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Complex Neuromuscular Changes Post-stroke Revealed by Clustering Index Analysis of Surface Electromyogram

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Abstract

The objective of this study was to characterize complex neuromuscular changes induced by a hemisphere stroke through a novel clustering index (CI) analysis of surface electromyogram (EMG). The CI analysis was performed using surface EMG signals collected bilaterally from the thenar muscles of 17 subjects with stroke and 12 age-matched healthy controls during their performance of varying levels of isometric muscle contractions. Compared with the neurologically intact or contralateral muscles, mixed CI patterns were observed in the paretic muscles. Two paretic muscles showed significantly increased CI implying dominant neurogenic changes, whereas three paretic muscles had significantly reduced CI indicating dominant myopathic changes; the other paretic muscles did not demonstrate a significant CI alternation, likely due to a deficit of descending central drive or a combined effect of neuromuscular factors. Such discrimination of paretic muscles was further highlighted using a modified CI method that

emphasizes between-side comparison for each individual subject. The CI findings suggest that there appears to be different central and peripheral processes at work in varying degrees after stroke. This study provides a convenient and quantitative analysis to assess the nature of neuromuscular changes after stroke, without using any special equipment but conventional surface EMG recording. Such assessment is helpful for development of appropriate rehabilitation strategies for recovery of motor function.

Index Terms—

Clustering index; interference pattern; muscle weakness; stroke rehabilitation; surface electromyogram.

I. Introduction

STROKE remains the leading cause of adult disability worldwide. The cardinal symptom of a hemispherical stroke is the motor impairment in contralesional side of the body (i.e., hemiparesis), including muscle weakness, spasticity, and abnormal movement coordination [1], [2]. The weakness for voluntary muscle contraction has been reported to be a primary cause of overall impairment [3] and a specific contributor to impaired motor control in stroke survivors [4]. Therefore, it is very important to explore the specific pathological mechanisms underlying muscle weakness post stroke, which is the prerequisite to an appropriate design of effective stroke rehabilitation protocols.

The interruption of the corticospinal tract after stroke is presumably a primary contributor to weakness [5]. However, it still remains ambiguous how a cerebral lesion affects motor unit (MU) survival and function, and whether/how the MU alternations are involved in the development of muscle weakness. Various studies have been performed to examine hemiparetic muscles, suggesting complex neural and muscular changes that collectively contribute to muscle weakness in stroke (given mixed observations). For example, some electrophysiological studies reported abnormal electromyogram (EMG) findings including the presence of spontaneous fibrillation potentials and positive sharp waves, increased muscle fiber density, and enlarged motor unit action potentials (MUAPs) in paretic muscles with needle EMG examination [6]–[12], as well as reduced compound muscle action potential (CMAP) and motor unit number estimate (MUNE) [13]–[15] by using intensive electrical stimulations. Such abnormal EMG findings have not been consistently observed by other studies [16]–[19].

In addition to the examinations associated with either invasive procedures or laborious electrical stimulations, surface EMG examination in stroke has also been reported through construction of EMG-force relation [20]–[22], peak amplitude distribution [23], and power spectrum analysis [24], [25], etc.

In this study, we presented a novel examination of stroke using clustering index (CI), a marker derived from surface EMG. The CI method was recently proposed by Uesugi *et al.* [26] and can be used to discriminate neurogenic and myopathic changes from surface EMG morphology. Highly clustered EMG interference patterns can be a sign of neurogenic

changes while those flat and dense EMG interference patterns might indicate myopathic changes. The diagnostic yield of the CI method for neuromuscular diseases has been reported in recent studies [26]–[28]. Taking advantage of its diagnostic power, our study presents the first application of the CI method in neurological injuries. We found that the CI analysis can assess and classify the complex nature of paretic muscle changes post stroke, which contributes to developing appropriate rehabilitation strategies or protocols. The noninvasive and convenient character of the CI method will further promote its application as an objective tool for examination of stroke.

II. Methods

A. Subjects

Seventeen subjects with stroke (S1–S17, age: 61 ± 11 years, mean $\pm$ standard deviation, range 46–82 years) and 12 age-matched healthy control subjects (C1–C12, age: 58 ± 10 years, range 48–72 years) participated in this study. All subjects with stroke were recruited from inpatient department of rehabilitation medicine in the First Affiliated Hospital of Anhui Medical University (Hefei, Anhui Province, China). Study inclusion criteria include: 1) age between 40–85 years old; 2) experience of first time stroke, initial onset > 14 days; 3) medically stable with clearance to participate; 4) experience of hemiparesis with mild to severe muscle weakness on the paretic side; 5) ability to fully or partially perform voluntary contraction of the paretic hand muscles; 6) inexperience of severe hand muscle spasticity, with modified Ashworth scale (MAS) no more than 1 for hand flexors. Stroke survivors with concurrent neurological disorders or other symptoms (such as neuropathy, radiculopathy, cervical spondylosis, and hyperglycemia, etc.) were excluded. Their motor function was evaluated based on the Brunnstrom stage and Fugl-Meyer test applied before the experiment. The detailed information of the 17 subjects with stroke, including their motor function evaluation, is presented in Table I. All the control subjects were healthy volunteers without any known history of neural or muscular disorder. The study was approved by the local ethic review boards. All subjects gave their informed consent before any experimental procedure.

B. Experiments

The abductor pollicis brevis (APB) muscle of the thenar eminence was examined in this study, because of its superficial location and the ease of performing thumb abduction (corresponding to the primary APB function). The thenar eminence was examined with Ag/AgCl disc surface electrodes (10mm in diameter, JK-1A, JunKang Inc., Shanghai, China). After the skin preparation with medical alcohol, two electrodes were firmly attached on the skin of APB muscle along the direction of muscle fibers. The center-to-center distance of the two electrodes was 20mm to produce a single-differential channel. A large round reference electrode (Dermatode; American Imex, Irvine, CA) was placed on arm fossa cubitalis on the same side. The surface EMG signal was amplified with a self-made surface EMG recording system with a two-stage gain of 2400 in total, and further digitized in a 24-bit A/D converter (ADS1299, Texas Instruments, TX) with a sample rate of 1 kHz. The data were monitored on the screen in real time, and were stored for offline analysis.

During the experiment, subjects were seated in a comfortable chair with their tested arm bent approximately 90 degrees and placed on a height-adjustable table. Alternatively, the subjects were also allowed to lie in an examination bed with their tested arm held still and put against the inside of the body. The palm of the tested hand was kept flat toward the body, while natural and slight finger flexion was allowed. The recording was performed on each side of a subject in a random order. The subject was asked to generate an isometric contraction by abducting the thumb at different levels. A resistant force was provided to the tested thumb by the experimenter to facilitate muscle force generation. In a single trial, the subject was instructed to perform thumb abduction from mild level to maximal levels, roughly corresponding to 10%, 30%, 50%, submaximal and maximal voluntary contraction (MVC), while the force was not accurately measured. At each contraction level, the subject was encouraged to remain as stable as possible for at least 3 second. Thus the recorded surface EMG from a single trial consisted of interference EMG patterns in a sequence of graded contraction levels. Such a trial was repeated at least 3 times to produce sufficient data. Sufficient rest was allowed between consecutive trials to avoid muscle fatigue.

C. Data Analysis

1) Data Preprocessing and Segmentation—The collected raw surface EMG signals were imported into Matlab (Version R2012a, MathWorks, Natick, MA, USA) for processing. A fourth-order Butterworth band-pass filter at 20–500 Hz and a set of second-order notch filters at 50-Hz power line interference and its harmonics were applied to all recordings for denoising purpose. For each tested thumb, the recorded surface EMG was divided into several non-overlapping epochs of 1-s duration (see Fig. 1). The epochs were selected from stable isometric muscle contraction [26], and those showing obvious varying force levels were discarded. At least two epochs were derived for each level, and approximately 30 epochs were derived from each side of a subject.

2) CI Analysis—CI is a non-invasive quantitative method for analyzing surface EMG interference patterns to differentiate neurogenic and myopathic changes [26]. It evaluates how the area of surface EMG signals is clustered into large MUAPs along the time-line.

To calculate CI, the signal in each epoch was divided into a series of non-overlapping consecutive windows with the same length. In our current study the window length was set to 15ms, which was regarded to approximately cover an individual MUAP [26], [28]. Assume that there are K windows derived in an epoch and the area of each window was A_i . Then differential sequences between every consecutive area value (DA_i), between every second area value (DB_i) and between every third area value (DC_i) can be calculated.

The CI of each epoch is defined as

$$CI = \left\{ \sum_{i=1}^{K-1} DA_i + \sum_{i=1}^{K-2} DB_i + \sum_{i=1}^{K-3} DC_i \right\} / 6 \cdot \sum_{i=1}^K A_i. \quad (1)$$

CI has values from 0 to 1, and the higher values are derived From highly clustered signals which appeared isolated large action potential spikes.

The degree of signal clustering expressed as the CI depended on the contraction level [26]: a lower CI value is related to a greater muscle contraction level. When the CI was plotted versus the area of the epoch (which was used as an estimate of the muscle contraction level), the relationship turned out to be approximately linear using double logarithmic scale. We found that this was also the case for the data in our current study. Consequently, each analysis epoch resulted in two values: a $\log(\text{Area})$ and a $\log(\text{CI})$, which can be expressed as a point in the CI-area plot (see Fig. 2). The points derived from a group of analysis epochs can be scattered to form a cloud over the CI-area plane.

In the analysis of EMG interference patterns, establishment of the normal data reference is the prerequisite to judgment of abnormality. To define the distribution of the normal cloud, linear regression analysis was performed on epochs (1 area 100uVsec) collected from the dominant side of all the control subjects and the unaffected/ contralateral side of all the stroke survivors for both $\log(\text{CI})$ and $\log(\text{area})$ (Fig. 2, $y = -0.203x - 0.570$). The regression line was used to account for the relationship between the CI and muscle contraction level. Then, for each epoch, the deviation of CI in the logarithmic scale from the linear regression line was calculated. An average of these deviation values over all the epochs was computed and denoted as a mean residual (R_m), which can then be used to assess the presence of abnormality for a tested muscle.

Afterwards, the mean μ_R and standard deviation (SD) σ_R of the R_m values for the contralateral side of all the subjects with stroke and the dominant side of all the controls were calculated. On this basis, a Z-score of the R_m ($(R_m - \mu_R)/\sigma_R$) Z-score was defined for a given side of a subject. In the routine CI method [26], [27], this Z-score was used as the final representative indicator for the diagnostic assessment. A Z-score outside ± 2.5 was defined as abnormal. Patient with a Z-score higher than $+2.5$ was diagnosed as being neurogenic and a Z-score lower than -2.5 indicated a myopathic change.

Considering that comparison between paretic and contralateral sides for individual subjects is a routine approach for stroke studies, a differential mean-residual, R_{md} , defined as the difference of R_m between two sides of a given subject, was further calculated. For a control subject, the R_{md} was obtained by subtracting R_m value of the dominant side from that of the non-dominant side. For a subject with stroke, the R_m value of the contralateral side was subtracted from that of the paretic side. The R_{md} was used to quantify the relative difference between both sides of a subject. Similarly, mean and SD values of the R_{md} values for all the controls were calculated, from which a Z-score of R_{md} , was obtained for a given subject. Such a Z-score of R_{md} was also used as a representative indicator for the diagnostic assessment.

D. Statistical Analysis—A two-way repeated-measure analysis of variance (ANOVA) was applied on the Z-score of R_m , with the subject group (2 observation levels: stroke and control) considered as a between-subject factor and the side (2 observation levels: contralateral vs. paretic for the subjects with stroke, and dominant vs. non-dominant for the control subjects) as a within-subject factor. Moreover, a student's T-test was made to compare the Z-scores of R_{md} derived from both subject groups (stroke and control). The level of statistical significance was set to $p < 0.05$ for all analyses. When necessary, post hoc

pairwise multiple comparisons with Bonferroni correction were used. All statistical analyses were completed using SPSS software (ver. 16.0, SPSS Inc. Chicago, IL).

III. Results

Fig. 1 shows examples of surface EMG signals measured from the dominant side of a control subject and both the paretic and contralateral sides of 3 subjects with stroke, respectively. For the control subject, a segment of surface EMG during varying muscle contraction levels is shown, from which a 1-s analysis epoch during a stable isometric muscle contraction level was selected for a closer demonstration. The representative epochs from both sides of 3 subjects with stroke are also shown. For this figure, the epochs with comparable area values (which were relatively low due to the substantial muscle weakness of the paretic muscles) were selected for a demonstration purpose.

Fig. 2 shows the results of the CI-area plot for both the contralateral and paretic muscles of all the subjects with stroke and the dominant side of all the control subjects. The data points from the non-dominant side of all the control subjects are not shown (to avoid making the figure messy). It can be visually inspected that the points derived from the stroke contralateral muscles were almost overlapped over those derived from the control subjects. The normal boundaries, pre-defined by ± 2.5 times the standard error (SE) of the linear regression of these points, were able to define the range of normal data, with few epochs (from the contralateral muscles) scattered outside the normal range.

Z-scores of Rm are present in Fig. 3 for both sides of all subjects. It was shown that Z-scores derived from the contralateral muscles (-0.303 ± 1.028 , mean $\pm$ SD) of the stroke group and both dominant muscles (0.430 ± 0.815) and non-dominant muscles (0.317 ± 0.799) of the control group were located in the predefined normal range ± 2.5 . For the paretic muscles (-0.587 ± 1.803), five subjects with stroke had Z-scores outside the normal range, being diagnosed as abnormal. Specifically, two subjects with stroke (namely S4 and S9 with the Z-scores 2.603 and 3.215, respectively) yielded Z-scores beyond +2.5, indicating neurogenic changes of the examined muscles. By contrast, three subjects with stroke (namely S3, S8 and S10 with the Z-scores -3.226 , -2.657 and -3.360 , respectively) had lower Z-scores below -2.5 indicating myopathic changes. For the other remaining 12 subjects with stroke, the Z-scores from their paretic muscles were located between a “normal” range from -2.5 to $+2.5$. The ANOVA reported no significant effect of either subject group ($F = 2.953$, $p = 0.063$) or side ($F = 0.455$, $p = 0.506$) on Z-scores of Rm and no significant interaction ($F = 0.084$, $p = 0.774$) between both factors. Although no significant difference in the mean values of Z-scores was found between two sides and two subject groups (stroke and control), variance of Z-scores was much larger for the paretic muscles compared with the contralateral and neurological intact muscles.

When comparing both sides of individual subjects, three different patterns of data point distribution were observed, as shown in Fig. 4. The three patterns are defined by relative distribution of data points from one side (paretic for a subject with stroke or non-dominant for a control) with respect to the other side (contralateral for a subject with stroke or dominant for a control). In each subplot of Fig. 4, there are two bold line segments parallel

with the regression line, describing a general distribution of data points for each side of a subject, respectively. For each line segment, its horizontal width denotes area range and its vertical distance from the regression line denotes the Rm of the data points. Thus, the differential mean-residual Rmd of each subject can be described as vertical distance between both parallel segments in each subplot. In Fig. 4a, the data points from the paretic side were generally located below those from the contralateral side. As a result, the Rm of the paretic muscle (-0.324) was much smaller than that of the contralateral muscle (0.096). Calculation of the Rmd indicated a negative value of -0.420 . The second distribution pattern was presented in the Fig. 4b, where similar data point distributions were observed in both sides. Computation of the Rm showed proximate values in the muscles on both sides. As a result, the Rmd was very close to 0. For the third distribution pattern as shown in Fig. 4c, the paretic side had higher Rm value (0.253) compared with the contralateral side (-0.062), which yielded a positive value of Rmd (0.315). Such distribution patterns were observed from the control group as well, as shown in Fig. 4d–4f. However, the vertical distances observed from the control subjects were not as large as those from the stroke survivors.

Alternatively, Fig. 5 shows the Z-scores of Rmd. As expected, it was found that all the control subjects had Z-scores of Rmd (0.000 ± 0.829) within the normal limits of ± 2.5 , whereas the subjects with stroke yielded Z-scores of Rmd (-0.210 ± 2.353) in a larger range from -5.180 to 4.133 . Five out of 17 subjects with stroke had abnormal Z-scores of Rmd outside the normal limits, including 2 subjects (S4 and S13 with the Z-scores 4.133 and 3.202 respectively) beyond upper boundary of $+2.5$ and 3 subjects (S3, S8 and S10 with the Z-scores -3.471 , -3.645 and -5.180 respectively) below lower boundary of -2.5 . Student's T-test reported no significant difference in the group mean value of Z-scores of Rmd between the control and stroke subjects ($T = -0.290$, $p = 0.774$). However, the SD of Z-scores of Rmd derived from the subjects with stroke was two times greater than that from the control subjects.

IV. Discussion

This study introduced a novel application of CI method to analyze surface EMG of stroke patients toward assessing their complex neuromuscular changes. For bilateral muscles of the stroke and healthy control subjects, CI was observed to decrease with increasing voluntary muscle contraction levels (described by the area of EMG). A linear regression analysis was then performed to model the relationship between the CI value and surface EMG area. Such an approach takes the force effect into account, thus making the CI method suitable for analyzing data from a variety of muscle contraction levels to provide diagnostic information.

When the Z-score of Rm or Rmd was employed to make a diagnostic decision, the paretic muscles showed a larger variance when compared with the contralateral or neurologically intact muscles. In 2 of the 17 subjects with stroke, we observed an abnormal increase in Z-score of the paretic muscles beyond the predefined upper normal limit of $+2.5$, indicating occurrence of neuropathic changes such as MU loss and muscle fiber reinnervation. MU loss was reported to occur as early as the second week after onset of stroke and may continue to 1 year (due to trans-synaptic degeneration of α -motor neurons) [14], [29]. Subsequent reorganization of the MU architecture including fiber type grouping [29], [30], muscle fiber

reinnervation [10], [12], [23], [24] could also occur in paretic muscle, resulting in enlargement of the remaining MUs [12]. Therefore, even at a weak contraction the paretic muscle would recruit these enlarged MUs, leading to large, scattered, isolated MUAPs in surface EMG, which would result in larger CI as well as greater Z-scores of Rm and Rmd. The increase of CI values in paretic muscles may also be attributed to impairment of MU control properties, such as compression of MU recruitment threshold [20], [23], increase of MUAP synchronization [31], [32] and reduction of MU firing rates [33]–[35], which have been reported to occur after stroke in previous studies.

We also found that 3 subjects with stroke had abnormally lower Z-scores of Rm and Rmd for the paretic side, indicating myopathic changes. This could be due to muscle fiber atrophy (i.e., wasting of muscle fibers). Atrophy of muscle fibers [29], especially type II fibers [6], was reported in stroke survivors. This could also be attributed to a selective degeneration of the large and superficial MUs in the paretic muscles [36]–[38]. These circumstances may induce flatter and denser surface EMG, leading to decreased CI Z-scores.

For the other 12 subjects with stroke, the resultant Z-scores of either Rm or Rmd for their paretic muscles were located within the normal range. Given the experience of substantial muscle weakness of these subjects, it is necessary to discuss why “normal” Z-scores were observed. First, this could be attributed to a deficit of descending central drive. For these subjects with stroke, the lower motoneurons and the muscles might still function more or less normally without significant degeneration. However, because of the upper motoneuron lesion, these subjects were only able to deliver a fraction of the normal voluntary drive, thereby suffering from corresponding muscle weakness, while the surface EMG still appeared normal. Alternatively, the “normal” Z-scores for these subjects with stroke might be a combined or cancelled effect of both neurogenic and myopathic processes. Since a variety of factors might affect CI in opposite ways, their collective effect probably drove the Z-scores of paretic muscles within the normal interval.

Comparison of the paretic and contralateral muscles (with similar limb lengths, dimension, etc.) of the same subject with stroke is more straightforward than muscles from different subjects. Therefore, the Z-score of Rmd was proposed to quantify the CI difference between the two sides for a given stroke subject, whereas the Z-score of Rm merely measured muscles on one side of the subject. The Z-score of Rmd can better overcome subject differences. It was observed that the resultant Z-scores of Rmd for subjects with stroke (Fig. 5) showed more dramatic variations than the Z-scores of Rm for the paretic muscles (Fig. 3). This demonstrated its advantage or improved sensitivity in revealing paretic muscle alternations post stroke.

It is of particular note that different decisions were made for two subjects with stroke when Z-scores of Rm or Rmd were used. They were S9 diagnosed as “neurogenic” and “normal”, and S13 diagnosed as “normal” and “neurogenic” by Z-scores of Rm and Rmd, respectively. For both subjects, the distributions of their data points in the CI-area plane are shown in detail in Fig. 6. Such conflicting results might be attributed to different statistical definitions of Z-scores of both Rm and Rmd.

For stroke patients, some potential factors that might influence CI analysis should also be acknowledged. For example, altered motor control or coordination after stroke may result in abnormal muscle activation (such as activation of remote intrinsic muscles other than the tested one). In such a situation, crosstalk contamination can be recorded by a conventional surface electrode with large uptake area (as used in the current study). This will induce low-frequency flat surface EMG, misleading the CI analysis (toward myopathic).

In general, our findings from the CI analysis were consistent to those from amplitude [23] or spectral [24] analyses of interference surface EMG signals in subjects with stroke. Although previous studies reported interference EMG amplitude or spectral alternations in paretic muscles compared with contralateral muscles, such alternations were only observed in a portion of the examined subjects with stroke, or dramatically varied across the tested subjects. These inconsistent surface EMG changes indicate that there appear different types of processes at work post stroke. Furthermore, the experimentally observed CI variations in hemiparetic muscles can be viewed as the overall or collective effects of these different factors.

In addition to its diagnostic power and the noninvasive feature, the CI method can be performed with very convenient experimental setup and protocols. It does not require accurate measurement of muscle force with a load cell, thus avoiding tedious and time-consuming protocols in previous surface EMG studies (such as EMG-force relation [20]–[22]). Instead, the CI method uses a simplified protocol to provide or adjust resistance forces to the examined muscle, which is similar to clinical routine.

It should also be noted that at the expenses of quick and convenient implementation, the CI has limited performance for quantifying specific MU properties. Although CI measurement can provide a valuable assessment of paretic muscles, more delicate analyses (such as EMG examination at motor unit or muscle fiber level, muscle imaging, muscle biopsy, etc.) are required to further discriminate/quantify the underlying complex factors contributing to the observed CI patterns. Nonetheless, given the clinically oriented design, the CI method still has a good potential of application for diagnosis and assessment of stroke sequelae (e.g., muscle weakness). Such applications can provide valuable information about paretic muscle changes after stroke. This will help the design of effective rehabilitation strategies or protocols toward improved motor function.

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Fig. 1. Illustration of recorded surface EMG interference patterns. (a) A surface EMG segment measured from a control subject C1 with graded isometric muscle contraction levels, from which (b) a 1-s analysis epoch was selected and shown in detail. (c)-(h) six 1-s analysis epochs from both sides of three subjects with stroke, namely S4, S16 and S3. The values of area and CI are also presented on the right side of each analysis epoch.

Fig. 2.

CI-area plot for the analysis epochs from both the contralateral side (black circle) and the paretic side (red dot) of all 17 subjects with stroke and the dominant side (blue diamond) of all 12 control subjects. A double logarithmic scale for both CI and Area was used. The regression analysis (dash dot line) was performed on normal data consisting of epochs (1 total area 100uVsec) from the dominant side of all control subjects and the contralateral side of all stroke survivors. The normal range (dashed lines) is presented within ± 2.5 times the standard error of the linear regression over the double logarithmic scale.

Fig. 3.

Z-scores of Rm for both sides of the two subject groups: stroke and control. The normal range (solid line) is presented within ± 2.5 of Z-scores.

Fig. 4.

Data point distribution in CI-area plot for three subjects with stroke and three control subjects, illustrating three different distribution patterns, respectively. In each subplot, there appear grey diamonds and circles representing the normal data cloud in Fig. 2, and two bold line segments in parallel with the regression line, representing relative distribution of data points from both sides for a subject, respectively. The vertical distance of each line segment from the regression line is the value of R_m and its horizontal width referred to the range of area in the corresponding side. The black line segment represented the contralateral side while the red line segment represented the paretic side of a subject with stroke. Different relative distribution patterns can be observed in subjects with stroke, namely the line segment from the paretic side being below (a), almost equal to (b), and beyond (c) that from the contralateral side. The blue line segment represented dominant side while the magenta line segment represented non-dominant side of a control subject. Three different distribution patterns were also illustrated accordingly in subplots (d)-(f).

Fig. 5.
Z-scores of Rmd for both sides of the two subject groups: stroke and control. The normal range (solid line) is presented within ± 2.5 of Z-scores.

Fig. 6. Data point distribution in CI-area plot for two subjects with stroke: S9 (left) and S13 (right), respectively. Both subplots are produced following the same manner as those in Fig. 4.

TABLE I

Demographic Information of Stroke Subjects

ID #	Sex	Age (year)	Duration (days)	Paretic side	B stage	F-M	MAS
S1	F	54	57	L	3	35	0
S2	M	63	42	L	4	45	0
S3	M	50	45	R	1	2	0
S4	M	57	41	L	4	44	0
S5	M	50	73	R	2	23	0
S6	F	59	409	L	5	50	0
S7	M	47	79	R	5	55	0
S8	F	76	223	L	3	21	0
S9	F	53	701	R	4	40	0
S10	M	65	56	R	2	4	0
S11	M	76	90	L	3	33	0
S12	M	73	27	L	2	15	0
S13	M	55	153	L	5	45	0
S14	M	82	35	L	5	55	0
S15	M	46	35	L	4	43	1
S16	M	58	71	L	4	21	0
S17	M	72	30	L	4	39	0

B stage: Brunnstrom stage

F-M: upper extremity Fugl-Meyer assessment

MAS: modified Ashworth scale