Interventional Spasticity Management for Enhancing Patient – Physician Communications

Improving Outcomes for Stroke Patients

Chiyuri NAGAYAMA
Department of Life Sciences
Graduate School of Ochanomizu University
Tokyo, Japan

***Abstract*—Stroke is the third most common cause of death in the Western world, behind heart disease and cancer, and accounts for over half of all neurologic admissions to community hospitals. Spasticity is commonly defined as excessive motor activity characterized by a velocity-dependent increase in tonic stretch reflexes. It is often associated with exaggerated tendon jerks, and is often accompanied by abnormal cutaneous and autonomic reflexes, muscle weakness, lack of dexterity, fatigability, and co-contraction of agonist and antagonist muscles. It is a common complication of central nervous system disorders, including stroke, traumatic brain injury, cerebral palsy, multiple sclerosis, anoxic brain injury, spinal cord injury, primary lateral sclerosis, and hereditary spastic hemiparesis. Leg muscle activation during locomotion is produced by spinal neuronal circuits within the spinal cord, the spinal pattern generator [central pattern generator (CPG)]. For the control of human locomotion, afferent information from a variety of sources within the visual, vestibular, and proprioceptive systems is utilized by the CPGs. Findings of this research can be applied to older adults in longitudinal home care who suffer spasticity caused by stroke.**

Keywords*—Electromyography; Spasticity; Internal Modeling; Viscoelasticity; Stroke Rehabilitation

I. INTRODUCTION

A. Stroke Rehabilitation

Stroke is the third most common cause of death in the Western world, behind heart disease and cancer, and accounts for over half of all neurologic admissions to community hospitals in Fig. 1. It is a common pre-cursor to placement in nursing homes or extended care facilities. Seven-hundred thousand new or recurrent cases of stroke are reported annually, and there are nearly 5.4 million stroke survivors currently in the USA. The estimated cost of care and lost income due to stroke in 2005 totaled $56.8 billion, of which costs due to lost income equaled $21.8 billion.

Comprehensive rehabilitation may improve the functional abilities of the stroke survivor, regardless of age and neurologic deficits, and may decrease long-term patient care costs [1]. Approximately 80% of stroke victims may benefit from inpatient or outpatient stroke rehabilitation. Ten percent of patients achieve complete spontaneous recovery within 8–12 weeks, while 10% of patients receive no benefit from any treatment. The literature suggests that intensive post stroke rehabilitation significantly improves functional outcomes. One meta-analysis of nine trials involving organized inpatient multi-disciplinary rehabilitation demonstrated significant reductions in death, death or institutionalization, and death or dependency. For every 100 patients receiving organized inpatient multi-disciplinary rehabilitation, an additional five returned home independently. Patients who were assigned to a specialized stroke service that included rehabilitation services had significantly greater survival rates at 1 year poststroke, better quality of life 5 years poststroke, and greater probability of surviving and living at home at 10 years poststroke. Stroke survivors admitted to inpatient rehabilitation facilities were more likely to return home than those admitted to traditional nursing homes, despite the higher costs. There is an association between the compliance with stroke guidelines and patient satisfaction, even after controlling for functional outcomes. While a small but statistically significant intensity-effect relationship exists between rehabilitation and functional outcomes, larger, more comprehensive studies are still needed to determine what aspects of rehabilitation are effective and why rehabilitation works (Fig. 2).

Motor recovery usually occurs in well-described patterns after stroke. Within 48 h of loss of movement, muscle stretch reflexes become more active on the involved upper and lower extremities in a distal-to-proximal direction. Onset of spasticity ensues thereafter, resulting in resting postures known as synergy patterns. Volitional movement returns in the same patterns, but eventually progresses to isolated movement. Spasticity decreases with increased volitional movement, but muscle stretch reflexes always remain increased despite total recovery [2]. Poor prognostic indicators for motor recovery include: proprioceptive facilitation (tapping) response greater than 9 days; prolonged flaccid period; onset of motion greater than 2–4 weeks; absence of voluntary hand movement greater than 4–6 weeks; and severe proximal spasticity.

Motor recovery occurs despite the presence of brain damage due to the unmasking of neural pathways and synapses not normally used for a given function that can be called upon to process the remaining input and ultimately replace the damaged system [3]. First described in the 1980s, researchers are gaining an understanding of the physiologic mechanisms of motor recovery after stroke. Positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have been helpful in identifying plasticity changes in association with improvements in motor function. The fMRI has demonstrated extended activation in the sensorimotor

cortex, premotor and dorsolateral prefrontal cortex, and around the perimeter of the infarcted area during rehabilitation interventions [4].

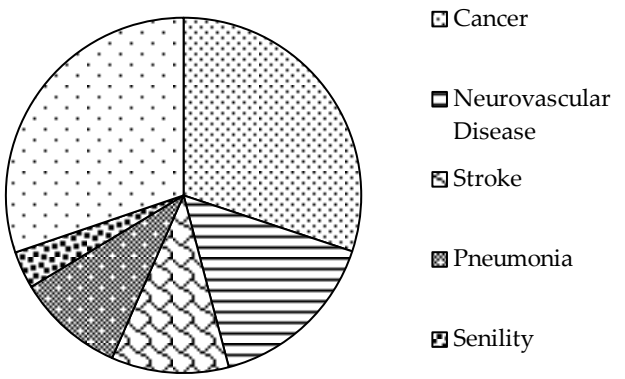

Fig. 1. Most Common Disease in Japan

Fig. 2. Passive Motion of Ankle Joint based on Daily Rehabilitation

B. Stroke with Cerebral Palsy

Cerebral palsy (CP) is the most common physical or motor disability affecting children in developed countries, the prevalence being 2.0–2.5 per 1000 live births. The prevalence has remained constant for decades, with improvements since the 1990s in live birth rates and the increased incidence of very pre-term births. Despite being a common and important clinical problem, there is still lack of precision in the definition of CP or as some prefer "the cerebral palsies". While the definition of CP has been refined from time to time, there appears to be a general agreement that the condition is characterized by "aberrant control of movement or posture appearing in early life, secondary to central nervous system lesion, damage, or dysfunction and not the result of a recognized progressive or degenerative brain disease".

Children with spasticity should be managed in a multi-disciplinary context, and a goal-oriented approach adopted based on clinical findings and the expectations and desires of the family and child. Careful selection of intervention methods for any muscle tone problem is very important. Some children with CP may depend on, and benefit from, primitive reflex patterns and increased muscle tone for weight bearing, transfers, and ambulation. However, in many other children eliminating spasticity enables them to be more functional. Treatment can also be classified as temporary (reversible) or permanent (irreversible) and as focal or generalized. Assessment of gait patterns is critical to help determine spasticity management, and physical therapy. Gait patterns can really only be precisely identified and categorized by three-dimensional (3D) gait motion analysis, but two-dimensional (2D) video recording with slow motion replay can also greatly enhance routine clinical observation.

C. Spasticity Management

Spasticity is commonly defined as excessive motor activity characterized by a velocity-dependent increase in tonic stretch reflexes. It is often associated with exaggerated tendon jerks, and is often accompanied by abnormal cutaneous and autonomic reflexes, muscle weakness, lack of dexterity, fatigability, and co-contraction of agonist and antagonist muscles. It is a common complication of central nervous system disorders, including stroke, traumatic brain injury, cerebral palsy, multiple sclerosis, anoxic brain injury, spinal cord injury, primary lateral sclerosis, and hereditary spastic hemiparesis. In many individuals, the presence of spasticity has negative consequences, interfering with mobility and activities of daily living. Disability may result from spasticity-related impairment of posture, abnormal quality of movement, painful spasms, and poor hygiene. In these patients treatment of spasticity is often considered (TABLE 1).

Muscle tone, defined as the resistance to externally imposed muscle movement, is modulated by central nervous system influences on the alpha motor neurons in the spinal cord. The pathways that regulate tone are similar to those that regulate voluntary and involuntary motor movements and, as a final common pathway, involve the spinal reflex arc. Alpha motor neurons that innervate muscle fibers are located in the ventral horns of the spinal cord, and comprise the efferent limb of this reflex arc. Afferent sensory impulses from muscle spindles are relayed to the spinal cord. Some of these fibers synapse directly on alpha motor neurons that innervate agonist muscles. This is a monosynaptic reflex pathway and allows for sensory feedback necessary for motor movements. There are several descending central nervous system pathways that synapse directly or indirectly on motor neurons and allow for the control of movement. Upper motor neuron lesions result in spasticity from a number of mechanisms. Collateral branches from these descending motor pathways excite inhibitory presynaptic interneurons. With an upper motor neuron lesion this excitation is removed leading to decreased "presynaptic inhibition" of the motor neuron pool and over-activity of the spinal reflex pathways.

Unfortunately, spinal injury is an increasingly common condition in Western societies, affecting over 100,000 citizens in the European Community. The pattern of incidence is largely due to accidental injury often in road traffic accidents and also reflects the relatively long life expectancy of these patients, which now approaches that of uninjured members of the community [5]. The spinal injury has many consequences such as loss of voluntary movement and sensation, muscle wasting, and urinary problems but the residual involuntary muscle tone and movements known as spasticity is the principle concern. Spastic contractions may be long lasting and lead to skeletal deformity or they may be brief and intense jerks of the limbs, which disturb sleep or make other activities, such as transfer from a bed to a wheel-chair difficult.

Spasticity is a major concern for those interested in rehabilitation of the spinal cord [6]. This is particularly true for those interested in reconstructing movements by electrical stimulation strategies, given that any programmed movement may be destroyed by an unexpected spasm. In addition, there is a fear than stimulation strategies may make spasticity worse. In general, physiotherapists, clinicians, and researchers believe that the frequency of spasms can be reduced following muscle training, but that the intensity of spasms may be greater. The increased force could be due to the same neural activity generating more force from the strengthened muscle. In addition, there were repeated comments from patients that their spasms were less troublesome, particularly during the night, resulting in their sleep being less disturbed. Whilst these statements were supported by patient diaries it was impossible to make any definitive statement about spasms and training. We hoped the repeated testing would answer this question but the results of the tests employed showed very little agreement and we conclude that there is still no single definitive measurement, which can define the intensity of muscle spasm.

TABLE I. BIOMEDICAL EVALUATION OF SPASTICITY CAUSED BY STROKE

	Grade	Degree of Muscle Tone
Ashworth Scale	1	No increase in tone
	2	Slight increase in tone
	3	More marked increase in tone
	4	Considerable increase in tone
	5	Affected part rigid in flexion or extension

II. EXPERIMENTAL SECTION

A. Passive Motion Device for EMG Measurement

One of the fundamental principles of electromyography (EMG) is the assessment of the peripheral nervous system's ability to conduct an electrical impulse. Nerve conduction studies and needle EMG are commonly referred to as EMG studies. Electromyography plays a crucial role in identifying disorders that affect the peripheral nerve, the dorsal root ganglia, the nerve root, or the anterior horn cell. Electromyography can also identify disturbances at the level of the neuromuscular junction and in the muscle. In addition, EMG studies can provide useful information in disorders involving the upper motor neurons or disorders of volition as well as evaluating gait. Thus, EMG serves as an important diagnostic and prognostic tool when applied within the context of the clinical neurologic examination. A detailed, focused history and neurologic examination should serve as the template upon which one designs and performs an EMG study. Data acquired during an EMG study must always be interpreted within the clinical context because the same data may have very different interpretations depending on the clinical situation. An EMG study performed in isolation of the clinical context may provide little useful information.

The primary goals of an EMG study are to localize the lesion, characterize the underlying nerve pathophysiology, quantitate the severity of the lesion, and assess the temporal course of the disorder. Localizing a lesion within the peripheral nervous system is best achieved with the use of an EMG study. An EMG study can be tailored in such a fashion as to specifically localize the lesion to the nerve roots, plexus, trunks, or individual peripheral nerves. A clinician designing an EMG study must have intimate knowledge of the anatomy of the peripheral nervous system for precise lesion localization. EMG studies can often identify the underlying pathologic process involving a nerve lesion, and can determine whether the pathology leading to the clinical deficit is secondary to axonal loss, demyelination, or if the underlying disorder is secondary to muscle disease or neuromuscular dysfunction. This differentiation between pathologic processes allows for a narrowing of the differential diagnosis. An EMG study can also assess the degree or extent of axonal loss versus demyelination, which then allows a clinician to predict the extent of recovery from a particular lesion and the expected time frame in which this recovery should take place. A lesion that primarily involves axonal loss will carry a worse prognosis, and therefore a less complete recovery would be expected. A lesion that is a result of demyelination can be expected to recover fully when given the required time for re-myelination. Lesions with mixed pathology will recover in an intermediate time frame. An EMG study can provide data on the temporal course and rate of recovery of a lesion. Abnormalities observed on EMG can separate lesions into acute or chronic. EMG must therefore take into account the patient's clinical time course for accurate interpretation of the abnormalities observed during the study.

B. Further Studies of Prosthetics with Stretch Reflex

BCI is a communication and control system that does not depend in any way on the brain's normal neuromuscular output channels [7]. Such a system involves an input stimulus such as a tactile stimulus. The user's intent is conveyed by brain signals (such as EEG) rather than by peripheral nerves and muscles, and these brain signals do not depend on neuromuscular activity for their generation.

Furthermore, as a communication and control system, BCI establishes a real-time interaction between the user and the outside world. The user receives feedback reflecting the outcome of the BCI's operation, and that feedback can affect the user's subsequent intent and its expression as brain signals. For example, if a person uses BCI to control the movements of a robotic arm, the arm's position after each movement is likely to affect the person's intent for the next movement and the brain signals that convey that intent. Thus, a system that simply records and analyzes brain signals, without providing the results of that analysis to the user in an online interactive fashion is not BCI.

Much popular speculation and some scientific endeavors have been based on the fallacious assumption that BCI is essentially a "wire-tapping" or "mind-reading" technology, a device for listening in on the brain, for detecting its intent, and then accomplishing that intent directly rather than through muscles. This misconception ignores the central feature of the brain's interactions with the external world: that the motor behaviors that achieve a person's intent, whether it be to walk in a certain direction, speak certain words, or play a certain piece on the piano, are acquired and maintained by initial and continuing *adaptive changes* in the central nervous system

(CNS) function. During early development and throughout later life, CNS neurons and synapses continually change both to acquire new behaviors and to maintain those already acquired. Such CNS plasticity underlies acquisition of standard skills such as locomotion and speech and more specialized skills as well, and it responds to and is guided by the results achieved. For example, as muscle strength, limb length, and body weight change with growth and aging, CNS adjusts its outputs so as to maintain and desired results.

TABLE II. UBIQUITOUS MONITORING BASED ON PROSTHETICS

	Example
Application of Human Numerical Modeling	Control Kitchen Tools
	Control Air Condition
	Control Robot
	Speaking with Child
	Control Wheelchair
	Communication

TABLE III. DEVELOPMENT OF A BIPEDAL ROBOT WITH NEUROPLASTICITY

	Example
Applications of Human Numerical Modeling	Help for Robot Walking
	Help for Robot Cooking
	Help for Robot Typing
	Help for Robot Dressing
	Help for Robot Eating
	Help for Robot Buying

This dependence on initial and continuing CNS adaptation is present whether the person's intent is accomplished in the normal fashion, i.e., through peripheral nerves and muscles, or through an artificial interface, BCI, which uses brain signals rather than nerves and muscles. BCI use depends on the interaction of two adaptive controllers: the user, who must generate brain signals that encode intent; and the BCI system, which must translate these signals into commands that accomplish the user's intent. Thus, BCI use is a skill that both user and system must acquire and maintain. The user must encode intent in signal features that the BCI system can measure; and the BCI system must measure these features and translate them into device commands. This dependence, both initially and continually, on the adaptation of a user to a system and a system to a user is the fundamental principle of BCI operation, and its effective management is the principal challenge of BCI research and development, shown in Table 2 and Table 3.

III. RESULTS AND DISCUSSION

A. EMG Activity Measurement

Difficulty in walking is reported by 10% of Americans. One-third report major difficulty. They are unable to walk or climb stairs or stand. The most rapid rates of increase occur after ages 54 and 74 years old.

Musculoskeletal and joint diseases account for 24% of cases of major difficulty, back pain for 8%, stroke for 5%, and multiple sclerosis for 2%. Falls affect 41% of these people yearly. Eleven percent never leave their home and only 32% get out of their home daily. By report, 25% receive some physical therapy during the year of major difficulty walking. At this level of difficulty, 48% with stroke use a cane, 28% use a walker, and 44% a wheelchair. Six months after a traumatic spinal cord injury (SCI), 2% of subjects graded by the American Spinal Injury Association (ASIA) scale as ASIA A (sensorimotor complete) are able to walk at least 25 feet 24 h after onset: this percentage is 30% for those graded ASIA B (motor complete), and 94% for those graded ASIA C.

Six months after stroke, 85% of patients with a pure motor impairment, 75% with sensorimotor loss, and 35% with sensorimotor deficits will recover the ability to walk at least 150 feet without physical assistance. These levels of recovery do not necessarily lead to the patient walking well enough to navigate outside of their home. Only 40% of patients who recover walking ability after stroke achieve community-walking velocities. More functional walking may also reduce risk factors for cardiovascular disease, recurrent stroke, and frailty by permitting more opportunity for exercise and fitness. The rehabilitation of walking poses some common questions about the services provided by clinicians [8]. How do we know when our patients have received enough goal-directed therapy for their level of motor control? What measures should we use to rate progress?

The study of movement control has relevance to our understanding of the brain and spinal cord function. However, it also has implications for various fields, such as neurology, cognitive neuroscience, rehabilitation medicine, and robotics. The understanding of movement disorders and their appropriate treatment critically depends on the knowledge of the neuronal mechanisms underlying functional movements. The study of movement disorders is a rapidly expanding field in medicine, leading to increasing costs for treatment and rehabilitation [9].

Locomotion is subconsciously performed everyday with high reproducibility. It is automatically adapted to the actual conditions, such as ground irregularities with a large security range. Changed locomotive characteristics are frequently the first sign of a central or peripheral lesion of the motor system. Neurological examination in such cases is characterized by changes in reflex excitability and muscle tone and leads to an appropriate diagnosis underlying the gait disorder. The physical signs obtained during the clinical examination can; however, give little information about the pathophysiology underlying the movement disorder: stretch reflex excitability and muscle tone are basically different in the passive (clinical examination) compared with an active motor condition (movement).

In addition, during a movement such as gait, several reflex systems are involved in its execution and control. Therefore, for adequate treatment of a movement disorder, we have to know about the function of reflexes in the respective motor task. A movement such as locomotion is determined by the stretch of EMG activation of antagonistic leg muscles as well as intrinsic and passive muscle properties.

The EMG activity recorded from the leg muscles (Fig. 3, Fig. 4, Fig. 5, and Fig. 6) reflects the action and interaction

between central programs and afferent inputs from various sources, which can only be separated to a limited degree [10]. For an assessment of the neuronal control of locomotion we have to record the EMG activity from several antagonistic leg muscles and the resulting biomechanical parameters such as joint movements and, eventually, of muscle tension. Using such an approach, it is possible to evaluate the behavior of neuronal and biomechanical parameters during a gait disorder. Any changes in the neuronal or biomechanical systems may lead to movement disorder.

Furthermore, impaired movement is not only the consequence of a defective central proprioception. Rather, the movement disorder also reflects secondary compensatory processes induced by the primary lesion. In many cases, the altered motor response can be considered as an optimal outcome for a given lesion of the motor system. The complexity of primary and secondary effects of a lesion requires a detailed analysis of the movement disorder to determine the target of any treatment.

Leg muscle activation during locomotion is produced by spinal neuronal circuits within the spinal cord, the spinal pattern generator [central pattern generator (CPG)]. For the control of human locomotion, afferent information from a variety of sources within the visual, vestibular, and proprioceptive systems is utilized by the CPGs. The convergence of spinal reflex pathways and descending pathways on common spinal interneurons appear to play an integrative role. The generation of an appropriate locomotive pattern depends on a combination of central programming and afferent inputs as well as the instruction for a respective motor condition.

This information determines the mode of organization of muscle synergies, which are designed to meet multiple conditions of stance and gait. Central mechanisms and afferent inputs interact in such a way that the strength of a reflex in a muscle or synergistic group of muscles is dependent on the actual task. The actual weighing of proprioceptive, vestibular, and visual inputs to the equilibrium control is context-dependent and can profoundly modify the central programming. Through this weighting, inappropriate responses are largely eliminated. Any evaluation of reflex function has to be assessed in connection with the actual motor programming, the biomechanical events (shown in Fig. 7), including their needs and their restraints.

Originality of this work is that measured spasticity of hemiparesis patients in stroke is different between spastic side and healthy side of soleus muscle. And in this work will further reveal the fact of internal modeling mechanism which is error function between spastic side and healthy side. We developed comparison between weak people and strong people are important for higher level education. So, we insist communication between patients with mental stress and people with skill of information technology enhance better learning in school.

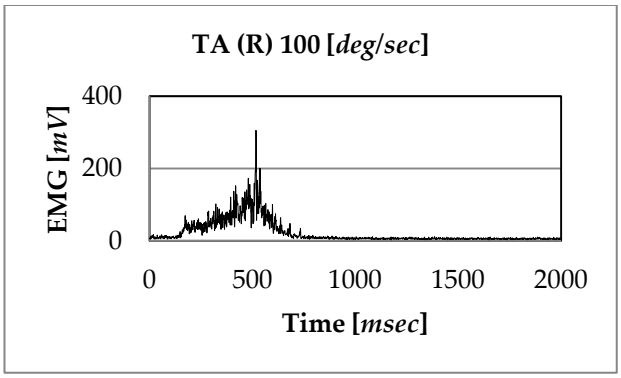

Fig. 3. EMG of the Tibialis Anterior Muscle of Spastic Limb

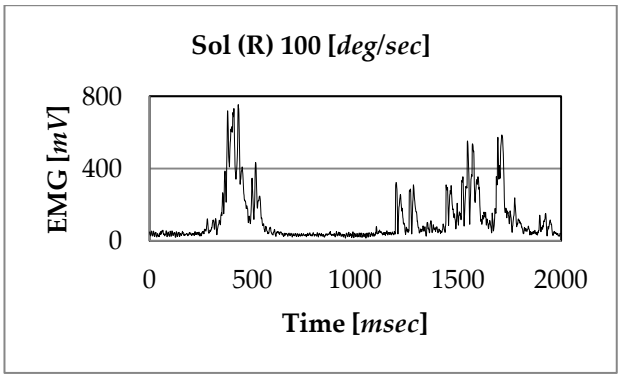

Fig. 4. EMG of the Soleus Muscle of Spastic Limb

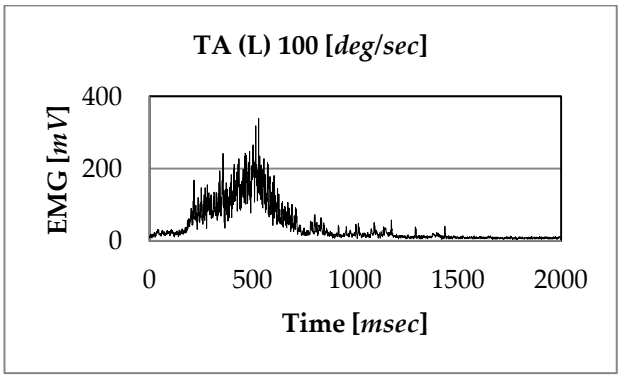

Fig. 5. EMG of the Tibialis Anterior Muscle of Healthy Limb

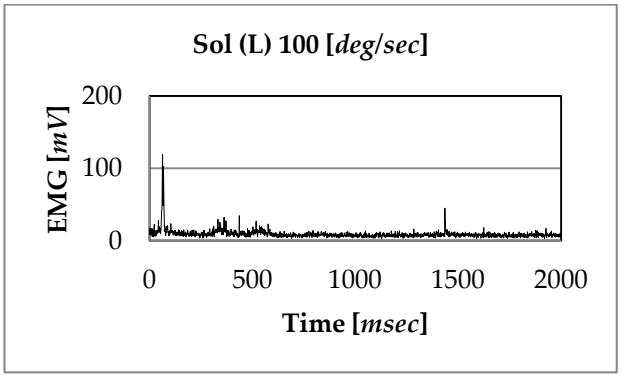

Fig. 6. EMG of the Soleus Muscle of Healthy Limb

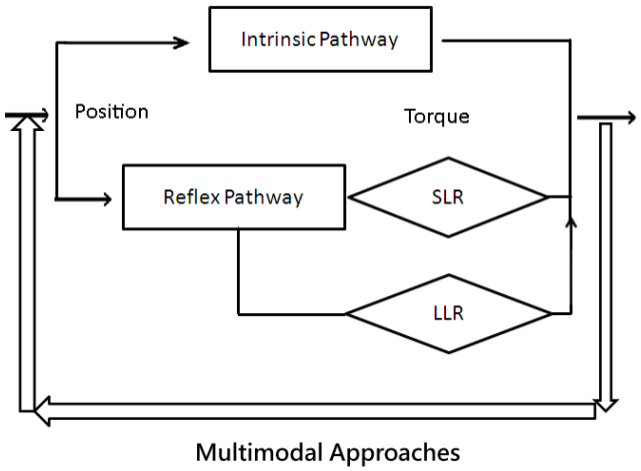

Fig. 7. Viscoelastic Properties of Spasticity for Motor Control System

B. Multimodal Approaches for Applications

It is known that the blind do experience mental imagery, although in the congenitally blind this cannot be visually based. In cases of acquired blindness, the ability to image visually decays over time [11]. A classic mental imagery task, originally introduced visually decays over time. A classic mental imagery task, originally introduced using visually presented stimuli, involves mental rotation. Blindness, particularly when congenital, shows performance of tasks requiring mental rotation of haptic stimuli. This suggests that visual imagery can facilitate haptic perception. ERP studies have shown that slow negativities recorded over the parietal scalp during mental rotation of haptic stimuli extend posteriorly over occipital areas in the blind. Similarly, relatively greater occipital negativity was observed in the blind than the sighted during mental imagery of textures felt with the fingertips. While the precise areas involved could not be localized, these studies indicate that the blind recruit their visual cortices for mental imagery in addition to the language functions discussed earlier. More recent PET studies show that early blind and sighted people activate rather similar areas during mental imagery.

Over the last few years, numerous investigations have revealed that visual cortical areas are active during tactile tasks even in sighted people. Although the tasks used in these studies differed from those employed to study the blind, and the precise nature of visual cortical processing in the blind and sighted remains to be elucidated, the findings raise the possibility that visual deprivation simply amplifies the normal range of cross-modal recruitment. The first report that visual cortical areas are active during tactile perception was based on a PET study. A contrast between an orientation discrimination task and a control task requiring discrimination of grating groove width yielded activation focused in the visual cortex. Others have shown that this focus was active during visual discrimination of spatial mental imagery of the grating. To rule out the possibility that cortical activation was merely an epiphenomenon, transcranial magnetic stimulation (TMS) was used to test whether blocking processing at this focus disrupts tactile perception. TMS applied directly over the locus of PET activation and at sites close to it (but not at more distant sites) significantly impaired performance in the grating orientation task.

These changes in performance and somatosensory cortex and visual deprivation are paralleled by both electrophysiology and autoradiography. An early indication of cross-modal plasticity in blind humans was provided by a report that used PET scanning to show that occipital cortical areas, generally considered to be visual in function, were more metabolically active in early blind individuals than in the late blind or sighted. This was interpreted as evidence of greater synaptic activity in the early blind, possibly reflecting incomplete developmental pruning of synapses. Subsequently, event-related potentials (ERPs) and MEG recordings were used to demonstrate occipital cortical recruitment in the blind during auditory discrimination of tones. However, because occipital activity was evoked in another ERP study during both tactile discrimination of line orientation and auditory tone discrimination, it has been suggested that this is the result of nonspecific attentional effects. Occipital cortical activity in the blind also occurs during sound localization, as first shown in ERP.

Considerable excitement has been generated by the observations that visual cortical regions of blind subjects are involved in reading Braille. One line of evidence for this comes from activation of neuroimaging studies. In interpreting these functional imaging studies, it is important to note that the underlying experimental design relies on measurement of a difference in the local hemodynamic response between an experimental condition of interest and a control condition. That the observed effects were due to finer movement was excluded by fMRI study of Braille character discrimination using stimulus presentations to the passive finger.

Activation of medical occipital cortex by Braille reading (relative to rest) occurs in early blind subjects, whereas these regions are deactivated in the late blind and sighted. A complementary approach to functional neuroimaging is provided by the technique of TMS. While the former method reveals brain areas that are active during a task, the latter can be used to transiently disrupt the function of a local cortical zone. If this interferes with performance of the task, it can be inferred that the cortical focus carries out processing that is necessary for the task. TMS over the medial occipital cortex impaired the ability of blind subjects to identify Braille or Roman letters and also distorted their subjective percepts of the stimuli. These effects (for Roman letters) were absent in sighted subjects, who were more susceptible than their blind counterparts to TMS over the sensorimotor cortex contralateral to the hand used. Thus, visual cortex is actually functionally involved in Braille reading.

This conclusion from the use of TMS is corroborated by the study of an early blind person who, after an infarct of the bilateral occipital cortex, developed alexia for Braille with otherwise normal somatosensory perception. Inactivation by TMS of the medial occipital cortex disrupted Braille-reading performance in the early blind but not late blind, implying that visual cortical involvement in Braille reading depends on cross-modal plasticity (shown in Fig. 8) that takes place during the critical period of visual development.

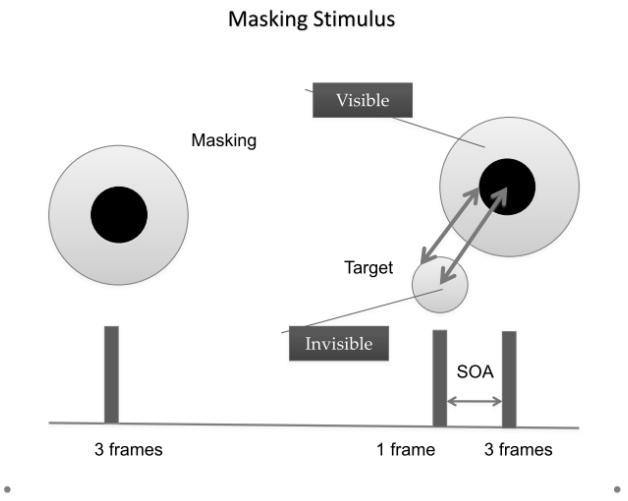

Fig. 8. Universal Design based on Internal Modeling of the Cerebellum

We have an idea about our practice that neighborhood walkability with visual light input (result of microwave). If people have impairments, we think they should go out and do daily works for their behavioral performance improvement. This functional recovery is also possible by wearable device such as Fig. 8. This kind of wearable device could be used in iPhone or PDA (smart phone) device.

IV. CONCLUSIONS

The individual computational elements of the nervous system—neurons—are physically small in diameter, allowing them to be packed together into dense nerve tracts and nuclei. To achieve biomimetic function, it is desirable to exchange information with neurons on a similar spatial scale. Microelectronic technology is just starting to work comfortably at the micron scale, but it remains difficult to engineer interfaces between electronic and biological structures with such small dimensions. Given, as yet, limited technology at our disposal, it has become necessary to look for situations in which relatively crude interfaces happen to produce clinically useful effects. In general, these consist of pathologies in which neural function is altered rather than absent; neural prosthetic treatment then consists of crudely modulating the residual activity to achieve a net benefit. This research should be applied for older adults in longitudinal home care who have spasticity caused by stroke. Author expects for readers that this study will practice in smart community project in Japan.

REFERENCES

[1] M. Gose, S. Plachta-Danielzik, B. Willié, M. Johannsen, B. Landsberg, and M. J. Müller, “Longitudinal influences of neighbourhood built and social environment on children's weight status,” Int. J. Environ. Res. Public Health, vol. 10, pp. 5083–5096, October 2013.

[2] M. Jerrett, et al., “Smart growth community design and physical activity in children,” Am. J. Prev. Med., vol. 45, pp. 386–392, October 2013.

[3] T. V. Calise, T. Heeren, W. DeJong, S. C. Dumith, and H. W. Kohl 3rd., “Do neighborhoods make people active, or do people make active neighborhoods? Evidence from a planned community in Austin, Texas,”Prev. Chronic Dis., vol. 10, pp. E102, June 2013.

[4] G. S. Lovasi, et al., “Neighborhood safety and green space as predictors of obesity among preschool children from low-income families in New York City,” Prev. Med., vol. 57, pp. 189–193, September 2013.

[5] Y. L. Michael, R.Gold, N. Perrin, T. A. Hillier, “Built environment and change in body mass index in older women,” Health Place, vol. 22, pp. 7–10, July 2013.

[6] S. J. Slater, L. Nicholson, J. Chriqui, D. C. Barker, F. J. Chaloupka, and L. D. Johnston, “Walkable communities and adolescent weight,” Am. J. Prev. Med., vol. 44, pp. 164–168, February 2013.

[7] Selzer et al., Textbook of Neural Repair and Rehabilitation, Cambridge University Press, 2006, vol. 1, pp. 602–615.

[8] M. Hoehner, S. L. Handy, Y. Yan, S. N. Blair, and D. Berrigan, “Association between neighborhood walkability, cardiorespiratory fitness and body-mass index,”Soc. Sci. Med.,vol. 73, pp. 1701–1716, December 2011.

[9] T. Hanibuchi, K. Kondo, T. Nakaya, K. Shirai, H. Hirai, and I. Kawachi, “Does walkable mean sociable? Neighborhood determinants of social capital among older adults in Japan,” Health Place, vol. 18, pp. 229–239, March 2012.

[10] L. Ada, W. Vattanasilp, N. J. O’Dwyer, and J. Crosbie, “Does spasticity contribute to walking dysfunction after stroke?” J. Neurol. Neurosurg. Psychiatry, vol. 64, pp. 628–635, May 1998.

[11] Selzer et al., Textbook of Neural Repair and Rehabilitation, Cambridge University Press, 2006, vol 1, pp. 180–194.