Implementation of Magnetic Pulse Shape Stimulation Device Suitable for Cerebral Cortical Modification of Alzheimer's Disease

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In general, when a brain neural stimulation device is used in brain disease or dementia, it does not reflect the pathophysiology, because regardless of the brain disease state, it stimulates only the motor cortex. In particular, it is necessary to be more cautious about the reactivity, plasticity, and connectivity of brain diseases and the dementia-related cortex. The brain neural stimulation device affects the distance and intensity of the stimulus coil from the initial brain axis of Alzheimer's Disease (AD). The volume of cerebrospinal fluid increased by cortical hyperactivity and cerebral atrophy changes the characteristics of the brain tissue, and the current induced in the magnetic stimulation device has a negative effect. The stimulation site is biochemically and metabolically invaded by areas other than the motor cortex. Changes in the motor cortex cause problems later on. The present research confirms the cerebral state in real time by the EEG sensor that grasps the cerebral state, and the EMG sensor that can grasp the nerve conduction state, for the treatment suitable for the modified cortical form of the brain disease patient. The optimal stimulation therapy pulse and optimal pulse-forming device suitable for lesion-modified cerebral morphology were used. EEG and EMG are monitored in real time by cell phones, computers, and the web to implement the neural stimulation device of therapy pulse suitable for the modified cerebral cortex. In addition, stimulation coils can be replaced or added depending on the performance and capacity of the power source device, and control of the pulse shaping device, in order to implement a therapy pulse corresponding to the modified cortical morphology of the brain disease patient.

Keywords : Magnetic field, cerebral cortical modification, magnetic stimulation

1. Introduction

The conventional transcranial magnetic stimulator (TMS) is being actively studied in various fields, including the rehabilitation area, because it can control the excitation degree of the brain by a noninvasive method. The therapeutic approach is easy, and it stimulates the cerebral cortex, depending on the stimulation condition [1]. The local changes in the cerebral cortex due to cranial magnetic stimulation have been reported in many studies to change the excitability of the cortex spinal cord in water for several hours, when stimulated in the primary motor area [2]. The effect of the cerebral cortex on the motor area at 5 Hz above the threshold value and the stimulation of the cervical space in the cervical vertebrae indicate that

the activity of the downward cortical spinal cord increases the magnitude of the motor-induced potential. In the case of subthreshold stimulation, there is no significant change in the motor-induced potential, and the short-intracortical inhibition (SICI) of the cerebral cortex is reduced [3, 4].

Repetitive stimulation of the motor area with low frequency increases the motor threshold. After single stimulation, the motor-induced potential decreases; and after the paired pulse, the cortical facilitation increases, and the cortical latency decreases [5]. In normal individuals, 1 Hz stimulation of the motor cortex and visual cortex lowers cortical excitability at the stimulation site, while high frequency of (5-20) Hz decreases the motor threshold at the stimulation site, and increases cortical excitability. This shows that the treatment can lead to a broader activity of the neural network involved than the stimulated region, showing that the neural network effect is functional, and shows a noninvasive brain stimulation, it

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is possible to try various changes of the deep part distant from the stimulation site, and stimulate the autonomic nervous system of the hypothalamus through cerebellum stimulation.

TMS stimulates the cerebral cortex in response to stimulation conditions. The effect of noninvasive brain stimulation is appropriate for the cerebral cortex [6]. Several studies have shown that short-term effects in neural networks due to repeated cranial magnetic stimulation can have more complex effects in cerebral lesions including stroke, and have a plastic effect.

Although studies to measure the effects of stimuli on neural networks are limited, changes in the motor area may induce a plasticity change when observed with functional magnetic resonance imaging before and after stimulation in stroke patients [7]. The stimulus activates the caudate nucleus, basal ganglia, and lesion side sagittal, as well as the lesional frontal and parietal lobes. The change of neural network not only affects exercise learning of normal adults, but can also play a role in promoting the brain plasticity mechanism of stroke patients [8]. In the case of some areas of the brain damaged by cranial magnetic stimulation, there is a possibility of achieving a functional recovery of the brain by using an undamaged neural network. Planning and applying brain recovery can be a therapeutic approach that can lead to functional recovery in the clinic [9].

In particular, depression, brain disease, dementia, and progressive irreversible cognitive decline in function, and changes in daily living function and neuropsychiatric behavior should be delayed and adapted for diagnosis and treatment [10]. Attempt at discovery is also needed before clinical symptoms occur.

2. Description of Magnetic Stimulation

In general, in brain diseases and dementia, when the neural stimulation device is used, it stimulates only the motor cortex irrespective of the brain disease state, and thus does not reflect the pathophysiology. Therefore, it is necessary to be more cautious about the reactivity, plasticity, and connectivity of brain diseases and dementiarelated cortex [1]. The cerebral ventricle itself is associated with hyperactivity of the cortex. The volume of cerebrospinal fluid changes the characteristics of brain tissue and affects the induced current. Biochemical and metabolic studies of AD show that stimulation sites are primarily involved in areas other than motor cortices, and changes in motor cortical areas occur later [5].

Alzheimer's early brain dynamics affect the intensity of the stimulus coil's distance. The volume of cerebrospinal fluid volume is increased in cortical hyperactivity, and cerebral atrophy changes brain tissue characteristics and negatively affects the induced current from magnetic stimulation devices [2]. The levels of amyloid- β , t-Tau protein in cerebrospinal fluid affect the effect of cranial nerve stimulation. According to Koch, there is a need to enhance the stimulation effect in patients with AD with high levels of t-Tau [1, 4]. Therefore, it may be necessary to control the levels of amyloid- β , t-Tau in cerebrospinal fluid during neuroimaging. In connection with the connectivity of the cortex, the linkage of the EEG and EMG sensor has the advantage of being able to distinguish between strengthening and suppression in the cortex [6].

A neuronal device and an EEG and EMG sensor linkage device that is capable of evaluating the physiological changes and connectivity of the brain in areas other than the motor cortex are required. Depending on the morphology of the cerebral cortex of patients with cerebral disease, a pulse-shaping device should be implemented to apply therapy pulse to the EEG and EMG sensor, and stimulus for treatment. In addition, real-time cell phone, computer, web, etc. can be monitored, and a pulse neural stimulation device that is suitable for various purposes should be implemented [8]. The present invention confirms the cerebral state in real time by the EEG sensor that grasps the cerebral state, and the EMG sensor that can grasp the nerve conduction state, in order to treat the cerebral disease patient's modified cerebral cortex shape. Optimal stimulation treatment pulse and optimal pulse shaping for cortical morphology modified by lesion were realized. We implemented the neural stimulation device with the therapeutic pulse that was suitable for the cerebral cortex modified by brain disease, while monitoring the EEG and EMG by cell phone, computer, and web in real time [2].

EEG and EMG sensors were added with removable coils and LEDs to detect abnormalities in the lesion area, and further, Short intra-cortical inhibition (SICI) stimulation pulses of (1-5) ms, long intra-cortical inhibition stimulation pulses of (20-200) mS, Intracortical facilitation stimulation pulses of (7-20) mS, Short afferent inhibition (median nerve) stimulation pulse of 20 mS, and other necessary therapy pulses were separately added [4]. In addition, stimulation coils can be replaced or added, depending on the performance and capacity of the power source device, and control of the pulse-shaping device, in order to implement a therapy pulse that corresponds to the modified cortical morphology of the brain disease patient. The EEG and EMG sensor modules consist of energy harvester, EEG, and EMG Front End (analog), and are light and small for convenience in carrying and moving. The microcontroller has an A/D converter, a memory, and a UART module, which can reduce power consumption, and a microprocessor module can be added (which is replaceable, depending on the DSP & FPGA performance). Also, BLE technology is supported by cellphones, it has an advantage that it can be widely used for IoT

3. Methods and Experiment Results

In Figure 1, The configuration of the therapeutic pulse cranial nerve stimulation suitable for cerebral cortical deformation is proposed in the present research [11]. The EEG and EMG sensor was used for precise diagnosis and treatment according to real-time cerebral cortex shape, and pulse for the stimulation suitable for cerebral cortical morphology modified by lesion was implemented. Symbol 1) indicates the process of processing the EEG data obtained from the sensor installed in the cerebrum, while symbol 2 indicates the process of transmitting the data obtained from the EEG sensor to the Bluetooth. Symbol ③ indicates the whole process that is monitored in real time. This involves computer, cell phone, EEG, and EMG. Symbol ④ indicates that the data is obtained from the Bluetooth connected to the microprocessor. Symbol 5 indicates the MSP 430 microprocessor used in this device, which controls the power. Symbol 6 indicates that the therapeutic stimulation pulse applied by the neuro-nerve conduction response in response to the human body can be confirmed. EEG and EMG sensors were added with removable coils and LEDs to detect abnormalities in the lesion area, and further, SICI stimulation pulses of 1-5 mS, long intra-cortical inhibition stimulation pulses of 20-200 mS, Intracortical facilitation stimulation pulses of 7-20 mS, Short afferent inhibition stimulation pulse of 20 mS, and other necessary therapy pulse are separately added.

In addition, stimulation coils can be replaced or added depending on the performance and capacity of the power source device, and control of the pulse-shaping device, in order to implement a therapy pulse corresponding to the modified cortical morphology of the brain disease patient. The EEG and EMG sensor modules consist of energy harvester, EEG, and EMG Front End (analog), and are light and small for convenience in carrying and moving. The microcontroller has an A/D converter, a memory, and a UART module, which can reduce power consumption, and a microprocessor module can be added (which is replaceable, depending on DSP & FPGA performance).

Peripheral-central Bluetooth was used for wireless communication, and monitoring was introduced for lesion monitoring by cell phone, computer, web, and neural stimulator. Sensors and stimulus coils can be added as needed for wire–wireless communication. In addition, the treatment data backup and accurate and intensive treatment were implemented.

In the present invention, signals (EEG signal, EMG signal, EEG and EMG measuring unit (analogue part)) measured in the cerebrum are processed in association



Fig. 1. (Color online) The configuration of the therapeutic pulse cranial nerve stimulation suitable for cerebral cortical deformation is proposed in the present research.

with the microcontroller unit and configured as a Bluetooth module. The signal amplified by the Instrumentation Amplifier is a 0.1 Hz RC circuit with a cutoff frequency, a DC filter is used to remove the DC component, and a Notch Filter is used to remove the 60 Hz power supply noise, after being amplified 50 times by the Instrumentation Amplifier. The signal amplified by the Instrumentation Amplifier is canceled by the low pass filter with the cutoff frequency of 100 Hz. Then, it is converted into digital signal by the built-in A/D converter, and then transmitted to the module through the UART module through the buffer, in the form of a Bluetooth ACL packet. BLE technology is supported by cellphones, it has an advantage that it can be widely used for IoT. The module used in the EEG and EMG signal measurement device transmits data to the monitoring module, the PC, and the Bluetooth module of the cell phone using the wireless communication and displays the signal on the LED by channel. Finally, the neural stimulator has high power consumption, and requires a high voltage power source. Therefore, a step size charging method was used to charge a high-voltage capacitor, and a small size DC power supply was implemented. The stimulation protocol drives the stimulation coil, which utilizes the phenomenon that an electric field is induced in the living body by applying a time-varying magnetic field to the living body.

In Figure 2, Symbol ① indicates the BMI module. Symbol ② indicates Multi-channel data processing mode. Symbol ③ is a block diagram of the EEG measurement and EEG processing for constituting pre/post processing software. The BMI sensor node consists of an electrode, analog front-end (AFE), and analog-to-digital converter (ADC). Electroencephalogram detection using a dry electrode is performed and converted into a digital signal through AFE and ADC. Symbol ④: Since the converted EEG does not have a large amount of data, it can be transmitted from the BMI sensor node to the data processing module by a serial communication method, such as I 2 C (inter-integrated circuit) of symbol (5). In the conventional technology, the EEG and EMG signal detected by the BMI sensor node is directly transmitted to the data processing module, and the data processing module converts the signal into a digital signal. There is also a technology for converting EEG signals into digital values and transmitting them to the data processing module at the BMI sensor node. This technique does not use a DSP for data preprocessing and filtering. The connection structure of the BMI sensor node in symbol 6 is the bus structure. The data processing module may use a commercial digital signal processor (DSP). In the present invention, primarily MSP 430 and high-performance DSP (DM8127) are used. We also implemented the EEG processing algorithm and brain feature extraction function.

In Figure 3, Symbol 1 is a transformer passed to the IGBT, symbol 2 is connected to the emitter-2 of the IGBT, symbol 3 is connected to the emitter-1 of the IGBT, and symbol 4 flows into the direct current via the bridge diode as an alternating current. Symbol 5 indicates



Fig. 2. (Color online) Symbol ① indicates the BMI module. Symbol ② indicates Multi-channel data processing mode.



Fig. 3. (Color online) Symbol 1 is a transformer passed to the IGBT, symbol 2 is connected to the emitter-2 of the IGBT, symbol 3 is connected to the emitter-1 of the IGBT, and symbol 4 flows into the direct current via the bridge diode as an alternating current.

a reference voltage, while symbol 6 indicates a PWM IC 494.

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In Figure 4, In the present invention, the cerebral state is detected by the EEG and EMG sensor, and sent to the Bluetooth nerve stimulation device indicated by the symbol (6). Symbol (1) indicates the driving power supply. The 2-Loop method is for a serious patient with brain disease, and is suitable for patients with dementia, severe depression, and acute stress. It is a method of implementing the treatment pulse according to symptom ② of the brain disease patient through the pulse-forming device indicated by symbol ③, according to the deformation state of the cerebral cortex. In the sensor indicated by the symbol ④, the required stimulation pulse type and stimulation capacity



Fig. 4. (Color online) In the present invention, the cerebral state is detected by the EEG and EMG sensor, and sent to the Bluetooth nerve stimulation device indicated by the symbol ⁽⁶⁾. Symbol ⁽¹⁾ indicates the driving power supply.



Fig. 5. (Color online) In the present invention, the cerebral state is detected by the EEG and EMG sensor (symbol 4), and then the cerebral cortex state is transmitted through Bluetooth.

are set according to the size and condition of the cerebral cortex (symbol (5)), and the brain nerve device is applied to the brain disease patient. Patients with mild symptoms of cerebral disease are treated with small stimulus pulses, while patients with severe Alzheimer's (AD) are stimulated after selecting a form with a large stimulus pulse. Symbol \bigcirc indicates a flyback type power supply device, the therapeutic stimulation coil can be additionally attached and detached from small size to large size, and it shows a 2-loop (two pulse) stimulation coil. Symbol (8) indicates the nerve conduction test and EMG monitored in real time. Electroencephalograms are recorded in the cerebral cortex through the electrodes attached to the scalp. The amplitudes are several μV and several hundred μV , and the frequencies are 0.1 Hz and several hundreds of Hz. The signal is classified into alpha wave, beta wave, gamma wave, delta wave, and theta wave, depending on the band.

In Figure 5, In the present invention, the cerebral state is detected by the EEG and EMG sensor (symbol ④), and then the cerebral cortex state is transmitted through Bluetooth. The operation of the power source drive device (symbol ①) causes the neural stimulation device to operate. The 1-loop device is suitable for patients with mild brain conditions (depression, stress disorder). This is a method to implement the treatment pulse according to the cerebral disease patient through the pulse-forming device indicated by symbol ③ according to the deformation state of the cerebral cortex. The required stimulation pulse type (symbol ⑤) and stimulation capacity are set according to the size and condition of the cerebral cortex, and the brain disease person is able to drive the brain nerve device. Patients with mild symptoms of cerebral disease are treated with small stimulus pulses, while patients with severe AD are stimulated after selecting a form with a large stimulus pulse. Therapeutic stimulation coil is implemented in a configuration that can be additionally attached and detached from small size to large size, and shows a 1-loop (one pulse) stimulation coil (symbol 2). Symbol (6) indicates information processing. (Fp1, Fp2, F3, F4, T3, T4, P3, and P4) are placed in the region containing a large amount of the frontal region. (F3, F4, C3, C4, P3, P4, O1, O2) are selected when the brain is related to the entire cerebral cortex, such as sleep, or anesthesia, At this time, the frontal lobe that is located close to the eye is excluded, as safety may be affected. Symbol (8) shows the nerve conduction test and EMG monitored in real time.

In Figure 6, Symbol ① indicates the pulse forming network, Symbol ② indicates data input, Symbol ③ indicates the stimulation coil, Symbol ④ indicates the Rogowski coil, Symbol ⑤ indicates the microprocessor, Symbol ⑥ indicates the switch device-1, Symbol ⑦ indicates switch device-1, Symbol ⑧ indicates the drivers.

Figure 6 is the power supply device of the stimulation device of a new sequential discharge control method using the PFN method. When SCR SC is turned on, energy is charged to the capacitor of PFN, and then S1, S2, S3, and S4 that are required for 1-MESH, 3 MESH, 4-MESH, and 5-MESH, respectively, are sequentially turned on to operate. At this moment, Energy stored in the capacitor of the PFN is transferred to the stimulation



Fig. 6. (Color online) Symbol ① indicates the pulse forming network, Symbol ② indicates data input, Symbol ③ indicates the stimulation coil, Symbol ④ indicates the Rogowski coil, Symbol ⑤ indicates the microprocessor, Symbol ⑥ indicates the switch device-1, Symbol ⑦ indicates switch device-1, Symbol ⑧ indicates switch device-1, Symbol ⑧ indicates the drivers.

coil to operate. The stimulation coil can operate variously as a single, multi-mesh circuit. These circuits can have the necessary current pulses when charging the discharge energy and sending it to the stimulation coil. The energy stored in the capacity C is the same as the following Eq. (1):

$$E_o = \frac{1}{2} C V_o^2 \tag{1}$$

The damping factor to determine the shape of the current pulse is given by Eq. (2):

$$\alpha = k_0 (V_0 Z_0)^{-\frac{1}{2}}$$
(2)

where, Z_0 is the characteristic impedance of the PFN.

The single-mesh PFNs generate sinusoidal pulses, while multiple-mesh PFNs have square-wave pulses that have approximately the same peak and average power. The ability to vary the shape of the treatment pulse can extend the treatment area of various lesions. The sequential discharge scheme is used in the present invention and provided in a way that the pulse width can be freed. The stimulation coil is sequentially driven by the control circuit, AVR, TI system AT80S8535. By fusing MSP430 onechip microprocessor technology and PFN, it is possible to create high-voltage pulse shapes with various pulse widths by actively nesting one-stage three-, four-, and five-stage circuits. The SCR1, SCR2, SCR3, SCR4 and SCR5 are sequentially turned on according to the desired delay time, using a one-chip microprocessor. Therefore, the energy stored in the capacitor of each PFN circuit is sequentially applied with a fixed delay time, and the current waveform of the stimulation coil is measured while changing the delay time of the trigger conduction angle of each SCR from (0 to 500) µS. Current and voltage were measured using Lecroy's high voltage probe (Probe X 6000 V) and Penmuk company's Rogowski current waveform transducer (CWT), respectively. Assuming that there is no loss of resonance or discharge circuit in the experimental set, there is no change in VC at the start of discharge and at the end of discharge. In an actual circuit, a loss occurs in the electrical resistance of the magnetic field generating coil and wiring, and discharge is completed. The voltage drops. The power loss of the discharge circuit due to these voltage drops is calculated by the following equation, and the power capacity of the high voltage power supply charging the discharge capacitor should be larger than the power loss. C was 100) µF 2000 V, and L was manufactured and configured at 100) µH. The same discharge action SCR used the module for exclusive use of



Fig. 7. (Color online) Presents the current pulses after triggering S1 at 0 s without delay, and the simultaneous triggering of S2, S3 (50 μ S delay), and S4 (100 μ S delay).

SEMIKRON SKT760, and the high-voltage high-current circuit connection used parts with sufficient current capacity and ability to withstand voltage. Since the voltage Vi at the initial stage of discharge is 1,000 V in the experiment, and the discharge completion voltage Ve = 760 v, the power loss capacity can be determined by the following equation: A time-varying magnetic field of 1 tesla was generated at a distance of 50 mm from the stimulus generation coil at a voltage of 1,000 V at the initial stage of discharge. In addition, even if the switching operation of the SCR was performed during the discharging operation, the parts of the voltage and current of the SCR peripheral circuit stabilized, without exceeding the rating.

In Figure 7, Symbol ① indicates pulse forming network, symbol ② indicates data input, symbol ③ indicates stimulation coil, symbol ④ indicates Rogowski coil, symbol ⑤ indicates microprocessor, symbol ⑥ indicates switch device-1, symbol ⑦ indicates switch device-1, symbol ⑧ indicates switch device-1, and symbol ⑨ indicates drivers. Figure 6 shows a short intra-cortical inhibition (SICI) with a condition stimulus smaller than the threshold, followed by a large stimulation of (1-5) mS. This activates inhibitory neuronal circuits in the cortex, inhibits action potentials, and the reduced MEP through SICI reflects inhibition at the motor cortex level; such inhibition of responsiveness is mediated by GABA receptors. Long-term intra-cortical inhibition (LICI) is the

process of applying a conditional stimulus greater than the threshold value, and then applying it after a long time of (20 to 200) mS. LICI is involved in SICI and other cortical cell populations, and the inhibitory process is transmitted through GABA-B receptors. Intracortical facilitation (ICF) is the process of applying a large stimulus of (7 to 20) mS after applying a conditional stimulus that is smaller than the threshold, and condition stimulation strengthens the MEP of the stimulus in the ICF. ICF delivers an excitatory neurotransmitter system through the NMDA receptor. Short afferent inhibition (SAI) is a conditional stimulus on the median nerve of the wrist, followed by an experimental stimulus on the motor cortex after 20 mS, which inhibits MEP. SAI is transmitted by acetylcholine, and SAI reflects the excitability of the cholinergic motor cortex. Frequently used therapy pulses are used as a basis, and a pulse-forming device is added. The basic configuration of the pulse forming apparatus is as follows: Symbol ① shows the current pulses when S1, S2, S3, and S4 were triggered simultaneously. The delay time was 0 s, and the pulse width was 240 µS. Symbol 2 presents the current pulses of the stimulation coil after the simultaneous triggering of S1 and S2 and the subsequent triggering of S3 (50 µS delay) and S4 (100 µS delay). The current pulses had three stages with a pulse width of 720 µs. Symbol ③ gives the current pulses after triggering S1 at 0 s without delay, S2 (50 μ S delay), S3 (100 μ S delay),



Fig. 8. (Color online) When an abnormal signal is detected in the EEG and EMG sensor No. 9 (symbol ①), it is transmitted to the brain nerve stimulation device through the Bluetooth indicated by ②, and the concept is operated as the nerve stimulation device indicated by ③.

and S4 (150 μ S delay). The pulses had four flat stages with a pulse width of 960 μ S. Symbol ④ gives the current pulses after the simultaneous triggering of S1 and S2 and of S3 and S4 (50 μ S delay). They had two thick stages with a pulse width of 480 μ S.

Figure 7 presents the current pulses after triggering S1 at 0 s without delay, and the simultaneous triggering of S2, S3 (50 μ S delay), and S4 (100 μ S delay). The pulses had three convex stages with a pulse width of 720 μ S.

In Figure 8, When an abnormal signal is detected in the EEG and EMG sensor No. 9 (symbol ①), it is transmitted to the brain nerve stimulation device through the Bluetooth indicated by ②, and the concept is operated as the nerve stimulation device indicated by ③. Data can be backed up in real-time to the cerebral cortex, the EEG and EMG sensor can be added as needed, and a lamp can be displayed to output anomalous signal if it deviates from the reference waveform. Symbol ② indicates that it is detected by the EEG and EMG sensor.

Symbol ③ indicates the communication process with the neural stimulator and the EEG and EMG sensor, and it is indicated by LED when abnormal. Symbol ④ shows the concept of the Bluetooth-based real-time measurement and operation. Symbol ⑤ shows the operation concept of the two-pulse neural stimulator that recognizes 9 or more information data received from Bluetooth. Symbol ⑥ shows the concept of the neural stimulation device that operates after recognizing the anomaly detection in sensor # 2. Symbol ⑧ indicates the electromyogram, which is a real-time monitored nerve conduction test.

In Figure 9, The most important role of Bluetooth protocol is HCI and L2CAP. L2CAP protocol is basically a Bluetooth protocol. L2CAP is located directly above the HCI layer, and it can send and receive data packets up to 64 Kb to upper protocol or application. In the present invention, the HCI packet and the L2CAP packet are used. Symbol ① indicates stimulation in the EEG measurement device, symbol 2 indicates a cell phone, the connection between the microcontroller and the Bluetooth module, the initialization PC connection, and data transmission. In order to receive Bluetooth Qualification from the Bluetooth Specialist Group (SIG), the L2CAP layer must be configured. The packet used is the H4 mode defined in Bluetooth Specification version 1.0B. The parameter value of Event-packet can be different for each Bluetooth module. After initializing the Bluetooth module, the EEG measuring device searches the PC side Bluetooth module for connection with the PC, which is called the inquiry process. The Bluetooth module on the PC side is set to slave mode, and inquiry scan is performed for connection with the EEG measurement device of symbol (3), and symbol (4) can be stimulated. Finally, if inquiry length has passed or num response is satisfied, an inquiry complete is generated, the host is informed that the inquiry process has been completed. With the address of the PC side Bluetooth module of the symbol 5 obtained



Fig. 9. (Color online) The most important role of Bluetooth protocol is HCI and L²CAP.

from the inquiry, the microcontroller of the EEG and EMG measurement device commands connection of its Bluetooth module with the PC side Bluetooth module

through the Connection command. When the connection to the PC is established, the biometric signal is sent to the payload of the data packet. When disconnecting the con-



Fig. 10. (Color online) Symbol 1 is a digital display unit, an interface section from analog to digital, and symbol 2 is an analog processing unit for digitally amplifying and processing an overcoming signal.

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Fig. 12. (Color online) Symbol ① indicates the stimulation coil of the neural stimulator, ② indicates the Bluetooth, and ③ gate module.

nection, the disconnection command is used to terminate the connection. Through the same process as symbol 6, the Bluetooth receiver and transmitter of symbol 7 are initialized and connected.

After this process, data exchange can be performed between them. After this process, the Bluetooth receiver and the transmitter are initialized and connected. After this process, data exchange can be performed between them.

In Figure 10, Symbol 1 is a digital display unit, an interface section from analog to digital, and symbol 2 is an analog processing unit for digitally amplifying and processing an overcoming signal.

In Figure 11, This is a block diagram of the sensor module and gateway module in hardware. The hardware segment is largely divided into sensor module and gateway module, and the sensor module consists of control and transmission block. The sensor block accepts the biological signals measured in the brain. The control and communication block digitize the analog signal received from the sensor block and transmits it to the smartphone or gateway through Bluetooth communication. The gateway module functions to connect the received biometric signal to the Internet using IPv4 or IPv6 based TCP/IP communication.

In Figure 12, Symbol ① indicates the stimulation coil of the neural stimulator, ② indicates the EEG sensor, and ③ indicates the abnormal signal detection LED. This shows the composition of the Microcontroller and Bluetooth, and MSP430 (TI) is used for the Microcontroller. Since the MSP430 has a 6-channel 12-bit ADC, one channel is used for the EEG. In addition, they were converted to 512 Hz and 12 bit, respectively. The communication module uses FB155BC (Firmtech), which supports Bluetooth V2.0 standard. The connection to the MCU is controlled by the SPI Interface.

In Figure 13, Symbol ① indicates the stimulation coil of neural stimulator, ② indicates the EEG and EMG sensor, and symbol ③ indicates the abnormality signal LED. The protocol structure of the gateway module for transmitting the measured bio-signals (EEG, EMG) to the Internet is shown. The information received through the Bluetooth module is interfaced to the Internet via the TCP/IP protocol. Bluetooth was implemented using the FB155BC chip described above, and the TCP/IP protocol stack was implemented using the W7200 (Wiznet, Korea) chip. In Figure 14, Symbol ① indicates the stimulation coil of the brain nerve stimulation device, symbol ②



Fig. 13. (Color online) Symbol ① indicates the stimulation coil of neural stimulator, ② indicates the EEG and EMG sensor, and symbol ③ indicates the abnormality signal LED.



Fig. 14. (Color online) Symbol ① indicates the stimulation coil of the brain nerve stimulation device, symbol ② indicates the EEG, EMG sensor, symbol ③ indicates the abnormal signal detection LED, Symbol ④ indicates signal monitoring, symbol

indicates the EEG, EMG sensor, symbol ③ indicates the abnormal signal detection LED. This is a diagram of the monitoring program and the driving program of the software sector, and the software section consists of the monitoring program and the driving program. In the structure of the PC version monitoring program, the data transmitted from one channel is composed of 12-bit data and 4-bit channel information. The channel information is

used to distinguish the EEG and EMG signals, and the configured biometric information is stored in the Signal Monitoring module and drawn as a waveform. The FFT module implements the time series data in the frequency domain and the FIR module in the digital filter, to process the biological signals in real time. The smartphone version performs a similar function to the PC version, but does not store biometric signals, due to limited resources.



Fig. 15. (Color online) Symbol ① indicates the stimulus coil of the neural stimulator, ② indicates the EEG and EMG sensor, ③ indicates the abnormal signal detection LED, and symbol ④ indicates the server platform

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In Figure 15, Symbol ① indicates the stimulus coil of the neural stimulator, 2 indicates the EEG and EMG sensor, ③ indicates the abnormal signal detection LED is constructed using Node.js, NoSQL, HTML5.0. In order to store a large amount of data, Node.js, which is an eventbased distributed web server, was used as a database server. In addition, we constructed Mongo DB among DBMSs that deal with atypical relational databases. The TCP/IP protocol stack including IPv4 and IPv6 should be implemented. In the present invention, a W7200 chip manufactured by Wiznet Corporation is used. A drive program is required for the initialization process and autoconfiguration and mode setting functions. The drive program initialization process performs address registration, SPI interface initialization, hardware reset function, and so on. Auto-configuration for IPv4 and IPv6 first checks whether it belongs to the IPv6 environment, or basically operates in the IPv4 environment. If it belongs to the IPv6 environment, it is checked for whether it is set manually or automatically. If it is set automatically, the IPv6 autoconfiguration process is performed, and the IPv6 address is assigned. For TCP/IP communication, it is necessary to decide whether W7200 module operates in UDP mode, TCP server mode, or Client mode. In UDP mode, it determines whether to transmit or receive after opening a socket and performs the UDP function accordingly. The TCP server mode waits for the client's SYN message after opening the socket, and performs a 3-way handshaking process to respond when the SYN message is received. In TCP Client mode, after opening a socket, it sends a SYN message to the server, receives a response, establishes a connection, and sends/receives data.

4. Discussion

Using the present invention, the volume of cerebrospinal fluid increased by cerebral atrophy is treated, so that the brain nerve stimulation is not biochemically or metabolically invasive to regions other than the motor cortex, as the characteristics of brain tissue are changed. In cerebral disease and dementia, the neural stimulation device can be used to stimulate only the target cortex, by implementing a customized therapy pulse according to the cerebral cortex type and disease progression, and accurately reflect the pathophysiology. In the present invention, an integrated device for real-time coordination and operation of a brain nerve stimulation device and an EEG and EMG sensor capable of evaluating brain physiological changes and connectivity in a region other than the motor cortex is implemented. The EEG and EMG sensor and the stimulation therapy were introduced for diagnosis and treatment according to the lesion. Low frequency and high frequency are possible, and exercise thresholds are increased by repetitive nerve stimulation. After single stimulation, the motor-induced potential decreases; and after the paired-pulse, the cortical facilitation increases, and the cortical latency decreases.

The stimulus is inserted in the correct position and state. In addition, according to the shape and response of the brain, therapeutic stimulation pulses can be arbitrarily applied to verify the effect, and physiological measurements are made possible. For accurate real-time diagnosis and treatment according to the symptoms, wired and wireless communication are introduced, and a method of adding a sensor and a magnetic pole coil as necessary is introduced. The local effect of the cranial magnetic stimulation is confirmed by the motor-induced potential, and the diagnostic device can confirm the change of the cerebral cortex activity. Combining both linear and nonlinear EEG and EMG analyses can be used to analyze temporal and spatial changes in the entire cortical neural network of a partially stimulated neural stimulator. In addition, the stimulation of the cerebral cortex in accordance with the condition provides a strengthening effect.

5. Conclusion

The local changes in the cerebral cortex can result in a rapid therapeutic effect through the excitation of the cortical spinal cord in the primary motor area. Nonlinear EEG and EMG analysis can be applied to the physiological state of the living body, heart rate, nerve activity, blood flow in the kidney, arterial pressure, EEG, EMG, and respiration signal. The cell phone, PC, web, etc., can process the diagnosis device and the measuring device by Bluetooth in real time, and the accurate power and frequency of the spectral value can be known. Nonlinear EEG and EMG analysis and the correlation dimension and Lyapunov exponent decrease the complexity of the EEG, EMG, and therapeutic stimulation works correctly. The EEG and EMG sensor modules consist of energy harvester, EEG, and EMG Front End (analog), and are light and small for convenience in carrying and moving. The microcontroller has an A/D converter, a memory, and a UART module, which can reduce power consumption, and can add a microprocessor module (which can be replaceable, depending on the DSP & FPGA performance). The development platform of the present research adopts the technology of Bluetooth low energy (BLE). Since BLE technology is supported by cellphones, it has an advantage that it can be widely used for IoT. We used peripheralcentral Bluetooth for wireless communication. Finally, the

introduction of monitoring for real-time lesion monitoring was designed to link cell phone, computer, web, etc. with the neural stimulation device.

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