

Beyond the Paralyzed Arm: Quantifying Intentional Motor Control Capacity in People with Severe Arm Impairment Post-Stroke with High-Density Surface EMG

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Research Article

Keywords: stroke, upper-limb rehabilitation, high-density surface EMG, myoelectric computer interface, motor control, patient stratification

Posted Date: October 18th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11189512/v1>

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Additional Declarations: No competing interests reported.

Abstract

Background

Standard clinical scales of motor impairment and activity limitation cannot detect whether a person with severe upper-limb impairment after stroke retains voluntary motor control. Consequently, the patient group that is most in need of active rehabilitation cannot be triaged for training using a/an biofeedback/intention-based interface. To address this gap, we tested whether people with stroke (PwS) with severe impairment (Action Research Arm Test [ARAT] ≤ 10) retain graded intentional motor control capacity (IMCC) that can be read from the myoelectric activity in the paretic arm.

Methods

Twelve severely impaired PwS (ARAT ≤ 10) and twelve healthy controls performed a movement-free myoelectric task in which high-density surface EMG (HD-sEMG) from two muscles belonging to distinct pathological synergies drove a cursor. Task success required genuine voluntary drive rather than spastic or synergetic co-activation. Principal-component analysis combined three performance measures (path length, radial target error, and Euclidean target error) into a composite IMCC score. We compared IMCC scores with those of healthy controls.

Results

PwS differed from controls in path length ($p = 0.005$) and radial target error ($p = 0.033$), yet the groups overlapped markedly: half of the PwS (6 of 12) produced IMCC scores inside the 85% confidence region of the healthy controls (the healthy reference range). This overlap was not predicted by ARAT or Modified Ashworth Scale scores.

Conclusions

Half of clinically non-functional arms retain IMCC within the healthy range. HD-sEMG allows measuring hidden intentional control capacity, providing a graded marker to identify which severely impaired PwS can be trained through an intention-based interface and possibly improving recovery prediction.

1. Background

Stroke is a leading cause of acquired disability worldwide: a 2022 global systematic analysis found that approximately 12.2 million people experience their first stroke each year, and over 101 million are currently living with its long-term consequences (1). Upper-limb motor impairment is one of the most frequent and disabling consequences of stroke, affecting approximately 80% of people with stroke (PwS) as part of the broader spectrum of post-stroke motor deficits. Its severity is strongly associated with

overall functional outcome. Recovery is often limited, with 30% to 66% of PwS with hemiplegia remaining without functional arm use six months after stroke (2–4).

A clinically distinct subgroup presents severe upper-limb impairment right after the stroke, and a recovery trajectory that is notably heterogeneous. Many severely impaired PwS show minimal recovery, while a subset achieves substantial gains (5, 6). Available clinical indicators do not fully predict this variability (7–9). Corticomotor pathway integrity, indexed by motor-evoked potential (MEP) status, is among the strongest predictors of superior upper-limb outcome in this subgroup, yet divergent outcomes occur even within the no-MEP group (2, 10, 11). The heterogeneity in recovery of severe PwS may be explained by significant loss of neural tissue that may or may not exceed a level that does not allow recovery. Standard clinical scores alone cannot make this distinction: they capture reduced voluntary motor-unit recruitment only through its behavioral consequence, movement. Thus, two PwS with identical, near-floor scores on conventional motor scales may therefore have markedly different residual capacities (12–14, 25).

This diagnostic gap is compounded by a therapeutic one: because the major evidence-based upper-limb interventions are built around active, repetitive movement, their reach is inherently limited for PwS who show little or no voluntary movement (15). Motor recovery is thought to be driven, at least in part, by Hebbian processes and therefore requires high-intensity, repetitive, task-specific practice (3, 4, 16, 17). However, this form of training requires a degree of residual voluntary movement that PwS with severe impairments often lack. Accordingly, most evidence-based upper-limb interventions impose minimum movement thresholds as eligibility criteria. Even those who claim to address the severely impaired population systematically exclude PwS with complete paralysis (18, 19). Biofeedback-based approaches were expected to close this gap by extending training to PwS who lack sufficient residual movement for conventional therapy, yet the evidence suggests they struggle to do so: a recent scoping review of 48 randomized controlled trials explicitly designed to target severe upper-limb impairment found that enrolled PwSs' Fugl-Meyer Assessment and Action Research Arm Test scores in fact spanned the full range of the scales, indicating that severely impaired PwS remain systematically underrepresented even in interventions designed for them (20). Furthermore, the dose actually delivered mirrors this gap: severely impaired PwS receive a median of 33 minutes of active practice per day, half that of mildly impaired PwS and roughly a quarter of the recommended two hours, despite spending more time in therapy, because that time is largely filled with passive content such as positioning, ranging and non-functional electrical stimulation (21). This prolonged inactivity of the paretic limb predisposes these PwS to a phenomenon termed learned non-use, in which suppression of movement in early stages leads to habitual (learned) non-use of the paretic arm (22). Taken together, this evidence indicates that PwS most severely impacted by stroke are those who do not engage in active rehabilitation, and therefore are dependent on their natural recovery alone and may not achieve their maximal recovery potential. Indeed, the prognosis for this group is poor: PwS with a flaccid arm who show no early return of voluntary motor control have only a 9–25% probability of regaining even minimal dexterity (ARAT $\geq$ 10) at six months (3, 23).

We suggest that this omission could be partially addressed by identifying and treating those with residual motor control capacity (IMCC) who could potentially benefit from active training using a myoelectric computer interface.

High-density surface electromyography (HD-sEMG) is an ideal system for extracting disrupted myoelectric signals. Despite the absence of visible movement, it can detect and measure voluntary muscle activation in the paretic limb, revealing voluntary drive that is otherwise invisible (24–26). Here, we use HD-sEMG because residual voluntary drive in the paretic limb is graded and subject-specific, rather than uniformly present or absent (27). Thus, reliably detecting and decoding this kind of sparse, individually variable voluntary drive requires spatial coverage that conventional one or two-channel sEMG cannot provide.

In this study, we developed a measure of Intentional Motor Control Capacity (IMCC) which is based on HD-sEMG recorded during a Myoelectric Computer Interface (MCI) task requiring simultaneous, graded activation of wrist extensors and elbow flexors, muscles thought to belong to different pathological synergies (28), whose coordinated activation reflects voluntary motor intent rather than the pathological synergy (29).

Two questions were evaluated: (1) Do severely impaired PwS ($\text{ARAT} \leq 10$) retain IMCC in their paretic arm? And (2) How much does this capacity vary across PwS?

2. Methods

2.1 Participants

Twelve PwS and twelve elderly controls participated in the study. PwS were recruited from the inpatient and outpatient departments of the Adi-Negev Rehabilitation Center. Inclusion criteria included a radiologically confirmed stroke and a score ≤ 10 on the Action Research Arm Test (ARAT) (30). Exclusion criteria were cognitive deficits precluding informed consent, pain or discomfort preventing task-relevant contraction, no difference between electrical activity recorded during maximal muscle contraction and rest, uncorrected visual impairment, wrist or elbow joint pain, cerebellar disorders, and reported perceptual dysfunctions such as Neglect and Pushers syndrome. The study was conducted at Lillian and David E. Feldman Research Center for Rehabilitation Sciences, Adi-Negev Nahalat-Eran, and was approved by the local institutional review board (0007-24-NGV). All participants provided written consent prior to their enrollment.

2.2 Clinical assessments

Motor activity and spasticity were assessed using the ARAT and the Modified Ashworth Scale (31), respectively. Assessments were administered before the first experimental session.

2.3 Experimental task

Participants sat on a chair in front of a laptop positioned on a desk, with their hand relaxed on the desk. Participants performed a myoelectric task in which EMG-derived signals controlled an on-screen cursor (central square canvas: 800×800 px on a 15-inch screen with a resolution of 2880 × 1620): the wrist-extensor envelope controlled the horizontal axis, and the elbow flexor envelope controlled the vertical axis. Healthy participants used their dominant hand, and PwS used their more affected (severely impaired) hand. We presented five targets radially arranged around the origin at fixed locations on the screen (see Table 1). A trial began when the cursor left the home base (A quarter-circle arc located at the left lower corner of the screen, with a radius of 240 pixels) and ended either when the cursor reached the target (a 100×100 px circle) and was held there for a pre-defined time (success) or after a 15 s time-out (failure). Both groups completed a single session. The task parameters were chosen to be challenging for neurologically intact performers as well. The task screen layout, the target arc, and the two reference radii used to compute the IMCC angular-error metric (§ 2.6) are shown in Fig. 1.

Table 1

Target positions in the reaching task. Five targets were arranged in a quarter-circle arc originating from the home position (bottom-left corner of the display), spanning 90° from the vertical to the horizontal axis in 22.5° increments. Each target was placed at a fixed radial distance R from the home position (default R = 560 pixels; canvas size = 800 pixels, radial factor (target-placement radius) = 0.70), adjustable via an experimenter-controlled slider. Target 1 (90°) constrained cursor movement to the vertical axis only, and Target 5 (0°) constrained cursor movement to the horizontal axis only, rendering these two targets effectively one-dimensional. Targets 2–4 permitted unconstrained two-dimensional cursor movement. Angular positions are reported in standard mathematical convention (0° = rightward horizontal, 90° = upward vertical).

Target #	Angle (°)	Direction
1	90°	Straight up
2	67.5°	Upper-left diagonal
3	45°	True diagonal
4	22.5°	Lower-right diagonal
5	0°	Straight right

2.4 High-density sEMG acquisition and processing

Muscle activity was recorded using the Sessantaquattro+ bioelectrical amplifier (OT Bioelettronica, Turin, Italy), configured for 64-channel monopolar acquisition at 2048 Hz. Two electrode grids, each comprising 32 channels arranged in an 8×4 matrix with 10 mm inter-electrode distance, were placed on

the affected side over the wrist extensors (common extensor compartment specified muscles; channels 1–32) and the biceps brachii (channels 33–64). These locations include muscles thought to belong to different pathological synergies. Wrist extensors are considered to be part of the extensor synergy, while the biceps brachii is part of the flexor synergy (32). Consequently, their coordination is unlikely to reflect a pathological synergy and is more likely to reflect voluntary action.

We processed raw signals online in the following order. First, within non-overlapping 25-sample windows, signal energy was computed per-channel, and the mean signal of the lowest-energy half of the channels was subtracted from all channels. The remaining signals were then high-pass filtered at 20 Hz and low-pass filtered at 500 Hz (fifth-order Butterworth filters, applied in cascade), followed by a second-order Butterworth band-stop (notch) filter (45–55 Hz) to attenuate 50 Hz power-line interference. The filtered signal was full-wave rectified and low-pass filtered at 2 Hz to obtain the signal's linear envelope (second-order Butterworth).

2.5 Calibration epoch (open-loop)

Each session began with an open-loop calibration epoch in which EMG data were acquired using a guided rest–movement protocol consisting of an initial 10-second rest, ten alternating cycles of 7 seconds of rest and 10 seconds of attempted contraction of the target muscle, and a final 15-second rest. On-screen visual cues indicated the instructed state. After calibration, the experimenter manually labeled contraction and relaxation segments. An automated channel-selection procedure based on functional SNR (contraction/relaxation) produced a binary channel mask per muscle, restricting the online control signal to the most informative channels. The mean envelope amplitude during labeled contraction and relaxation segments defined each muscle's control parameter.

Then, a familiarization and tuning epoch began, where two subject-specific gain parameters were defined per muscle– one determining the effect of the EMG envelope on the movement of the cursor away from the origin and the other on the movement toward the origin. EMG envelope amplitude was mapped to cursor velocity using a linear normalization bounded by calibrated rest and contraction values:

$$v(t) = G \left(\frac{2 \left[e(t) - \alpha \bar{e}_{\text{rest}} \right]}{\beta \bar{e}_{\text{MVC}} - \alpha \bar{e}_{\text{rest}}} - 1 \right)$$

where $e(t)$ is the EMG envelope, $\bar{e}_{\text{rest}}$ and $\bar{e}_{\text{MVC}}$ are the mean envelope amplitudes during rest and maximal voluntary contraction recorded at calibration, and G is a fixed velocity gain. The parameters α and β are per-subject gain factors applied to the rest and contraction periods, respectively. Gain parameters were tuned individually during the familiarization epoch to account for inter-subject variability in EMG amplitude. Positive v drives cursor movement toward the target; negative v drives return movement toward the home base. Both α and β remained constant throughout the main task.

2.6 Intentional Motor Control Capacity (IMCC)

We nominally define intentional motor control capacity (IMCC) as the ability to control the cursor on the screen using muscle activation patterns taken from two muscle groups. Operationally, IMCC depends on the ability to grade the level of voluntary muscle activation, and to coordinate the activation of multiple muscle.

IMCC was estimated using three performance variables: (i) angular error - the mean absolute angular deviation between the position of the cursor and the target at target radii (closest and furthest from the home base). The 500 radius crossover points are considered when the cursor moves away from the home base (representing an attempt to reach the target using muscle contraction). The 627 radius crossover points considered when the cursor moves towards the homebase (representing an attempt to reach the target using muscle relaxation); (ii) Path length (excursion) in pixels from start to end of the trial (target reach or the 15 s time-out); and (iii) Euclidean error - the minimal distance (in pixels) between the cursor and the target during the trial. Successful trials, in which the cursor reached and held the target, were coded as Euclidean error = 0. Lower values indicate better control for all three measures.

We combined the three variables into a single composite IMCC score based on the two-dimensional PCA space that was derived from the healthy control subjects. PCA was performed on z-transformed values of the three performance variables obtained from the healthy control group. We then projected PwS performance into the healthy-derived PCA space.

IMCC scores were defined in two complementary ways: (i) inclusion in 85% confidence ellipse fit to the healthy participants in the PC1–PC2 plane using the chi-square quantile with two degrees of freedom and (ii) the Euclidean distance of each participant from the healthy centroid in the PC1–PC2 plane.

2.7 Statistical analyses

Between-group differences were tested with the two-sided Mann–Whitney U test ($\alpha < 0.05$) and reported the rank-biserial coefficient as an effect size. We compared ARAT and MAS scores between participants whose IMCC scores fell inside or outside the HC ellipse using a two-sided Mann–Whitney U test.

Healthy-vs-PwS classification based on IMCC performance was computed using leave-one-out cross-validated logistic-regression ROC AUCs. To verify that this classification was robust to the sampling of recruited healthy controls, we performed a leave-one-healthy-out (LOHO) sensitivity analysis. In each of 12 iterations, one healthy participant was held out and the entire pipeline, z-scoring, PCA, and the 85% confidence ellipse, was refit on the remaining 11 healthy participants; every PwS was then reclassified against the new ellipse.

3. Results

We tested the central hypothesis that severely impaired PwS ($\text{ARAT} \leq 10$) may retain a graded IMCC. After describing participant characteristics (§ 3.1), the results are organized in three parts: validation of

the three performance variables against the healthy group (§ 3.2); depicting the composite IMCC score and the resulting group overlap (§ 3.3); and the separation between the IMCC score and standard clinical measures of activity (§ 3.4).

3.1 Participant characteristics

Twelve PwS and twelve HC participated in the study. PwS were 41 to 70 years old (mean $\pm$ SD: 60.6 $\pm$ 9.2). They were tested 1.8 to 132.7 months after the stroke (mean 15.4 $\pm$ 37.0), and all presented severe upper-limb impairment (ARAT scores ranging from 0 to 10, mean 3.4 $\pm$ 3.8). Their MAS scores ranged from 0 to 3 (mean 1.3 $\pm$ 1.4; Table 2). Controls were 66–94 years old (mean 77.0 $\pm$ 8.8).

Table 2
Demographic and clinical characteristics of the 12 PwS. Columns: subject ID, age in years, time post-stroke in months, ARAT score (Action Research Arm Test; range 0 to 57; lower scores indicate more severe impairment), and MAS score (Modified Ashworth Scale; range 0 to 5, recoded from the standard 0, 1, 1+, 2, 3, 4 grading; higher scores indicate greater spasticity). The bottom row gives the cohort mean $\pm$ SD across the 12 PwS.

Subject	Age (yrs)	Time post-stroke (months)	ARAT (0–57)	MAS (0–5)
NC28	62	2.1	0	0
NC29	45	5.6	3	3
NC30	41	7.1	3	3
NH144	66	3.7	0	2
NH146	63	2.7	4	0
NH148	62	8.0	10	1
NC23	61	6.4	8	3
NH157	69	1.8	0	0
NH135	70	7.9	4	0
NC32	56	3.4	0	3
NH149	63	3.4	9	1
NC35	69	132.7	0	0
Mean $\pm$ SD	60.6 $\pm$ 9.2	15.4 $\pm$ 37.0	3.4 $\pm$ 3.8	1.3 $\pm$ 1.4

3.2 Validation of performance variables

Controls’ success rate was 78.3 $\pm$ 22.9% (33.3%-100%). This variance indicates that the task is challenging even for neurologically intact performers, with maximum scores being rare. All three performance variables (Table 3) showed worse performance in the PwS group compared to the HC

group, with significantly worse control in PwS, as indicated by greater path length and radial target error scores, and by greater, non-significant scores for Euclidean target error (Fig. 2).

Table 3

Group-level summary of the 12 PwS and 12 healthy controls. Each row gives group mean $\pm$ SD. ARAT and MAS group means are given for the PwS cohort only; healthy controls were clinically unimpaired by design and have no ARAT or MAS score (dashes in the table). Radial target error, path length, and Euclidean target error are the three IMCC metrics defined in § 2.6; lower values reflect better IMCC. Success rate is the percentage of main-task trials in which the cursor reached and held the target. We tested group differences with two-sided Mann–Whitney U tests ($n = 12$ per group); the test statistic (U), p -value, and rank-biserial effect size (r) are given in the last three rows. Asterisks denote significance (* $p < 0.05$, ** $p < 0.01$); path length and radial target error differed significantly between groups, whereas Euclidean target error and success rate did not (n.s.). ARAT and MAS were assessed only in the PwS cohort.

Group	N	ARAT	MAS	Radial target error (°)	Path length (px)	Euclidean target error (px)	Success rate (%)
Healthy	12	–	–	18.9 ± 7.0	874.7 ± 528.7	40.7 ± 49.4	78.3 ± 22.9
PwS (ARAT ≤ 10)	12	3.4 ± 3.8	1.3 ± 1.4	25.5 ± 6.0	1457.5 ± 527.6	81.4 ± 56.0	65.8 ± 23.6
Mann–Whitney U		–	–	34	23	41	98
p -value		–	–	0.033 *	0.005 **	0.078	0.140
Effect size (r)		–	–	0.52	0.68	0.43	0.36

To validate the relevance of all three variables for the IMCC model, we computed leave-one-out cross-validated ROC AUCs (using logistic regression on each feature in turn) for each of the three IMCC single measures, asking how well each one discriminates healthy controls from PwS. Of the three variables, path length provided the strongest healthy-vs-PwS discrimination (AUC = 0.79); radial target error and the composite IMCC score performed comparably (AUC = 0.70 and 0.69, respectively), and Euclidean target error alone was the weakest (AUC = 0.60; Table 4).

Table 4
Healthy-vs-PwS classification performance for each single performance variable, reported as leave-one-out cross-validated logistic-regression ROC AUC (n = 12 per group). A higher AUC indicates better discrimination between PwS and healthy controls.

Variable	LOOCV AUC (healthy vs PwS)
Radial target error alone	0.70
Path length alone	0.79
Euclidean target error alone	0.60

All distributions nevertheless overlapped substantially (Fig. 2), supporting our hypothesis that some PwS retain control capabilities that are hidden by motor impairment.

3.3 Composite IMCC score and group overlap

Next, we developed a composite controllability score. We projected performance variables from the healthy control group into a PCA-derived space (§ 2.6). The first and second principal captured 87.4% of the healthy variance (PC1 = 58.1%, PC2 = 29.3%); on PC1, all three metrics loaded with the same positive sign and comparable magnitudes (radial target error + 0.68, path length + 0.49, Euclidean target error + 0.55), so PC1 can be read as a general IMCC axis on which higher values correspond to worse performance on every variable simultaneously. On PC2, signs shifted for radial target error and Euclidean target error metrics (radial target error – 0.029, path length 0.76, Euclidean target error – 0.64).

When projecting the PwS into the healthy-derived PCA space (determined by covering 85% of subjects), six of twelve PwS (50%) were positioned inside the healthy zone cutoff ellipse and the remaining six (50%) are outside (Fig. 3).

To assess the robustness of the PCA space, we conducted LOHO analysis. Eleven of the 12 PwS retained their classification across all 12 iterations (5 always inside, 6 always outside); one PwS (NC29) was classified inside in 11 of the 12 iterations and outside in the remaining iteration. This confirms that the inside/outside split is not driven by the specific sample of healthy controls, supporting the model's consistency and the relative homogeneity of the healthy reference group.

3.4 IMCC versus clinical measures

To better estimate the association between IMCC and clinical measures of impairment, we compared differences in clinical measures as a function of IMCC classification (inside vs. outside the ellipse). While the clinical measures showed worse outcomes for the outside ellipse subjects, the effects were not consistent (ARAT: inside 5.0 ± 4.2 , outside 1.8 ± 1.9 , Mann–Whitney U $p = 0.28$; MAS: inside 1.8 ± 1.2 , outside 0.8 ± 1.2 , $p = 0.15$; Table 5). The association between clinical-scale score and ellipse classification was not deterministic also at the individual level: NC32 and NC35, both with ARAT = 0

(NC32 additionally with MAS = 3), fell inside the healthy ellipse, whereas NH146 (ARAT = 4) fell outside. The MAS likewise did not strongly predict ellipse classification: three of the six PwS inside the healthy ellipse had high spasticity scores (NC32, NC23, and NC29; MAS = 3), whereas only one of the six outside-ellipse PwS presented spasticity (NC30; MAS = 3). The absence of a strong MAS–IMCC association indicates that IMCC is not overridden by spasticity

Table 5

Clinical-scale scores (ARAT and MAS) by ellipse sub-group. The "Inside (n = 6)" and "Outside (n = 6)" columns give the mean $\pm$ SD of the score across the six PwS classified inside the 85% healthy ellipse and the six PwS classified outside, respectively. The "Inside range" and "Outside range" columns give the observed minimum and maximum of the score in each sub-group. The p-value column reports two-sided Mann–Whitney U tests between the inside and outside sub-groups; with n = 6 per sub-group, the tests have limited statistical power.

Clinical measure	Inside (n = 6)	Outside (n = 6)	Inside range	Outside range	p-value (MW-U)
ARAT	5.0 $\pm$ 4.2	1.8 $\pm$ 1.9	0–10	0–4	0.279
MAS	1.8 $\pm$ 1.2	0.8 $\pm$ 1.2	0–3	0–3	0.149

4. Discussion

This study tested whether severely impaired stroke PwS ($\text{ARAT} \leq 10$) retain graded intentional motor control capacity (IMCC) in the paretic arm, and its variability across individuals. Using a movement-free myoelectric cursor task, we quantified IMCC from high-density surface electromyography through three metrics - radial target error, path length, and Euclidean target error- combined into a composite score. Although the 12 PwS performed worse than the 12 healthy controls on all three metrics, the distributions overlapped substantially: projected into a healthy-derived reference space, 6 of the 12 PwS (50%) fell inside the 85% confidence region of the healthy controls, a classification that held under leave-one-healthy-out cross-validation. Neither ARAT nor the Modified Ashworth Scale predicted this inside/outside division. Within the limits of a small, cross-sectional sample, these findings indicate that a substantial subgroup of clinically non-functional arms retain measurable, healthy-comparable IMCC that conventional scales miss.

Not all severely impaired PwS are alike, and binary markers cannot tell them apart

These findings carry a clinical implication: severely impaired PwS, even those with no visible movement, are not a homogeneous group. The proportional-recovery literature reaches the same conclusion from a different direction: Prabhakaran et al. (7) and Winters et al. (9) found that PwS with near-floor motor impairment scores in the early sub-acute stage after stroke comprise at least two distinct subpopulations in terms of their recovery trajectory ("fitters" and "non-fitters"). The tools currently used to distinguish these sub-groups are limited. Corticospinal integrity, indexed by motor-evoked potential (MEP) status, is among the strongest predictors of upper-limb outcome (10, 11), but MEP is a binary readout, present versus absent, that reports whether a descending pathway is detectable rather than

grading how much voluntary control or movement a patient can express. Furthermore, Powell et al. (33) documented meaningful gains even in MEP-negative PwS. Additionally, measuring MEP requires transcranial magnetic stimulation and specialized expertise that are not routinely available at the bedside. The IMCC score is designed to address both gaps: rather than a single binary marker, it provides a graded, multivariate index of the quality and level of residual voluntary control, obtained from HD-sEMG during a task that does not require active movement, and requires a mobile surface EMG interface - an assessment more readily deployable in routine clinical settings than TMS-based mapping.

Reading intention in non-moving PwS: the case for high-density sEMG

Residual voluntary drive in a paralyzed limb can be read from several signal sources.

Electroencephalography (EEG)-based brain-computer interfaces (BCIs) paired with motor imagery detect movement intention directly from cortical activity and can engage sensorimotor areas even without muscle contraction (34, 35). However, the low signal-to-noise ratio of EEG, its non-stationarity, and its cumbersome setup limit use outside controlled laboratories (36). Corticomuscular coherence (CMC), which records EEG and EMG simultaneously to confirm voluntary effort, is similarly informative but depends on continuous, costly neural recording and is degraded by lesion location and by the low contraction forces typical of severe stroke, which makes it impractical for routine clinical use (37, 38).

Surface electromyography (sEMG) takes the opposite approach: it records voluntary drive at its functional endpoint, the muscle, rather than inferring it upstream from the cortex. This carries both physiological and practical advantages. Physiologically, sampling the signal at the target muscle stays on the pathway that produces real movement, so the measured activity corresponds directly to the motor output the patient is attempting to generate. This also makes the interface intuitive and easier to control. Practically, sEMG is comparatively inexpensive, non-invasive, simple to apply even by non-technical users, applicable in patients with severe movement disorders, and free of neural-recording hardware (39). While a single bipolar electrode pair yields only a coarse, position-sensitive reading that struggles to distinguish voluntary activation from the involuntary discharge associated with spasticity and abnormal muscle tone, high-density sEMG (HD-sEMG) records spatial as well as temporal muscle activity across a grid of closely spaced electrodes (40), which allowed us to distinguish spatially consistent voluntary activity from background noise and to select the most informative channels for each participant (§ 2.5).

Clinical implications: diagnosis and treatment

The IMCC score has two potential clinical applications: diagnostic and treatment. The diagnostic application is to detect, among PwS who look uniformly non-functional on clinical examinations, those who retain intentional motor control that could be leveraged for rehabilitation. Half of our severely impaired sample (6 of 12) produced IMCC scores within the healthy range, a distinction invisible to ARAT and MAS. Identifying these PwS matters because chronic severe-to-moderate PwS can still gain on the Fugl-Meyer Assessment and ARAT through intensive task-oriented training (33). A graded, movement-

free readout of who holds this capacity offers a way to stratify severely impaired PwS for active rehabilitation rather than defaulting them to spontaneous recovery alone.

The treatment application follows from the same setup. Motor recovery depends on high-intensity, repetitive, task-specific practice (3, 4, 16, 17), but the major evidence-based upper-limb interventions require a minimum of voluntary movement and therefore exclude PwS with none (18, 19), leaving them vulnerable to learned non-use (22). Because the IMCC task reads intentional control directly from paretic-limb sEMG and returns it as real-time cursor feedback, it closes the loop between intention and outcome without requiring overt movement. The same interface that diagnoses residual control can thus deliver active, intention-driven practice to a limb that cannot yet move, extending to non-movers the closed-loop training that conventional rehabilitation provides only once movement is present. Establishing whether such training improves outcomes will require a prospective trial.

Limitations

Several factors bound the generalization of these estimates. First, the sample is small: the proportion of PwS falling within the healthy range, and the precise extent of the measured overlap, may shift in a larger or differently composed population, yet the existence of the overlap does not depend on sample size. Second, the control task is cognitively demanding, requiring sustained attention and the mental transformation of muscle activity into cursor motion, so part of the performance variance may reflect the task difficulty rather than motor capacity alone. Third, the PwS differed in lesion location, stroke type, and time since stroke, and the sample was not stratified along these dimensions. Fourth, the healthy controls were older than the PwS (66–94 vs. 41–70 years). Because myoelectric control performance is expected to decline with age, an older reference group defines a more permissive healthy region, so the proportion of PwS classified inside it may be somewhat overestimated relative to an age-matched reference. Future work employing larger, stratified cohorts with more stringent selection criteria would refine these estimates and disentangle the cognitive and clinical sources of variance from motor control capacity itself.

5. Conclusions

Half of the severely impaired PwS in this sample ($\text{ARAT} \leq 10$) produced a composite IMCC score inside the healthy reference range on a movement-free HD-sEMG cursor task, a split that was not predicted by ARAT or MAS scores. HD-sEMG thus provides a graded, clinically deployable marker of residual intentional motor control that conventional scales cannot detect. The measure could be used to stratify severely impaired PwS for active, intention-driven rehabilitation and, pending prospective validation, to improve prediction of recovery.

Abbreviations

ARAT

Action Research Arm Test

AUC
area under the curve
BCI
brain–computer interface
CMC
corticomuscular coherence
EEG
electroencephalography
EMG
electromyography
HC
healthy controls
HD-sEMG
high-density surface electromyography
IMCC
intentional motor control capacity
LOHO
leave-one-healthy-out
LOOCV
leave-one-out cross-validation
MAS
Modified Ashworth Scale
MCI
myoelectric computer interface
MEP
motor-evoked potential
MVC
maximal voluntary contraction
MW-U
Mann–Whitney U test
PC
principal component
PCA
principal-component analysis
PwS
people with stroke
ROC
receiver operating characteristic
SD
standard deviation

sEMG
surface electromyography
TMS
transcranial magnetic stimulation.

Declarations

Ethics approval and consent to participate

The study was approved by the Helsinki Committee of Adi-Negev Nahalat-Eran Rehabilitation Center (approval no. 0007-24-NGV). All participants provided written informed consent prior to enrollment.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Funding

This research was supported by the Israel Science Foundation (grant 1244/22 to L.S.), the ABC Robotics Initiative of Ben-Gurion University of the Negev, and the Lillian and David E. Feldman Research Fund. The funders had no role in the design of the study, in data collection, analysis or interpretation, or in writing the manuscript.

Author Contribution

K.P contributed to the conception and design of the study, developed the myoelectric interface, collected and analysed the data, and drafted the manuscript. N.G contributed to the study design and the development of the myoelectric interface. A.L.H, G.M and H.I collected the data and helped revise the manuscript. T.M supervised the clinical conduct of the study. L.S contributed to the conception and design of the study and supervised the work. All authors read and approved the final manuscript.

Acknowledgement

The authors thank the participants and the staff of the Adi-Negev Nahalat-Eran Rehabilitation Center. This research is affiliated with the Lillian and David E. Feldman Research Fund and the ABC Robotics Initiative of Ben-Gurion University of the Negev.

Data Availability

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

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Figures

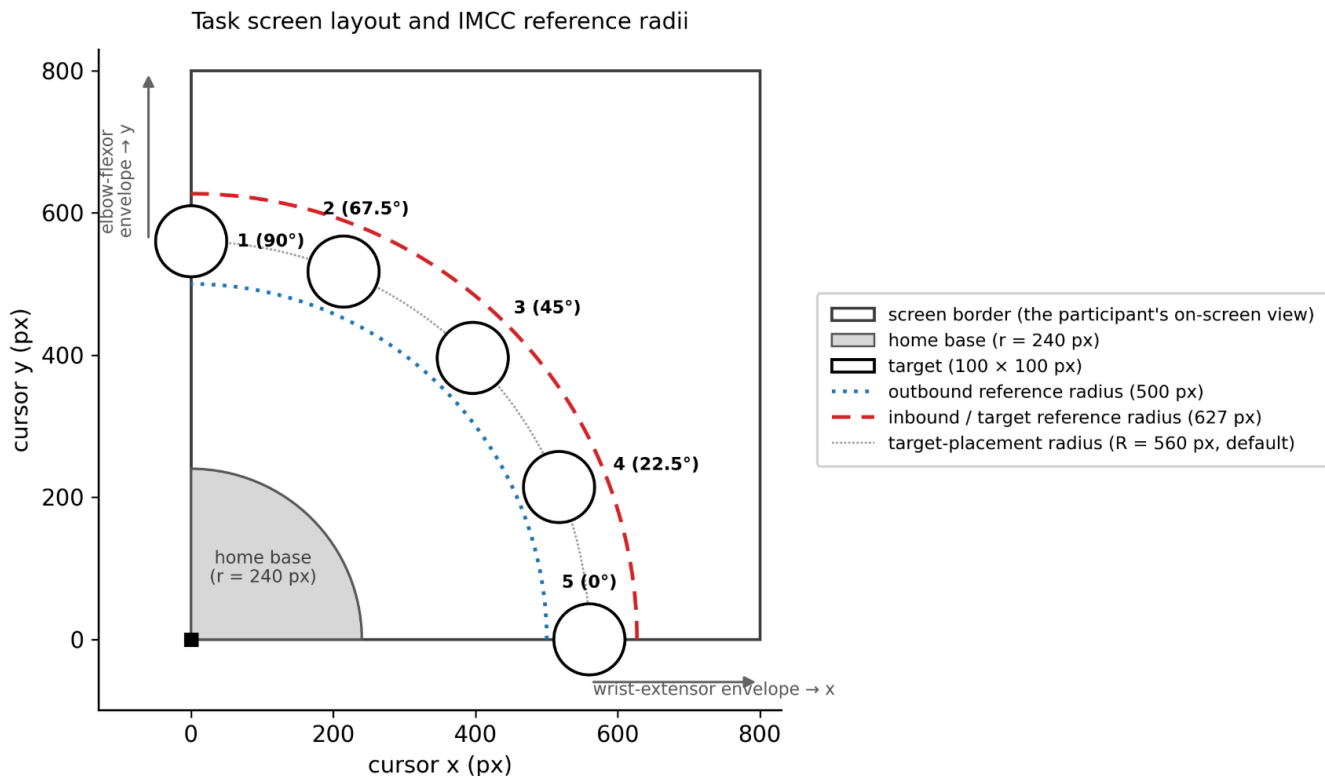


Figure 1

Task screen layout and reference radii used for the IMCC angular-error metric. The outer square marks the screen border exactly as seen by the participant (800×800 px canvas), with the home base (quarter-circle arc, $r = 240$ px, lower-left corner), the five targets (100×100 px circles) arranged along the target-placement arc (default $R = 560$ px; radial factor = 0.70), and the two reference radii used to score angular error (§2.6): the outbound reference radius (500 px, dotted) and the inbound/target reference radius (627 px, dashed). Cursor x was driven by the wrist-extensor envelope and cursor y by the elbow-flexor envelope.

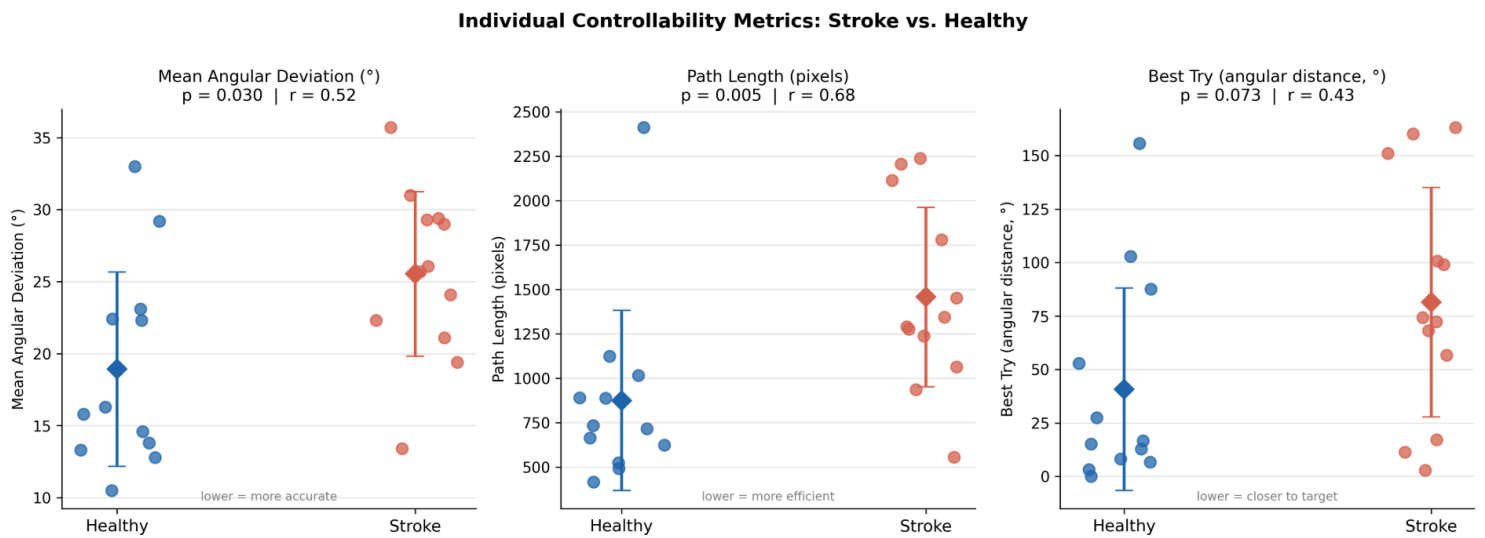


Figure 2

Individual IMCC metrics by group. Each panel shows one of the three task-derived IMCC metrics (radial target error, path length, Euclidean target error; defined in §2.6) for the healthy controls (blue) and the PwS group (red). Individual participants are shown as jittered circles; diamonds with whiskers indicate group mean ± 1 SD. The p-values shown on each panel are from two-sided Mann–Whitney U tests, and r is the rank-biserial effect size. Lower values reflect better performance in every panel.

IMCC PCA Presentation (normalised to healthy; 85 % confidence ellipse)

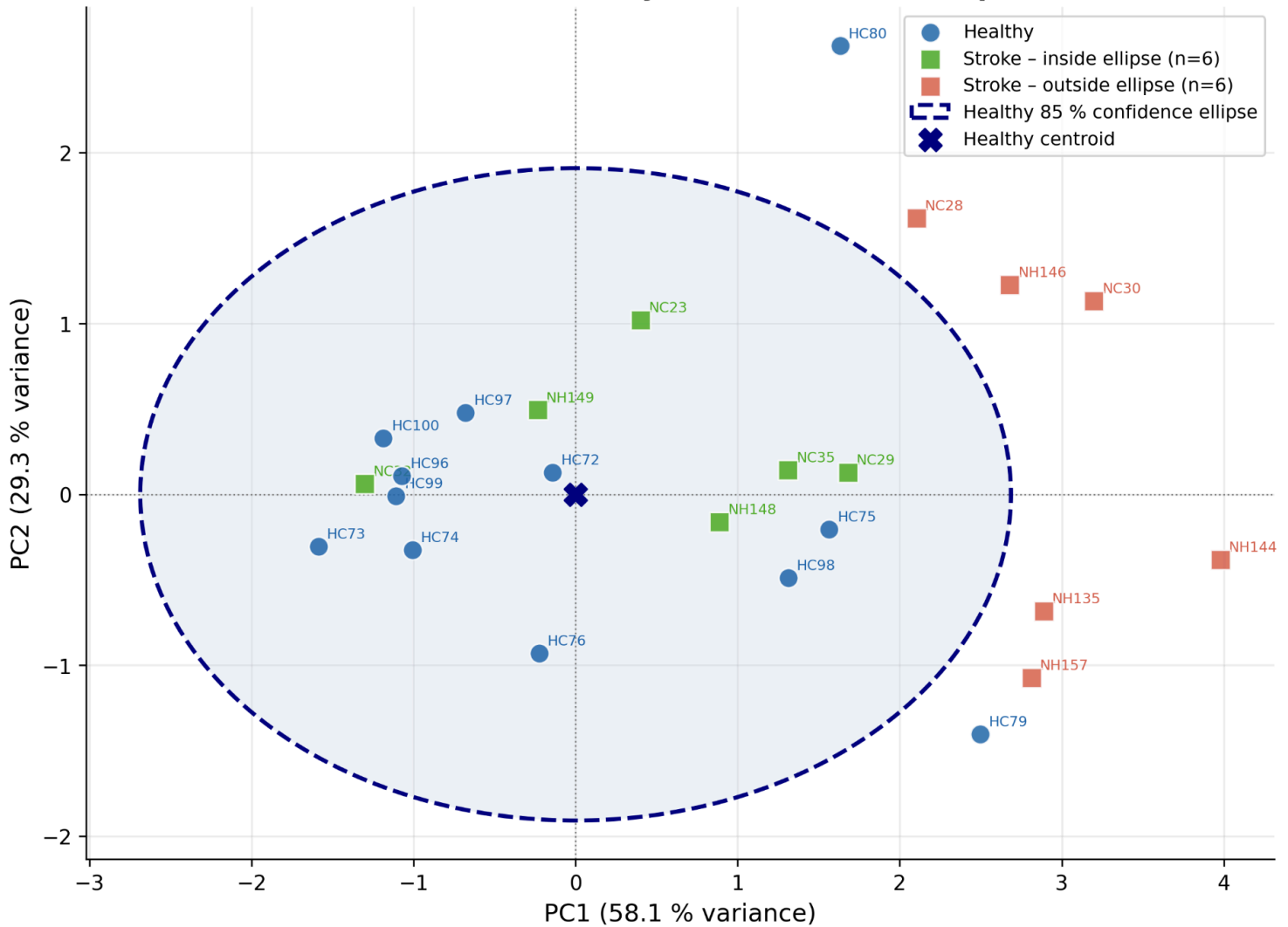


Figure 3

Composite IMCC score in the PC1–PC2 plane with the 85 % healthy confidence ellipse. The PCA was fit on the 12 healthy participants only (after z-scoring features using the healthy mean and SD); PwS were projected onto the same axes without contributing to their direction. PC1 and PC2 together explain 87.4% of the healthy variance (PC1, 58.1%; PC2, 29.3%). Healthy controls are shown as blue circles, PwS classified inside the healthy ellipse as green squares, and PwS classified outside the ellipse as red squares. The navy "X" marks the healthy centroid. The dashed ellipse encloses the 85% chi-square confidence region of the bivariate healthy distribution; PwS are classified inside or outside this region.