ANALOG CMOS MODELING OF INVERTEBRATE LEARNING

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Abstract

Analog CMOS circuits which model synaptic habituation, sensitization and classical conditioning found in the marine mollusc *Aplysia* are presented. These circuits are expected to be useful in ANNs with higher-order synapses and learning rules that perform temporal association of multiple inputs. Our investigation illustrates that biological synaptic learning in invertebrates involves more elaborate mechanisms than those found in most current ANN models.

I. Introduction

In previous studies we have described implementations of artificial neural networks (ANNs) using low-accuracy analog CMOS transconductance circuits [4,5]. These ICs implement conventional ANN architectures, with Hebbian [7] and contrastive Hebbian [9] learning circuitry at each synapse. We briefly outline our work with contrastive Hebbian ANNs in section II. For the balance of this paper, we take a slightly different approach to the implementation of ANNs: rather than designing circuits to implement an ANN architecture, we propose circuits which are suggested by the behavior of a *biological* neural network.

Our circuits model three learning paradigms present in biological synapses: habituation, sensitization and classical conditioning. Specifically, we are modeling the behavior of the marine mollusc *Aplysia*, which has been studied extensively -- see for example, [1,2]. It must be emphasized that, although our circuits are more biologically plausible than most current ANNs, they still present a highly simplified picture of biological neural networks. More complex aspects of biological synapse operation, such as those involving extinction and spontaneous recovery [2], are omitted from the circuits described below. In doing so we achieve a balance between accurate modeling of neural biology, and mainstream ANNs.

This work draws upon two of our previous investigations, [3], in which CMOS circuits implementing habituation, sensitization and classical conditioning with EEPROM weight storage are described, and [4], which deals with analog CMOS Hebbian learning circuits using capacitive weight storage. Other approaches to ANN implementation with learning circuitry at each synapse have been reported recently, including [6]. Below we begin by describing our work with contrastive Hebbian ANNs. Then, in section III, biological synaptic learning is reviewed. Section IV presents a simplified mathematical model of this learning behavior, which is followed by a description of the proposed analog CMOS synaptic circuit, as well as simulation results, in section V.

II. Contrastive Hebbian ANNs

In this section we describe an analog CMOS implementation of a fully connected ANN with *contrastive* Hebbian learning [9] circuitry at each synapse.

Networks with contrastive Hebbian learning are intended for supervised learning applications, where a set of training patterns is used for weight learning. A key feature of contrastive Hebbian learning is that it can be used to train weights in networks with *hidden neurons*, that is, neurons whose activations are neither network inputs nor outputs.

The architecture of the ANN under consideration is a fully-connected Hopfield-type arrangement of neurons and synapses, in which there is a synaptic connection between each pair of neurons. Thus, a network of N neurons has $N(N-1)$ synapses, and synaptic circuitry occupies the majority of silicon chip area.

Manhattan contrastive Hebbian learning (CHL) is a two-phase weight learning process, governed by:

$$V_i = f\left(\sum_j W_{ij} V_j\right) \tag{1}$$

$$\Delta W_{ij} = a \cdot \mathrm{sgn}(V_i^+ V_j^+ - V_i^- V_j^-) \tag{2}$$

where V_i^+ and V_j^+ are the activations of the i^{th} and j^{th} neurons in the *clamped* [9] phase, V_i^- and V_j^- are the activations of the i^{th} and j^{th} neurons in the *unclamped* phase, and a is a small positive constant which determines the weight learning rate.

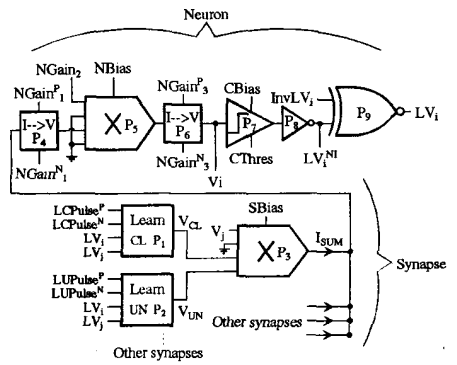

Figure 1 *System block diagram. $N-1$ synaptic circuits are connected to each neuron circuit.*

Fig. 1 is a block diagram of the ANN system: the circuit is an analog CMOS approximation to (1) and (2). Both neuron activations and synaptic weights may take on analog values in the range $[-V,V]$. Multipliers P_3 and P_5 are transconductance multipliers, whose inputs are voltages and output is a current. P_4 and P_6 are linear current to voltage converters, functioning as a resistor to $(Vdd + Vss)/2$. The comparator P_7 outputs either Vdd or Vss, depending on whether its input is above or below its threshold reference,

and P_8 and P_9 are conventional digital gates. Lastly, P_1 and P_2 are Manhattan CHL circuits, containing weight update circuitry and weight storage capacitors. Synaptic weights are represented by the differential voltage $V_{CL} - V_{UN}$.

The circuit of Fig. 1 approximates equations (1) and (2), representing ideal network behavior, as follows. The synaptic weight W_{ij} is represented by the differential voltage, $V_{CL} - V_{UN}$. Then, the product $W_{ij}V_j$ in (1) is implemented by P_3 as $I_{SUM} = b(V_{CL} - V_{UN})V_j$, and the summation operation is performed as current summation at the input to P_4. P_4 - P_6 realize a variable gain neuron, whose transfer function may be adjusted in a variety of ways, using the *NGain* control signals.

Since we implement Manhattan learning, only the *sign* of neuron activations are required for learning. P_7 - P_9 generate LV_i, a binary version of each neuron activation for learning.

Figure 2 *Photomicrograph of 19 neuron, 342 synapse network (4.16 x 2.76mm², 1.2μm CMOS).*

Network operation proceeds as follows. First, the network inputs are set for a particular training pattern, while output and hidden neurons remain free (ie. unclamped). The network is allowed to settle to its unclamped phase minimum energy state, LV_i^- and LV_j^- (the binary learning activations generated by the i^{th} and j^{th} neurons) settle, and control signals $LUPulse^P$ and $LUPulse^N$ are pulsed briefly. The binary product $LV_i^-LV_j^-$ determines whether a small quantity of charge is added to, or removed from, the storage capacitor connected to the unclamped learning circuit. Next, network outputs are set to training pattern values, the network settles in the clamped phase, and learning control signals $LCPulse^P$ and $LCPulse^N$ are pulsed briefly. As before, the binary product $LV_i^+LV_j^+$ determines whether charge is added to, or removed from the clamped storage capacitor. This two phase procedure is repeated for each training pattern. Typically, many passes through the entire set of training data is required to learn network weights. Fig. 2 is a photomicrograph of the multi-project die containing a 19 neuron, 342 synapse test network.

III. Basic Biological Synaptic Learning

Kandel et al [1], in their study of the marine mollusc *Aplysia*, have shown that chemical changes in individual synapses are responsible for three important types of learning: habituation, sensitization and classical conditioning. In this section, biological synaptic function is described at the behavioral level; for a description of the electro-chemical processes involved, see [1]. Fig. 3 shows a simplified schematic diagram of the portion of *Aplysia's* 10^5 neuron nervous system responsible for its protective gill withdrawal reflex. The neural network connecting the gill (*Aplysia's* respiratory organ), siphon (a small spout for expelling sea water), mantle and tail allows the mollusc to withdraw its gill when a stimulus is applied to the tail, siphon or mantle. In nature, this reflex has obvious evolutionary advantages, as it allows *Aplysia* to protect its sensitive gill at the first sign of attack. *Aplysia's* response to tail, mantle and siphon stimuli may be altered dramatically by what it has learned about these stimuli in the past. In Fig. 3, the three sensory neurons SN_1, SN_2 and SN_3 receive stimuli from the mantle, siphon and tail, respectively. SN_1 and SN_2 synapse directly with motor neuron MN (S_1 and S_2), so siphon and mantle stimuli can trigger gill withdrawal. In addition, SN_3 synapses with the facilitating interneuron FN, which in turn synapses with *synapses* S_1 and S_2 (through F_1 and F_2). These synapses on synapses, F_1 and F_2, play a critical role in learning, as described below. For the purposes of neural modeling, the pairs S_1 and F_1, and S_2 and F_2 may be regarded as single *ternary* synapses (synapses between three neurons), in which the synaptic weight is determined by the interaction of two external signals. This is the approach that we take in the following section. Note that the schematic of Fig. 3 is highly simplified; in particular, each neuron illustrated represents approximately 6 to 24 neurons operating in parallel.

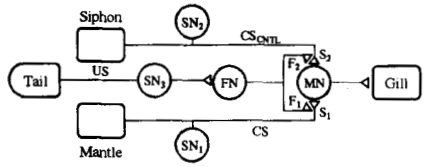

Figure 3 *Simplified schematic diagram of the portion of Aplysia's nervous system responsible for gill withdrawal reflex.*

Habituation may be defined as "a decrease in the strength of a behavioral response that occurs when an initially novel eliciting stimulus is repeatedly presented" [1]. Kandel et al have shown that repeated mild tactile stimuli to the mantle cause the gill withdrawal reflex to habituate, as the animal learns that the stimuli pose no danger. This learning behavior has been traced to chemical changes in the synaptic connection between mantle sensory neurons and gill motor neurons (S_1): the effective weight of the synapse S_1 is reduced.

The sensitization mechanism is somewhat more complex. *Sensitization* is defined as, "the enhancement of an animal's reflex response as a result of the presentation of a strong or noxious stimulus" [1]. In the case of *Aplysia*, noxious stimuli to the tail result in enhanced subsequent gill withdrawal in response to mild mantle stimuli. Again the locus of learning is the synapse S_1, in this case through *presynaptic facilitation* from synapse F_1. The mechanism is as follows: tail sensory neuron firing (SN_3) causes the facilitating interneurons (FN) to fire, which in turn cause F_1 to

alter the chemistry of S_1, such that the synaptic weight of S_1 is increased. Not surprisingly, sensitization can reverse the effects of habituation.

"In classical conditioning, an initially weak or ineffective conditioned stimulus (CS) becomes highly effective in producing a behavioral response after it has been paired in time with a strong unconditioned stimulus (US)" [1]. Classical conditioning is a form of associative learning, by which an organism learns a *predictive* relationship between two stimuli. *Aplysia* can be classically conditioned by applying a mild tactile stimulus to the mantle (CS), followed approximately $\frac{1}{2}$ second later by a strong stimulus to the tail (US). Again F_1 plays an important role. In classical conditioning, recent activity in S_1 due to the CS results in *activity-dependent enhancement of presynaptic facilitation*. Thus classical conditioning uses the same mechanism as sensitization (presynaptic facilitation), the effect of which is enhanced by the arrival of the CS $\frac{1}{2}$ second before. In classical conditioning, the time between the CS and the US is critical: if the US arrives *before* the CS, no classical conditioning occurs. This makes good survival sense, since *Aplysia* is concerned about learning when a mild stimulus (CS) predicts a potentially threatening one (US). In Kandel's experiments with *Aplysia*, CS_{CNTL} (siphon stimulus) was used to differentiate between the effects of sensitization and classical conditioning.

As the above discussion shows, classical conditioning is a form of associative learning, and is therefore related to Hebbian learning [7] and its variants. However, classical conditioning is *non commutative*, because the CS must precede the US by a critical interval.

Kandel's investigation of synaptic learning shows that *Aplysia* uses learning rules which are much more elaborate than those used by most ANNs. *Aplysia* employs non-commutative timing-critical associative learning, as well as two forms of non-associative learning, habituation and sensitization. In the following section, an abstraction of these aspects of learning in *Aplysia* is presented. Note that the above description of biological synaptic function omits many interesting aspects of learning in *Aplysia*; see [2] for a more detailed discussion.

IV. Model of Synaptic Learning

As a starting point for the development of this learning model, we use a standard ANN model with Hebbian learning described in [4]. In this model, both synaptic weights and neuron activations can take on values in the range $[-V,V]$. Network operation is governed by the following equations:

$$V_i = f\left(\sum_j W_{ij} V_j\right) \tag{3}$$

$$\frac{dW_{ij}}{dt} = AV_i V_j - BW_{ij} \tag{4}$$

where V_i and V_j are the current activations of the i^{th} and j^{th} neurons, W_{ij} is the weighting factor determining the effect that the j^{th} neuron has on the i^{th} neuron's activation, and A and B are small constants. $f(\cdot)$ is a sigmoidal saturating non-linear function.

The current activation of the i^{th} neuron is computed from a weighted summation of the current activations of all neurons which synapse on neuron i (3). Network learning, or adaptation, is governed by (4), a common form of the Hebbian learning rule [7].

The neural network model presented above differs from neuron biology in two important ways. Neuron activations are represented as analog values in the model, rather than as neuron firing rates. Secondly, biological neurons only have positive (unipolar) activations and synaptic weights, whereas the above model allows both positive and negative (bipolar) activations and weights.

System behavior in the model of biological synaptic function proposed in the present paper is governed by the equations:

$$V_i = f\left(\sum_j \sum_k W_{ijk} V_j\right) \tag{5}$$

$$\frac{dW_{ijk}}{dt} = C\,\mathrm{d}(V_j)V_k - D \mid V_j \mid W_{ijk} + E \mid V_k \mid W_{ijk} \tag{6}$$

The ternary nature of these synapses is evident in (5) and (6): W_{ijk} is the weighting factor determining the effect that correlation between the activations of the j^{th} and k^{th} neurons will have on the activation of the i^{th} neuron. Hebbian learning, as described by (4), may be regarded as a special case of (6), in which $i = j$. Using the notation of the previous section, the unconditioned stimulus $US = V_{US} = V_k$, the conditioned stimulus $CS = V_{CS} = V_j$, and (6) may be rewritten (for motor neuron MN) as:

$$\frac{dW}{dt} = C\,\mathrm{d}(V_{CS})V_{US} - D \mid V_{CS} \mid W + E \mid V_{US} \mid W \tag{7}$$

where the synaptic weight W is a shorthand notation for $W_{ijk} = W_{MN,CS,US}$, and indices CS and US represent *neurons* SN_1 and FN_1 respectively, rather than their activations. The term $C\,\mathrm{d}(V_{CS})V_{US}$ implements classical conditioning, where $\mathrm{d}(V_{CS})$ is a delayed, time-averaged version of V_{CS}, representing the presynaptic facilitation function of F_1 in Fig. 3. Note that (7) differs from Hebbian learning, as it involves a correlation of two inputs, rather than an input and an output activation. V_{CS} must precede V_{US} by a critical interval for classical conditioning to cause a change in synaptic weight W, as in *Aplysia*, and the size and direction of the weight change are determined by the signs and magnitudes of V_{CS} and V_{US}, as in the system described by (4). Thus the system can learn *predictive* relationships between V_{CS} and V_{US}. Note that in addition to creating a delay, the function $\mathrm{d}(V_{CS})$ is *gated* by $V_{US} \neq 0$, such that classical conditioning will not occur when V_{US} precedes or coincides with V_{CS} (absence of reverse conditioning).

The second term in (7) represents habituation. Habituation occurs when V_{CS} is non-zero, and always causes W to decay towards zero. This extension of habituation to the bipolar case makes intuitive sense, since it causes non-associative weight decay when V_{CS} is active, as in habituation in *Aplysia*. The form of the expression is also appropriate, in light of Mead's observation, "A great deal of inhibitory feedback in biological systems depends on activity in sensory input channels, but does not depend on the *sign* of the input" [8].

Finally, the third term in (7) $E \mid V_{US} \mid W$ is the sensitization term. V_{US} activity causes an increase in the magnitude of the weight, resulting in increased sensitivity to subsequent V_{CS} signals.

In general, $C > E > D$, so associative learning dominates, and sensitization and habituation result in smaller non-associative weight changes. The role of habituation as a form of inhibitory (negative) feedback is apparent from

(7). Sensitization causes weight values to increase: however, the natural saturating behavior of any practical realization of (7) will limit the effects of sensitization.

V. Synaptic Learning Circuit

The analog CMOS circuits which we use to implement our synaptic learning circuit are compact, low-accuracy amplifiers, multipliers, absolute value circuits, etc [5]. The decision to use analog, rather than digital computation results in large silicon chip area savings, at the expense of computational accuracy and repeatability. However, the biological machinery of *Aplysia* is even less precise, so any neural network architecture that requires high accuracy components is not biologically plausible, and therefore not of interest in the present work.

The components which we are using are *transconductance* circuits, where the input signals are voltages and the output is a current. Many of these circuits are variants of the circuits used in [8], but unlike in Mead's work, the transistors in our circuits are operating above threshold ($V_{GS} > V_T$).

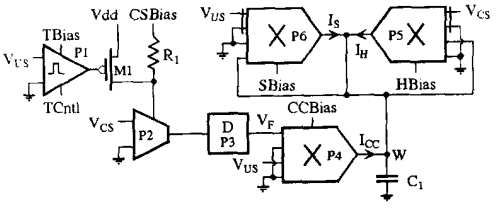

Figure 4 *Synaptic circuit.*

Fig. 4 is a bipolar synaptic learning circuit, built from simple analog components. This circuit approximates (7), incorporating classical conditioning, habituation, and sensitization. The synaptic weight is stored as the voltage W on capacitor C_1, and weight changes are governed by:

$$\frac{dW}{dt} = \frac{I_{CC} + I_H + I_S}{C_1} \qquad (8)$$

$$= \frac{cV_F V_{US} - d \mid V_{CS} \mid W + e \mid V_{US} \mid W}{C_1}$$

where I_{CC}, I_H and I_S are, respectively, the classical conditioning, habituation and sensitization weight change components. P_1 - P_4, M_1 and R_1 implement classical conditioning. Inhibition of presynaptic facilitation (prevention of reverse conditioning) is achieved as follows: when $\mid V_{US} \mid > V_{THRES}$, P_1 turns on M_1, which turns off P_2 by setting its bias current to zero. As a result, when $\mid V_{US} \mid > V_{THRES}$, V_{CS} has no effect on V_F, and therefore no classical conditioning can occur. Conversely, when $\mid V_{US} \mid < V_{THRES}$, P_2 is active, and classical conditioning can occur.

Absolute value multipliers P_5 and P_6 approximate habituation and sensitization. Note that W is connected to the negative input of P_5, since habituation drives W towards zero. Below, two examples are presented which illustrate the learning behavior of this synaptic circuit. For the purposes of illustration, higher than typical learning rates are used, so that weight changes are evident over the relatively short interval of these simulations.

A. Classical Conditioning

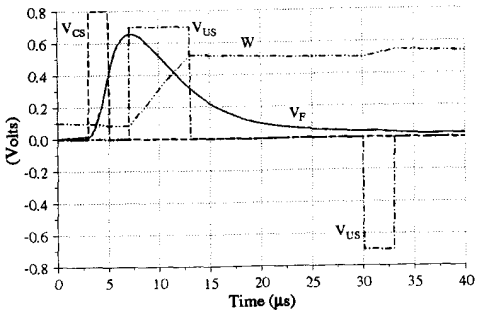

Figure 5 *Classical conditioning and sensitization. V_{CS} precedes V_{US} by critical interval, so classical conditioning occurs.*

Fig. 5 shows the synaptic circuit's response to a typical classical conditioning event. At $t = 3\mu s$, a $2\mu s$ conditioned stimulus (V_{CS}) pulse initiates a presynaptic facilitation pulse (V_F). At $t = 7\mu s$, the unconditioned stimulus (V_{US}) is presented, resulting in an increase in the weight W, as the correlation relation between V_{CS} and V_{US} is learned. The critical period for classical conditioning, that is, the time after V_{CS} occurs in which V_{US} must be presented for conditioning to occur, is approximately 2 to $8\mu s$. The shape of V_F can easily be changed by choosing different component values for the delay circuit, P_3.

At $t = 30\mu s$, V_{US} is briefly pulsed negative. The presynaptic facilitation signal V_F has decayed to almost zero, and thus no classical conditioning occurs. However, V_{US} does cause a slight increase in W, through the sensitization mechanism. Notice that despite V_{US} being negative at $t = 30\mu s$, W *increases*, since sensitization is independent of the sign of V_{US}. The slow decay of W from 13 to $30\mu s$ is due to a small component mismatch in P_4, P_5 and P_6.

B. Reverse Conditioning

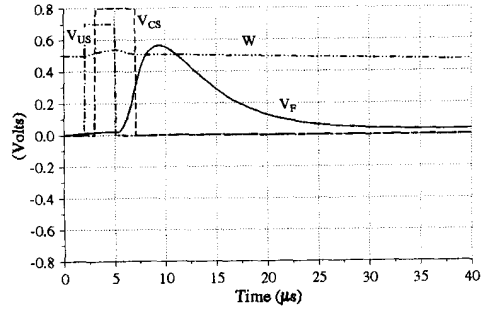

Figure 6 *Reverse conditioning. V_{US} occurs before V_{CS}, so no classical conditioning occurs.*

In the example of Fig. 6, V_{US} occurs before V_{CS}, and

thus no classical conditioning can occur. For $3\mu s < t < 5\mu s$, the presence of V_{US} is preventing V_{CS} from generating a V_F pulse, through the gating mechanism implemented with P_1 in Fig. 4. At $t = 5\mu s$, V_{US} is set to zero and V_F begins to increase. If a second V_{US} pulse were presented, for instance at $t = 10\mu s$, then classical conditioning would occur.

The small weight changes from $t = 2\mu s$ to $7\mu s$ are due to sensitization and habituation. During the interval $2\mu s < t < 3\mu s$, sensitization is occurring, during the interval $3\mu s < t < 5\mu s$, sensitization and habituation occur (the rate of weight increase is reduced), and during $5\mu s < t < 7\mu s$, only habituation occurs (the weight decays towards zero). Habituation also occurs during $3\mu s < t < 5\mu s$ in Fig. 5, but the effect on W was smaller, because the rate of habituation is determined by the product $DW \mid V_{CS} \mid$. Thus, the rate of habituation is always a fraction of the synaptic weight, rather than a fixed value.

VI. Implications for ANNS

The development of our biology-motivated learning model raises a number of interesting questions, including, whether the biological learning details presented in this paper are important in ANN models. We began with a conventional Hebbian model, generalizing and extending it to incorporate the functions of synaptic learning found in *Aplysia*. Thus Hebbian learning (4) is a special case of (7), in which the delay of d(·) is zero, $i = j$, and reverse conditioning is allowed.

The effect of non-zero delay in d(·), together with inhibition of reverse conditioning, is that learning becomes non-commutative. This "symmetry breaking" would result in non-symmetric weights ($W_{ijk} \neq W_{ikj}$) in the case of a fully-connected, Hopfield-type network. This type of non-commutative learning could be incorporated into networks using contrastive Hebbian learning [9], again resulting in non-symmetric weights. Further work is planned to investigate this possibility.

The non-associative learning found in biological systems, habituation and sensitization, may also have a role to play in ANNs. Their importance, and how to go about incorporating them into an artificial system is less clear than with non-commutative learning, because non-associative learning is not used in many ANNs. However, they are obviously important in biological neural networks, as shown by the study of a variety of animals, and therefore must be considered for inclusion in ANNs.

An important issue is whether the biological synaptic learning presented in this work depends upon a certain degree of "hard-wiring". For example, *Aplysia* has been "wired" so that it can learn a predictive relationship between mild mantle stimuli and noxious tail stimuli. If the neural network of Fig. 3 were missing the facilitating interneuron FN, then neither sensitization nor classical conditioning could occur. There are two interpretations of this "pre-wiring": it is evidence that the biology of *Aplysia* is not of interest to ANN researchers, because it represents a special case in which pre-determined network architecture plays a major role; or, it is an indication that tailoring a network architecture for a particular purpose is an essential part of neural networks, whether natural or artificial. Our tendency is towards the latter view, although the issue is far from settled.

VII. Conclusion

This work has demonstrated that biological details, which are omitted from most current ANN models, may be efficiently implemented with analog CMOS circuits. Three types of learning, habituation, sensitization and classical conditioning, are incorporated in the synaptic learning circuit which we have developed. Habituation and sensitization are types of non-associative learning, and are not often included in ANN models. Classical conditioning is a form of associative learning, related to Hebbian learning. It differs from Hebbian learning in temporally correlating two inputs at ternary synapses (synapses between three neurons), rather than the input and output of binary synapses. Classical conditioning is also more complex, as it is non-commutative, resulting in a type of "symmetry breaking" in the neural network system. Our work shows that biological synaptic learning is substantively different from current ANN learning, both in terms of learning paradigms employed, and in terms of the extensive use of "hard-wiring" of connections in biological neural networks. Further research is required to determine whether these aspects of neural biology are a critical part of information processing in biological and artificial neural networks.

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