VCO} is proportional to the input of VCO and inversely proportional to the threshold voltage V_{th}, resistance R_{Osc}, and integrating capacitor C_{Osc}. The proportional relationship between the frequency of the output pulse and the input of *VCO* imitates the biological phenomenon of strong stimulation because the biological neuron exhibits higher frequency of action potential in strong stimulation scenario [2]. Moreover the neurophysiological phenomenon of synaptic plasticity such as long term potentiation or long term depression [2] [13] can be imitable by adjusting the frequency controlled parameters of VCO (V_{th}, R_{Osc}, and C_{Osc}).

The output of the VCO is connected to the input of pulse shaping circuit which converts the output pulse V_{Rec} to the shape of action potential (V_{A_AP}) as shown in Fig. 2. The shape, amplitude, and the values of undershoot of the artificial APs (V_{A_AP}) can be controlled by changing the values of the resistors and capacitors of the pulse shaping circuit. The summation amplifier circuit is used to match the artificial APs (V_{A_AP}) with the biological action potential and denoted as V_{B_AP}.

[1] The mathematical model and the computational derivation of the frequency response of VCO circuit is elaborately describe in [12].

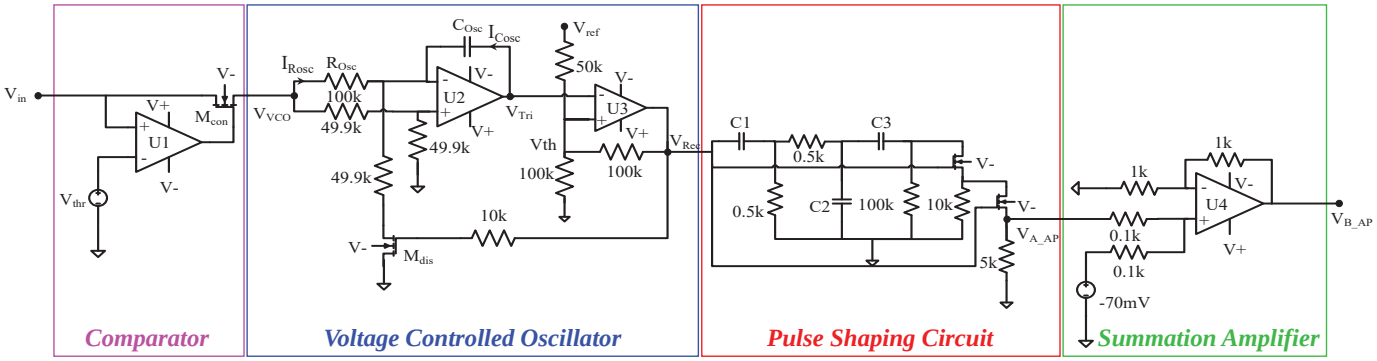

Fig. 2. Artificial circuit of voltage-dependent action potential generator.

IV. Results and Simulations

The simulations and results of the proposed voltage-dependent action potential generator circuit is represented in this section. The proposed architecture is verified with different input waveforms as well as with the biological distinctiveness.

The response of the proposed artificial circuit for a sawtooth input is shown in Fig. 3. The input voltage V_{in}, the VCO input V_{VCO}, the output of the VCO integrator circuit V_{Tri}, and the output of Schmitt trigger of VCO V_{Rec} is shown with blue, magenta, green, and red waveforms, respectively, in Fig. 3(a). The blue and magenta waveforms in Fig. 3(b) represent the artificially generated action potentials V_{A_AP} and the biologically matched action potential V_{B_AP}, respectively. Observe from Fig. 3(a) and (b) that the proposed architecture doesn't generate action potential over the time period 0 – 25ms as $V_{in} < V_{thr}$. Moreover, as the input voltage increases over the time periods the proposed architecture generates V_{Rec} output pulse more frequently and eventually the frequency of the AP is higher than the initial time period. Furthermore, the magnitude range of the biologically matched action potentials (V_{B_AP}) in Fig. 3(b) is similar to the biological action potential, typically from -110mV to +40mV, shown in Fig. 1.

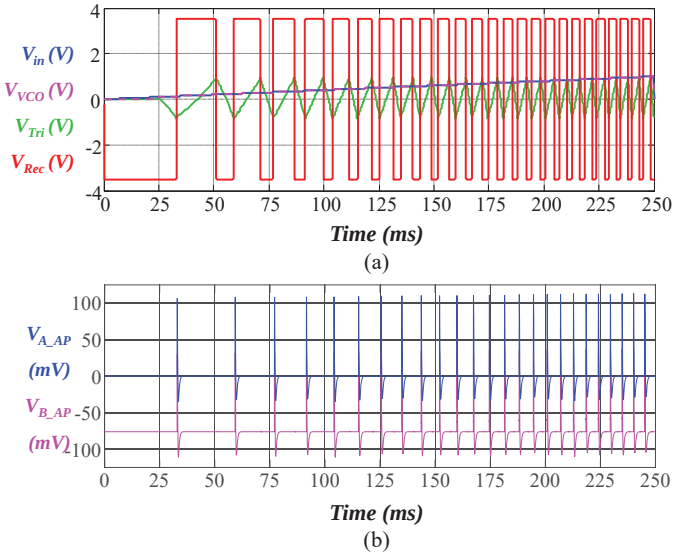

Fig. 3. Waveforms generated from proposed voltage-dependent action potential circuit for a sawtooth input. (a). Sawtooth input V_{in}, corresponding input of VCO V_{VCO}, integrator output V_{Tri}, and output of VCO V_{Rec}. (b). Artificial action potential (V_{A_AP}) and biologically matched action potential (V_{B_AP}).

Comparison between the proposed architecture waveforms and the waveforms represented in [5] illustrates that the proposed architecture performs better than the architecture presented in [5] as the frequency of the proposed architecture is higher than that of [5]. Moreover, for the same sawtooth input, the initial time delay of the VCO output is less than the VCO output of [5].

To demonstrate the versatility of our proposed artificial architecture we stimulated the artificial circuit with a pulse input $V_{in} = 1V$ which is shown in Fig. 4. Fig. 4(a) represents the V_{in}, V_{VCO}, V_{Tri}, and the V_{Rec} in blue, magenta, green, and red waveforms, respectively. The artificial action potential V_{A_AP} and the biologically matched action potential V_{B_AP} are shown in blue and magenta, respectively, in Fig. 4(b). Scrutinizing the input V_{in} (blue) and the VCO input V_{VCO} (magenta) waveforms in Fig. 4(a) reflect that the VCO circuit produces voltage-dependent pulse output only in the positive cycle of the V_{in} due to the comparator controlled switch M_{con} as shown in Fig. 2. Moreover, for each input pulse the proposed artificial architecture successfully generates the action potentials.

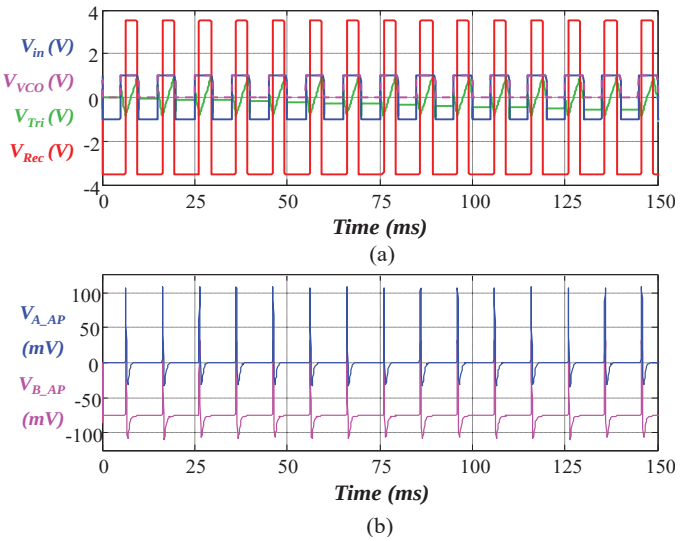

Fig. 4. Response of the artificial action potential generator circuit for a pulse input. (a). Pulse input $V_{in} = 1V$, corresponding input of VCO V_{VCO}, integrator output V_{Tri}, and output of VCO circuit V_{Rec}. (b). Artificially generated action potential (V_{A_AP}) and biologically matched action potential (V_{B_AP}).

To reveal whether the proposed artificial circuit can satisfies the biological distinctiveness we stimulated the proposed architecture with a strong stimulating pulse input with $V_{in} = 2V$

as shown in Fig. 5. Fig. 5(a) shows the waveforms of V_{in}, V_{VCO}, V_{Tri}, and the V_{Rec} in blue, magenta, green, and red, respectively, and Fig. 5(b) shows the artificially generated action potentials V_{A_AP} and the biologically matched action potential V_{B_AP} in blue and magenta waveforms, respectively. Observe from Fig. 4 and Fig. 5 that the frequency of the generated action potentials in Fig. 5 is higher than that of Fig. 4 although the circuit structure and the parameters are exactly same in both simulations. The reason behind such an increment in the frequency of the action potentials is the strong simulating input, which is similar to the attributes of biological neurons.

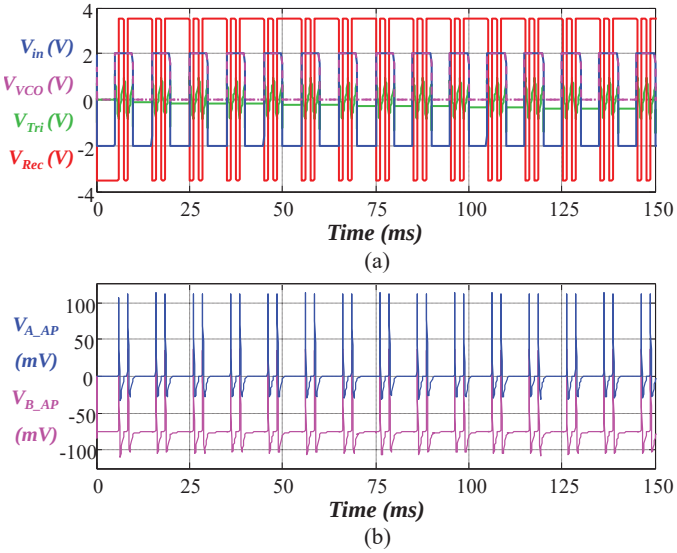

Fig. 5. Simulation results of the voltage-dependent artificial action potential generator circuit for strong stimulation. (a). Pulse input V_{in} =2V, corresponding input of VCO V_{VCO}, output of integrating circuit V_{Tri}, and output of VCO V_{Rec}. (b). Artificially generated action potential (V_{A_AP}) and biologically matched action potential (V_{B_AP}).

V. Conclusion

This paper presents an artificial structure of voltage-dependent action potential generator circuit for biological neuron. For strong stimulating signal the proposed architecture exhibits the similar biological attributes of a neuron by generating higher frequency action potentials. One of the major advantage of the proposed circuit is that it can truncate the negative part of the bipolar input which prevents the discharging phenomenon of the integrating capacitor for a negative input. It is important to prevent such types of discharging phenomenon as the discharging phenomenon of the integrating circuit in negative cycle consequently reduces the frequency of the VCO. The action potential generating architecture presented in [5] exhibits such type of capacitor discharging problem if driven by a bipolar input. Moreover, compare to [5] the proposed architecture provides more flexibility to control the frequency of the VCO by controlling the threshold of the VCO (V_{th}) with a reference signal (V_{ref}).

The proposed bioelectronics device can be used in academia to imitate the role of action potential generator according to membrane potential of biological neuron by connecting the input port of the artificial architecture with the voltage clamp experimental method [13].

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