Studying the Behaviour of Model of Mirror Neuron System in Case of Autism

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***Abstract*— Several experiment done by the researchers conducted that autism is caused by the dysfunctional mirror neuron system and the dysfunctions of mirror neuron system is proportional to the symptom severity of autism. In the present work those experiments were studied as well as studying a model of mirror neuron system called MNS2 developed by a research group. This research examined the behavior of the model in case of autism and compared the result with those studies conducting dysfunctions of mirror neuron system in autism. To perform this, a neural network employing the model was developed which recognized the three types of grasping (faster, normal and slower). The network was implemented with back propagation through time learning algorithm. The whole grasping process was divided into 30 time steps and different hand and object states at each time step was used as the input of the network. Normally the network successfully recognized all of the three types of grasps. The network required more times as the number of inactive neurons increased. And in case of maximum inactive neurons of the mirror neuron system the network became unable to recognize the types of grasp. As the time to recognize the types of grasp is proportional to the number of inactive neurons, the experiment result supports the hypothesis that dysfunctions of MNS is proportional to the symptom severity of autism.**

***Keywords*— Autism, MNS, mirror neuron, neural network, BPTT**

I. INTRODUCTION

Autism is a highly variable neurodevelopment disorder that first appears during infancy or childhood, and generally follows a steady course without remission. Overt symptoms gradually begin after the age of six months, become established by age two or three years, and tend to continue through adulthood, although often in more muted form. It is distinguished not by a single symptom, but by a characteristic triad of symptoms: impairments in social interaction; impairments in communication; and restricted interests and repetitive behavior. Other aspects, such as atypical eating, are also common but are not essential for diagnosis. Autism's individual symptoms occur in the general population and appear not to associate highly, without a sharp line separating pathologically severe from common traits [1].

According to the study in press at the journal Cognitive Brain Research, electroencephalograph (EEG) recordings of 10 individuals with autism show a dysfunctional mirror neuron system: Their mirror neurons respond only to what they do and not to the doings of others. Mirror neurons are brain cells in the premotor cortex area of the brain. First identified in macaque monkeys in the early 1990s, the neurons -- also known as "monkey-see, monkey-do cells" -- fire both when a monkey performs an action itself and when it observes another living creature perform that same action. Though it has been impossible to directly study the analogue of these neurons in people (since human subjects cannot be implanted with electrodes), several indirect brain-imaging measures, including EEG, have confirmed the presence of a mirror neuron system in humans. The human mirror neuron system is now thought to be involved not only in the execution and observation of movement, but also in higher cognitive processes -- language, for instance, or being able to imitate and learn from others' actions, or decode their intentions and empathize with their pain. Because autism is characterized, in part, by deficits in exactly these sorts of social interaction and communication skills, previous research has suggested that a dysfunctional mirror neuron system may explain the observed pathology. The current findings, the researchers say, lend substantial support to the hypothesis [2].

This paper introduces mirror neuron system (MNS) model's behavior in case of autism. To do so we studied the model of MNS named MNS2 developed by James Bonaiuto · Edina Rosta · Michael Arbib [3] and implement it to observe its behavior in case of several percentages of inactive mirror neurons and realize whether it is allied with autism or not. The results are also compared with the different study results done previously.

II. STUDIES RELATING MNS AND AUTISM

Several studies have been done to identify the source of autism. Many of those studies have found the deficient mirror neuron system. The hypothesis of a deficient Mirror neuron system (MNS) in autism was first introduced in 1999 by Rittia Haris,s group. Two years later Williams et al. published the first review on imitation, mirror neuron and autism. They offered a series of predictions that flows to the hypothesis of deficient MNS in autism. Anatomical and functional studies have been done for the past four years that support their proposition [4].

A. Anatomical studies

The anatomical substrates of Autism are still unknown. A group with Hadjikhani, Joseph, Snyder and Tager-Flusberg conducted a MRI study in a group of autistic adults in 2006. They found that adults with autism have significantly reduced cortical thickness in the areas of MNS. In addition the degree of cortical thickness decrease was correlated with the severity of communicative and social symptoms of the subjects [4].

B. Magnetoencephalographic Studies

Magnetoencephalography (MEG) is a method which allows measuring the minute magnetic field changes associated with

brain electrical activity non-invasively with a millisecond resolution.

The first study testing the hypothesis of a deficient MNS in autism was performed using MEG by Hari's group (Hamalainen, Hari, Ilmoniemi, knuutilaand Lounasmaa) in Finland. The result of this study was negative and found no differences between autism subjects and controls. Later in 2003 the same group pursued this hypothesis and showed in a behavioral experiment that autistic subjects unlike normal controls did not profit from mirror-image movement of others during an imitation task. A year later they published another MEG study [5] showing delayed and weaker activation of the inferior frontal lobe (IFL) and primary motor cortex (PMC) in Asperger subjects providing evidence of dysfunctional MNS.

C. Electroencephalographic Studies

Two electroencephalographic (EEG) studies have been done so far to examine the associations of MNS with autism. First Lindsay M. Oberman and his group performed the EEG studies on MNS in 2005 [6]. They observe EEG oscillations in the mu frequency (8–13 Hz) over sensorimotor cortex. It is established that mu power is reduced in typically developing individuals both when they perform actions and when they observe others performing actions. From the study they found that In case of autistic individuals mu power is reduced during action performance but it became unchanged during action observation. This results in a support to the hypothesis of dysfunctional MNS in autism. In 2006 Lepage JF and Théoret H. have found the same result examining the EEG on children with autism [7].

D. Functional MRI Studies

Several functional MRI (fMRI) have been done to examine the MNS dysfunction in autism. A study by Hadjikhani, Joseph, Snyder and Tager-Flusberg examined the facial emotional expression during observation and imitation in autistic children and compared with typically developing children [8].The study showed that both groups were able to perform the task but only the typically developing child have the activation in the pars opercularis of inferior frontal gyrus. On the other hand the autistic child had no mirror neuron activity in this area. The study also showed that the activation of MNS is inversely proportional to the symptom severity in the social domain.

III. MODEL OF MNS

A model of mirror neuron system was first introduced by Oztop and Arbib in 2002 which define the MNS (Mirror Neuron System) model of F5 and related brain regions. The connectivity of the model is constrained by the existing neurophysiology and neuroanatomy of the monkey brain, but except for AIP and F5 the anatomical localization of schemas is not germane to the simulations. The F5 grasp-related neurons are divided between (i) F5 mirror neurons which are, when fully developed, active during certain self-movements of grasping by the monkey and during the observation of a similar grasp executed by others, and (ii) F5 canonical neurons, namely those active during self-movement but not during the observation of grasping by others. They complemented the visual pathway via AIP by pathways directed toward F5 mirror neurons, which allow the monkey to observe arm-hand trajectories and match them to the affordances and location of a potential target object. They then showed how the mirror system may learn to recognize actions already in the repertoire of the F5 canonical neurons. In short, they provided a mechanism whereby the actions of others are "recognized" based on the circuitry involved in performing such actions [9].

Later in 2006 James Bonaiuto, Edina Rosta and Michael Arbib introduced mirror neuron system II (MNS2), a new version of the MNS model of action recognition. The new model used a recurrent architecture that is biologically more plausible than that of the original model. Moreover, MNS2 extends the capacity of the model to address data on audio-visual mirror neurons and on the response of mirror neurons when the target object was recently visible but is currently hidden. The system diagram for the MNS2 model (updating the MNS model of Oztop and Arbib 2002) is shown in fig. 1.

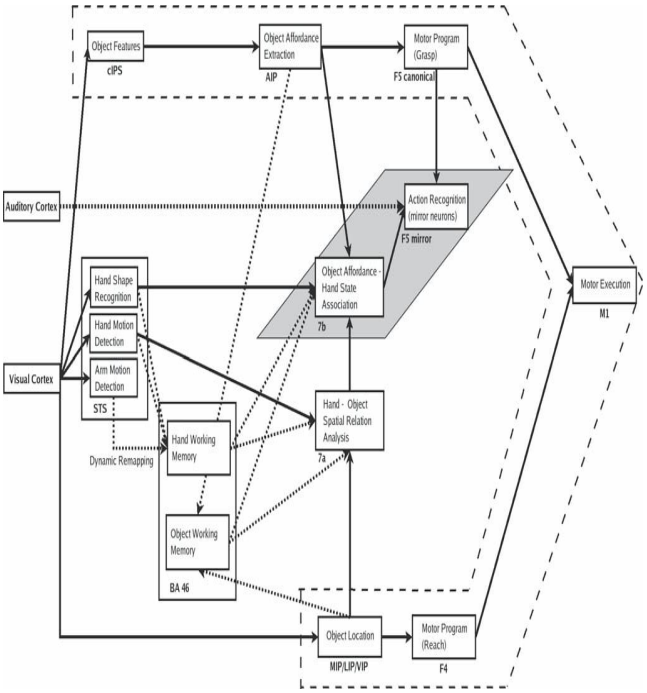


Fig. 1 System diagram of MNS2 model [3].

The main recurrent network, models the areas 7b and F5mirror, shown here in the gray parallelogram, by the activity of its hidden and external output layers, respectively. The audio recurrent network models the Auditory Cortex. The dotted arrows denote the connections unique to the MNS2 model [3].

IV. THE METHODS

A. Network Design

Here we represent a neural network expressing the main recurrent model of MNS2. Here we consider the input from only the visual cortex not the auditory cortex. The network is used to recognize three different types of grasping of an object by hand. The three types of grasps are differentiated according to their movement time and those are faster grasp, normal grasp and slower grasp. The model has 4 input neurons, 3 recurrent input neurons and 3 output neurons. The model takes different hand states and object size as input.

A hand reaching and grasping an object requires coordination of three components: hand/wrist transport, grip

aperture, and hand orientation. The 'transport component' consists of a single phase that involves the movement of the hand/wrist from an initial position to a final position that is close to the object being reached. The 'aperture component' consists of two sequential phases. 'Preshaping' opens the grip, slowly and monotonically, to a maximum aperture, and 'enclosing' reduces the aperture quickly until the fingers contact the object. The third component, 'hand orientation', quantifies changes, in alignment of the hand axes that make it convenient for the hand to grasp the object [10].

So the network takes as input the grip aperture, the wrist velocity, object distance and object size. The whole grasping process is divided into a series of time steps and the inputs at every time steps are fed into the network. The model has 15 hidden neurons and 3 output neurons. The output neurons are connected recursively with the recurrent input neurons to reflect the mirror neurons functionality. All the neurons of one layer are internally connected to all the neurons of the next layer. The networks output is a three dimensional vector each elements of which encodes a type of grasp (faster, normal, slower). The most active element in the networks output unit indicates the classification of grasp. Fig. 2 shows the pictorial representation of the neural network.

B. Implementation Technique

Backpropagation through time learning algorithm is used to implement the model. Backpropagation through time (BPTT) is a learning method for recurrent neural networks expanding on the backpropagation learning method for feedforward networks [11].

In BPTT, the network is "unfolded" for a number of time steps *L* into a large feedforward network with connections between copies of the network replacing the recurrent connections. After running the network forward for *L* time steps, the output layer error is propagated backwards "through time" along the unfolded network [3].

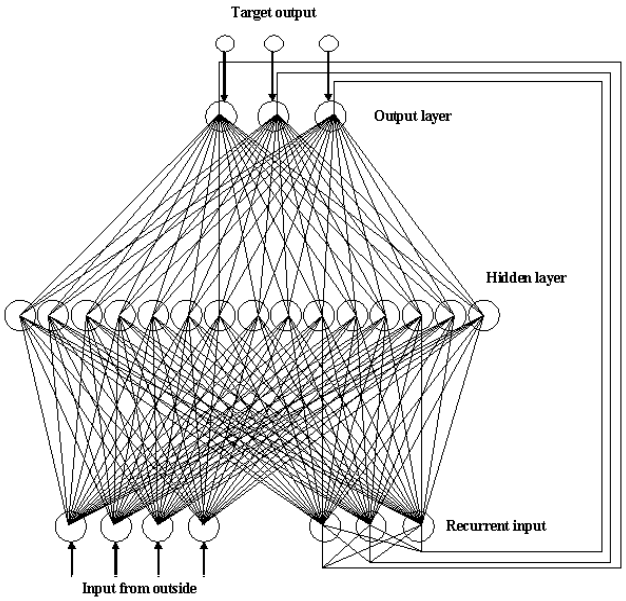


Fig. 2 The Neural Network designed to implement the model of MNS.

The following formulations are used for this network.

$$O_i(t) = g\left(\sum_{j=1}^{15} W2_{ij}\, g\left(\sum_{k=1}^{4} W1_{jk} IO_k(t) + \sum_{i=1}^{3} W1_{ji} IR_i(t)\right)\right) \quad (1)$$

$$IR_i(t+1) = g\left(\sum_{i=1}^{3} W3_{ii} O_i(t)\right) \quad (2)$$

Here, $O(t)$ represents the model's output at time t. $IO(t)$ represents the input vector given to the network from outside at time t. $IR(t)$ represents the recurrent inputs at time t. W1 is a 15×7 matrix of real numbers representing the input layer to hidden layer weights. W2 is a 3×15 matrix of real numbers representing the hidden to output layer weights. W3 is a 3×3 matrix of real numbers representing the output layer to recurrent input layer weights. g is the activation function. The activation function taken for this network is $g(x) = \frac{1}{1+e^{-x}}$, which bounds each units activity between zero and one.

The network is run in the feed forward mood for length of L. the value of L is chosen from the entire length of the time steps. The activation of each unit is saved at each time steps. At the end of the time steps the error is propagated backward through the network and the weights are updated according to the average weight change over all time steps.

Learning weights from hidden to output layer are as follows:

$$\delta W2_{ij}(t) = g'\left(\sum_{j=1}^{15} W2_{ij}\, g\left(\sum_{k=1}^{4} W1_{jk} IO_k(t) + \sum_{i=1}^{3} W1_{ji} IR_i(t)\right)\right)\left(O_i(t) - O_i(t)\right) \quad (3)$$

$$W2_{ij} = W2_{ij} + \eta \frac{1}{L} \sum_{t=1}^{L} \delta W2_{ij}(t) \quad (4)$$

Here η is the learning rate. $\delta W2_{ij}(t)$ is the weight change from hidden to output layer at each time step. Finally the updated weight is calculated by summing the average weight change multiplied by the learning rate with the previous weight.

Learning weights from input to hidden layer are as follows:

$$\delta W1_{jk}(t) = g'\left(\sum_{k=1}^{4} W1_{jk} IO_k(t)\right)\left(IO_k(t)\right) \quad (5)$$

$$W1_{jk} = W1_{jk} + \eta \frac{1}{L} \sum_{t=1}^{L} \delta W1_{jk}(t) \quad (6)$$

Learning weights from recurrent input to hidden layer are as follows:

$$\delta W1_{ji}(t) = g'\left(\sum_{i=1}^{3} W1_{ji} IR_i(t)\right)\left(IR_i(t)\right) \quad (7)$$

$$W1_{ji} = W1_{ji} + \eta \frac{1}{L} \sum_{t=1}^{L} \delta W1_{ji}(t) \quad (8)$$

Learning weights form output to recurrent input units are as follows:

$$\delta W3_{mi}(t) = g'\left(\sum_{m=1}^{3} W3_{mi} O_i(t-1)\right)\left(O_i(t-1)\right) \quad (9)$$

$$W3_{mi} = W3_{mi} + \eta \frac{1}{L} \sum_{t=1}^{L} \delta W3_{mi}(t) \quad (10)$$

C. Inputs

The model is experimented with several data sets based on the speed of grasping. We organized the data sets in three ways: For faster grasp, for normal grasp and for slower grasp.

Data are taken based on the experiment done by Roy, Paulignan, Farne, Jouffrais, & Boussaoud in 2000. They showed that Maximum grip aperture occurs at about 60–80% of movement time. Wallace and Weeks (1988) instructed subjects to grasp a small object (3 mm) at different distances (30 and 15 cm) and within a specified movement time (200 and 400 ms). Subjects were told to grasp the target object with a pinch grip as accurately as possible. Results of this experiment showed occurrences of maximum grip apertures between 61 and 67.8% of movement time. Similarly,

Jeannerod (1984) found a small band of relative times of occurrence of maximum aperture (74–81%) by using object widths between 2 and 8 cm, at distances of 25, 32, and 40 cm. Further, Paulignan and Jeannerod (1996) reported maximum aperture occurrence at 70–80% of movement time, and Jakobson and Goodale (1991) noted the peak aperture to occur right after two-thirds of movement time. Thus, the grip aperture of a reach–grasp movement invariably peaks at about 60–80% of movement time [10].

Faster reach–grasp movements lead to larger maximum grip apertures. Wing, Turton, and Fraser (1986) instructed subjects to grasp objects at two speeds, normal and fast. The normal speed was chosen by the subject and the fast one was 'as fast as possible' without dropping the object. The size of the object was 2.8 cm and it was located at 28 cm from the hand's initial position. The mean movement times obtained in this task were 376 ms for the fast movement, and 735 ms for the normal movement. Larger maximum apertures were observed for the faster movements. Thus, faster reach– grasp movements lead to larger maximum grip apertures. Reach–grasp movements that start with an open grip aperture show a tendency of the hand grip to partially close before achieving its maximum aperture [10].

According to these experiment described above the input data for the MNS model are organized. The data are organized as follows. The object size was taken 2.8 cm. The distance between hand to object was taken 28 cm. The whole grasping process is divided into 30 time steps. As the slower grasp requires the maximum time and it is almost 900 ms so each time steps of grasping process corresponds to 30 ms. And the data in each time steps are taken for the input of MNS model. Figure 3 gives the wrist velocity curve according to time step for all of the three types of grasps.

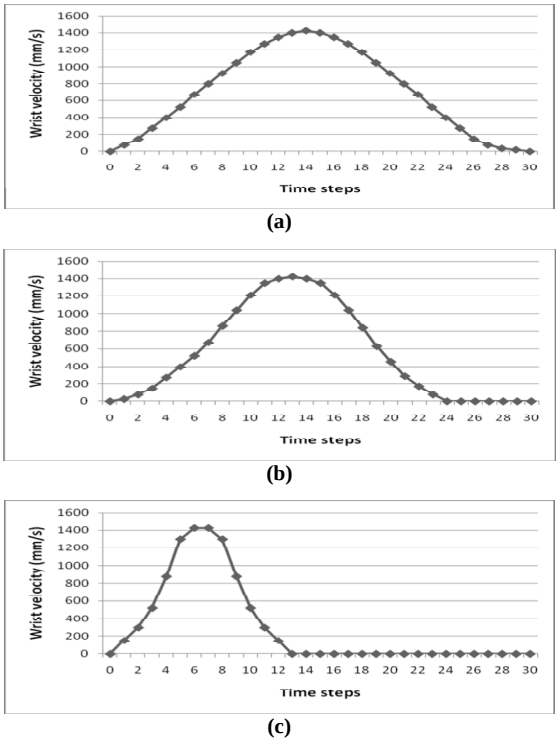


Fig. 3 Wrist Velocity at each time step for (a) slower grasp (b) normal grasp and (c) Faster grasp.

From fig. 3 it can be seen that the wrist velocity goes zero at 30 time steps for slower grasp that is the hand touched the object at 30 time steps. For normal grasp it goes zero at 24 time steps and for faster grasp at 13 time steps. The data are organized in this way because grasping requires almost 900 ms for slower movement, 735 ms for normal movement and 376 ms for faster movement. Fig. 4 shows the grip aperture curve with respect to time steps for each of the three types of grasps.

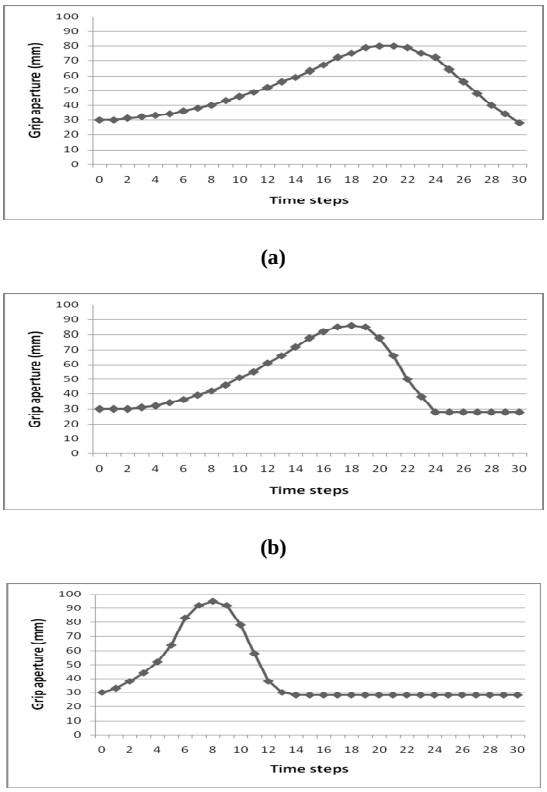


Fig. 4 Grip Aperture at each time step for (a) slower grasp (b) normal grasp and (c) Faster grasp.

We know that grip aperture goes to its maximum level at about 60-80% of the total movement time and maximum grip aperture found in the faster grasp. The data are organized based on this theory. From figure it can be seen that the maximum grip aperture occurs at faster grasp. In all of the three cases the grip aperture goes to its maximum level at about 60-80% of time steps. And as the object size was 2.8 cm the grip aperture for each of the three cases finished with 2.8 cm.

The object distance at each time steps for all of the three grasps were taken according to the same theory described above. Fig. 5 shows the object distance at each of the time steps for each of the three grasps. Initially the object distance was 28 cm. The object distance decreased gradually as the hand goes closer to the object. And when the hand touched the object the distance was taken zero.

These data are normalized with the maximum value of each vector element at each unit. Then these data are fed into the network for all 30 time steps. And the activation of each unit is saved and the errors are calculated for each unit. After

finishing the feed-forward mood for the all time steps the errors are then sent backward and the weights are updated.

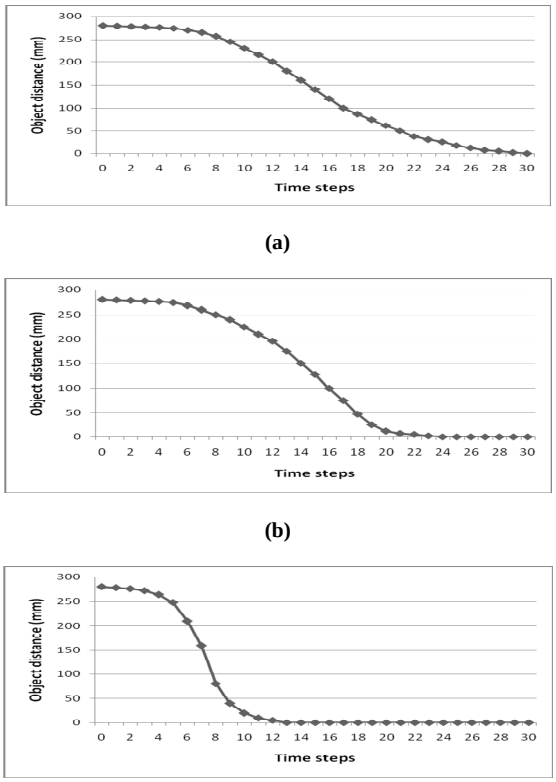


Fig. 5 Object distance at each time step for (a) slower grasp (b) normal grasp and (c) Faster grasp.

V. RESULTS AND DISCUSSION

The model is implemented using C#. And the model became successful to recognize all of the three types of grasps. The model gave ambiguous results initially for most of the grasps but eventually resolved it before the hand contact the object. Fig. 6 shows the network's output unit activity for faster, normal and slower grasps respectively. It can be observed from figure that the network recognized the faster grasp after 12 to 13 time steps. The normal grasps were recognized after around 16 time steps. And finally for slower grasps the network recognized it after around 19 time steps.

To observe the behavior of the MNS model the network was implemented with several percentages of neurons keeping inactive. At first 20% of the neurons in the network's hidden layer were kept inactive that is only 80% of the hidden neurons kept participating on the network. Fig. 7 shows the output neurons activity of the network with only 80% active hidden neuron.

The network became successful to recognize all of the three types of grasp but it took more times than in normal case. From fig 7 can be observed that the network could recognize the faster grasp after about 16 time steps whereas the network with all the neurons active took only 13 time steps. In this case the network took 20 time steps to recognize the normal grasp and about 23 time steps to recognize the slower grasp.

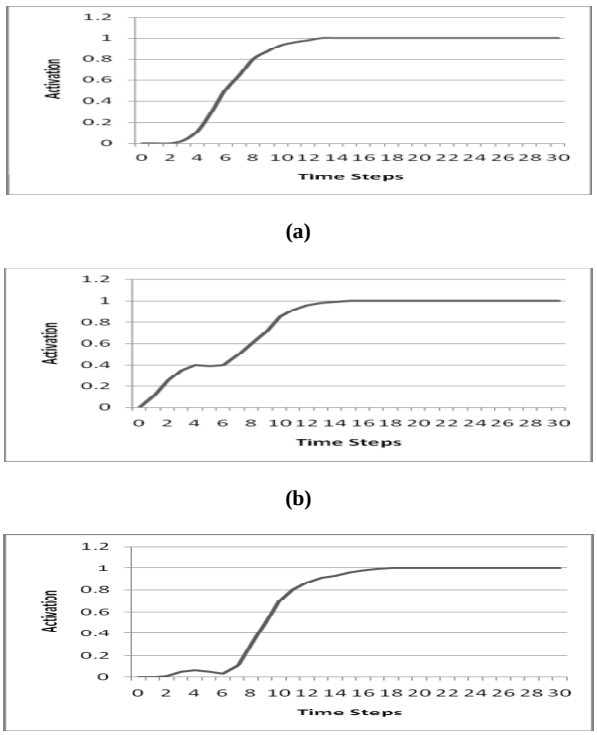


Fig. 6 Output unit activation for (a) Faster grasp, (b) Normal grasp and (c) Slower grasp.

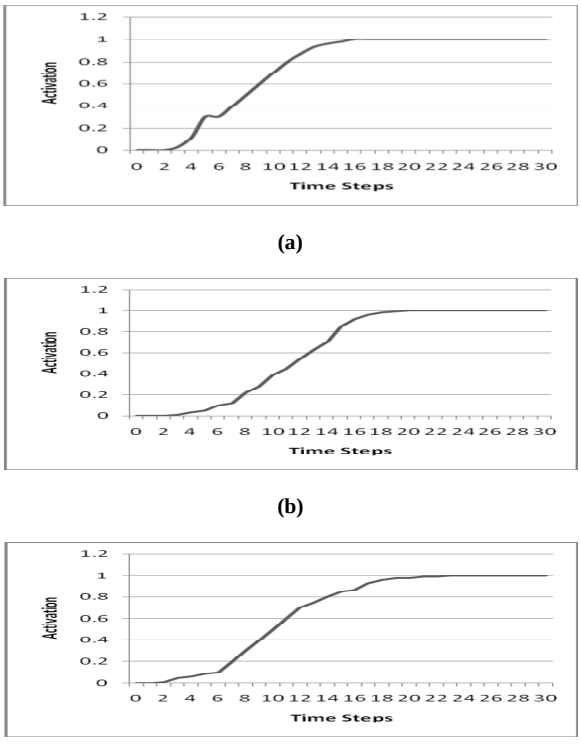
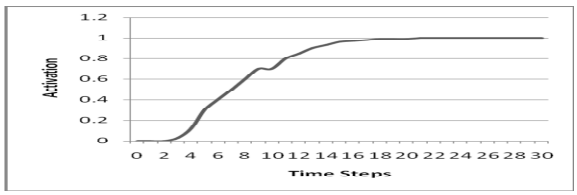


Fig. 7 Output unit activation for (a) Faster grasp, (b) Normal grasp and (c) Slower grasp keeping 20% hidden neurons inactive.

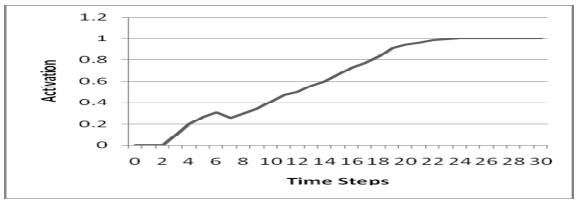
The network was experimented again keeping 60% of the hidden neurons inactive. Fig. 8 shows the networks behavior in this case. The network was able to recognize all of the three types of grasp in this case also but it took more time steps to recognize. The faster grasps are recognized after 20 time steps that means after the hand contact the object. And the normal

and slower grasps are recognized after 24 and 26 time steps respectively.

Lastly the network was experimented with keeping 80% hidden neurons inactive that is only 20% percentages of hidden neurons were kept participating on the network. Fig. 9 shows the networks behavior in this case. The network was able to recognize only the slower grasp in this situation. The network became unable to recognize the faster and normal grasp at all. And it could recognize the slower grasp almost at about 30 time steps.



(a)



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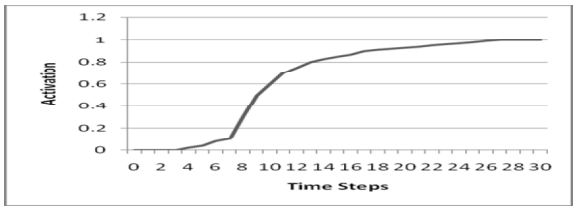
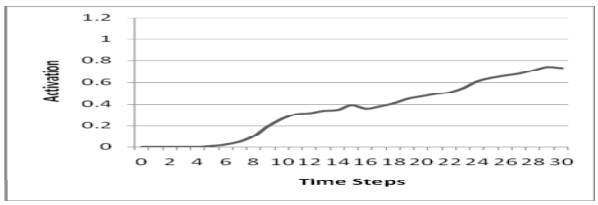


(c)

Fig. 8 Output unit activation for (a) Faster grasp, (b) Normal grasp and (c) Slower grasp keeping 60% hidden neurons inactive.

VI. COMPARISON WITH DIFFERENT STUDIES

Several studies showing the dysfunctions of MNS in autism discussed earlier. Now a comparison between those studies and the experimented results are shown here. The following table shows the comparison between different study result and the result of the experiment done here. The anatomical studies stated that the degree of cortical thickness in the area of MNS decrease is correlated with communicative and social symptoms like delayed learning, weaker social communication etc. The cortical thickness decreasing results the weaker activation or inactiveness of mirror neurons. Fig. 10 shows the comparison of the MNS network's behavior at normal case (100% active neurons) and autistic case (keeping 20%, 60% and 80% hidden neurons inactive).



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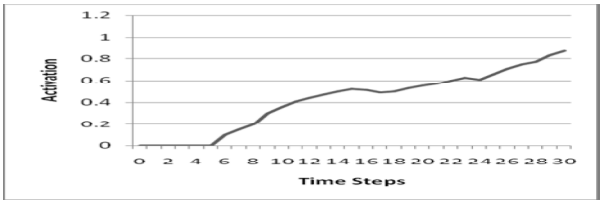

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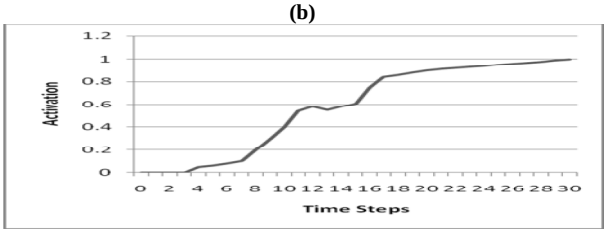
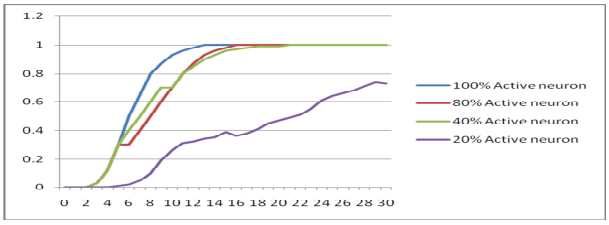
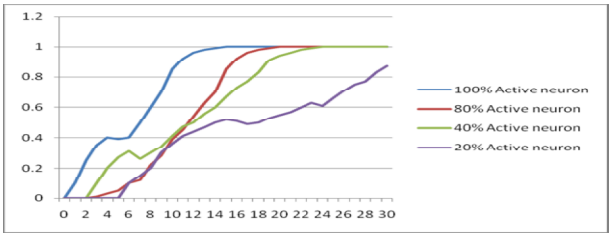


(c)

Fig. 9 Output unit activation for (a) Faster grasp, (b) Normal grasp and (c) Slower grasp keeping 80% hidden neurons inactive.



(a)



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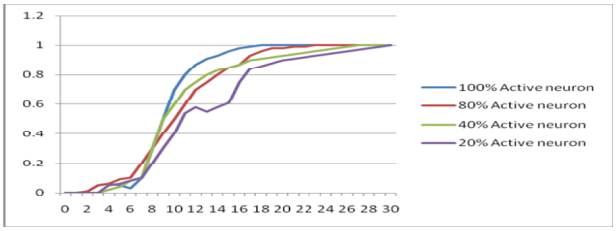

(c)

FIG. 10 THE COMPARISON OF THE MNS NETWORK'S BEHAVIOR AT NORMAL CASE (100% ACTIVE NEURONS) AND AUTISTIC CASE (KEEPING 20%, 60% AND 80% HIDDEN NEURONS INACTIVE).

TABLE I
COMPARISON BETWEEN DIFFERENT STUDY RESULT AND THE EXPERIMENT RESULT

Study Name	Study Result	Experiment Result
Anatomical studies	The degree of cortical thickness in the area of MNS decrease is correlated with the severity of communicative and social symptoms of the subjects.	As the number of inactive neurons increases the grasp recognition time of the network also increases.
Magnetoence phalographic Studies	Delayed and weaker activation of the inferior frontal lobe (IFL) and primary motor cortex (PMC) area of the brain.	Delayed activation of the output unit.
Functional MRI Studies	The symptom severity in the social domain is inversely proportional to the activation of mirror neurons.	The activation of output unit is inversely proportional to the no of inactive neurons.

The experiment results that increasing number of inactive neurons leads to increasing time for grasp recognition which can be correlated with the anatomical study result in a sense that as the number of inactive neurons in MNS increases the autistic behavior like delayed learning also increases. Also Magnetoencephalographic studies found the delayed and weaker activation of area IFL and PMC in where MNS exists. The delayed activation of neurons in our experiment can be allied with this study result. All of these studies summarizes to a point that deficient MNS is responsible for autism and the severity of autism symptoms are proportional to the deficiency rate of MNS. As with the increasing number of inactive neurons increases the grasp recognition time, the experiment also summarizes to the same point that the symptom severity of autism is proportional to the number of inactive mirror neurons.

VII. CONCLUSIONS

When one observes some action, the mirror neurons fire and they fire also when s/he acts that task. Based on this theory the MNS2 model was implemented. The inputs of the model are taken from the simulated data of a hand grasping an object with faster, normal and slower speed. The model was implemented to distinguish these three types of grasp. And it was successful to classify all of the three types of grasp which reflects the MNS activity. Finally the model was implemented with keeping several percentages of neurons inactive. The time to recognize the types of grasp required more times as the percentages of inactive neurons increased. And in case of very little percentages of active neurons the model was failed to recognize them. And finally the result was compared with different studies relating MNS with autism.

So, from the experiment it has been found that if the mirror neurons don't work properly then the learning may be delayed. And as the number of dysfunctional mirror neurons increases the required learning time also increases. It also might be possible that the learning remains incomplete in case of dysfunctions of majority of mirror neurons. Since the autistic people have some difficulties in learning and which varies among different autistic people so it can be said that the learning of autistic people is cramped because of dysfunctional MNS.

So the study provides support to the hypothesis of dysfunctional mirror neuron system in autism. The indecent functionality of MNS could be one of the reasons of autism. And the severity of autism depends on the severity of dysfunctions of MNS.

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