

Conjoint Contribution of Cognition and Emotion on the Control of Thought and Behavior Among Learners by Artificial Neural Networks

Zeny Maureal*

*Bukidnon State University, Malaybalay City, Bukidnon, Philippines, <https://orcid.org/0009-0005-9817-7932>, zenymaureal@buksu.edu.ph

Abstract: The study developed new neural models for integrating cognition and emotion and assessing the conjoint contribution of the integrated neurons on the control of outputs (thoughts and behaviors) of learners. The neural model is then fed into different learning mechanisms, notably, error-minimization learning & Hebbian learning to determine the results of integration. Results revealed that (i) (i) in general, the integration of cognition and emotion in a single neuron requires a greater number of training samples to achieve the same level of learning efficiency as in pure cognition models; (ii) a coupled neuron in which the emotion and cognition activities are inseparable leads to learning cycles and divergence in which the student never learns regardless of the learning mechanism used; and (iii) an uncoupled neuron on which the affective and cognitive neurons are integrated with separate synaptic weights can be trained but with more training samples than is normally required for cognitive learning. In the latter case, the restriction $w_{j,i} > \eta_{j,i}$ implies that the teacher must begin by motivating the learner in order to compensate for a negative affective neuron and must consistently do so throughout the lesson in order for the neural synapses to be trained, i.e., for the learners to understand the subject matter. In more practical terms, the results also indicate that in terms of pedagogy, more teacher-learner contacts will be required to achieve acceptable learning efficiency levels. Further, at each contact, the synaptic weights corresponding to positive emotions will need to be increased while the synapses corresponding to negative emotions will need to be decreased.

Keywords: artificial neural networks, synaptic weights, cognition-emotion interaction, pedagogy

1. INTRODUCTION

The development of a child's cognitive abilities has been the focus of most efforts in the current educational process, largely neglecting the important affective dimension of the individual learner. In the past, these two dimensions (cognitive and affective) have been treated as separate concerns in education, but current research has demonstrated the strong interdependence between these two. At some point of processing, functional specialization is lost, and emotion and cognition conjointly and equally contribute to the control of thought and behavior (Gray et al., 2002). In other words, many behaviors can be reasonably characterized by cognitive-emotional interactions, which allow for some separation between emotion and cognition, but in many situations, true integration of both emotion and cognition may also occur. The latter further blurs the distinction between cognition and emotion. (See Duncan and Barret (2007) for a similar view.)

How do emotion and cognition integrate to control a learner's thoughts and behavior? The approaches to derive partial answers to this question have been diverse: social science approaches, mainly educational psychology, to the more scientific neuroscience approach (see a review by Pessoa (2009)). Each approach used in the past had its distinctive strengths and weaknesses, and what the researcher is proposing to do in this research is to examine the same question from an artificial neural networks (ANN) point of view. Out of such a mathematical approach to the problem, learning theories can be derived. The study of learning theories has two chief values: one is in providing us with vocabulary and a conceptual framework for interpreting the examples of learning that we observe. The other is in suggesting where to look for solutions to practical problems. The theories do not give us solutions, but they do direct our attention to those variables that are crucial in finding them.

The potential implications of this study are significant, particularly in the realms of education, artificial intelligence, and psychological interventions. By integrating cognition and emotion into artificial neural network (ANN) models, this research could revolutionize personalized learning systems in educational technology, enabling adaptive platforms that respond to both the cognitive progress and emotional states of learners. For instance, e-learning platforms could detect and respond to frustration or anxiety in students, adjusting instructional methods to maintain engagement and reduce stress. Additionally, such models could improve human-computer interaction by making AI systems more empathetic and responsive, fostering trust and usability. In psychology and mental health, these integrated models could aid in developing tools for understanding and predicting emotional-cognitive interactions, potentially guiding interventions for emotional regulation or cognitive impairments. Moreover, industries relying on behavior prediction, such

as marketing or customer service, could benefit from ANN systems that account for emotional dynamics, creating more effective and personalized user experiences.

Artificial neural networks (ANN) reduce the activities in the human brain to an analysis of the behavior of artificial neurons and the synapses that connect them to one another. Mathematical models of learning are then imposed on these quantities, which, to date, have been limited to error-minimization learning, Hebbian learning, competitive learning, Boltzmann learning, and distance-minimization learning, all of which are cognitive models. However, they neglect the important component of human emotions in learning. This study, therefore, proposes to modify some of these neural network models of learning to reflect the integration of cognition and emotion in learning. A chief advantage of such an approach is the ease with which we may be able to manipulate the parameters of the mathematical models derived to predict outcomes and to control behaviors of the system.

2. State of Research on Cognition-Emotion Interaction

With the advent of functional MRI (fMRI), it seems that cortical regions of the brain are engaged by cognitive processes (Gazzaniga et al. 2008). While there is clear discussion about what constitutes cognition, the same cannot be said about emotion. Some incorporate emotions into the concepts of drive and motivation. They view it as a state elicited by rewards and punishment or related to the conscious (or unconscious) evaluation of events like appraisals (Rolls, 2007; Arnold, 1960). Some approaches concentrate on basic emotions (fear, anger, happiness) (Ekman, 1992) or an extended set of emotions (e.g., pride, envy), including moral ones (Haidt, 2003; Moll et al., 2005). There is also a strong evidence linking emotions to the body (Damasio, 1994). Brain structures associated with emotion are often subcortical, such as the amygdala, hypothalamus, and ventral striatum, which are often considered evolutionarily conserved, or primitive. They are also assumed to operate fast and in an automatic manner. Accordingly, an individual may be unaware of a stimulus that caused brain responses in an affective brain region, such as the amygdala. For discussion, see (Ohman, 2002; Pessoa, 2005). In the human brain, the amygdala is known to be a critical structure for the enhancement of memory by emotion, mediating many aspects of emotional learning and behaviour (Salzman, 2022). Recent studies have begun to delineate some of the specific functions of this structure. For example, it is believed that the right amygdala is highly engaged in emotional memory formation, whereas the left amygdala is involved in the retrieval of those memories (Sergeje et al 2006). This suggests a potential hemispheric dissociation of amygdala involvement at different stages of emotional memory. Furthermore, amygdala responses are also linked to a novelty effect on memory tasks. This means that it can classify items as new rather than old (Sergeje et al., 2007).

Another important line of studies that has investigated cognitive-emotional interactions is cognitive emotion regulation, particularly cognitive appraisal (Ochsner and Gross, 2005; Ochsner and Gross 2008). This involves rethinking the meaning of affectively charged stimuli or situations in terms that will affect them emotionally. Reappraisal appears to depend upon interactions between prefrontal and cingulate regions that are frequently involved in cognitive control and systems like the amygdala and insula that have been connected in emotional responding. Remarkably, thinking about stimuli that keep or increase emotion may boost amygdala activity, while having the thought to decrease emotion may lessen it. Furthermore, changes in emotional experience and autonomic responding may correlate with the increase or decrease of prefrontal or amygdala activity. Considering the anatomical information will also help us understand the relationship between emotion and cognition. Developments in the understanding of brain connectivity suggest that a given brain region is only a few synapses away from every other brain region (Sporns et al. 2004; Sporns and Zwi, 2004). The brain is configured according to a small-world topology in which the path length between nodes is small—typically cortical areas are connected directly or via just one or two intermediate areas (Hilgetag et al., 2000; Sporns et al., 2000) and nodes are highly clustered (Sporns, 2006). As shown in figure 1, the amygdala was connected to all but 8 cortical areas. These connections involved multiple region clusters, which suggests that the amygdala is not only highly connected, but also its connectivity topology might be consistent with that of a hub that links multiple functional clusters. It looks like the amygdala is very well situated to integrate and distribute information. In this manner, the amygdala may be important for the integration of cognitive and emotional information. Hence, a careful consideration of brain connectivity is informative and significant in understanding potential cognitive-emotional interactions.

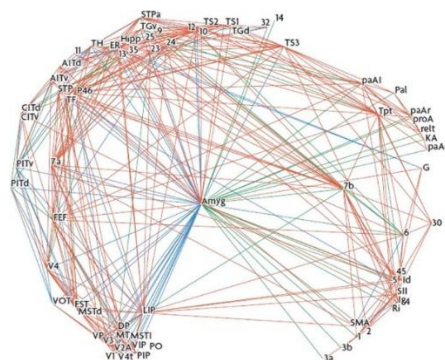


Figure 1. Brain Connectivity Graph. Figure reproduced from Young et al (1994) with permission from Freund Publishing House Ltd. Analysis of connectivity: Neural systems in the cerebral cortex, Reviews in the Neurosciences; copyright (1994).

It is also informative to consider connectivity of the hypothalamus (Risold et al., 1997). It has long been acknowledged for its importance in emotional behaviours (Swanson, 2000, 2003). In particular, via its descending connections that excite brainstem motor systems, this structure is believed to have a crucial role in the implementation of goal-directed behaviors. Hypothalamic signals also can be delivered to the cortex, frequently through the thalamus. Critically, prefrontal cortical territories project directly to the hypothalamus. Thus, the hypothalamus appears to be organized in such a way that it can generate both relatively reflexive behaviors and behaviors that are voluntarily triggered by inputs from the cerebral cortex (Swanson, 2000). Overall, this structure appears to be connected with all levels of the nervous system, including the neocortex (Swanson, 2000), enabling important hypothalamic regulatory signals to have widespread effects on the brain.

For past years, emotion and cognition have been viewed as separate entities. However, recent research suggests that such a view is likely inadequate and that, in order to understand how complex behaviors are carried out in the brain, an understanding of the interactions between the two may be necessary. In fact, some suggested not only focusing on their interactions but also their integration. That is, understanding how cognition and emotion are effectively integrated in the brain. Whereas many behaviors may be reasonably well characterized in terms of cognitive-emotional interactions such that emotion and cognition are partly separable, true integration of emotion and cognition can also occur in many situations. This emphasized the obscure distinction between cognition and emotion.

3. Methods

This study utilized an artificial neural network (ANN) model consisting of input, hidden, and output layers with connected neurons, which emulate a biological neural network, specifically simulating the electrical activity of the brain. Neural models considered here are coupled and uncoupled neuron models. These neural models will be inputted to the learning mechanisms, namely, the error-minimization learning pattern and the Hebbian learning mechanism.

4. The Proposed Neural Models and Learning Mechanisms

The notations to be adopted are: English letters are used for cognition neurons and synapses; thus, $\{x_j\}$ denotes the input neurons and $\{y_i\}$ are the output neurons while $\{w_{j,i}\}$ denotes the synaptic weights from the neuron j to neuron i . Greek letters will be used for affective neurons, thus, (θ_j) denotes the input affective neurons, and the synaptic weights are $(\eta_{j,i})$ from neuron (θ_j) to output $\{y_i\}$.

a. Neural Model 1
(Coupled Neuron Model)

In the first model, neuron i receives inputs from neuron j in the form of a cognition-emotion interaction $x_j\theta_j$ with synaptic weight $w_{j,i}$. The activation function at neuron i is :

$$v_i = g(\sum_j w_{j,i}x_j\theta_j) \quad (1)$$

The neuron θ_j is a binary variable +1 for positive emotions and -1 for negative emotions. The activation function $g(.)$ can be the threshold function or the sigmoidal function.

b. Neural Model 2.
(Uncoupled Neuron Model)

In this model, a neuron i receives inputs from the two sources: the cortical (or cognition) neurons x_j and subcortical (emotion) neurons θ_j . The summator at neuron i performs the uncoupled summing:

$$\eta_{j,i}\theta_jx_j = \sum_j (w_{j,i}x_j + \eta_{j,i}\theta_jx_j) \quad (2)$$

This neuron model says that information passed on to neuron i consists of two parts: the pure cognition part $w_{j,i}x_j$ and the conjoint part $\eta_{j,i}\theta_jx_j$. Note that the number input cognition neuron x_j need not equal to the number of input affective neuron θ_j . We assume that whenever there is no affective input to a cognitive input, then $\theta_j = 1$ and $\eta_{j,i} = 0$.

c. Error-Minimization
Learning Pattern (EML)

We will now input these two neuron models into our learning mechanisms. The first mechanism is the EML, in which the learner's objective is to minimize:

$$E_i = \frac{1}{2}(y_i - v_i)^2 = \frac{1}{2}(y_i - g(s_i))^2 \quad (3)$$

Inputting a Coupled Neuron Model

When $s_i = \sum_j w_{j,i} \theta_j x_j$, equation (3) becomes

$$E_i = \frac{1}{2}(y_i - v_i)^2 = \frac{1}{2}\left(y_i - g\left(\sum_j w_{j,i} \theta_j x_j\right)\right)^2 \quad (4)$$

To update the synaptic weights $w_{j,i}$, we need to compute the derivative:

$$\frac{\partial E_i}{\partial w_{j,i}} = (-Err_i)(g'(s_i)\theta_j x_j) \quad (5)$$

Where $Err_i = (y_i - v_i)$ and $g'(s_i)$ is the derivative of the activation function. Let $\Delta_i = (-Err_i)(g'(s_i))$, then (5) becomes

$$\frac{\partial E_i}{\partial w_{j,i}} = \Delta_i \theta_j x_j \quad (6)$$

From this, we derive the updating rule:

$$w_{j,i}(new) = w_{j,i}(old) + \alpha \Delta_i \theta_j x_j \quad (7)$$

Where α is the learning rate.

The impact of the affective neuron θ_j on the updating rule can be easily seen from (6). If it happens to equal +1, then the usual iterations or gradient descent method will be able to train the synapse $w_{j,i}$. However, if $\theta_j = -1$, then the gradient descent method can diverge. In other words, no matter how many trainings are conducted, the synapse $w_{j,i}$ will not stabilize and the individual never learns.

Illustrative Example 1. We illustrate what happens when $\theta_j = -1$ for learning the rule $y_i = 2x_j$. Column shows the fast back propagations algorithm when $\theta_j = +1$ while column 2 shows what happens when $\theta_j = -1$. The learning rule is $\Delta w_{j,i} = \frac{\nabla E_n}{\nabla E_{n-1} - \nabla E_n} \nabla w_{j,i}$.

	Column 1: $\theta_j = +1$						Column 2: $\theta_j = -1$					
	x_i	y_i	w_i	E_i	$\Delta w_{j,i}$	w_{new}	x_i	y_i	w_i	E_i	$\Delta w_{j,i}$	w_{new}
	2	4	0.4	3.2	0.4	0.8	2	4	0.4	4.8	0.4	0.8
	2	4	0.8	2.4	-0.6	0.2	2	4	0.8	5.6	2.8	3.6
	4	2	0.2	3.6	1.8	2	2	4	3.6	11.2	5.6	9.2
	2	4	2	4	0	0	2	4	9.2	22.4	11.2	20.4

	$w_{new} = 0$ means the network learned in 4 th iteration Synaptic weight =2	$\vdots$ As $i \rightarrow \infty$, divergence detected
--	---	---

A student with a negative “affect” can, potentially, never learn. This is not necessarily because he is dull, but his emotional disposition prevents him from learning. This is particularly useful in mathematics education, where most students already “dislike” mathematics. A student’s dislike for mathematics can be due to (a) frustrating experiences where he seldom gets the correct answer, (b) teachers who are severe or not knowledgeable enough, or (c) pre-conditioning based on experiences of other students. Also note that when the neuron model inputted in the learning mechanism is neuron model 1, there is no way by which the negative impact of an affective neuron can be controlled by the teacher.

i. Inputting the Uncoupled Neuron

In the coupled neuron model, we see that the educative process can do very little in adjusting the synaptic weights of the affective neurons. Consider what happens when the uncoupled neuron is inputted to the activation function. We obtain:

$$g(s_i) = g(\sum_j (w_{j,i} + \eta_{j,i}\theta_j) x_j) \quad (8)$$

Let us simplify (8) by letting $w_{j,i}^* = w_{j,i} + \eta_{j,i}\theta_j$ to obtain

$$(s_i) = g(\sum_j w_{j,i}^* x_j) \quad (9)$$

Additionally, we put the restriction $w_{j,i} - \eta_{j,i}\theta_j \geq 0$. Expression (9) is completely analogous to the pure cognition model. The last restriction states that the affective synaptic weight should be less than the cognition synapse, for otherwise, divergence will happen again and no learning will take place. The derivative of the error E_i with respect to $w_{j,i}^*$ is :

$$\frac{\partial E_i}{\partial w_{j,i}^*} = (-Err_i)g'(s_i) \cdot [1 + \sum_j \eta_{j,i}\theta_j]x_j \quad (10)$$

$$\frac{\partial E_i}{\partial w_{j,i}^*} = \Delta_i \cdot \left[1 + \sum_j \eta_{j,i}\theta_j \right] x_j$$

From which:

$$w_{j,i}^*(new) = w_{j,i}^*(old) + \alpha \Delta_i \left[1 + \sum_j \eta_{j,i}\theta_j \right] x_j$$

Illustrative Example 2. We use the sample problem earlier, which tries to discover the simple rule $y = 2x$ for a student with negative affect $\theta_j = -1$. Recall that in illustrative example 1, this led to a divergence.

Iteration i	x_i	y_i	w_i	η_i	E_i	* $w_{j,i}$
1	2	4	0.4	0.2	1.6	0.2

* $w_{j,i}(new) = 0.2 + 0.2 = 0.4$ where * $w_{j,i}(new) = w_{j,i}(new) - \eta_i(new)$. In the next stage of the iteration, we consider only the value of the updated synapse * $w_{j,i}$ in the calculations.

Iteration i	x_i	y_i	$w_{j,i}$	E_i	* $w_{j,i}$	* $w_{j,i}(new)$
2	2	4	0.4	3.2	0.4	0.8
3	2	4	0.8	2.4	-0.6	0.2
4	2	4	0.2	3.6	1.8	2.0
5	2	4	2.0	0		

In the 5th iteration, the error will be zero, and the neuron has learned. The final synaptic weight is 2.0.

We note that when there is no affective component in the EML, the same problem required only n=2 iterations. However, when the affective component is inputted, we needed at least n=3 iterations. The first training, actually, took care of the affective component, i.e., the first contact should aim to stabilize the affective/emotional disposition of the student.

If we now compare the two types of input neurons to an EML learning process, we see that the second neural model allows us to control the impact of a negative affective neuron whereas we are unable to do so in the first neural model. Biologically, we can imagine a cortical neuron firing and sending electrical signals directly to an output neuron and also to a subcortical neuron where the affective and cognitive signals are conjoined and sent to the output neuron. This happens at the amygdala, as reviewed earlier.

In terms of pedagogy, the teacher's strategy must always begin with motivation to ensure that the negative affective synaptic weight will always be less than the cognitive synaptic weight in magnitude. While this was practiced long ago in lesson planning, there was no scientific reason provided. As a matter of fact, motivation was not recognized as a central part of the lesson plan (it was treated as a one-paragraph introduction). Now we realized that the entire learning process can potentially result in no learning if this aspect is not properly done. Secondly, the restriction that $w_{j,i} > \eta_{j,i}$ at each iteration implies that motivation and interests must be maintained all throughout the lesson. This puts a lot of pressure on the teachers who need to devise ways of keeping students' interests high at every stage of the lesson given that the attention span of a typical student is less than 10 minutes.

d. Hebbian Learning Mechanism

There is evidence that biological neurons, particularly those observed in the hippocampus, behave in a Hebbian learning fashion. Consider two neurons j (the input or pre-synaptic neuron) and I (the output or post-synaptic neuron). When the pre-synaptic neuron fires and the post-synaptic neuron fires as well, then the synaptic connection between the two neurons is strengthened. This is the essence of Hebb's hypothesis. In a nutshell, according to Hebb, a positive correlation between the two neurons increases the synaptic weight, while a zero or negative correlation decreases the strength of the synaptic connection.

Hebbian synapses are highly localized such that spatio-temporal contiguity of neurons is required. Additionally, Hebbian synapses are time-dependent, meaning that the synchronicity of firing among contiguous neurons is essential. Let $\Delta w_{k,j}(n)$, $y_k(n)$ and $x_j(n)$ denote the change in synaptic weight, output neuron and input at time n . A Hebbian modification of the synaptic weight consists of a function F of the input and output neurons at time n .

$$\Delta w_{k,j}(n) = F(y_k(n), x_j(n)) \quad (11)$$

Hebb's hypothesis claims that the function $F(\cdot)$ is

$$\Delta w_{k,j}(n) = \alpha y_k(n) x_j(n), \text{ for } n = 1, 2, \dots, k \text{ where } \alpha \text{ is the learning rate} \quad (12)$$

The last equation is called the Activity Product Rule in the literature. This synaptic weight equation is sometimes modified by the covariance hypothesis.

$$\Delta w_{k,j}(n) = \alpha (y_k - \bar{y})(x_j - \bar{x}) \quad (13)$$

where $\bar{x}$ is the time-averaged value of x_j

$\bar{y}$ is the time-averaged value of y_k

Illustrative Example 3. Hebbian learning can be illustrated for the same simple problem of discovering $y = 2x$.

x_i	y_i	$w_{j,i}$	E_i	Δw	$w_i(\text{new})$
2	4	0.4	3.2	1.6	2.0

Following Hebb's hypothesis and assuming $\alpha = 1$, we see that the synapse is trained after one iteration.

Inputting Neuron Model 1

If we input neuron model 1 into the Hebbian hypothesis, we obtain:

$$\Delta w_{k,j} = \alpha (w_{k,j} x_j \theta_j) x_j \quad (14)$$

$$w_{\text{new}} = w_{k,j} + \alpha w_{k,j} \theta_j x_j^2$$

If $\theta_j = -1$ is a negative affective neuron then (14) becomes:

$$\alpha x_j^2) w_{k,j} \quad w_{\text{new}} = (1 - \quad (15)$$

Example 4. Let us examine what happens when this neuron is inputted into the previous problem with

$\alpha = 1$.

iteration	x_i	y_i	w_j	E_i	Δw	$w_i(new)$
1	2	4	0.4	4.8	-1.6	-1.2
2	2	4	-1.2	1.6	4.8	3.6
3	2	4	3.6	11.2	-14.2	-11.2

Again, we see the phenomenon of cycling and divergence. The student with a negative affective neuron does not learn even under the more efficient Hebbian learning mechanism. Further, because the affective and cognitive neurons are coupled, the teacher cannot intervene in the process.

Inputting Neuron Model 2

We next input neuron model 2 into the Hebbian learning mechanism. In this case, Hebb's hypothesis becomes:

$$\Delta_{w_{k,j}}(n) = \alpha[w_{k,j} + \eta_{k,j}\theta_j]x_j \cdot x_j$$

$$\Delta_{w_{k,j}}(n) = \alpha[w_{k,j} + \eta_{k,j}\theta_j]x_j^2 \quad (16)$$

$$w_{new} = w_{k,j} + \alpha[w_{k,j} + \eta_{k,j}\theta_j]x_j^2 \quad (17)$$

For $\theta_j = -1$ and $\alpha = 1$:

$$w_{new} = [1 + \alpha x_j^2]w_{k,j} - \eta_{k,j}x_j^2 \quad (18)$$

Prior to applying this learning rule, we introduce the concept of 'clamping' synaptic weights. This concept is applied in another learning rule called Boltzmann learning in statistical mechanics. This idea can be stated as follows. We adjust the synaptic weights of the cognitive and affective neurons separately. When the cognitive synaptic weights stabilize, we clamp it to that value. When the synaptic weight exceeds unity, we clamp it to the value before it. We will discuss the significance of this clamping technique after the example.

Illustrative Example 5. We utilize the same parameters as before, as summarized in the table below. Here, O_n and O_w are the outputs of affective and cognitive neurons computed separately.

x_i	y_i	w_j	η_j	θ_j	O_n	O_w	O	Error Cognitive	Error Affective	Error Total
2	4	0.4	0.2	-1	-0.4	0.8	0.4	3.2	0.4	3.6
$\Delta w = 2(0.8) = 1.6$ $w_{new} = 0.4 + 1.6 = 2.0$								$\Delta \eta = 2(0.4) = 0.8$ $\eta_{new} = 0.2 + 0.8 = 1.0$ Clamped		
Clamped $w_{new} = 2.0$									Error	
x_i	y_i	w_j	η_j	θ_j	O_n	O_w	O	Cognitive	Affective	Total
2	4	2.0	1.0	-1	-2.0	4.0	2	0	2	2

This illustrative example shows that one can control the cognitive and affective synaptic weights separately. If there were no affective neuron involved, the Hebbian learning mechanism correctly identifies the weight $w = 2$. However, the affective neuron prevents the correct identification of this cognitive synapse, and consequently, the optimal (minimum error) is 2. Further iteration only serves to increase the error exponentially.

In practice, however, we would only observe the pairs (x_i, O_i) , e.g. (2,4), (2,2), and (2,10) had we continued the iterations above. We are asked to determine the appropriate synaptic weights that train both the affective and cognitive neurons. Since the training output is known to be $y = 4$, the network can compute the successive errors $(4 - 0.4) = 3.6$, $(4 - 2) = 2$, and $(4 - 10) = -6$, which says that the second output has the least error (in magnitude). Thus the output (2,2) is the output of interest. At this output, we assume that the affective synaptic weight is clamped to one, that is, the value of $\eta = 1$. It follows that:

$2(w - \eta) = 2 \rightarrow w = 2$ is the correct cognitive synaptic weight. This means that if we can successfully remove the negative affect of a student, the correct output would be:

$$\text{Output} = wx = 4$$

which tallies with the training sample output perfectly.

Summary of Findings of Interest in Educational Theory:

1. The configuration of the neurons in the brain is such that the cortical (cognitive) neurons and the subcortical (affective) neurons may or may not be received as coupled information by a post-synaptic neuron.
2. In the coupled case, the *amygdala* sends a perfect integrated cognitive-affective information $w_{j,i}\theta_j x_j$ to the *hippocampus*, while in the uncoupled case, a cortical neuron sends a cognitive information $w_{j,i}x_j$ and an integrated cognitive-affective information $\eta_{j,i}\theta_j x_j$ to the *hippocampus*.
3. If the individual's neuron configuration is coupled, then regardless of the learning style or mechanism used to learn, the synaptic weights cannot be trained. In the case of the error-minimizing learning style (EML), the errors will fluctuate and ultimately diverge; in the case of a Hebbian learning mechanism, the errors will grow exponentially without bound.
4. If the individual's neuron configuration is uncoupled, the learning takes place but at a slower pace than when the affective domain is neglected. In the case of the EML learning style, provided that the cognitive synaptic weights are kept greater than the affective synaptic weights at each stage of the lesson, the student ultimately learns the subject matter; in case of the Hebbian learning style, the affective synaptic weight must be clamped to one through the teacher's active engagement of the learner, and eventually, the cognitive synaptic weights are trained so that the student learns as well.
5. These findings have implications for lesson planning. Motivation, the first part of every lesson, is an underrated part of a lesson plan. The findings of the study show that this is the most important part of the lesson plan for without it, there is a large possibility that the student might never learn the subject matter. Furthermore, each part of the lesson plan must take into account the affective dimension, ensuring that the student's interest never wanes. An attention span of 10 minutes for a typical student implies that the teacher needs to change activities once every 10 minutes.

Acknowledgement:

The author highly acknowledged the late Dr. Roberto N. Padua for the significant input and motivation he gave to the researcher and the research. Additionally, heartfelt gratitude to BukSU-Center for Mathematical Innovations for the financial support.

References:

1. Adolphs R., Cahill L., Schul R., Babinsky R (1997) Impaired declarative memory for emotional material following bilateral amygdala damage in humans. *Learn Mem* 4: 291-300
2. Alheid GF, Heimer L. (1988) New perspective in basal forebrain organization of special relevance for neuropsychiatric disorders: the striatopallidal, amygdaloid, and corticopetal components of substantia innominate. *Neuroscience* 27:1-39
3. Amaral DG, Price JL, Pitkanen A, Carmichael ST (1992) Anatomical organization of the primate amygdaloid complex. In: *The Amygdala neurobiological aspects of emotion, memory, and mental dysfunction* (Aggelton J., ed), pp 1-66. New York: Wiley-Liss.
4. Anderson AK (2005) Affective influences on the attentional dynamics supporting awareness. *J Exp Psychol Gen* 134:258-281.
5. Anderson AK, Phelps EA (2001) Lesions of the human amygdala impair enhanced perception of emotionally salient events. *Nature* 411: 305-309
6. Anderson AK, Christoff K, Panitz D, De Rosa E, Gabrieli JD (2003) Neural correlates of the automatic processing of threat facial signals. *Journal of Neuroscience* 23: 5627-5633.
7. Arnold MB (1960) *Emotion and Personality*. New York: Columbia University Press.
8. Aron AR, Robbins TW, Poldrack RA (2004) Inhibition and the right inferior frontal cortex. *Trends Cogn Sci* 8: 170-177.
9. Barbas H (1995) Anatomic basis of cognitive-emotional interactions in the primate prefrontal cortex. *Neuroscience and Biobehavioral Reviews* 19:449-510.
10. Bargh JA (1997) The automaticity of everyday life. In: *Advances in social cognition* (Wyes Jr. RS, ed) pp 1-61. Mahwah, NJ: Erlbaum
11. Bishop SJ (2007) Neurocognitive mechanism of anxiety: an integrative account. *Trends Cogn Sci* 11: 307-316.
12. Bishop SJ, Duncan J, Lawrence AD (2004) State Anxiety modulation of the amygdala response to unattended threat-related stimuli. *J Neurosci* 24: 10364-10368.
13. Bishop SJ, Jenkins R, Lawrence AD (2007). Neural processing of fearful faces: effects of anxiety are gated by perceptual capacity limitations. *Cereb Cortex* 17: 1595-1603.
14. Bornstein R. (1989). Exposure and affect: Overview and meta-analysis of research, 1968-1987 *Psychological Bulletin* 106: 265-289.
15. Bradley MM, Greenwald MK, Petry MC, Lang PJ (1992) Remembering pictures, pleasures and aroused in memory. *J Exp Psychol Learn Mem Cogn* 18: 379-390.

16. Cahill L, Haier RJ, Fallon J, Alkire MT, Tang C, Keator D, Wu J, McGaugh JL (1996). Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proc Natl Acad Sci USA* 93: 8016-8021
17. Damasio AR (1994) *Descartes' error: Emotion, reason, and the human brain*. New York: G.P. Putnam
18. Damasio AR (1999). *The feeling of what happens: body and emotion in the making of consciousness*. New York: Harcourt Brace.
19. Dolan R (2003) Emotion, cognition and behavior. *Science* 298: 1191-1194.
20. Duncan S, Barrett LF (2007). Affect is a form of cognition: A neurobiological analysis. *Cognition and Emotion* 21:1184-1211
21. Eastwood JD, Smilek D, Merikle PM (2001). Differential attentional guidance by unattended faces expressing positive and negative emotion. *Percept Psychophys* 63: 1004-1013.
22. Ekman P (1992) An argument for basic emotions. *Cognition and Emotion* 6:169-200.
23. Etkin A, Klemmehagen KC, Dudman JT, Rogan MT, Hen R, Kandel ER, Hirsch J (2004) Individual differences in trait anxiety predict the response of the basolateral amygdala to unconsciously processed fearful faces. *Neuron* 44: 1043-1055.
24. Fazio R, Sanbonmatsu D, Powell M, Kardes F (1986) On the automatic activation of attitudes. *Journal of Personality and Social Psychology* 50: 229-238.
25. Funayama ES, Grillon C, Davis M, Phelps EA (2001) A double dissociation in the affective modulation of startle in humans: effects of unilateral temporal lobectomy. *J Cogn Neurosci* 13: 721-729
26. Fuster JM, Alexander GE (1971) Neuron activity related to short-term memory. *Science* 173: 652-654
27. Gazzaniga MS, Ivry RB, Mangun GR (2008). *Cognitive neuroscience* (3rd edition) W.W. Norton & Company.
28. Goldstein M, Brendel G, Tuescher O, Pan H, Epstein J, Beutel M, Yang Y, Thomas K, Levy K, Silverman M, Clarkin J, Posner M, Kernberg O, Stern E, Silbersweig D (2017). Neural substrates of the interaction of emotional stimulus processing and motor inhibitory control: an emotional linguistic go/no-go fMRI study. *Neuroimage* 36: 1026-1040.
29. Gray JR, Braver TS, Raichle ME (2002). Integration of emotion and cognition in the lateral prefrontal cortex. *Proceedings of the National Academy of Sciences USA* 99: 4115-4120.
30. Haidt J (2003) The moral emotions. In: *Handbook of Affective Sciences* (Davidson RJ, Scherer KR, Goldsmith HH, eds) pp 852-870. Oxford: Oxford University Press.
31. Heimer L, Van Hoesen GW (2006). The limbic lobe and its output channels: implications for emotional functions and adaptive behavior. *Neurosci Biobehav Rev* 30: 126-147.
32. Hilgetag CC, Burns GA, O'Neill MA, Scannell JW, Young MP (2000) Anatomical connectivity defines the organization of clusters of cortical areas in the macaque monkey and the cat. *Philos Trans R Soc Lond B Biol Sci* 355: 91-110
33. Hsu SM, Pessoa L (2007). Dissociable effects of bottom-up and top-down factors on the processing of unattended fearful faces. *Neuropsychologia* 45: 3075-3086.
34. Hugdahl K, Ohman A (1977) Effects of instruction on acquisition and extinction of electrodermal responses to fear-relevant stimuli. *J Exp Psychol [Hum Learn]* 3: 608-618.
35. Kubota K, Niki H (1971) Prefrontal cortical unit activity and delayed alternation performance in monkeys. *Journal of Neurophysiology* 34: 337-347.
36. Kunst-Wilson WR, Zajonc RB (1980) Affective discrimination of stimuli that cannot be recognized. *Science* 207:557-558.
37. Lazarus RS (1984) On the primary of cognition. *American Psychologist* 39: 124-129.
38. LeDoux JE (1996) *The emotional brain*. New York: Simon & Schuster.
39. Leventhal H, Scherer KR (1987). The relationship of emotions to cognition: A functional approach to a semantic controversy. *Cognition & Emotion* 1: 3-28.
40. Moll, Jorge & de Oliveira, Ricardo & Zahn, Roland & Grafman, Jordan. (2008). The cognitive neuroscience of moral emotions. *Moral psychology*. 3.
41. Öhman, A. (2002). Automaticity and the amygdala: Nonconscious responses to emotional faces. *Current Directions in Psychological Science*, 11(2), 62–66. <https://doi.org/10.1111/1467-8721.00169>
42. Pessoa, Luiz (2005). To what extent are emotional visual stimuli processed without attention and awareness? *Current Opinion in Neurobiology* 15 (2):188-196.
43. Pessoa, Luiz (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences* 13 (4):160-166.
44. Rolls, ET (2007). Reward- and punishment-related learning; emotion and motivation. *Memory, Attention, and Decision-Making: A Unifying Computational Neuroscience Approach*. (Oxford, 2007; online edn, Oxford Academic, 1 Sept. 2009)
45. <https://doi.org/10.1093/acprof:oso/9780199232703.003.0003> accessed 29 Nov. 2022.
46. Salzman, C. Daniel (2022, November 18). amygdala. *Encyclopedia Britannica*. <https://www.britannica.com/science/amygdala>
47. Storbeck, J., Robinson, M. D., & McCourt, M. E. (2006). Semantic processing precedes affect retrieval: The neurological case for cognitive primacy in visual processing. *Review of General Psychology*, 10(1), 41–55. <https://doi.org/10.1037/1089-2680.10.1.41>
48. Whalen, P. J., Kagan, J., Cook, R. G., Davis, F. C., Kim, H., Polis, S., McLaren, D. G., Somerville, L. H., McLean, A. A., Maxwell, J. S., & Johnstone, T. (2004). Human amygdala responsivity to masked fearful eye whites. *Science (New York, N.Y.)*, 306(5704), 2061. <https://doi.org/10.1126/science.1103617>
49. Young MP, Scannell JW, Burns GAPC, Blakemore C (1994) Analysis of connectivity: Neural systems in the cerebral cortex. *Reviews in the Neurosciences* 5:227-249.
50. Zajonc, R. B. (1994). Emotional expression and temperature modulation. In S. H. M. van Goozen, N. E. Van de Poll, & J. A. Sergeant (Eds.), *Emotions: Essays on emotion theory* (pp. 3–27). Lawrence Erlbaum Associates, Inc.