

Providing a better life for Amyotrophic Lateral Sclerosis Patient or Spinal Cord Injured Patient by Artificial Neural Network or BrainGate

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Abstract—BrainGate is a brain implant system built and previously owned by Cyberkinetics, currently under development and in clinical trials, designed to help those who have lost control of their limbs, or other bodily functions, such as patients with amyotrophic lateral sclerosis (ALS) or spinal cord injury. The sensor, which is implanted into the brain, monitors brain activity in the patient and converts the intention of the user into computer commands. BrainGate is a path to a better way of life for severely motor-impaired individuals. Through years of advanced research, BrainGate enables these people with the ability to communicate, interact and function through thought. BrainGate's mission is to further the advancement of this life-changing technology to promote wider adoption to help impaired individuals communicate and interact with society. For instance, the Cyberkinetic's BrainGate Neural Interface is currently the subject of a pilot clinical trial being conducted under an Investigational Device Exemption (IDE) from the FDA. The system is designed to restore functionality for a limited, immobile group of severely motor-impaired individuals. It is expected that people using the BrainGate System will employ a personal computer as the gateway to a range of self-directed activities. These activities may extend beyond typical computer functions to include the control of objects in the environment such as a telephone, a television and lights.

Keywords—BrainGate, Artificial Neural Network, Medical Science, amyotrophic lateral sclerosis (ALS), Sensor, Cyberkinetics, neurons.

I. INTRODUCTION

BrainGate consists of a sensor implanted in the brain and an external decoder device, which connects to some kind of prosthetic or other external object. The sensor uses 100 hair-thin electrodes that sense the electromagnetic signature of neurons firing in specific areas of the brain, for example, the area that controls arm movement. The sensor translates that activity into electrically charged signals, which are then sent to an external device and decoded in software. The decoder connects to and can use the brain signals to control an external device, such as a robotic arm, a computer cursor, or even a wheelchair. In essence, BrainGate allows a person to manipulate objects in the world using only the mind [1]. In addition to real-time analysis of neuron patterns to relay movement, the BrainGate array is also capable of recording electrical data for later analysis. A potential use of this feature would be for a neurologist to study seizure patterns in a patient with epilepsy. BrainGate was originally developed by researchers in the Department of Neuroscience at Brown University in conjunction with bio-tech company Cyberkinetics, Inc. Cyberkinetics later spun off the device manufacturing to Blackrock Microsystems, who now manufactures the sensors and the data acquisition hardware. The BrainGate Company purchased the intellectual property and related technology from Cyberkinetics and continues to own the intellectual property related to BrainGate. Addition to real-time analysis of neuron patterns to relay movement, the BrainGate array is also capable of recording electrical data for later analysis. A potential use of this feature would be for a neurologist to study seizure patterns in a patient with epilepsy.

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II. ROLE OF NEURONS

Neurons are cells that use a language of electrical impulses to communicate messages from the brain to the rest of the body. BrainGate is a controller which work as a sensor, transmitter and analyzer of neurons. We are developing products to restore function, as well as to monitor, detect, and respond to a variety of neurological diseases and disorders. The platform technology is based on the results of several years of research and development at premier academic institutions. BrainGate offers a systems approach which applies the language of neurons in both short and long-term settings.



Fig.1 Non-Working Model Of Brain Gate

III. SENSOR NETWORK

BrainGate's unique technology is able to simultaneously sense the electrical activity of many individual neurons. Braingate sensor consists of a silicon array about the size of a baby aspirin that contains one hundred electrodes, each thinner than a human hair. In the BrainGate Neural Interface System, the array is implanted in the area of the brain responsible for limb movement and for other body processes. The array is implanted on the surface of the brain.

IV. HUMAN BRAIN AND SUPER COMPUTER

The human brain is a super computer with the ability to instantaneously process vast amounts of information. BrainGate's technology allows for an extensive amount of electrical activity data to be transmitted from neurons in the brain to computers for analysis. In the current BrainGate™ System, a bundle consisting of one hundred gold wires connects the array to a pedestal which extends through the scalp. The pedestal is connected by an external cable to a set of computers in which the data can be stored for off-line analysis or analyzed in real-time. Signal processing software algorithms analyze the electrical activity of neurons and translate it into control signals for use in various computer-based applications [3].

V. ARTIFICIAL NEURAL NETWORK (ANN)

In machine learning and cognitive science, artificial neural networks (ANNs) are a family of models inspired by biological neural networks (the central nervous systems of animals, in particular the brain) and are used to estimate or approximate functions that can depend on a large number of inputs and are generally unknown. Artificial neural networks are generally presented as systems of interconnected "neurons" which exchange messages between each other. The connections have numeric weights that can be tuned based on experience, making neural nets adaptive to inputs and capable of learning [4]. For example, a neural network for handwriting recognition is defined by a set of input neurons which may be activated by the pixels of an input image. After being weighted and transformed by a function (determined by the network's designer), the activations of these neurons are then passed on to other neurons. This process is repeated until finally, an output neuron is activated. This determines which character was read. Like other machine learning methods – systems that learn from data – neural networks have been used to solve a wide variety of tasks that are hard to solve using ordinary rule-based programming, including computer vision and speech recognition.

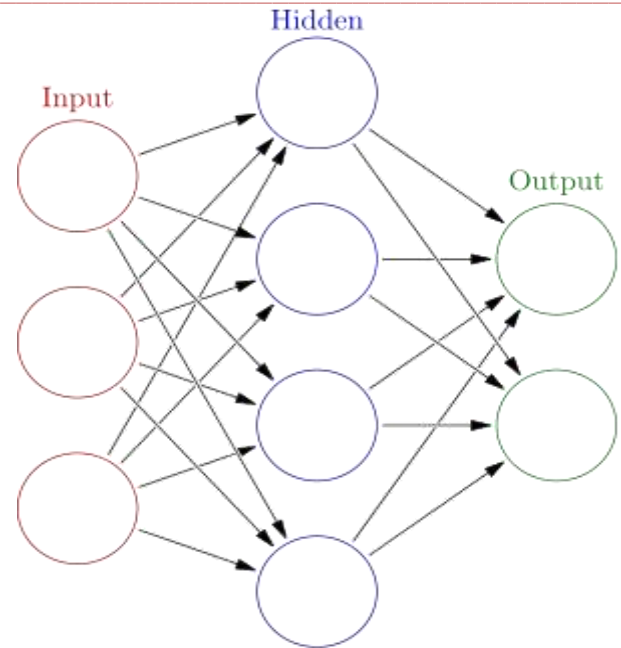


Fig.2 Artificial Neural Network

A. Network Function

The word network in the term 'artificial neural network' refers to the inter-connections between the neurons in the different layers of each system. An example system has three layers. The first layer has input neurons which send data via synapses to the second layer of neurons, and then via more synapses to the third layer of output neurons. More complex systems will have more layers of neurons, some having increased layers of input neurons and output neurons. The synapses store parameters called "weights" that manipulate the data in the calculations.

An ANN is typically defined by three types of parameters:

- 1) The interconnection pattern between the different layers of neurons.
- 2) The learning process for updating the weights of the interconnections.
- 3) The activation function that converts a neuron's weighted input to its output activation.

Mathematically, a neuron's network function is defined as a composition of other functions, which can further be defined as a composition of other functions. This can be conveniently represented as a network structure, with arrows depicting the dependencies between variables. A widely used type of composition is the nonlinear weighted sum, where, where (commonly referred to as the activation function) is some predefined function, such as the hyperbolic tangent. It will be convenient for the following to refer to a collection of functions as simply a vector [5].

VI. AMYOTROPHIC LATERAL SCLEROSIS (ALS)

Amyotrophic lateral sclerosis (ALS) is a specific disorder that involves the death of neurons [6]. In a number of countries the term motor neuron disease (MND) is commonly used while others use that term for a group of five conditions of which ALS is the most common [7], ALS is characterized by stiff

muscles, muscle twitching, and gradually worsening weakness due to muscles decreasing in size. This results in difficulty speaking, swallowing, and eventually breathing [8]. The cause is not known in 90% to 95% of cases. About 5–10% of cases are inherited from a person's parents. About half of these genetic cases are due to one of two specific genes. It results in the death of the neurons that control voluntary muscles. The diagnosis is based on a person's signs and symptoms with testing done to rule out other potential causes. There is no known cure for ALS [9]. A medication called riluzole may extend life expectancy by about two to three months. Non-invasive ventilation may result in both improved quality and length of life. The disease usually starts around the age of 60 and in inherited cases around the age of 50. The average survival from onset to death is three to four years. About 10% survive longer than 10 years. Most die from respiratory failure. In much of the world, rates of ALS are unknown. In Europe and the United States, the disease affects about 2 people per 100,000 per year [10]. Descriptions of the disease date back to at least 1824 by Charles Bell. In 1869, the connection between the symptoms and the underlying neurological problems were first described by Jean-Martin Charcot, who in 1874 began using the term amyotrophic lateral sclerosis. It became well known in the United States in the 20th century when it affected the baseball player Lou Gehrig and later when Stephen Hawking gained fame for his scientific achievements. In 2014, videos of the ice bucket challenge went viral on the internet and increased public awareness [11].

A. SIGN AND SYMPTOMS

The disorder causes muscle weakness and atrophy throughout the body due to the degeneration of the upper and lower motor neurons. Individuals affected by the disorder may ultimately lose the ability to initiate and control all voluntary movement, although bladder and bowel function and the muscles responsible for eye movement are usually spared until the final stages of the disorder [12]. Cognitive function is generally spared for most people, although some (about 5%) also develop front temporal dementia. A higher proportion of people (30–50%) also have more subtle cognitive changes which may go unnoticed, but are revealed by detailed neuropsychological testing. Sometimes, ALS coexists in individuals who also experience dementia, degenerative muscle disorder, and degenerative bone disorder as part of a syndrome called multisystem proteinopathy. Sensory nerves and the autonomic nervous system are generally unaffected, meaning the majority of people with ALS maintain hearing, sight, touch, smell, and taste [13].

B. INITIAL SYMPTOMS

The start of ALS may be so subtle that the symptoms are overlooked. The earliest symptoms of ALS are muscle weakness and/or muscle atrophy. Other presenting symptoms include trouble swallowing or breathing, cramping, or stiffness of affected muscles; muscle weakness affecting an arm or a leg; and/or slurred and nasal speech. The parts of the body affected by early symptoms of ALS depend on which

motor neurons in the body are damaged first. About 75% of people contracting the disorder first experience weakness or atrophy in an arm or leg and this is known as "limb-onset" ALS. Awkwardness when walking or running or even tripping over or stumbling may be experienced and often this is marked by walking with a "dropped foot" which drags gently on the ground. Or if arm-onset, difficulty with tasks requiring manual dexterity such as buttoning a shirt, writing, or turning a key in a lock may be experienced. Occasionally, the symptoms remain confined to one limb for a long period of time or for the duration of the illness; this is known as monomeric amyotrophic [14]. About 25% of cases begin as progressive bulbar palsy termed "bulbar-onset" ALS. Initial symptoms will mainly be of difficulty speaking clearly or swallowing. Speech may become slurred, nasal in character, or quieter. There may be difficulty in swallowing and loss of tongue mobility. A smaller proportion of people experience "respiratory-onset" ALS, where the intercostal muscles that support breathing are affected first. A small proportion of people may also present with what appears to be front temporal dementia, but later progresses to include more typical ALS symptoms. Over time, people experience increasing difficulty moving, swallowing (dysphagia), and speaking or forming words (dysarthria). Symptoms of upper motor neuron involvement include tight and stiff muscles (spasticity) and exaggerated reflexes including an overactive gag reflex. An abnormal reflex commonly called Babinski's sign also indicates upper motor neuron damage. Symptoms of lower motor neuron degeneration include muscle weakness and atrophy, muscle cramps, and fleeting twitches of muscles that can be seen under the skin (fasciculation) although twitching is not a diagnostic symptom and more of a side effect so twitching would either occur after or accompany weakness and atrophy. Around 15–45% of people experience pseudobulbar affect, a neurological disorder also known as "emotional lability", which consists of uncontrollable laughter, crying, or smiling, attributable to degeneration of bulbar upper motor neurons, resulting in exaggeration of motor expressions of emotion [Citation needed] For ALS to be diagnosed, symptoms of both upper and lower motor neuron damage that cannot be attributed to other causes must be present.

C. LAST STAGE

Although respiratory support can ease problems with breathing and prolong survival, it does not affect the progression of ALS. Most people with ALS die from respiratory failure, usually within three to five years from the onset of symptoms. The median survival time from onset to death is around 39 months, and only 4% survive longer than 10 years. Guitarist Jason Becker has lived since 1989 with the disorder, while physicist Stephen Hawking has survived for more than 50 years, but they are considered unusual cases [15]. Difficulty in chewing and swallowing makes eating very difficult and increases the risk of choking or of aspirating food into the lungs. In later stages of the disorder, aspiration pneumonia can develop, and maintaining a healthy weight can become a significant problem that may require the insertion of a feeding tube. As the diaphragm and intercostal muscles of the rib cage that support breathing weaken, measures of lung

function such as vital capacity and inspiratory pressure diminish. In respiratory-onset ALS, this may occur before significant limb weakness is apparent. Most people with ALS die of respiratory failure or pneumonia. In late stages, the oculomotor nerve that controls the movements of the eye can be affected as can the extraocular muscles (EOMs). The eye movements remain unaffected largely until the later stages due to differences in the extraocular muscles compared to the skeletal muscles that are initially and readily affected. In the disease's final stages, a person's condition may resemble locked-in syndrome.

D. SOME OTHER FACTORS

Where no family history of the disease is present – i.e., in around 90% of cases – no cause is known for ALS. Possible associations for which evidence is inconclusive include head trauma, military service, frequent drug use, and participation in contact sports [25]. Studies also have focused on the role of glutamate in motor neuron degeneration. Glutamate is one of the neurotransmitters in the brain. Scientists have found, compared with healthy people, people with ALS have higher levels of glutamate in their serum and spinal fluid [26]. Riluzole is currently the only FDA-approved drug for ALS and targets glutamate transporters. It only has a modest effect on survival, however, suggesting that excess glutamate is not the sole cause of the disease. Certain studies suggested a link between sporadic ALS, specifically in athletes, and a diet enriched with branched-chain amino acids, a common dietary supplement among athletes, which cause cell hyper excitability resembling that usually observed in people with ALS. The proposed underlying mechanism is that cell hyper excitability results in increased calcium absorption by the cell, and thus brings about cell death of neuronal cells, which have particularly low calcium buffering capabilities. Some evidence supports superoxide dismutase 1 (SOD1) protein misfolding propagates between molecules in a similar fashion to prions [27]. Similarly, it has been proposed that incorporation of the cyanobacterial toxin β -methylamino-L-alanine (BMAA) leads to another prion-like protein misfolding propagation. Another very common factor associated with ALS is a lesion to the motor system in areas such as the front temporal lobes. Lesions in these areas often show signs of early deficit, which can be used to predict the loss of motor function, and result in the spread of ALS [28]. The mechanisms of ALS are present long before any signs or symptoms become apparent. Before any muscular atrophy becomes apparent during ALS, roughly one-third of the motor neurons must be destroyed [29]. Many other potential risk factors including chemical exposure, electromagnetic field exposure, occupation, physical trauma, and electric shock, have been investigated, but are without consistent findings.

VII. RESEARCH METHODOLOGY

A number of clinical trials are underway globally for ALS; a comprehensive listing of trials in the US can be found at ClinicalTrials.gov. A large genetic study, called project Min E, initiated by two people with ALS is going on currently. It is a crowdfunded research project with many countries involved

to discover more genes. A division of the US Centers for Disease Control and Prevention maintains a registry of Americans with ALS. A phase-II trial on tirasemtiv has been completed with a follow-on phase-IIb study in progress under the name "BENEFIT-ALS". Results of the first study are available here. The current trial is an international, placebo-controlled, multiple-center study on 680 participants. This makes it one of the largest studies to date. A phase-II trial on ozanezumab is in progress. It is a large multiple-site international research project sponsored by GlaxoSmithKline. A phase-II clinical trial is being conducted by BrainStorms Cell Therapeutics at the Hadassah Medical Center in Israel and interim results "demonstrated a tendency toward stabilization in some parameters in the ALS Functional Rating Scale." People in the trial have bone marrow stem cells removed and differentiated in a clean room into cells that express neurotropic factors. The cells are injected back into the same person by intrathecal and intramuscular injections. A second phase-II trial is expected to open in the United States at several institutions including the Mayo Clinic.

The initial clinical trial of BrainGate, led by researchers at Massachusetts General Hospital, Brown University, and the Department of Veterans Affairs, ran from 2004 to 2006 and studied four patients with tetraplegia. The results, published in a 2006 article in the journal *Nature*, showed that a human with tetraplegia was able to control a cursor on a computer screen just by thinking, enabling him to open emails, and to operate devices such as a television. One participant, Matt Nagle, had a spinal cord injury, whilst another had advanced ALS [16] [17]. In July 2009, a second clinical trial (dubbed "BrainGate2") was initiated by researchers at Massachusetts General Hospital, Brown University, and the Providence VA. In November 2011, researchers from the Stanford University Neural Prosthetics Translational Laboratory joined the trial as a second site. This trial is ongoing [18]. In May 2012, BrainGate researchers published a study in *Nature* demonstrating that two people paralyzed by brainstem stroke several years earlier were able to control robotic arms for reaching and grasping [19][20]. One participant, Cathy Hutchinson, was able to use the arm to drink coffee from a bottle, the first time she was able to drink unaided in 15 years [21]. This took place on site at The Boston Home in Dorchester, Massachusetts, a specialized residence where Ms. Hutchinson resided [22]. The study included researchers at Brown University, the Department of Veterans Affairs, Massachusetts General Hospital, Harvard Medical School, and the German Aerospace Center [23][24].

VIII. BRAIN – COMPUTER INTERFACE

A brain-computer interface (BCI), sometimes called a mind-machine interface (MMI), direct neural interface (DNI), or brain-machine interface (BMI), is a direct communication pathway between an enhanced or wired brain and an external device. BCIs are often directed at researching, mapping, assisting, augmenting, or repairing human cognitive or sensory-motor functions [30]. Research on BCIs began in the 1970s at the University of California, Los Angeles (UCLA) under a grant from the National Science Foundation, followed

by a contract from DARPA. The papers published after this research also mark the first appearance of the expression brain-computer interface in scientific literature [31]. The field of BCI research and development has since focused primarily on neuroprosthetic applications that aim at restoring damaged hearing, sight and movement [32]. Thanks to the remarkable cortical plasticity of the brain, signals from implanted prostheses can, after adaptation, be handled by the brain like natural sensor or effector channels. Following years of animal experimentation, the first neuroprosthetic devices implanted in humans appeared in the mid-1990s [33].

IX. CONCLUSION

With the help of Artificial Neural Network or BrainGate there is a path for providing a better life for Amyotrophic Lateral Sclerosis patient and spinal cord injured patient. With the help of sensors present in BrainGate, movement of neurons are sensed which provide the message produce by the brain to the rest of the body. On using Brain Machine Interface there is a direct path formed between a human brain and a computer for communication.

REFERENCES

- [1] Brain Gate gets a new lease on life, The Boston Globe, August, 2009.
- [2] McCulloch, Warren; Walter Pitts (1943). "A Logical Calculus of Ideas Immanent in Nervous Activity". *Bulletin of Mathematical Biophysics*.
- [3] Anurag Kumar Sisodia, Piyush Kumar and Deepak Punetha, "Design, analysis and optimization to mount different logic families in a single IC," in *IEEE International Conference on Advances in Computing & Communications Engineering (ICACCE 2015)*, Dehradun, India, pp: 388-391.
- [4] Hebb, Donald (1949). *The Organization of Behavior*. New York: Wiley.
- [5] Dominic, S., Das, R., Whitley, D., Anderson, C. (July 1991). "Genetic reinforcement learning for neural networks". *IJCNN-91-Seattle International Joint Conference on Neural Networks. IJCNN-91-Seattle International Joint Conference on Neural Networks*. Seattle, Washington, USA: IEEE. doi:10.1109/IJCNN.1991.155315. ISBN 0-7803-0164-1. Retrieved 29 July 2012.
- [6] Kelly, Evelyn B. (2013). *Encyclopedia of human genetics and disease*. Santa Barbara, Calif.: Greenwood.
- [7] "Motor neurone disease". <http://www.nhs.uk/>. Retrieved 2 January 2015.
- [8] Ellison, edited by Seth Love, David N. Louis, David W. (2008). *Greenfield's neuropathology (8th Ed.)*. London: Hodder Arnold.
- [9] "Motor Neuron Diseases Fact Sheet". National Institute of Neurological Disorders and Stroke (NINDS). Retrieved 7 November 2010.
- [10] Kiernan, MC; Vucic, S; Cheah, BC; Turner, MR; Eisen, A; Hardiman, O; Burrell, JR; Zoing, MC (12 March 2011). "Amyotrophic lateral sclerosis."
- [11] Miller, RG; Mitchell, JD; Moore, DH (14 March 2012). "Riluzole for amyotrophic lateral sclerosis (ALS)/motor neuron disease (MND)."
- [12] Dugdale DC, Hoch DB, Zieve D (27 August 2010). "Amyotrophic lateral sclerosis". *A.D.A.M. Medical Encyclopedia*.
- [13] Taylor JP (August 2015). "Multisystem proteinopathy: Intersecting genetics in muscle, bone, and brain degeneration."
- [14] Castrillo-Viguera C, Grasso DL, Simpson E, Shefner J, Cudkowicz ME (2010). "Clinical significance in the change of decline in ALSFRS-R". *Amyotroph Lateral Scler (Journal Article)*.
- [15] James L. Bernat: *Ethical Issues in Neurology*.
- [16] Neuronal ensemble control of prosthetic devices by a human with tetraplegia, *Nature*, July 2006.
- [17] *Mind Control*, *Wired*, March 2005.
- [18] *BrainGate - Turning thought into Action*.
- [19] Stanford joins BrainGate team developing brain-computer interface to aid people with paralysis, Nov 11, 2011.
- [20] Stanford School of Medicine - Stanford joins BrainGate team developing brain-computer interface to aid people with paralysis.
- [21] Reach and grasp by people with tetraplegia using a neurally controlled robotic arm, *Nature*, May 2012.
- [22] People with paralysis control robotic arms using brain-computer interface, *Brown University*, May 2012.
- [23] Mind-controlled robot arms show promise, *Nature*, May 2012.
- [24] People with paralysis control robotic arms using brain-computer interface, *Stanford University*, May 2012.
- [25] Holtcamp, W. (2012). "The emerging science of BMAA: do cyanobacteria contribute to neurodegenerative disease?"
- [26] Rodgers KJ (March 2014). "Non-protein amino acids and neurodegeneration: The enemy within".
- [27] Rosenbohm, A., Kassubek, J., Weydt, P., Marroquin, N., Volk, A., Kubisch, C., Huppertz, H., & Weber, M. (2014). Can lesions to the motor cortex induce amyotrophic lateral sclerosis?
- [28] Walling A (1999). "Amyotrophic lateral sclerosis: Lou Gehrig's disease".
- [29] Sutedja NA, Fischer K, Veldink JH, van der Heijden GJ, Kromhout H, Heederik D, Huisman MH, Wokke JJ, van den Berg LH (2009). "What we truly know about occupation as a risk factor for ALS: a critical and systematic review". *Amyotrophic Lateral Sclerosis*.
- [30] Vidal, JJ (1973). "Toward direct brain-computer communication".
- [31] J. Vidal (1977). "Real-Time Detection of Brain Events in EEG" (PDF). *IEEE Proceedings* 65 (5)
- [32] Levine, SP; Huggins, JE; Bement, SL; Kushwaha, RK; Schuh, LA; Rohde, MM; Passaro, EA; Ross, DA; Elisevich, KV; et al. (2000). "A direct brain interface based on event-related potentials". *IEEE transactions on rehabilitation engineering: a publication of the IEEE Engineering in Medicine and Biology Society* 8 (2): 180-5. doi:10.1109/86.847809. PMID 10896180.
- [33] Wolpaw, J.R. and Wolpaw, E.W. (2012). *Brain-Computer Interfaces: Something New Under the Sun*. In: *Brain-Computer Interfaces: Principles and Practice*, editors: Wolpaw, J.R. and Wolpaw, E.W. Oxford University Press.

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