during a relaxed state. It is attenuated by movement execution and motor imagery. That phenomenon is referred to as event-related desynchronization (ERD) (Arroyo et al. 1993). The ERD of the mu rhythm, named mu ERD, is interpreted as the desynchronized activities of the activated neurons, and it appears around the motor area during motor execution, preparation and motor imagery (Pfurtscheller and Aranibar 1977; Pfurtscheller and Lopes da Silva 1999). Recently, electroencephalogram (EEG)-based BCI was applied to patients with chronic stroke (Daly et al. 2009; Shindo et al. 2011). These EEG-based BCIs controlled an orthotic device to extend their paretic fingers. The EEG-based BCIs detected mu ERD during motor imagery with EEG and reported that BCI training improved hand motor function in patients with chronic stroke.

However, the application of BCI to patients with severe motor disabilities has been limited, because of the difficulty detecting stable brain signals (Platz et al. 2000; Leocani et al. 2006). If it is possible to potentiate ERD, it would be easier to apply BCI to these patients.

Matsumoto et al. (2010) reported that tDCS could modulate mu ERD in healthy persons. Anodal tDCS (10 min, 1 mA) increased the magnitude of mu ERD in M1. If tDCS could also increase mu ERD in patients with severe hemiparetic stroke, it may be useable as a conditioning tool to facilitate the detection of more stable ERD for BCI application. Therefore, the aim of this study was to test whether anodal tDCS could increase ERD in patients with severe hemiparetic stroke.

Methods

Participants

Six patients with chronic hemiparetic stroke (4 males and 2 females) participated in this study. Inclusion criteria consisted of the following: (1) first unilateral subcortical stroke, not involving sensorimotor cortex as confirmed with brain MRI; (2) time from the stroke onset more than 6 months; (3) moderate to severe hemiparesis (participants could not move their paretic fingers individually); and (4) no motor improvement in the last 1 month before starting the intervention as confirmed by physicians and patients' testimonies. Exclusion criteria were as follows: (1) history of major psychiatric or previous neurological diseases, including seizure; (2) cognitive impairment precluding informed consent; (3) use of central nervous system-active drugs; and (4) implanted pacemaker or other metallic object. Participants' mean age was 56.8 ± 9.5 years. The mean time from the onset was 70.0 ± 19.6 months. The mean score on the Fugl-Meyer assessment of upper extremity motor score was 30.8 ± 16.5 (Fugl-Meyer et al. 1975), and the median score of the modified Ashworth scale for finger flexors was 1+ (range = 1+ to 2) (Bohannon et al. 1987). All participants were right handed. Clinical details of the participants are shown in Table 1. Additionally, seven age-matched healthy persons were recruited. All were right handed. Their mean age was 54.4 ± 6.1 years. We found no significant difference in the age between the stroke and age-matched healthy participants (unpaired t test, $P = 0.593$). The purpose and procedures of the study were explained to the participants, and written informed consent was obtained. The study was approved by the institutional ethics review board and performed in accordance with the Declaration of Helsinki.

Measurement of event-related desynchronization (ERD)

We assessed mu ERD during imagery of extension of the affected fingers just before and after anodal and sham tDCS over the motor area of the affected hemisphere in the stroke participants. The order of the stimulations was randomized,

Table 1 Clinical details of participants

Participant (sex)	Age	Dx	Lesion	Paretic side	Time from onset (months)	FM U/E	Modified Ashworth scale
1 (M)	67	CH	L thalamus	R	96	35	1+
2 (M)	44	CH	L putamen	R	64	50	1+
3 (M)	63	CH	R thalamus	L	49	12	2
4 (M)	46	CI	R corona radiata	L	48	49	1+
5 (F)	61	CI	R putamen	L	85	24	1+
6 (F)	60	CI	L putamen	R	78	15	2
Mean	56.8				70	30.8	1+[a]
SD	9.5				19.6	16.5	

CI cerebral infarction, *CH* cerebral hemorrhage, *FM U/E* Fugl-Meyer assessment score upper extremity motor score, *L* left, *R* right

[a] median value

and the interval between the stimulations was more than 2 days. In the healthy participants, we assessed mu ERD during imagery of right finger extension before and after anodal tDCS over the left motor area.

EEG signals were recorded with 15 Ag/AgCl disk electrodes (1 cm in diameter) with binaural references according to the international 10-20 system of electrode placement (FC3, FC1, FCz, FC2, FC4, C3, C1, Cz, C2, C4, CP3, CP1, CPz, CP2, CP4) with the average of bilateral earlobe references. Impedance for all channels was maintained below 10 kΩ throughout the experiment. Electromyograms (EMGs) were simultaneously recorded from the bilateral extensor digitorum communis muscles (EDC) with surface Ag/AgCl disk electrodes (1 cm in diameter) to monitor EMG activities during the imagery task to avoid unexpected muscle contraction. EEG and EMG were amplified, digitized with sampling frequency of 1,000 Hz and band-pass filtered (EEG 0.53–100 Hz, EMG 20-1 kHz) using a commercially available biosignal recorder (Neurofax EEG-9100, Nihon Kohden Corporation, Japan).

The participants sat in an upright position in an armchair with their eyes open facing the computer monitor showing the task. The monitor was placed approximately 0.5 m in front of the subjects at eye level. One trial started with an 8-s period of relaxation during which the word "Rest" was shown on the monitor. After that, the word "Ready" was shown for 2 s, then the word "Start" was presented for 5 s, and the participants were asked to imagine extension of their affected fingers. The trial ended when the word "Rest" reappeared, and the next trial began. They were given no feedback regarding EEG changes to avoid a learning effect. One session consisted of 20 trials. Before and after tDCS, three sessions were conducted with approximately 5 min of rest between each session. All three sessions were completed within 30 min (Fig. 1).

Quantification of ERD

Event-related trials lasting 5 s during motor imagery were selected for off-line data processing. All trials were visually assessed. The trials with artifacts resulting from eye movement and the trials with increased EMG activities were excluded. All trials were segmented into successive 1-s windows with 900 overlapping samples, and the Fourier transform with the Hanning window was applied in each segment. The power spectrum densities of each segment were estimated over the trials by Welch's averaged periodogram method (Welch et al. 1967).

The mu ERD was expressed as the percentage of the power decrease in relation to the 1-s reference interval before the direction of "Ready." The ERD at a certain frequency was calculated for each time (resolution = 0.1 s) and frequency (resolution = 0.98) according to Eq. (1).

$$\mathrm{ERD}\ (f,t) = \{[R(f) - A(f,t)]/R(f)\} \times 100\,(\%) \qquad (1)$$

where $A(f, t)$ is the power spectrum density of the EEG at a certain frequency band f[Hz] and time t[s] since the imagery task was started, and $R(f)$ is the power spectrum at the same frequency f[Hz] of the baseline period (a 1-s interval before the direction of "Ready" was displayed). The largest power decrease during motor imagery was selected as the value of mu ERD. The values of mu ERD before tDCS application were compared in all adjacent pairs of bipolar derivations of EEG and determined the electrode pairs showing the strongest value of mu ERD for individuals. The values of mu ERD in two stimulation conditions (anodal and sham stimulation) were calculated from the same bipolar derivation of EEG. All off-line analyses of EEG data were performed using MATLAB (The MathWorks, Inc. USA).

Transcranial direct current stimulation (tDCS)

The tDCS was applied through rectangular saline-soaked sponge electrodes (50 × 70 mm) with a battery-driven stimulator (CX-6650, Rolf Schneider Electronics, Gleichen, Germany). In the stroke participants, the position of M1 of the affected hemisphere was determined as the symmetrically opposite side of M1 of the unaffected

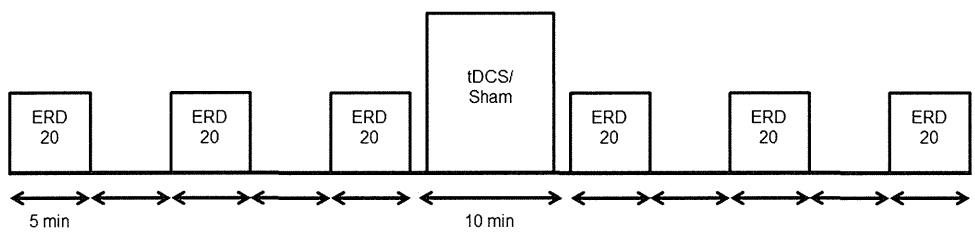

Fig. 1 The paradigm of the experiment. We assessed the ERD during imagery of the affected fingers extension just before and after the anodal and sham tDCS over the motor area of the affected hemisphere. The order of the stimulations was randomized, and the interval between the stimulation was more than 2 days. One ERD assessment session consisted of 20 trials. One trial consisted of an 8-s period of relaxation, a 2-s period of ready state, and a 5-s period of imagery. Before and after tDCS or sham stimulation, three sessions were conducted with approximately 5 min of rest between each session

hemisphere confirmed by the induction of the largest motor-evoked potentials (MEPs) in the unaffected EDC muscle with constant stimulus intensity using TMS with a figure-eight stimulation coil connected to a Magstim 200 magnetic stimulator (Magstim, Whitland, UK). This is because the MEPs could be evoked in the unaffected hemisphere but not in the affected hemisphere in all stroke participants. The anode was placed over M1 of the affected hemisphere, and the cathode was placed over the opposite side in the supraorbital region. In the active condition, tDCS was applied for 10 min with a current intensity of 1 mA. Participants sat awake in a comfortable armchair during the stimulation. In the sham stimulation, the electrodes were arranged similarly to the anodal stimulation and applied stimulation within the first 10 s only to mimic the transient skin sensation at the beginning of actual tDCS without producing any conditioning effects on the brain (Furubayashi et al. 2008). In the healthy participants, the anode was placed over the left M1 determined by TMS, and the cathode was placed over the right supraorbital region. TDCS was applied to them for 10 min with a current intensity of 1 mA. To place the tDCS electrodes on the head, 3 to 4 EEG electrodes over the stimulus site were removed after marking the scalp. After the stimulation, the EEG electrodes were set again on the same position as before. Because it took less than 3 min for electrode replacement, the effect of elapsed time after tDCS on the ERD measurement was limited.

Statistical analysis

To analyze the difference in mu ERD value and baseline EEG power spectrum with stimulation (both anodal tDCS and sham stimulation), Wilcoxon signed-rank test was used. To compare the mu ERD value between the stroke and healthy participants, Mann–Whitney test was used. Statistical analysis was performed with SSPS 18.0 J (SSPS Japan).

Results

None of the participants reported any adverse effects during or after the experiment. All participants showed mu ERD during motor imagery. The changes of ERD with tDCS in the stroke and healthy participants were shown in Fig. 2. Anodal tDCS significantly increased mu ERD in both the stroke ($P = 0.028$) and healthy participants ($P = 0.018$), though we did not find any significant change of mu ERD in the stroke participants with sham stimulation ($P = 0.084$). The mean (SD) values of mu ERD in the stroke participants were 21.2 % (11.7) before anodal tDCS and 21.4 % (15.8) before sham stimulation, and there was no significant difference in the baseline mu ERD values ($P = 0.818$). The mean mu ERD value (SD) before tDCS in the healthy participants was 28.0 % (7.2). The mu ERD values before tDCS in the healthy participants were relatively larger than those in the stroke participants, though the difference was not significant ($P = 0.317$).

We found no significant difference in the baseline EEG power spectrum between before and after tDCS in both the stroke and healthy participants. The mean (SD) value of the baseline EEG power spectrum was 0.46 μV^2 (0.24) before anodal tDCS and 0.47 μV^2 (0.26) after anodal tDCS in the stroke participants ($P = 0.715$), and 0.23 μV^2 (0.12) before anodal tDCS and 0.27 μV^2 (0.15) after anodal tDCS in the healthy participants ($P = 0.398$).

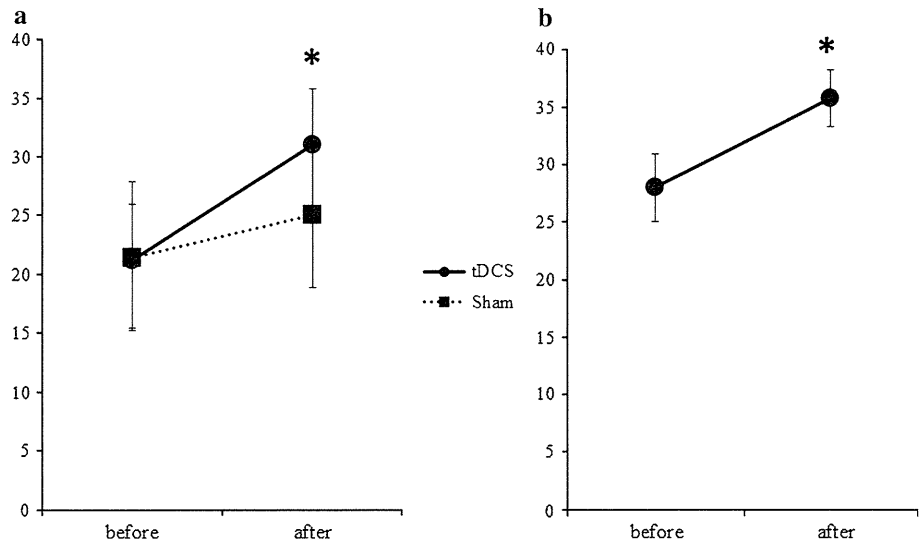

Fig. 2 Changes of mu ERD during the motor imagery with tDCS in the stroke participants (**a**) and the age-matched healthy participants (**b**). The *circle* shows the mean ERD before and after anodal tDCS, and square shows the mean ERD before and after sham stimulation. *Error bars* are standard errors. *Wilcoxon signed-rank test $P < 0.05$

Discussion

We found that anodal tDCS was able to increase ERD during imagery of extension of the affected fingers in patients with chronic severe hemiparetic stroke as same as age-matched healthy persons. This result was similar to younger healthy persons as demonstrated by Matsumoto et al. (2010). It has been reported that anodal tDCS increases cortical excitability (Nitche and Paulus 2000). It was supposed that anodal tDCS increased spontaneous neuronal firing (Bindman et al. 1964; Purpura and McMurtry 1965) and depolarization of the resting membrane potentials (Bindman et al. 1964; Nitche and Paulus 2001; Nitsche et al. 2003). The mechanism of ERD is thought to be a decrease in synchrony of the underlying neuronal population (Pfurtscheller and Lopes de Silva 1999). Therefore, modulation of ERD with tDCS could be explained by changes in the oscillatory behavior of cortical neurons, such as membrane potentials in the primary motor area, and the neurons firing according to input signals in response to motor imagery. An increase in cortical excitability, such as depolarization of the membrane potential of the cortical neurons in the M1, will result in more activated and desynchronized neurons, based on the input signals from motor imagery, which will strengthen ERD.

EEG patterns in patients with stroke are different from those in healthy subjects. Platz et al. (2000) showed that stroke patients with somatosensory deficits had reduced alpha centroparietal ERD during movement preparation and execution. We found that the baseline ERD values of the stroke patients in this study were relatively smaller than the values of the age-matched healthy participants. Anodal tDCS may lead to normalization of the pattern of EEG by increasing ERD of the affected hemisphere in patients with severe hemiparetic stroke. Since ERD was fully detected in every patient before tDCS, it might be interesting to replicate this study in more severe patients in order to fully test for the interest of the present findings.

There are several limitations to be considered in this study. First, we determined the position of M1 of the affected hemisphere using the symmetrical opposite side as a marker, that is, M1 of the unaffected hemisphere. This is not the exact position decided by motor-evoked potential (MEP) of the affected EDC by directly stimulating the affected hemisphere. This is because MEP could not be evoked from the affected EDC. Second, because we used fairly large (5 cm $\times$ 7 cm) electrodes for tDCS, we could not exclude the aftereffect of the premotor cortex or sensory motor cortex. Thirdly, there is the possibility that some participants did not imagine well before the stimulation. This is very difficult to assess. It could be supposed that tDCS directly influenced the attention (Kang et al. 2009). Further study of the relationships between modulation of ERD and stimulation site among patients with stroke is needed.

In conclusion, anodal tDCS can increase mu ERD of the affected hemisphere in patients with severe hemiparetic stroke as well as in healthy persons. Therefore, it could be a conditioning tool for EEG-based BCI to make detection of ERD easier.

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上肢機能治療戦略の最前線

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片麻痺上肢機能障害に対するアプローチは，ともすると能力低下の代償が強調されていた．しかし近年，上肢機能障害に対するさまざまなアプローチが報告され，その有効性も示されている．本稿では，近年報告されている上肢機能障害へのアプローチを概説するとともに，われわれが開発したHANDS therapyならびにBrain-Machine Interfaceを紹介する．

Key Words: 脳卒中（stroke），片麻痺（hemiparesis）

Ⅰ．はじめに

脳卒中片麻痺患者において実用手を獲得するのは，リハビリテーション（以下，リハ）病院入院患者の約3～40％である．発症から2～3カ月以内にそれぞれの手指の屈曲伸展が可能となるいわゆる手指の分離運動が出現する例ではその90％以上が回復期のリハにより実用的な手の機能の獲得が可能であるが，分離運動が出現していない例では実用性の獲得が非常に限られているのが現状である．

また片麻痺患者の上肢機能障害に対しては，その機能障害の程度によらず能力低下へのアプローチに重きが置かれるあまり，健側による代償が強調されて，機能障害へのアプローチが十分されず，いわゆるlearned non-useを作っているとの指摘もある[19)]．また，実用的な機能を獲得していても，実際の生活においては非麻痺側を使用してしまい，learned non-useを作っている場合もある．その反省に基づき，近年は従来の運動療法ならびに作業療法に加えて，Constraint-induced movement therapy（CIMT）[19, 20)]，装具の使用[5, 18)]，治療的電気刺激（therapeutic electrical stimulation: TES）[1, 3, 4)]，Hybrid Assistive Neuromuscular Dynamic Stimulation（HANDS）therapy[6, 10, 14)]，反復経頭蓋磁気刺激（rTMS），経頭蓋直流電気刺激（tDCS）[8)]，Brain-Machine Interface（BMI）[2, 15)]などのさまざまな機能障害へのアプローチが報告されている．

本稿では，近年新たに報告されているこれらの治療の実際ならびに効果につき概説する．

Ⅱ．Constraint-induced movement therapy（CIMT）

脳損傷後の四肢の機能回復におけるlearned-non useの存在がTaubらにより指摘され，従来，

ともすると健側上肢による代償による能力低下へのアプローチが優先され，機能障害へのアプローチが十分になされていなかったのではないかとの反省に基づきWolfらにより提唱された治療法である[19]．日中，健側上肢を拘束することによる麻痺側上肢の強制使用を促すいわゆるforced useにさらにshapingを基に訓練士による1対1での訓練を1日6時間行うCI療法に発展した．

CI療法の効果機序としては，いわゆるlearned-non useの解消ならびにuse-dependent plasticityが主に挙げられている．

米国においてThe EXCITE Randomized Clinical Trialが行われ，2006年にその結果がJAMAに発表されている[20]．CI療法は通常のリハに比し有意に上肢機能の改善を認め，麻痺側上肢の日常での使用頻度も増加を認め，その効果は1年後にも持続されていた．実際の臨床場面では1日6時間もの1対1対応ができる施設は限られ，また麻痺に関しても，長時間の健側の拘束に耐えうる麻痺肢の機能が要求され，適応には限りがあるのが現状である．そこでもう少し時間や頻度を減らしたmodified CI療法も開発されているが，簡便性の問題と適応の限界は存在する．

しかしながら，CI療法は，慢性期の片麻痺患者においても，十分な訓練量により日常生活での麻痺手の使用を増やすことが可能であり，機能障害の改善が見込まれるということを示した点で，上肢機能障害へのリハに対して，非常に重要なインパクトを与えたことは言うまでもない．CI療法の出現以降，上肢機能障害に対するアプローチが次々と発表され，上肢機能障害も回復しうるという認識が広まりつつある．

Ⅲ．装具（図1）

片麻痺患者における上肢装具は古くから用いられており，上肢筋緊張の抑制への効果が報告されている．

Fujiwaraらは長時間使用に耐えうる簡便で通気性の良いwrist-Hand splint（**図1A**）の装着により，屈筋共同運動パターンの患者で随意運動時の屈筋群の過剰な筋活動を抑制できることを報告し，さらに日中8時間の装着により自動運動可動域ならびに痙縮の改善を認めることを報告している[5]．

日中の活動時に装着することにより日常生活の諸動作による上肢筋緊張の増強を抑制し，連合反応などの出現も抑制することが可能である．痙縮の抑制効果は手関節のみならず，手指，肘，肩にも及ぶ．機序に関しては持続伸張によるmonosynaptic spinal reflexの抑制のみならず，type 2 afferentを介するpolysynaptic spinal reflex pathwayの関与も示唆されている[5, 18]．

また手指機能の改善には，C barによる母指の外転，対立位の保持や掌側支えによる手掌アーチの再建も有効である（**図1B**）．手指集団屈曲レベルでも，これらにより，母指，示指によるlateral pinchを再建することにより補助手としての機能の獲得が可能な例も存在する．

Ⅳ．電気刺激療法

従来より，筋再教育目的に低周波刺激は用いられており，適切な量の電気刺激は麻痺肢の回復を促すとされており，CauraughらによるEMG trig-

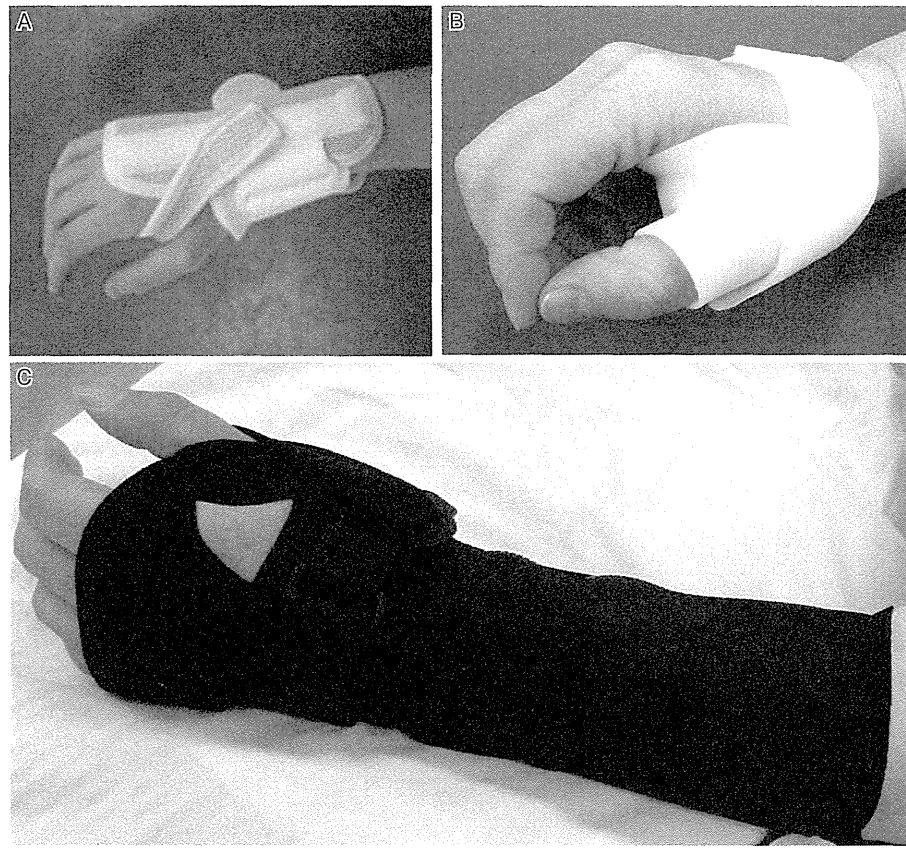

図1　装具
A：手関節固定装具，B：短対立装具，C：長対立装具
通気性に優れた素材でできた既製品．長時間の使用が可能．

gered Neuromuscular electrical stimulation (EMG-NMES) の効果も報告されている[3]．EMG-NMESは麻痺肢の筋活動をトリガーとして一定の電気刺激を行うものである．通常の電気刺激がpassiveな刺激であるのに比し，刺激の開始は麻痺肢の随意収縮によるため，随意性を高めると考えられている．de Kroonらは脳卒中麻痺側上肢に対する電気刺激療法のSystematic reviewを行い，通常の電気刺激を行うよりもEMG-NMESのほうが効果が出る可能性が高いと報告している[4]．Boltonらも Meta-analysis における EMG-NMES の効果を報告している[1]．

また，Cauraughらのグループでは EMG-NMES を非麻痺側上肢との両手運動訓練と併用することにより効果を挙げている．

Ⅴ．Hybrid Assistive Neuromuscular Dynamic Stimulation（HANDS）

Khaslavskaiaらは随意収縮単独や電気刺激単独と比較して，電気刺激に合わせて随意収縮を行

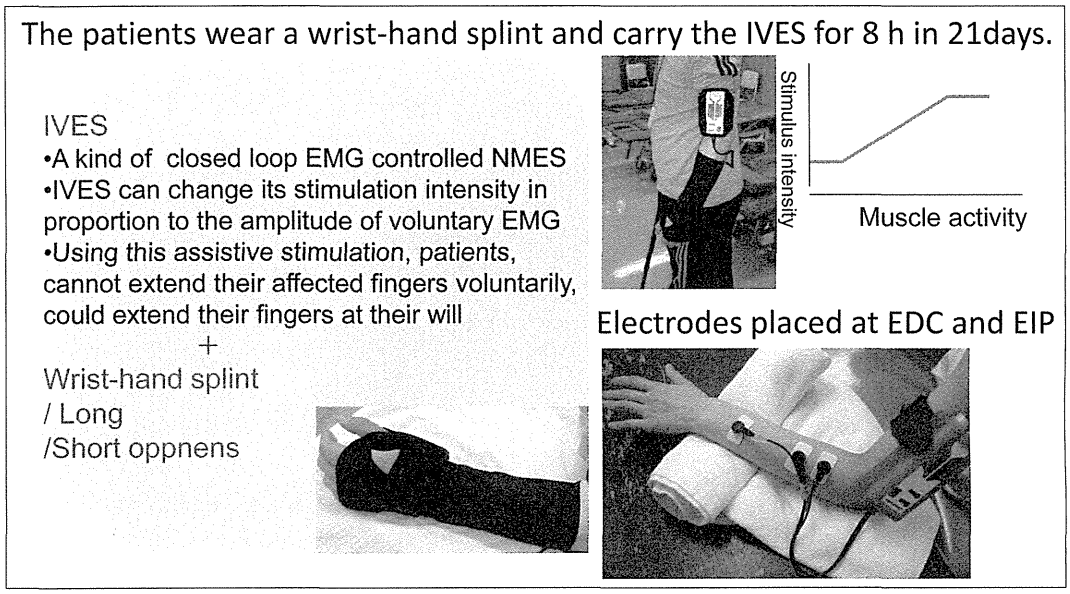

図2 Hybrid Assistive Neuromuscular Dynamic Stimulation (HANDS) therapy
Integrated Volitional control Electrical Stimulation (IVES) と長対立装具を8時間装着し，刺激装置はアームバンドに収納し携帯．訓練のみならず，日常生活での麻痺肢の使用を積極的に促す．

った場合に皮質運動野の興奮性が有意に増加することを示している[11]．よって電気刺激を行う場合でも，afferent stimulation を passive に与えるだけでなく随意運動による central command を同時に入力することが motor cortex の plasticity をより引き起こすと考えられる．さらに刺激自体も随意運動によってコントロールできれば，その効果はさらに増強することが考えられる．

村岡ら[13]は，随意運動介助型電気刺激装置(Integrated Volitional control Electrical Stimulator：IVES) を開発した．IVES では標的筋の随意筋電量に比例した電気刺激が可能であり，随意収縮の強弱のコントロールや，運動の中止，収縮後の脱力の学習が可能である．また本刺激装置は一度条件設定を行えば，本体に記憶させることが可能で小型で携行可能である．Hara ら[7]はこの装置を脳卒中片麻痺上肢の訓練に応用し，その有効性を報告している．

Fujiwara らは IVES と手関節固定装具（図 1B, C）を日中8時間着用して日常生活における麻痺側上肢の使用を促す Hybrid Assistive Neuromuscular Dynamic Stimulation (HANDS) 療法を開発した（図2）[6, 10]．

IVES の刺激兼導出電極は麻痺側総指伸筋(EDC) 上に置き，刺激強度は安静時には感覚閾値下程度の刺激を加え，EDC 随意収縮時には指の伸展運動が十分に認められる強さを最大とする．装着中は刺激装置をアームバンドに収納し，日中施行中は携帯させる．介助刺激なしには，指の伸展が不十分な例においても，刺激により，随

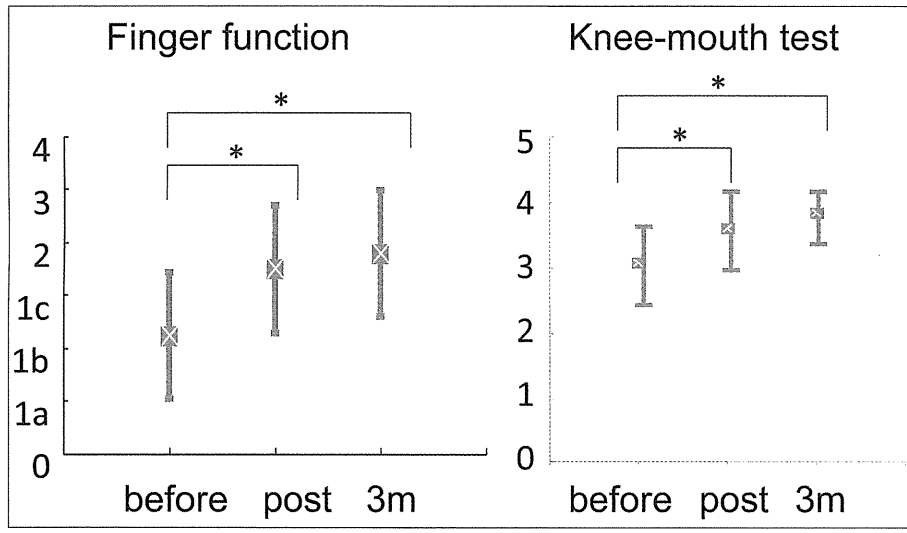

図3　HANDS therapy 前，後，治療終了3カ月後の SIAS finger function test, knee-mouth test score

Repeated measure ANOVA $p<0.001$

* Post hoc Bonferroni $p<0.001$

治療終了後3カ月後にも機能障害の改善は維持されている．

意的な指の伸展運動を促し，麻痺肢による grip & release を容易にし，日常での使用頻度を増加させることが可能である．

発症後150日以上経過した脳卒中片麻痺患者20例において3週間の HANDS 療法により麻痺側手指機能の有意な改善が認められ，その効果は治療終了後3カ月後まで持続されていた（図3）．また，痙縮，上肢実用性に関しても同様に有意な改善を認めた．さらに，電気生理学的にも麻痺側前腕屈筋群の相反性抑制の改善が認められ，経頭蓋磁気刺激二重刺激による皮質内抑制の検討においても，手指伸展筋群における損傷半球での皮質内抑制の脱抑制が認められた[6]．

上肢機能改善の機序としては，現在のところ，使用頻度の増加による dose dependent な機能回復，電気刺激，装具着用による脊髄レベルでの相反性抑制の改善による痙縮の改善，さらには随意運動と電気刺激による中枢性の機能再構築が考えられている．また，Shindo らは Randomized Control Trial（RCT）を亜急性期の患者で行い，Fugl-Meyer 上肢運動項目の改善は装具のみを使用した対照群と比較し有意な改善を認め，特に手指機能の顕著な改善を認めたと報告している[14]．

HANDS 療法は電気刺激により随意運動を補助することにより，日常生活での長時間の使用が可能であり，比較的重度の麻痺にも適応が可能である．適応の基準としては，表面電極において標的とする手指伸筋群に筋活動を認めることが必要である．

Ⅵ. rTMS, tDCS

脳卒中における皮質興奮性の変化として現在考えられている一つのモデルとして，損傷半球における興奮性の低下と非損傷半球における興奮性の増大がある[14]．損傷半球における興奮性の低下の概念は比較的わかりやすいが，一部の患者では非損傷半球の過剰な興奮性の増加が，逆に損傷半球への過剰な半球間抑制（IHI）を引き起こし，損傷半球の活動を妨げている可能性も示唆されている．

このモデルに基づいてrTMSおよびtDCSの治療的応用を考えると，①損傷半球の興奮性を増加させる刺激，②非損傷半球の興奮性を低下させる刺激を用いることが考えられる．①に関しては，損傷半球運動野へのanode（陽極）tDCS，high frequency rTMS，PAS，iTBSが行われている．②に対してはlow frequency rTMSおよびcathodal（陰極）tDCSによる報告がある．しかしながらいずれの研究においても対象はいわゆる分離運動が可能な運動麻痺の軽度な例が対象であり，電気生理学的な変化や運動課題に要する時間の短縮などの変化にとどまり，いわゆるreal-lifeでの改善の報告はほとんどないのが現状である．また，非損傷半球の興奮性の増加が損傷半球を過剰に抑制しているとする説に関してもこれがすべての例において認められるとは限らず注意が必要である．Lotzeらは皮質下病変をもつ亜急性期の脳卒中患者では，運動機能の改善には非損傷半球の運動野，運動前野，上頭頂葉が関与しており，これらの部位を運動課題中に磁気刺激により干渉するとパフォーマンスの低下を認めたと報告している[12]．

その点では損傷半球の興奮性を高める刺激を用いるほうが好ましいと思われるが，てんかん等の発生には注意が必要である．しかしながら，rTMS，tDCSのみによる麻痺の回復には限りがあり，リハを上回る効果は期待できないのが現状である．そこでrTMSなどをconditioningとして用い，作業療法，運動療法と併用することが検討されつつあり，その効果が近年，報告されている[9, 16, 17]．

Ⅶ. Brain-Machine Interface（BMI）

BMIは脳と機械を連動させるシステム全般のことを言う．今までは，障害者の能力低下を代償する目的でのいわゆる機能代償型BMIの開発が中心に行われていたが，近年はBMIを重度麻痺患者の運動機能の回復を目的とする機能回復型BMIが注目されている．

Buchら[9]は脳磁図を用いた機能回復型BMIを慢性期の脳卒中患者に用いている．麻痺肢運動イメージ時のμ律動の変化を利用して麻痺側手指に装着した装置を動かす訓練を行った．結果は装置のコントロールは改善したが，実際の麻痺肢運動機能の改善は認めなかった．しかしながら，機能回復型のBMIを用いた訓練により，脳活動の賦活，運動企図を繰り返すことによる，使用依存性の可塑性の誘導の可能性などが示唆され，特に重度麻痺患者の上肢機能回復において，その可能性が期待された．慶應義塾大学理工学部の牛場らが開発した非侵襲的脳波型BMIを用いてShindoらは表面電極による脳波を用いて，運動イメージ

時の運動野近傍における事象関連脱同期（event related desynchronization：ERD）を感知して電動装具により手指伸展を行うBMI訓練を脳卒中片麻痺患者に行い，機能回復ならびに手指伸展筋群の筋活動の増加を報告している[15]．

われわれは，麻痺側手指伸展筋群に筋活動を認めない重度麻痺例においてBMI訓練を行うことにより筋活動の出現を認め，前述したHANDS therapyへの移行が可能となった症例を経験している．重度片麻痺患者においてもBMI，HANDS therapyを組み合わせることにより，従来は得られなかった機能の回復を得られるようになってきている．

Ⅷ. まとめ

近年は，上肢機能障害への新たなアプローチが報告されており，慢性期の患者においても上肢機能障害の改善が得られる可能性が出ている．代償手段による能力低下の改善だけでなく，しっかりとした機能障害の評価に基づく上肢機能障害への適切なアプローチを考える必要がある．

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