

A hidden signal in the resting hand: dissociable bilateral activation after stroke

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Abstract

Background

During upper-limb assessment after stroke, attention usually focuses on the hand asked to move; the resting hand is considered informative only if it visibly mirrors the action. We asked whether it carries covert physiological information: whether bilateral activation is a single output-related overflow signal, or whether different features also reflect how skilled actions are represented.

Methods

In this cross-sectional study, we studied 18 participants with chronic left-hemisphere stroke (> 6 months post-stroke; 41–79 years, mean 56.9 ± 10.7 ; 8 women) and 13 controls (21–73 years, mean 45.5 ± 16.9 ; 10 women). The stroke cohort comprised 11 apraxia-dominant participants with relatively preserved strength and 7 paretic participants with marked hemiparesis. Participants performed a 49-gesture praxis battery spanning imitation, intransitive gestures, pantomimed tool use and real-object actions while bilateral high-density surface electromyography and motion capture were recorded. Coupling was defined as resting-hand muscle activity exceeding participant-specific baseline and temporally locked to active-hand movement. We quantified coupling rate (how often coupling occurred) and composite intensity (how strong and temporally coherent it was when present).

Results

During coupling events, median resting-hand wrist displacement was 0.64 mm; 80% were < 2 mm and 93% < 5 mm. Coupling rate decreased as movement extent increased in controls and the apraxia-dominant group, but increased in the paretic group (group $\times$ extent $\chi^2(2) = 19.8$, $P = 4.9 \times 10^{-5}$), persisting after adjustment for active-hand muscle output, movement speed and gesture category. Composite intensity differed: controls generated higher resting-hand amplitude for real-object than internally generated actions, whereas the apraxia-dominant group showed elevated, context-insensitive amplitude, and the paretic group showed low amplitude with a further reduction for real-object actions (group $\times$ category $\chi^2(6) = 35.9$, $P < 0.0001$). Clinical arm function related to coupling rate in a gesture-dependent manner.

Conclusions

Covert bilateral activation after stroke is therefore not a single feature. Coupling rate reflects when the resting hand is recruited and relates to movement extent and paretic output constraints; coupling intensity reflects how strongly and selectively it is recruited and relates to apraxia-dominant disruption of action representation. High-density surface electromyography reveals physiological structure that

routine observation misses, offering a candidate bridge between clinical assessment and mechanism-based rehabilitation stratification.

Background

Involuntary activation of one limb during intended movement of the other, variously described as ‘mirroring’, ‘motor overflow’ or ‘synkinesia’, is recognised across developmental, movement-disorder and acquired neurological conditions, and has attracted particular interest after stroke because of its relationship to impairment and recovery (1). In clinical practice, this phenomenon is usually recognised only when the resting limb visibly moves. Yet the absence of visible movement does not imply absence of physiological coupling: low-amplitude, time-locked muscle activity may remain below the threshold of bedside observation while still carrying information about how the damaged motor system is organised. This distinction matters because mirror activity after stroke has generally been treated as a unitary manifestation of released descending drive, whereas the content of the resting-hand signal has rarely been examined.

Interhemispheric imbalance after stroke has been studied with transcranial magnetic stimulation and functional neuroimaging, linking altered interactions between the damaged and intact hemispheres to motor impairment and recovery (2–4). Bilateral activation has therefore been proposed as both a biomarker of recovery potential and a therapeutic target (5). Several mechanisms have been proposed, including failed transcallosal inhibition (2, 6), upregulation of bilaterally organised subcortical pathways released from cortical constraint (7–9), and residual ipsilateral corticospinal projections (6, 10). These accounts differ in substrate, but share a common prediction: bilateral activation should scale with force, effort or the demands placed on descending motor output. Isometric contractions and grip tasks have been well suited to testing this prediction. What they do not address is whether the representation that specifies what action to perform, as distinct from the descending command that executes it (11), can independently contribute to the bilateral signal. Here, we use action representation to refer to the learned knowledge that specifies the identity, form and contextual constraints of a skilled gesture, and descending motor output to refer to the effector-level drive that implements the selected action.

Limb apraxia provides a direct way to separate disruption of skilled-action representation from corticospinal output failure. After left-hemisphere stroke, ideomotor apraxia reflects damage to parietofrontal praxis networks that generate, select and lateralise skilled action (12, 13). These processes are partly dissociable from the motor system that executes them (11, 14, 15). We therefore contrasted the apraxia-dominant group, whose participants had relatively preserved strength, with the paretic group, in which motor output failure dominated the presentation, recognising that apraxia and paresis commonly co-occur. This allowed us to ask whether different features of the same covert bilateral signal relate preferentially to skilled-action representation or to impaired motor output.

High-density surface electromyography (HD-EMG) with synchronised motion capture is well suited to this problem because it samples a physiological level that lies between bedside observation and brain

imaging. Clinical examination and upper-limb scales score the visible endpoint of movement, such as weakness, abnormal movement, impaired gesture or loss of dexterity, yet behaviour is generated by physiological activity that may remain measurable even when no overt movement is apparent. This is analogous to cardiovascular assessment, where inspection and palpation are complemented by quantitative physiological measures such as blood pressure and the electrocardiogram. In motor assessment after stroke, high-density surface electromyography provides a comparable physiological readout at the muscle level: it detects distributed, time-locked recruitment across extensor and flexor compartments during natural actions, while motion capture establishes whether that recruitment produces observable movement. Combining the two methods therefore allowed us to treat the resting hand not simply as a source of visible mirror movements, but as a physiological signal with two separable dimensions: how often bilateral coupling occurs, and how strongly the resting hand is recruited when coupling is present.

We recorded bilateral forearm HD-EMG during a 49-gesture praxis battery in participants with chronic left-hemisphere stroke with apraxia-dominant or paretic profiles and in age-comparable controls. The naturalistic gesture battery allowed us to examine the resting hand during goal-directed actions that varied in movement extent, praxic demand and object support. We reasoned that if the resting-hand signal reflected only effector-level drive, coupling should scale similarly with movement extent across groups. By contrast, if the signal also carried information about how the action is represented, its rate or intensity should vary with gesture context and clinical phenotype. We examined three questions: whether bilateral coupling rate scales with movement extent and whether this differs between the apraxia-dominant and paretic groups; whether the intensity of the bilateral signal is modulated by gesture context; and how these measures relate to clinical arm function.

Materials and methods

Participants

This was a cross-sectional study comparing bilateral EMG coupling across three groups (controls, apraxia-dominant stroke, and paretic stroke), each assessed at a single research session. Thirty-eight participants were screened: 17 healthy volunteers and 21 people with chronic (> 6 months) stroke recruited from the IC3-Apraxia cohort (Rounis et al., in preparation; a subset had previously completed the IC3 study protocol (16)), a cross-sectional study of post-stroke apraxia at Imperial College Healthcare NHS Trust. Recruitment and HD-EMG/motion-capture assessment for the present study took place between July and December 2026. Analyses were restricted a priori to left-hemisphere lesions, given the strongly left-lateralised organisation of the praxis network (Fig. 1, (12)). Seven participants were excluded: three with stroke (two not meeting this criterion and with concurrent technical artefact, one without confirmed ideomotor apraxia on pre-selection testing) and four controls (two left-handed, one outside the age-matching range, one with baseline recording artefact), leaving 13 controls (mean age 45.5 [SD 16.9] years; 10 female) and 18 participants with stroke (mean age 56.9 [SD 10.7] years; 8 female; median time since stroke 55 months [IQR 27–96]; see Table 1 for group values). Groups did not

differ significantly in age (Kruskal–Wallis $P = 0.23$) or sex ($\chi^2 P = 0.19$). The study received ethical approval from Imperial NHS Research Ethics Committee (REC Reference 23/SW/0140, IRAS 330409). Written informed consent was obtained from all participants according to the Declaration of Helsinki.

Table 1

Participant characteristics by group. Group labels: Control, healthy volunteers; NPA, apraxia-dominant stroke group with relatively preserved strength (analysis label); Paretic, paretic-dominant stroke group.

Characteristic	Controls (n = 13)	NPA (n = 11)	Paretic (n = 7)
Age, years, mean (SD)	45.5 (16.9)	57.5 (12.5)	56.0 (7.9)
Sex, female n (%)	10 (77%)	5 (45%)	3 (43%)
Months post-stroke, median [IQR]	—	41 [23–62]	92 [43–127]
mRS, median [range]	—	2 [0–3]	2 [2–4]
ARAT, median [IQR]	—	51.0 [49.2–56.5]	1 [0–2.5]
MVC ratio, mean (SD)	—	0.98 (0.22)	0.12 (0.06)
Aphasia, n	—	4	5
<i>Transitive</i> accuracy, % (SD)	—	70.7 (18.0)	74.1 (11.8)
<i>Objects</i> accuracy, % (SD)	—	84.9 (11.3)	83.8 (8.9)

Participants with stroke were classified into an apraxia-dominant group (confirmed ideomotor apraxia with relatively preserved strength, defined by the dominant clinical phenotype and, where maximum voluntary contraction (MVC) data were available, supported by an MVC ratio ≥ 0.50 , defined as affected-to-unaffected peak isometric grip force); $n = 11$; mean age 57.5 [SD 12.5] years; median modified Rankin Scale (mRS) score 2 [0–3]; median Action Research Arm Test (ARAT) score 51.0 [IQR 49.2–56.5]) and a paretic group (complete hemiparesis or MVC ratio < 0.30 ; $n = 7$; mean age 56.0 [SD 7.9] years; median mRS 2 [2–4]; median ARAT 1 [IQR 0–2.5]). Three participants with moderate hemiparesis but missing MVC data were retained in the apraxia-dominant group based on clinical presentation; sensitivity analyses reassigning them did not alter the primary results. ARAT was lower in the paretic than the apraxia-dominant group by design ($p < 0.001$). Aphasia was present in 9/18 participants with stroke (4/11 apraxia-dominant, 5/7 paretic). Participant characteristics are given in Table 1.

Definition and operationalisation of the apraxia-dominant group. Because apraxia and corticospinal paresis frequently co-occur after left-hemisphere stroke, the two stroke groups were defined by dominant clinical phenotype rather than by a single apraxia severity score. Ideomotor apraxia was confirmed by a consultant neurologist on standardised clinical examination, and apraxia status was established from a separate pre-selection praxis assessment administered independently of the 49-gesture EMG protocol, including gesture production, single- and multi-object use, meaningless imitation and gesture decision (17, 18). Because several subtests were compressed toward ceiling in this cohort,

apraxia status was determined clinically rather than by a single cut-score. The paretic group was not assumed to be free of apraxia: most participants in the paretic group in this cohort were also apraxic. The groups therefore differ in which deficit dominates the motor phenotype, not in the presence or absence of apraxia.

No formal a priori sample-size calculation was performed. Study size was determined by the number of eligible participants available within the parent IC3-Apraxia cohort during the recruitment period and by the feasibility of the intensive HD-EMG and motion-capture protocol. The design prioritised dense within-participant sampling, with each participant completing up to three repetitions of 49 gestures, analysed using mixed-effects models that account for repeated observations within participants and gestures.

Gesture battery

Participants performed 49 gestures across four categories of graded praxic demand, each repeated three times, while keeping the contralateral arm relaxed. *Imitation* comprised 10 meaningless hand (four) and finger (six) configurations copied from the examiner (17, 18). *Intransitive* comprised 10 communicative gestures without objects (e.g. wave, thumbs-up). *Transitive* comprised 14 pantomimed tool-use gestures without the object (e.g. hammering, writing). *Objects* comprised 15 actions with the real object present (e.g. using a screwdriver, cutting with scissors). The battery incorporated items from established praxis assessments (17, 18), supplemented by gestures developed for the present study, and was designed to span dimensions relevant to the apraxia-paresis dissociation: reliance on stored praxic representations and the presence or absence of sensorimotor object affordances (15, 19). The full gesture list is provided in Table 2.

Table 2
Gesture battery

Gesture Category	Gesture
Intransitive	Thumbs Up
	Military Salute
	Stop Gesture
	Finger Snap
	Waving Hand
	Silence Gesture
	Blowing a Kiss
	Scratching Head
	Index Pointing
	Swatting Gesture
Transitive (pantomime – no object in hand)	Drinking from a Glass
	Sprinkling Salt
	Playing a Trumpet
	Writing with a Pen
	Eating a Small Fruit
	Combing Hair
	Hammering a Nail
	Playing a Guitar
	Cutting with a Knife
	Using a Lighter
	Screwing in a Light Bulb
	Eating an Apple
	Shaving with a Razor
	Cutting with Scissors
Objects (with real objects in hand)	Grasping Cup by Handle
	Grasping Cup from Top (Handle on Left)
	Grasping Cup from Top (Handle on Right)

Gesture Category	Gesture
	Using a Screwdriver
	Locking a Door with a Key
	Sprinkling Salt
	Clipping with a Foldback Clip
	Removing a Staple
	Cutting with Scissors
	Drinking with a Straw
	Cutting with a Knife
	Combing Hair
	Using a Whisk
	Using a Nail Clipper
	Eating with a Fork
Imitation	Hand held vertically in front of the mouth, palm sideways
	Hand raised to the side of the head
	Moving to a flat hand above the forehead
	Hand covering mouth horizontally, palm towards mouth
	Thumb and little finger extended
	Thumb, index and little finger extended
	Thumb and fingers forming a C shape
	Closed fist with little finger pointing downward
	Index and thumb extended
	Index, ring and little finger extended, middle and thumb flexed

Bilateral HD-EMG and motion capture

Four high-density surface EMG grids (Novecento, OT Bioelettronica; 96 electrodes per grid in a 6×16 layout, 8-mm inter-electrode distance, 2000 Hz) were placed bilaterally over forearm flexor and extensor compartments (Fig. 2). A synchronised infrared motion-capture system (VICON Nexus 2.18, 28-camera Vantage, 200 Hz; 18×4 mm markers on the hand and 4×14 mm markers per forearm) provided

kinematic references. Synchronisation was achieved using Arduino-generated pulses, with matched pulse times used offline to correct inter-system clock drift. EMG signals were band-pass filtered (20–450 Hz, fourth-order zero-phase Butterworth) and notch filtered (50 Hz); per-channel sliding root-mean-square (RMS) envelopes (50 ms window) were computed at the 2000 Hz sampling rate, averaged across all 96 channels of each grid, and smoothed with a 200 ms moving-average window. High-density grids were used to sample the entire extensor and flexor compartments, so that coupling was detected wherever it arose within the muscle rather than being contingent on bipolar electrode placement; averaging then yields a compartment-level estimate robust to the precise location of active motor units. An automated integrity sweep flagged flat channels (near-zero variance) as rare (median 0, mean 1.5 of 96 channels per grid; 71% of grids had none).

Bilateral coupling measures

Bilateral coupling was quantified along two dimensions: how often gesture-locked activity appeared in the resting hand (coupling rate) and how strong it was when present (intensity). For every gesture repetition we analysed the resting (task-inactive) hand. Repetitions were delimited within each manually annotated gesture by detecting peaks in the VICON speed signal or by equal subdivision. The *Coupling rate* metric was the proportion of repetitions meeting two joint criteria: C1 (amplitude), requiring that the peak resting-hand extensor RMS within the repetition exceeded a participant-specific threshold set at the median plus two standard deviations of that participant's quiet-baseline samples, derived from dedicated rest recordings with fallback to inter-gesture rest segments; and C2 (temporal coupling), requiring that the positive peak cross-correlation, computed over the full repetition, between the resting- and active-hand extensor envelopes within a ± 150 ms lag window exceeded $r = 0.5$. Requiring both C1 and C2 restricted the measure to amplitude events that were also time-locked to the active gesture, excluding both sub-threshold fluctuations and tonic or temporally unrelated resting activity. *Composite intensity* was the product of the resting-hand peak extensor RMS and its positive peak cross-correlation with the active hand during coupling events, capturing the magnitude of gesture-locked bilateral EMG on a single scale while down-weighting temporally incoherent activity. RMS-derived amplitude indices have been used to quantify bilateral EMG overflow magnitude in stroke (20), and cross-correlation has been used to quantify temporal coupling between EMG signals (21); we combined them multiplicatively since the product suppresses cases where either factor is near zero (high amplitude without temporal lock, or spurious correlation at near-baseline amplitude). *Movement extent* was the mean active-hand forearm markers path length (mm), per repetition from VICON.

Statistical analysis

All models were linear mixed-effects models (LME; Python statsmodels MixedLM) with random intercepts, estimated by restricted maximum likelihood (REML). Fixed-effect significance was assessed from Wald z-tests; nested models were compared by likelihood-ratio test (LRT) re-estimated under maximum likelihood. Regression coefficients (β) are fixed-effect estimates for the corresponding model

term, reported with the p-value from its Wald z-test unless stated as a likelihood-ratio p-value for a nested-model comparison. We report a random-intercept-only structure; models with random slopes were singular at this sample size, so we retained the most complex structure the data reliably supported. All tests were two-sided at $\alpha = 0.05$. The three interaction tests corresponding to the study's pre-specified hypotheses (H1, H2 and H3) were controlled for family-wise error using the Holm-Bonferroni procedure. Since an interaction term shows only that an effect differs across groups, we recovered each group's own slope by re-fitting the model with each group as the reference category.

H1 (coupling rate $\times$ movement extent). Given that the coupling event is binary, the coupling rate was modelled at the repetition level as a linear probability model: coupling rate $\sim$ extent_centred $\times$ group + (1|participant) + (1|gesture), with movement extent mean-centred and group a three-level factor.

H2 (intensity $\times$ gesture context). Composite intensity was modelled as intensity $\sim$ group $\times$ gesture_category + (1|participant), with the omnibus group $\times$ category interaction tested by LRT; left- and right-active repetitions were modelled separately because the paretic group contributed no right-active data; the two were additionally compared in a combined model of controls and apraxia-dominant group carrying a group $\times$ category $\times$ active-hand interaction.

H3 (clinical correlates). As ARAT is a between-person predictor, the ARAT $\times$ gesture-type interaction was fit on participant $\times$ gesture-type mean coupling rates (18 participants $\times$ 4 categories) rather than at the repetition level, which would pseudo-replicate a single-valued participant-level trait across $\sim 2,600$ repetitions (the alternative random-slope correction was singular at this sample size). Coupling rate was modelled as coupling rate $\sim$ ARAT $\times$ gesture_type + (1|participant), Imitation reference, with the ARAT $\times$ Transitive interaction as the pre-specified contrast. In the MVC subset ($n = 13$), MVC-normalised resting-hand amplitude was correlated with ARAT. For the H3 secondary analysis, the same coupling-rate procedure (criteria C1–C2) was applied to the flexor compartments, yielding an analogous flexor coupling-rate measure.

The H1 interaction was subjected to convergent robustness checks: leave-one-out re-estimation across the paretic group, re-computation across C2 thresholds of 0.3, 0.5 and 0.7, addition of kinematic covariates (active-hand peak speed, resting-hand path length) and active-hand EMG power, a logistic mixed model on the binary outcome with crossed participant and gesture intercepts, and a model additionally adjusted for gesture category. In a secondary, exploratory analysis (not pre-specified and not included in the family-wise correction), movement extent was split into each participant's average extent for a given gesture and each repetition's departure from that average, to ask whether the relationship operated primarily between participant-gesture averages or between repetitions of the same gesture.

During manuscript preparation, the authors used large language model tools for editorial assistance with wording and structure. The authors reviewed, edited and approved all resulting text, and take full responsibility for the content of the manuscript.

Results

Bilateral coupling is predominantly subthreshold for visible movement

Bilateral EMG coupling was detected in all three groups. During EMG-detected coupling events the resting hand was essentially stationary: its net wrist displacement had a median of 0.64 mm, and 80% of events showed displacement below 2 mm (93% below 5 mm; Fig. 3). Movement of this magnitude would not be reliably detected on routine visual inspection at the bedside. The large majority of bilateral coupling was therefore subclinical, small enough to be missed by standard bedside assessment, though readily quantified by motion capture.

H1. Coupling rate: movement-extent slope reversal is Paretic-specific

If the bilateral signal reflects movement demand alone, coupling should scale with how much movement is required; if it also reflects the action plan, it need not. The relationship between movement extent and bilateral coupling rate differed by group (Fig. 4, Row A), in a model carrying random intercepts for both participant and gesture. Controls and the apraxia-dominant group showed negative slopes of similar magnitude (Controls: $\beta = -1.02$ pp per 100 mm, 95% CI $[-1.80, -0.24]$, $P = 0.010$; apraxia-dominant: $\beta = -0.71$ pp per 100 mm, 95% CI $[-1.32, -0.10]$, $P = 0.023$): reduced movement extent was associated with higher bilateral coupling rate, with no significant difference between the two groups (β difference = $+0.31$ pp per 100 mm, 95% CI $[-0.64, +1.27]$, $P = 0.52$). The paretic group reversed to a positive slope ($\beta = +0.92$ pp per 100 mm, 95% CI $[+0.30, +1.55]$, $P = 0.004$): gestures with greater movement extent were associated with more frequent bilateral coupling (group $\times$ extent interaction $\chi^2 = 19.8$, $df = 2$, $P = 4.9 \times 10^{-5}$). Coupling increased with a participant's average extent for a given gesture ($+1.05$ pp per 100 mm, 95% CI $[0.39, 1.71]$, $P = 0.002$), whereas variation between repetitions of the same gesture showed no detectable association (-0.23 pp per 100 mm, 95% CI $[-2.27, 1.80]$, $P = 0.82$); with only two to three repetitions per gesture the latter estimate is imprecise, and the two components did not differ significantly ($P = 0.24$).

Overall coupling rates did not significantly differ between groups (Controls 16.5%, apraxia-dominant 15.4%, paretic 13.9%, Kruskal-Wallis $P = 0.71$). Movement extent and active-hand extensor EMG power were only weakly related ($r = 0.03$), indicating that larger gestures were not systematically associated with greater active-hand extensor output. Adding active-hand extensor EMG power as a covariate left unchanged the group $\times$ extent interaction ($\beta = 1.94 \rightarrow 1.92$ percentage points per 100 mm; $P = 0.0007 \rightarrow 0.0008$), as did movement speed. The result survived leave-one-out exclusion of each paretic participant and was reproduced by a logistic mixed model on the binary coupling outcome (odds ratio = 1.14 per 100 mm, 95% CI $[1.06, 1.23]$, $p = 5.8 \times 10^{-4}$), and by a model additionally adjusted for gesture category. The interaction remained significant at C2 cross-correlation thresholds of 0.3 and 0.5, but attenuated at the most stringent threshold of 0.7 where few coupling events qualified.

H2. Intensity: elevated and context-insensitive in apraxia-dominant stroke

We next asked whether the magnitude of the resting-hand signal is sensitive to gesture context beyond differences in active-hand muscle output. On the intensity dimension, for left-hand-active (right-hand-resting) trials the three groups showed qualitatively different patterns of category modulation (Fig. 4, Row B, left panel; omnibus group $\times$ category LRT: $\chi^2 = 35.9$, $df = 6$, $P < 0.0001$); the corresponding right-hand-active analysis was not significant ($\chi^2 = 0.8$, $df = 3$, $P = 0.854$). This left-right difference was confirmed directly in a combined model (group $\times$ category $\times$ active-hand interaction, $\chi^2 = 12.75$, $df = 3$, $P = 0.005$; secondary analysis, not pre-specified and not included in the family-wise correction), and was carried by the apraxia-dominant group (category $\times$ active-hand $\chi^2 = 11.29$, $df = 3$, $P = 0.010$) with no such effect in controls ($\chi^2 = 4.06$, $df = 3$, $P = 0.26$).

In Controls, composite intensity was modulated by gesture context, with real-object actions producing a higher amplitude than all internally generated categories (1.38 $\times$, 95% CI [1.20 $\times$, 1.58 $\times$], $P < 0.001$). This is consistent with the idea that when a real object is present the intended action is more strongly constrained by object affordances, and that more of the selected action is expressed in the resting limb. In the apraxia-dominant group, coupling intensity was uniformly elevated across gesture categories, with no significant difference between Objects and the internally generated categories ($P = 0.29$). In the paretic group, composite intensity was uniformly low with a further reduction during Objects ($P = 0.004$), consistent with impaired descending output limiting bilateral signal generation particularly when precise object manipulation was required.

This effect was not attributable to differences in active-hand muscle output: adding active-hand extensor EMG power as a covariate left unchanged both the omnibus group $\times$ category interaction ($\chi^2 = 35.9$ with or without the covariate) and the apraxia-dominant Objects-vs-internally-generated contrast ($P = 0.29 \rightarrow 0.30$).

Both dissociations reproduced in flexors, attenuated (not pre-specified; outside the Holm family): group $\times$ extent $\chi^2 = 10.5$, $P = 0.005$; group $\times$ category $\chi^2 = 16.4$, $P = 0.012$. Only the Paretic Objects reduction was extensor-specific (1.02 $\times$, $P = 0.22$).

H3. Clinical arm function relates to bilateral coupling rate

Finally, we asked whether either dimension of the bilateral signal carries clinically meaningful information about arm function. There was no global ARAT main effect on coupling rate (Fig. 5), consistent with the opposing gesture-specific slopes seen in the group analyses. The relationship between ARAT and bilateral coupling rate was gesture-type-dependent (ARAT $\times$ Transitive interaction $P < 0.001$ for extensors, $P = 0.018$ for flexors). The Transitive simple slope was negative but did not reach significance alone ($P = 0.072$), while Imitation moved in the opposite direction ($P = 0.002$). Full model parameters are given in Fig. 5. In the subset of participants with stroke for whom MVC data were available ($n = 13$), MVC-normalised resting-hand amplitude correlated negatively with ARAT ($r = -0.84$,

95% CI [– 0.95, – 0.53], $P < 0.001$), showing that greater MVC-normalised resting-hand amplitude was associated with poorer arm function.

All three primary interaction tests remained significant after Holm-Bonferroni correction (adjusted $P = 8.7 \times 10^{-6}$, 9.84×10^{-5} , and 0.0014 for H2, H1, and H3 respectively).

Discussion

We used bilateral high-density surface EMG during a naturalistic gesture battery to ask what an apparently still resting hand contains after chronic left-hemisphere stroke. The central finding was not simply that bilateral activation was detectable below the threshold of clinical observation, but that this hidden signal separated into two dimensions. In the paretic group, coupling rate increased with movement extent. In the apraxia-dominant group, coupling rate remained similar to healthy controls, but coupling intensity was elevated and lost its normal modulation by gesture context. Thus, rate and intensity did not provide duplicate measures of overflow; they provided different windows onto the resting-hand signal (summarised in Table 3). Unlike force- or amplitude-matched reaching paradigms, this naturalistic battery captured bilateral activation during goal-directed actions varying in praxic demand and object support, closer to the behaviours assessed clinically and used in daily life.

Table 3

The coupling rate/intensity double dissociation. Group labels: Control, healthy volunteers; NPA, apraxia-dominant stroke group with relatively preserved strength (analysis label); Paretic, paretic-dominant stroke group.

NPA vs Controls	Coupling rate (extent slope)	Intensity (composite RMS × xcorr)
	Same (both negative, both significant)	Elevated, context-insensitive (affordance modulation abolished; $P < 0.001$)
Paretic vs Controls	Reversed positive slope; group × extent interaction $P < 0.001$	Low, with reduction in Object category ($P = 0.004$)
Interpretation	Consistent with effector-level overflow (extent-scaled)	Consistent with representation-level contribution (context-insensitive)

The two measures ask different questions. Coupling rate asks when, or how often, the resting hand is recruited during an intended unilateral action; coupling intensity asks how strongly and selectively it is recruited once coupling occurs. The paretic abnormality was therefore one of recruitment probability, tied to movement extent; the apraxia-dominant abnormality was one of contextual tuning, in which the resting hand was not recruited more often but the signal reaching it was stronger and less appropriately shaped by gesture context. We consider these two patterns in turn.

Two dimensions, two candidate mechanisms

The paretic pattern is best understood as an abnormality of recruitment probability: larger movements were more likely to trigger bilateral coupling, even though movement extent was not simply a proxy for

greater active-hand extensor output ($r = 0.03$). The coupling rate-extent slope reversal was specific to the paretic group. In these participants, greater movement extent across gestures was accompanied by more frequent bilateral coupling events, a pattern consistent with the reticulospinal overflow account developed by Ejaz and colleagues (7) and extended by Dai and colleagues (20) into more ecological tasks. Primate work has demonstrated that corticospinal lesions upregulate reticulospinal drive to bilateral extensor motoneurone pools (8, 9, 22), providing an anatomically plausible substrate for the extent-scaled component we observe. Our data do not resolve whether the bilateral signal in the paretic group originates from ipsilateral corticospinal projections, from reticulospinal drive, or from a combination, but the extent-dependent profile, reproduced across extensor and flexor compartments, is compatible with the picture that has emerged from that literature. Because each gesture was repeated two to three times, the present design could not establish whether the extent-coupling relationship also operates from one execution to the next within the same gesture.

The apraxia-dominant pattern was different: the abnormality lay not in recruitment probability, but in the contextual tuning of signal intensity. In controls, real-object actions produced the highest bilateral amplitude, consistent with models in which object affordances support action selection and stored manipulation knowledge (15, 19, 23). The apraxia-dominant group largely lost this modulation, with elevated coupling amplitude that did not scale with gesture category. One interpretation is that damage to the ventro-dorsal praxis network compromises the lateralisation of internally-specified motor commands, so that the selected action is expressed bilaterally with amplitude that no longer depends on whether sensorimotor affordances scaffold the action. The intensity effect was direction-specific, with the group $\times$ category interaction evident for left-hand-active but not right-hand-active trials; we therefore frame the intensity result as an elevated and context-insensitive signal rather than as a fully symmetric bilateral phenomenon.

Affordance modulation and the dual-route praxis framework

The category-specific intensity pattern aligns with the dual-route model of praxis (14, 15, 24). Real-object actions engage online, affordance-supported control (15, 19, 25, 26, 33), whereas pantomime and imitation place greater demands on learned action representations and praxis networks (27–32). The affordance-competition framework proposes that object properties specify candidate actions online, which must be selected and parameterised according to current task demands (26, 33). One possibility is that in apraxia, impaired use or control of this online information leaves action output relatively more dependent on stored or stereotyped action representations and less effectively scaled to the current object. At the muscular level, this would be expected to reduce context-dependent modulation of motor recruitment. The loss of object-related modulation of bilateral EMG in the apraxia-dominant group is compatible with this account, although the present data do not directly measure object-specific scaling of the active-hand motor command. The same pattern could in principle arise from a more general loss of context sensitivity in the praxis system, or from an altered gain of bilateral cortico-cortical or corticoreticular projections when the parietofrontal praxis network is lesioned. Jeannerod and

colleagues' (34) distinction between pragmatic and semantic components of grasping, and Osiurak and Badets' (35) related dissociation between reasoning- and manipulation-based accounts of tool use, both suggest that the intensity signal we measure may index the integrity of the stored representation rather than online sensorimotor control alone.

This interpretation also helps explain why the apraxia-related effect was expressed on the intensity rather than the rate dimension. Transitive pantomime and meaningless imitation place high demands on internally generated action representations, whereas object-present actions provide online affordance support. In controls, this support modulated bilateral coupling intensity; in the apraxia-dominant group, that modulation was lost, leaving an elevated and context-insensitive signal across gesture categories. The intensity measure may therefore index the integrity and lateralisation of the stored action representation as it is expressed bilaterally, even when overall coupling rate remains control-like.

In the paretic group, intensity was uniformly low, with a small further reduction during real-object action. This is compatible with the descending pathway itself constraining bilateral signal amplitude, particularly when the object-manipulation task demands the most precise and dexterous output.

Relation to prior work

The increase in bilateral EMG with movement extent in the paretic group is broadly consistent with the effort-dependent account proposed by Ejaz and colleagues (7). Although movement extent is not equivalent to force, both studies show that bilateral activity scales with a parameter of the movement: force in their paradigm and movement extent in ours. This convergence may reflect increasing engagement of bilaterally organised subcortical pathways when corticospinal motor output is impaired, although our data do not identify the pathway responsible. Movement extent is also not equivalent to effort: perceived effort during reaching depends on movement duration and force rather than distance per se (36). We therefore interpret the rate–extent relationship as compatible with an output-linked component, rather than as a direct measure of effort. Our findings also extend the surface-EMG observations of Dai and colleagues (20) to naturalistic whole-hand gestures and complement neuroimaging evidence of ipsilesional and contralesional motor-cortical activation during stroke recovery (3, 4, 37).

The present paradigm allowed this relationship to be examined across gestures that differed not only in movement extent but also in their praxic requirements, including pantomime and actual object use. Analysing the apraxia-dominant and paretic groups separately further showed that the bilateral signal did not vary uniformly across stroke phenotypes. Controls and the apraxia-dominant group showed similar scaling with movement extent despite differing in overall signal intensity, whereas paretic group showed a distinct scaling pattern. This dissociation suggests that the magnitude of bilateral activity and its modulation by movement extent may reflect partly different influences, potentially related to praxic dysfunction and motor impairment.

Gesture-execution accuracy, scored on a subset of participants by a single blinded rater, was positively associated with bilateral coupling rate in the apraxia-dominant group. Because scored gestures were available for seven participants and were rated by a single assessor, we treat this observation as hypothesis-generating rather than confirmatory: it is consistent with the idea that patients who reproduce the intended action more accurately have more coherent signal to broadcast bilaterally, and it identifies multi-rater or automated gesture scoring as a natural target for follow-up work.

Clinical implications

Standard upper-limb scales compress many mechanisms into a single behavioural score. Two individuals may perform equally poorly for different reasons: one because descending output is weak, another because the skilled action representation is degraded or poorly contextualised. The clinical value of the rate–intensity dissociation is that it begins to separate these possibilities physiologically: coupling rate captures the likelihood that the resting hand is recruited during attempted action, whereas intensity and contextual tuning capture how that recruitment is shaped by the intended gesture.

If bilateral activation after stroke is not a unitary phenomenon, then interventions that use the less-affected hand as a trigger, cue, mirror or training model for the affected hand may not be mechanistically interchangeable. Some may primarily harness output-linked coupling, whereas others may depend on the integrity of the skilled action representation being transferred. Kim and Lee (38) demonstrated that biofeedback functional electrical stimulation, synchronised to non-paretic hand EMG and combined with mirror therapy, improved upper-limb motor recovery. The logic of this approach aligns closely with the extent-driven bilateral coupling that our paretic group displays. More generally, transfer of motor skill learning from the intact to the affected hand has been demonstrated after stroke (39); our findings raise a potential caveat, however, since such transfer may depend on an intact action representation. In the apraxia-dominant group, where the left-lateralised praxic representation is itself degraded, approaches that rely on transferring the skilled action representation may therefore be less effective. Whether elevated context-insensitive bilateral coupling identifies patients less likely to benefit from such approaches warrants prospective investigation. For patients in whom the bilateral signal appears more consistent with representation-level contributions, complementary strategies targeting the praxic representation itself, such as gesture-specific training, motor imagery, and systematic retraining of the action hierarchy (40, 41), may warrant investigation. The sign of the coupling rate–extent relationship could, in principle, offer a physiological handle for stratifying patients toward mechanism-matched interventions; establishing this would require prospective validation in a substantially larger sample.

Limitations

The study prioritised physiological resolution and dense within-participant sampling over cohort-scale recruitment: each participant contributed repeated measurements across a 49-gesture battery, enabling robust estimation of extent–coupling relationships. The cohort size limits precision for finer subgroup and lesion-level analyses, although the primary rate–extent interaction was stable across participant-level resampling, leave-one-out exclusion, alternative coupling thresholds, logistic mixed modelling,

gesture-level modelling, and adjustment for kinematic covariates. Because apraxia and corticospinal paresis frequently co-occur after left-hemisphere stroke, the groups should be interpreted as dominant clinical phenotypes rather than pure mechanistic categories; sensitivity analyses based on case reassignment and continuous clinical measures supported the same pattern. The active-hand dependence of the intensity effect could be tested in controls and the apraxia-dominant group, but not in the paretic group because its participants could not contribute right-hand-active trials; its anatomical basis therefore remains open. Participants were recruited from a single tertiary UK stroke service and were able to attend a research session involving a lengthy bilateral recording protocol; findings may not generalise to acute-phase patients, individuals with more severe impairment unable to participate, or settings with different rehabilitation pathways.

Conclusions

Bilateral forearm EMG during naturalistic gesture reveals patterns consistent with two dissociable contributions to the resting-hand signal after stroke: extent-scaled coupling rate in paretic stroke, and elevated context-insensitive coupling amplitude in apraxia-dominant stroke. Whether these correspond to the output-level and representation-level contributions suggested by dual-route models of praxis will require further work, but the observations reported here suggest that the bilateral signal is not a single quantity and that the resting hand may carry more information than standard clinical assessment reveals.

Abbreviations

ARAT: Action Research Arm Test

EMG: electromyography

HD-EMG: high-density surface electromyography

LME: linear mixed-effects model

LRT: likelihood-ratio test

MVC: maximum voluntary contraction

mRS: modified Rankin Scale

REML: restricted maximum likelihood

RMS: root-mean-square

Declarations

Ethics approval and consent to participate

The study received ethical approval from the South Central–Berkshire Research Ethics Committee (IRAS 19720) and the Imperial College Research Ethics Committee (REC Reference 23/SW/0140, IRAS 330409). All participants gave written informed consent in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable; no individually identifiable participant data are presented.

Availability of data and materials

Aggregate source data underlying the results and figures presented in this article, together with the analysis code, are archived at Zenodo (<https://doi.org/10.5281/zenodo.22231074>). Additional de-identified derived data supporting the reported analyses may be made available to researchers upon reasonable request to the corresponding author, subject to appropriate ethical and institutional data-sharing requirements. Participant-level raw HD-EMG recordings are not publicly available because these high-dimensional physiological data may retain a risk of participant re-identification.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Rossana Lovecchio and Elisabeth Rounis conceived and designed the study. Elisabeth Rounis, Rossana Lovecchio, and Nga Man Ting led data acquisition, with Pranav Mamidanna, Matthew Banger and Alison McGregor contributing to data acquisition and experimental support. Rossana Lovecchio, Elisabeth Rounis, Dario Farina, and Pranav Mamidanna led data processing, analysis, validation, and interpretation, with Pasquale Ferrentino and Felicia Tassone contributing to data processing and validation. Paul Bentley, Fatemeh Geranmayeh, and Vishal Rawji provided clinical, ethical, and regulatory support. Elisabeth Rounis, Dario Farina, Matthew A. Lambon Ralph, Tom Verstraten, and Bart Jansen contributed to funding acquisition and supervision. Rossana Lovecchio drafted the manuscript with Elisabeth Rounis. All authors contributed to writing – review and editing, approved the final version, and agree to be accountable for the work.

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Figures

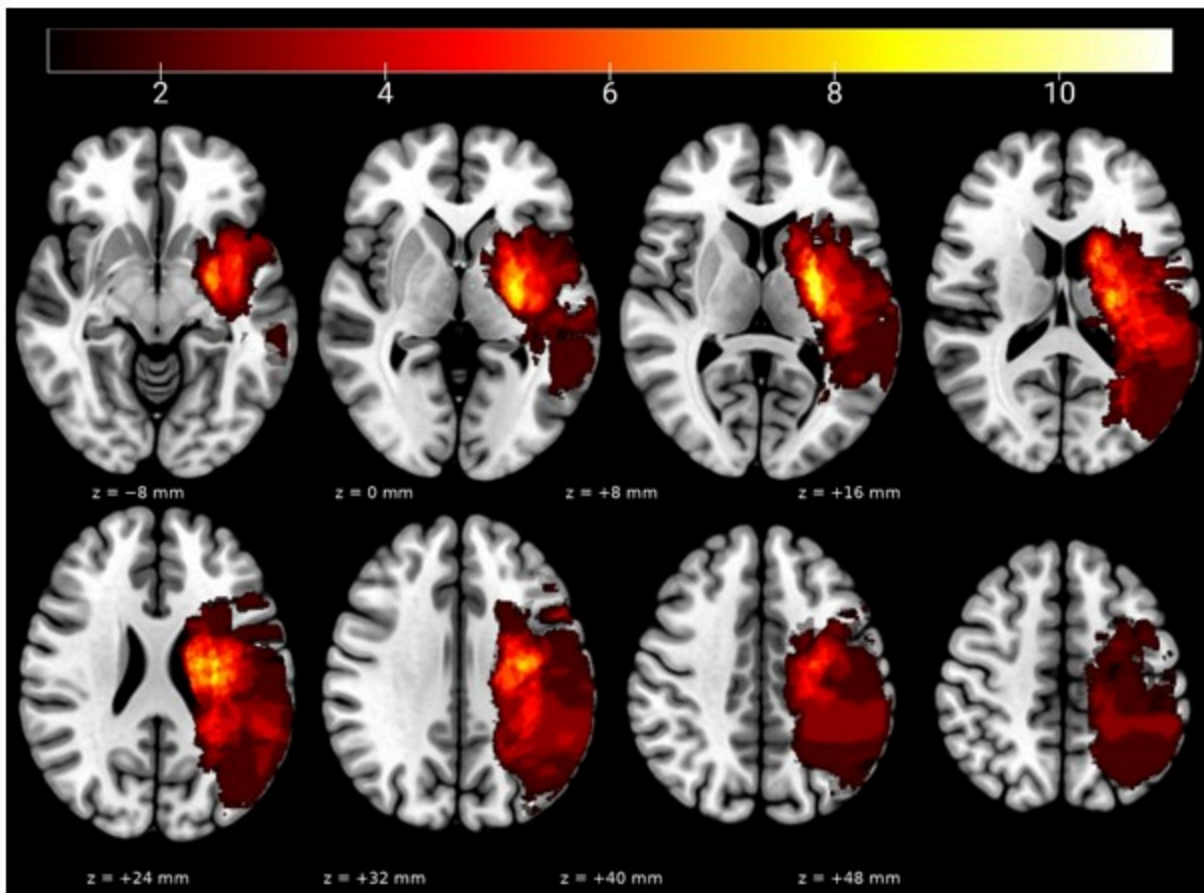


Figure 1

Lesion distribution in the stroke cohort.

Voxelwise overlap of lesion masks from the 18 participants with left-hemisphere stroke included in the physiological analyses (11 apraxia-dominant and 7 paretic), displayed on the MNI152 template. Axial slices are shown at $z = -8, 0, +8, +16, +24, +32, +40$, and $+48$ mm. Colour indicates the number of patients with a lesion at each voxel, with warmer/lighter colours indicating greater overlap and a maximum overlap of 11 participants.

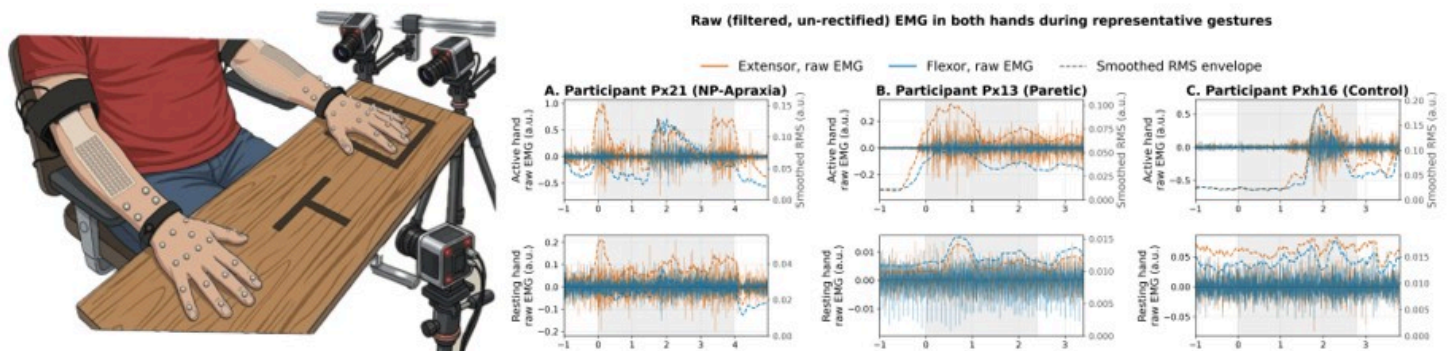


Figure 2

Experimental setup. Group labels: Control, healthy volunteers; NPA, apraxia-dominant stroke group with relatively preserved strength (analysis label); Paretic, paretic-dominant stroke group.

Participants performed a 49-gesture battery with the active hand while keeping the resting hand still on the table. Bilateral forearm muscle activity was recorded using four 6×16 high-density surface EMG grids (two per forearm), simultaneously with upper-limb kinematics from 22 VICON optical markers. The setup enabled co-registration of resting-hand EMG coupling events with motion-capture data to assess whether detected activation crossed the threshold for overt movement. Representative single-repetition traces (one HD-sEMG channel per muscle group, bandpass-filtered 20–450 Hz, notch-filtered at 50 Hz, un-rectified) for the active hand (top row) and resting hand (bottom row) during an Imitation gesture, in one participant per group: (A) Px21, apraxia-dominant; (B) Px13, Paretic; (C) Pxx16, Control. Grey shading marks the labelled gesture window. Dashed lines show the smoothed RMS envelope (right axis) from which all coupling statistics are derived. Peak cross-correlation and composite bilateral coupling intensity (resting-hand RMS × peak cross-correlation) for the depicted repetition are reported below each column.

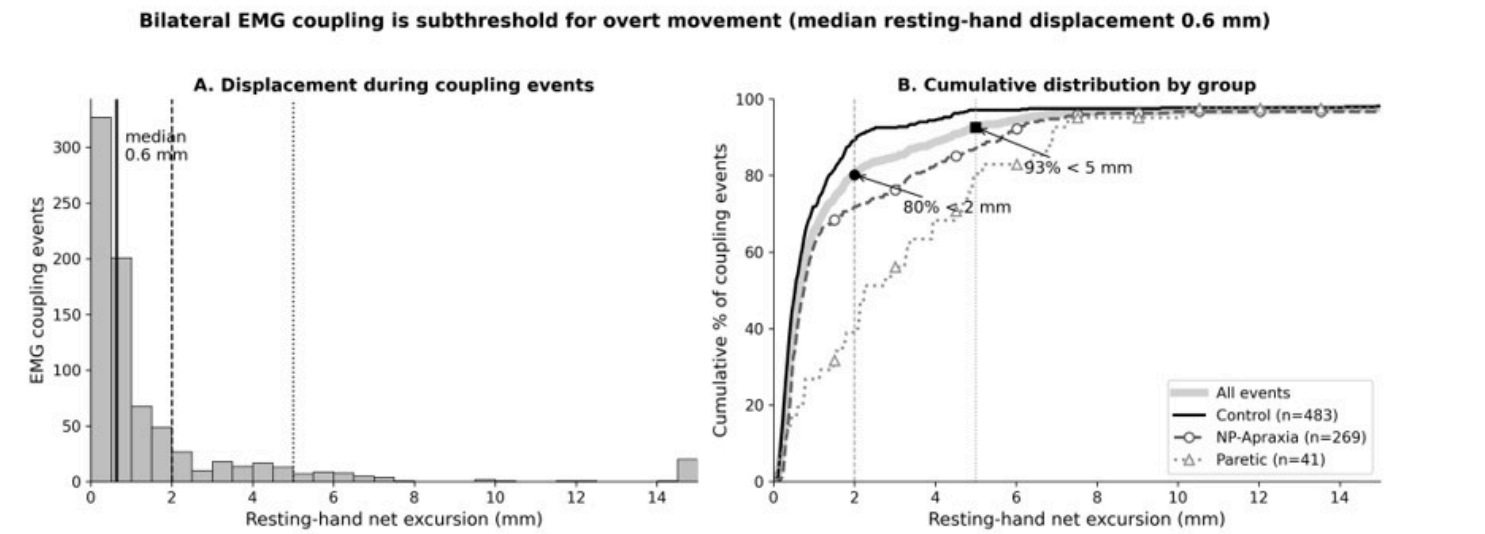


Figure 3

Bilateral EMG coupling is subthreshold for overt movement. Group labels: Control, healthy volunteers; NPA, apraxia-dominant stroke group with relatively preserved strength (analysis label); Paretic, paretic-dominant stroke group.

(A) Distribution of resting-hand net wrist excursion (mm) during all EMG-detected coupling events. Median displacement 0.6 mm; 80% of events fall below 2 mm. (B) Cumulative distribution of resting-hand displacement by group; 80% of events show displacement below 2 mm, and 93% below 5 mm.

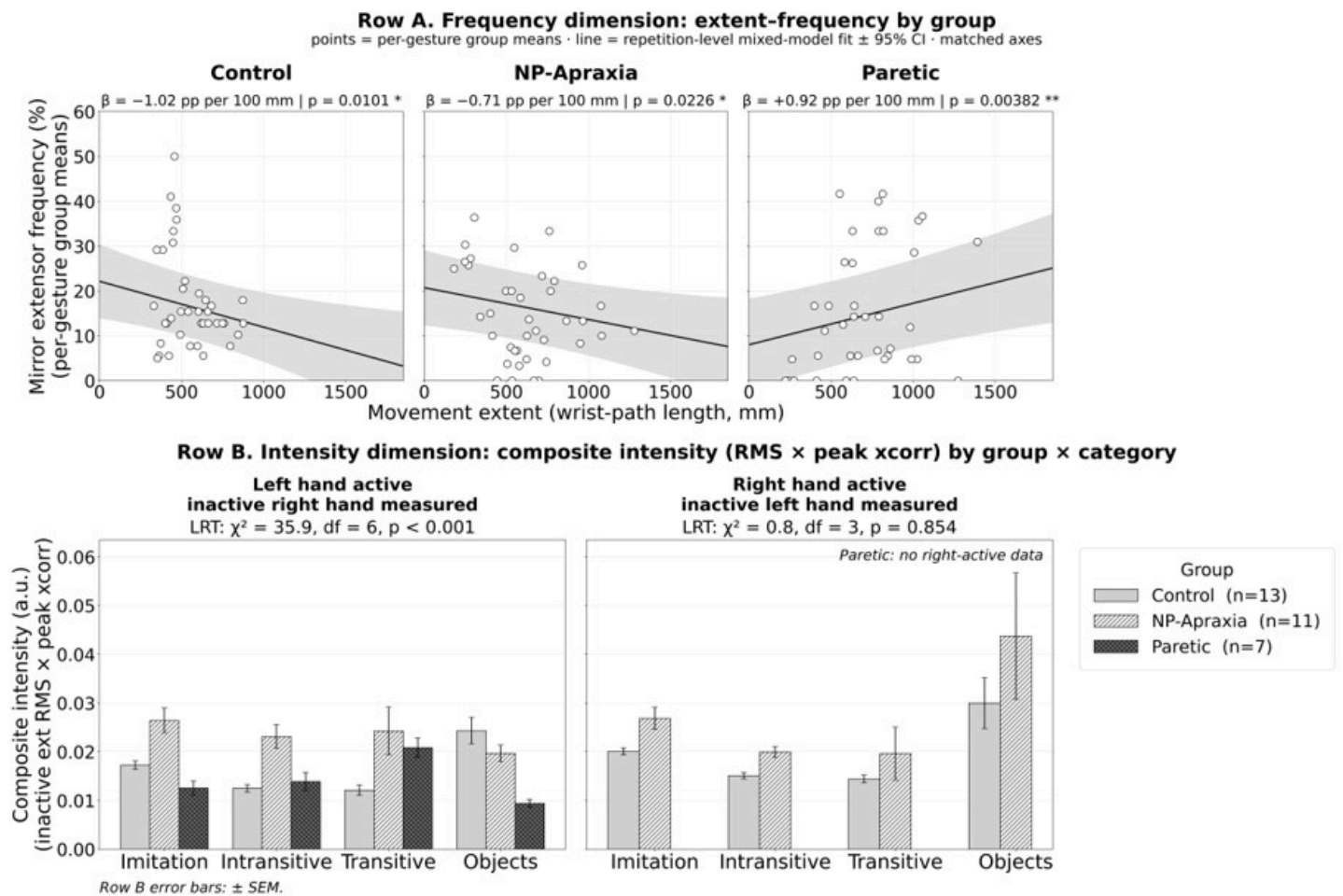


Figure 4

The coupling rate/intensity double dissociation. Group labels:Control, healthy volunteers; NPA, apraxia-dominant stroke group with relatively preserved strength (analysis label); Paretic, paretic-dominant stroke group.

Row A. Coupling rate dimension. Per-gesture group-mean extensor bilateral-coupling rate versus VICON wrist-path length (mm). Fitted lines are predictions from the repetition-level linear probability mixed model with random intercepts for participant and gesture (shaded band = 95% CI); points are per-gesture group means. Controls ($\beta = -1.02$ pp per 100 mm, 95% CI $[-1.80, -0.24]$, $P = 0.010$) and NPA ($\beta = -0.71$ pp per 100 mm, 95% CI $[-1.32, -0.10]$, $P = 0.023$) show negative slopes of similar magnitude; Paretic reverses to positive ($\beta = +0.92$ pp per 100 mm, 95% CI $[+0.30, +1.55]$, $P = 0.004$; group $\times$ extent $\chi^2 = 19.8$, $df = 2$, $P = 4.9 \times 10^{-5}$). The sign reversal was specific to the Paretic group. Row B. Intensity dimension. Composite intensity (inactive-hand max RMS $\times$ peak cross-correlation; extensor channel) by group $\times$ gesture category. Left panel: left hand active (inactive right hand measured); omnibus group $\times$ category LRT $\chi^2 = 35.9$, $P < 0.0001$. Controls show affordance modulation (Objects amplitude 1.38 $\times$, 95% CI $[1.20 \times, 1.58 \times]$, $P < 0.001$); NPA shows uniformly elevated intensity across all categories (internally-generated vs Objects: $P = 0.29$); Paretic shows uniformly low intensity with Objects reduction ($P = 0.004$). Individual data points overlaid. Right panel: right hand active (inactive left hand measured); paretic group

contributed no right-hand-active trials. The difference between panels is supported by a group $\times$ category $\times$ active-hand interaction ($\chi^2 = 12.75$, $df = 3$, $P = 0.005$; post-hoc).

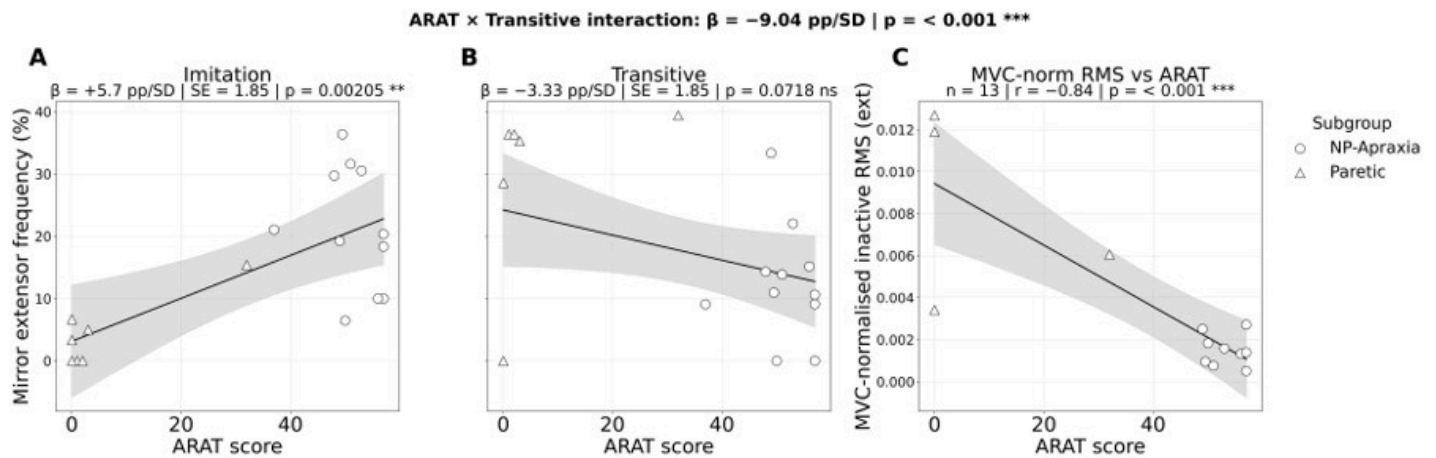


Figure 5

Clinical arm function relates to bilateral coupling rate. Group labels: Control, healthy volunteers; NPA, apraxia-dominant stroke group with relatively preserved strength (analysis label); Paretic, paretic-dominant stroke group.

(A) Imitation: ARAT slope positive ($\beta = +5.7$ pp/SD, 95% CI [+2.08, +9.33], $P = 0.002$).

(B) Transitive: ARAT slope negative but non-significant ($\beta = -3.33$ pp/SD, 95% CI [-6.96, +0.30], $P = 0.072$); ARAT $\times$ Transitive interaction $\beta = -9.04$ pp/SD, 95% CI [-13.55, -4.52], $P < 0.001$. (Slopes are per SD of ARAT [z-scored]; x-axes are shown in raw ARAT score for readability – same fit, relabelled axis.)

(C) MVC-normalised resting-hand RMS versus ARAT ($r = -0.84$, 95% CI [-0.95, -0.53], $P < 0.001$, $n = 13$).