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Upper limb motor dysfunction is associated with fragmented kinetics after brain injury

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ABSTRACT

Background: Characterization of motor deficits after brain injury is important for rehabilitation personalization. While studies reported abnormalities in the kinematics of paretic and non-paretic elbow extension for patients with brain injuries, kinematic analysis is not sufficient to explore how patients deal with musculoskeletal redundancy and the energetic aspect of movement execution. Conversely, interarticular coordination and movement kinetics can reflect patients' motor strategies. This study investigates motor strategies of paretic and non-paretic upper limb after brain injury to highlight motor deficits or compensation strategies.

Methods: 26 brain-injured hemiplegic patients and 24 healthy controls performed active elbow extensions in the horizontal plane, with both upper limbs for patients and, with the dominant upper limb for controls. Elbow and shoulder kinematics, interarticular coordination, net joint kinetics were quantified.

Findings: Results show alterations in kinematics, and a strong correlation between elbow and shoulder angles, as well as time to reach elbow and shoulder peak angular velocity in both upper limbs of patients. Net joint kinetics were lower for paretic limb and highlighted a fragmented motor strategy with increased number of transitions between concentric and eccentric phases.

Interpretation: In complement to kinematic results, our kinetic results confirmed patients' difficulties to manage both spatially and temporally the joint degrees of freedom redundancy but revealed a fragmented compensatory motor strategy allowing patients upper limb extension despite quality alteration and decrease in energy efficiency. Motor rehabilitation should improve the management of this fragmentation strategy to improve the performance and the efficiency of active movement after brain injury.

1. Introduction

Brain injury is a major cause of motor disorders around the world (Hattem et al., 2016). A common deficit observed after brain injury is hemiparesis of the contralesional upper limb (Cramer et al., 1997). While motor recovery can allow patients to regain autonomy in the activities of daily living (Thrane et al., 2020), around 30% of patients only recover reduced mobility of their contralesional upper limbs (Nakayma et al., 1994).

In the activities of daily living, elbow extension is an essential movement, even for simple gripping tasks such as lifting a glass, tying shoelaces, etc. Many studies reported abnormalities in the kinematics of paretic elbow extension for brain-injured patients when compared to that of healthy subjects: the movement takes longer and presents smaller amplitude and lower smoothness (Thrane et al., 2020; van Dokkum et al., 2014), while the magnitude of elbow peak angular velocity is lower and is reached earlier, increasing the deceleration phase (Murphy and Häger, 2015). Noteworthy is that some of these deficits are also

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found in the non-paretic ipsilesional upper limb, but are less obvious (Bustrén et al., 2017). Ipsilesional deficits can be explained by the fact that approximately 30% of the cortico-spinal tract fibers descend on the ipsilateral side and that there are functional connections between the contralesional and ipsilesional cerebral hemispheres (De Paiva Silva et al., 2018).

While it is undeniable that kinematic analysis provides important information to understand the extent and nature of patients' motor deficits, it does not fully allow to investigate adaptations of the underlying motor strategies, i.e., how joint and muscle redundancies are managed (Buma et al., 2016; Murphy and Häger, 2015; Thrane et al., 2020). Conversely, the net torque reflects the collective behavior of the muscles (McCrea et al., 2003) chosen by each subject to solve the motor problem (Centomo et al., 2007; Winter and Eng, 1995). In addition to kinematic variables, kinetic analyses are therefore mandatory to investigate adaptation of motor strategies. The net extension torque at the elbow may be reduced in the paretic and non-paretic limbs after brain injury, due to lack of agonist muscle activation (Gowland et al., 1992), excessive agonist/antagonist co-contraction (el-Abd et al., 1993), increase in the sensitivity of the myotatic reflex (el-Abd et al., 1993) or modification of muscle properties (Gray et al., 2011). However, previous studies investigated the net joint torque alteration in brain-injured patients only for isometric (Ellis et al., 2005; McCrea et al., 2003; Riley and Bilodeau, 2002) or isokinetic contractions (Lum et al., 2004), or

constrained movements in an apparatus (Beer et al., 2000), thus offering somewhat limited insights into the motor control strategies leading to changes in inter-articular coordination between the shoulder and the elbow (Cirstea et al., 2003a; Murphy and Häger, 2015) and in energy efficiency of paretic and nonparetic upper limb movements.

The present study aims at assessing and investigating motor strategies of the paretic and non-paretic upper limb after brain injury, with the clinical goal to better tune patients' rehabilitation. We quantified the kinematics, net elbow and shoulder torques and the associated powers during an elbow extension movement at spontaneous speed. In line with previous results, we expected brain-injured patients to produce less ample and less smooth extension movements of both upper limbs, with lower and earlier peak velocity, in comparison to movements performed by healthy subjects with their dominant upper limb. We hypothesized that these kinematic alterations may be associated with increased fragmented kinetics, i.e., lower energy efficiency characterized by an increased number of transitions between concentric and eccentric power phases.

2. Methods

2.1. Participants

Twenty-six hemiparetic brain injured patients in the chronic phase (more than six months after brain injury) and twenty-four healthy

Table 1
Participant's description.

Subject	Sex	Age (years)	Time Since brain injury (months)	Fugl-Meyer /66	Tardieu scale score /4	Type of brain injury	Location of brain injury
1	M	57	14	45	2	Ischemic stroke	Left, posterior limb of the internal capsule
2	M	75	26	26	2	Ischemic stroke	Left, cortical, and subcortical territories of MCA
3	M	48	8	49	2	Ischemic stroke	Left, cortical, and subcortical territories of MCA
4	M	65	116	30	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
5	M	49	13	53	2	Ischemic stroke	Predominant right, Pons (paramedian)
6	F	33	9	45	2	Ischemic stroke	Predominant left, Pons, and middle cerebellar peduncles
7	M	61	28	60	2	Ischemic stroke	Predominant right, Medulla oblongata
8	M	57	18	41	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
9	M	56	34	29	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
10	M	76	12	50	2	Ischemic stroke	Left, subcortical territories of MCA.
11	M	74	34	23	2	Ischemic stroke	Right, Pons (paramedian)
12	M	67	6	47	2	Ischemic stroke	Left, internal capsule
13	M	43	15	46	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
14	M	72	43	36	2	Hemorrhagic stroke	Right, thalamus
15	M	41	15	58	0	Ischemic stroke	Right, subcortical territories of MCA
16	M	52	36	41	2	Ischemic stroke	Left, internal capsule
17	M	54	12	27	2	Hemorrhagic stroke	Right, internal capsule and thalamus
18	F	39	52	63	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
19	M	71	244	25	2	Cerebral angioma	Left
20	M	61	51	38	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
21	M	59	18	46	2	Hemorrhagic stroke	Right, subcortical territories of MCA
22	F	69	19	44	2	Ischemic stroke	Right, Pons (paramedian)
23	M	65	75	32	2	Hemorrhagic stroke	Left, basal ganglia
24	M	50	30	42	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
25	M	54	27	47	2	Ischemic stroke	Right, cortical and subcortical territories of MCA
26	F	50	122	32	2	Hemorrhagic stroke	Left, cortical, and subcortical territories of MCA
Patients	22 M/4F	58 ± 9	41 ± 32	41 ± 9	96% of 2 4% of 0		
Controls	10 M/ 14F	51 ± 14	/	/			

subjects participated in this study (see Table 1 for detailed participants' demographics). To be included, patients had to present a functional inconvenience during elbow extension linked to hyperactivity of the flexor muscles. Their active elbow extension amplitude had to be at least 20° but be limited by at least 15°. Participants had no elbow pain or cognitive impairment limiting the understanding of instructions, or progressive neurological pathology. Six patients and nine healthy subjects were included in a study approved by the Research Ethical Committee of Toulouse University Hospitals (No. 07–0716). Twenty patients were included in a routine care protocol (No. ID-RCB: 2017-A01616–47) and fifteen healthy subjects in an interventional protocol (No. ID-RCB: 2017-A01616–47). All volunteers gave written informed consent prior to participation and all procedures complied with the Declaration of Helsinki.

2.2. Protocol

The data used in the present study is part of the database of a research project aim at evaluating the effect of botulinum toxin injections into the elbow flexors on the reduction of spastic co-contractions during an elbow extension movement in brain-injured patients (Chalard et al., 2020; Delcamp et al., 2022). During the protocol, the patients received botulinum toxin injections in at least one of the main elbow flexors (i.e., biceps brachii, brachialis or brachioradialis), with the main functional goal to improve the reaching movement. The analysis performed in the present study relies on the data collected during the first session of this protocol, before botulinum toxin injection. The experimental procedure included ten active self-paced full elbow extensions for the paretic (PAR) and non-paretic (N-PAR) upper limbs of patients and the dominant upper limb (DOM) of healthy subjects. As an initial position, participants were seated in a chair with their back secured to the backrest, shoulder flexed at 80° with 90° internal rotation, elbow flexed at 90°, and forearm pronated. During the extension movement, the participant's upper limb was kept at a consistent distance from the table to achieve in-plane movement. Between each extension movement, the participant had a rest period ranging from eight to fifteen seconds. Kinematic data of reflective markers placed on the acromion, lateral epicondyle, ulnar styloid and 2nd metacarpus of both sides of the body were recorded at 125 Hz (system OptiTrack; NaturalPoint Inc., Corvallis, OR, USA).

2.3. Data analysis

All analyses were conducted with Matlab (R2019b, 9.7.0, The Mathworks Inc., Natick, MA).

Preprocessing: Prior to any other processing, the raw continuous kinematic data were filtered using a low-pass Butterworth filter (0-lag, 4th order) at the cut-off frequency of 6 Hz (Cahouët et al., 2002).

Kinematic analysis: For each movement, joint angle was computed in the horizontal plane from the Cartesian coordinates of the anatomical landmarks, using the sign convention that flexion direction is positive (Fig. 1). Spline functions adjusted to changes in joint angles were determined using the 'csaps' function in MATLAB, then derived once for velocity and a twice for acceleration for each trial. The joint angle, velocity and acceleration were normalized to hundred data points and averaged over the ten trials. As recommended by Rohrer et al. (2002), movement smoothness was quantified as the number of peaks in the acceleration profile, normalized by the mean angular velocity. The time to peak velocity was defined as the percentage of movement where maximal peak velocity occurs.

Kinetic analysis: The net joint torques at the shoulder (Ts), elbow (Te) and wrist (Tw) were calculated by solving the problem of inverse dynamics using the Lagrange formalism. As in Amarantini and Martin (2004) and Hufenus et al. (2006), we define the resultant torque vector T_{inv} (T_s, T_e, T_w) by (1)

$$T_{inv} = A(\theta) \cdot \ddot{\theta} + B(\theta) \cdot \dot{\theta} + C(\theta) \cdot \dot{\theta}^2 \quad (1)$$

Where $\theta = \langle \theta_s, \theta_e, \theta_w \rangle^T$ is the angular position vector, $\dot{\theta} = \langle \dot{\theta}_s, \dot{\theta}_e, \dot{\theta}_w \rangle^T$ et $\ddot{\theta} = \langle \ddot{\theta}_s, \ddot{\theta}_e, \ddot{\theta}_w \rangle^T$ are angular velocity vectors and $\ddot{\theta} = \langle \ddot{\theta}_s, \ddot{\theta}_e, \ddot{\theta}_w \rangle^T$ is angular acceleration vector; and with A corresponding to the matrix of the inertial component, B for the Coriolis component and C for the centrifugal component (see detailed equations in Supplementary Materials). Net joint power was computed at each joint as the scalar product of angular velocity and net joint torque (Centomo et al., 2007). A negative power value indicates that the net torque acts in the opposite direction of the angular velocity allowing to infer the dominant contraction of eccentric muscles absorbing energy; a positive power value indicates dominant contraction of concentric muscles to generate energy (Winter and Eng, 1995). For comparison between subjects, joint torque and power values were normalized by body mass. Movement fragmentation was defined as the number of transitions between concentric and eccentric phases.

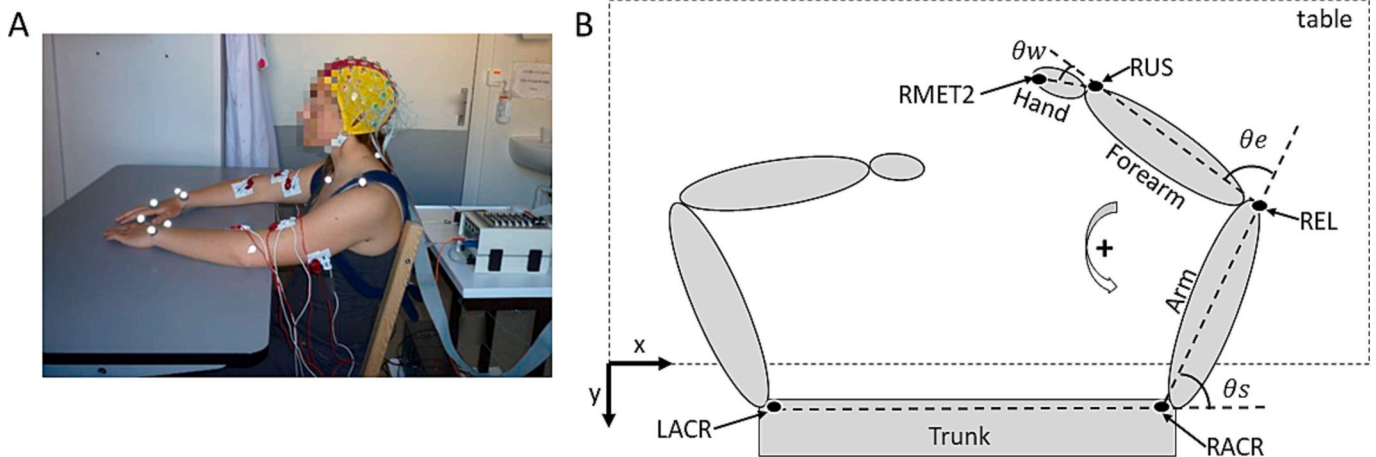


Fig. 1. Experimental set-up photograph (A) and schematic representation of the protocol (B). The elbow extensions were realized in the horizontal plane and were modelled in two dimensions (X vs. Y). The black circles show the position of the reflective markers: LACR for left acromion, RACR for right acromion, REL for right lateral epicondyle, RUS for right ulnar styloid and RMET2 for right 2nd metacarpus. On the left side, markers are placed symmetrically but are not represented here. Shoulder (θ_s), elbow (θ_e) and wrist (θ_w) angles were calculated in the horizontal plane.

2.4. Statistical analysis

Elbow and shoulder were the joints subjected to statistical analysis.

Student's *t*-tests were conducted to assess the differences in time to peak velocity between PAR and N-PAR with DOM. Additionally, equivalent non-parametric Mann-Whitney tests were employed to compare maximal angular velocity, acceleration, deceleration, movement smoothness, movement fragmentation, maximal net joint torque, and power between PAR and N-PAR with DOM. Adjusted *p*-values for multiple comparisons were calculated using the procedure proposed by Benjamini and Hochberg (1995). To complete the information provided by the *P*-values, the effect sizes (ES) and their 95% confidence interval were calculated for each comparison (Cumming, 2014).

To consider the temporal aspect of motor strategies and to limit the risk of misinterpretation due to the fact that statistical analysis of discrete data from continuous variables over time significantly increases the risk of false positives (Pataky et al., 2016), we also analyzed the variations of kinematic and kinetic data over the entire extension movement using statistical non-parametric mapping (SnPM). A SnPM two-tailed nonparametric independent *t*-test was used to compare kinematic and kinetic data variables of both elbow and shoulder of PAR and N-PAR with DOM as a function of time. The scalar output statistic, $\text{SnPM}\{t\}$, was calculated separately at each individual time node. To test our null hypothesis, we calculated the critical threshold that only 5% of smooth random curves would exceed (Adler and Taylor, 2007). SnPM uses the supra-threshold cluster size to calculate cluster *P*-values (Pataky, 2016). All SnPM analyses were implemented using the open source *spm1d* code (v.0.4.8, www.spm1d.org) in Matlab.

The significance threshold was set at 0.05 for all statistical analysis. Considering the small sample size of our study and independently of the *p*-values, we considered an effect size with confidence interval not including zero as a meaningful difference (Cumming, 2014).

3. Results

Only the significant results are presented above.

3.1. Alteration in movement execution (Fig. 2, Table 2)

Mean elbow angle was lower for PAR than for DOM at 6–45% of extension movement time, and higher at 71–100% of the extension movement time ($P < 0.001$ for both clusters).

Mean shoulder angle was lower for PAR than for DOM at 72–96% of the extension movement time ($P = 0.02$). Furthermore, mean shoulder angle was lower for N-PAR than for DOM for the entire duration of the extension movement ($P = 0.003$).

Mean elbow velocity was lower for PAR than for DOM at 3–7% ($P = 0.01$) and 25–93% ($P < 0.001$) of the extension movement time with an earlier peak velocity. Mean elbow angular peak velocity was 51.57% lower for PAR than DOM with a large effect size and occurred at 30.29% of the extension movement time for PAR versus 56.20% for DOM. The peak velocity is also earlier for N-PAR at 47.71% of the extension movement time.

Mean shoulder velocity was lower for PAR than for DOM at 87–100% ($P < 0.001$) of the extension movement time. The evolution of this velocity over time for PAR shows small amplitudes of variation compared to DOM. Mean shoulder angular peak velocity for PAR was earlier than for the DOM with a large effect size and occurred at 36.60% of the extension movement time for PAR versus 52.54% for DOM.

The elbow movement smoothness was 89.26% lower for PAR compared to DOM, with a large effect size. Likewise, the shoulder movement smoothness was 65.05% lower for PAR than for DOM, with a large effect size.

3.2. Correlations

Correlations between elbow and shoulder angles were all significant ($\text{Tau} = 1$ $P < 0.001$ for both patients' upper limbs; $\text{Tau} = 0.858$ $P < 0.001$ for healthy subjects) and greater for patients than for healthy subjects ($z > 18.4$ $P < 0.001$). Correlations between the times to peak velocity at the elbow and shoulder were significant only for PAR ($r = 0.438$ $P = 0.025$) and N-PAR ($r = 0.565$ $P = 0.003$) (Table 3).

3.3. Kinetics (Fig. 3, Table 2)

Mean net elbow torque was lower for PAR than for DOM at 6–43% ($P < 0.001$) and 73–98% ($P < 0.001$) of the extension movement time. The evolution of net elbow torque over time for PAR shows small amplitudes of variation, compared to DOM which presents a biphasic profile with well-marked positive phase and negative phase. In addition, the maximum negative net elbow torque was 14.28% lower for PAR compared to DOM with a large effect size.

Mean net shoulder torque was lower for PAR than for DOM at 19–59% ($P < 0.001$) and 77–98% ($P < 0.001$) of the extension movement time. The evolution of net shoulder torque over time for PAR shows small amplitudes of variation, compared to DOM. Moreover, the maximum positive net shoulder torque was 37.04% lower for PAR compared to DOM with a large effect size. The maximum positive net shoulder torque was 25.93% for N-PAR with a large effect size.

Mean net elbow power was lower for PAR than for DOM at 10–32% ($P < 0.001$) and 76–93% ($P < 0.001$) of the extension movement time. The evolution of net elbow power over time for PAR shows small amplitudes of variation, compared to DOM which presents a biphasic profile with well-marked positive phase and negative phase. In addition, the maximum positive net elbow power was 42.86% lower, and the maximum negative net elbow power was 57.14% lower for PAR compared to DOM with a large effect size.

Mean net shoulder power was lower for PAR than for DOM at 10% ($P = 0.02$), 11–15% ($P = 0.01$), 16–18% ($P = 0.02$), 37–40% ($P = 0.02$) and 84–96% ($P < 0.001$) of the extension movement time. The maximum negative net shoulder power was 50% lower for N-PAR with a large effect size.

The “movement fragmentation”, i.e., the number of power alternations at the elbow, was 68.78% higher with a large effect size for PAR compared to DOM. Likewise, the movement was 63.27% more fragmented for the PAR shoulder than for the DOM shoulder with a large effect size.

4. Discussion

This study investigated the motor strategies used during in-plane elbow extension movements after brain injury for both paretic and non-paretic patients' upper limbs. To this aim, we compared upper limbs' kinematics of brain-injured patients to those of healthy subjects. Our results suggest that kinematic alterations may be associated with fragmented kinetics in brain-injured patients.

4.1. Motor impairment

Our results highlight a decrease in elbow angular range of motion and a lower and earlier elbow angular peak velocity for PAR than for DOM, together with a longer deceleration phase. These results are in agreement with previous findings on elbow kinematics after brain injury (Fauvet et al., 2021; Murphy and Häger, 2015; Thrane et al., 2020). These deficits can be attributed to a difficulty in reaching a peak of adequate velocity linked to sensorimotor impairment and greater reliance on feedback in a pointing and grasping task (Thrane et al., 2020). However, in the absence of target to reach in the present study, the feedback was less important in the movement realization and the kinematics alteration could be related to the lower production of initial

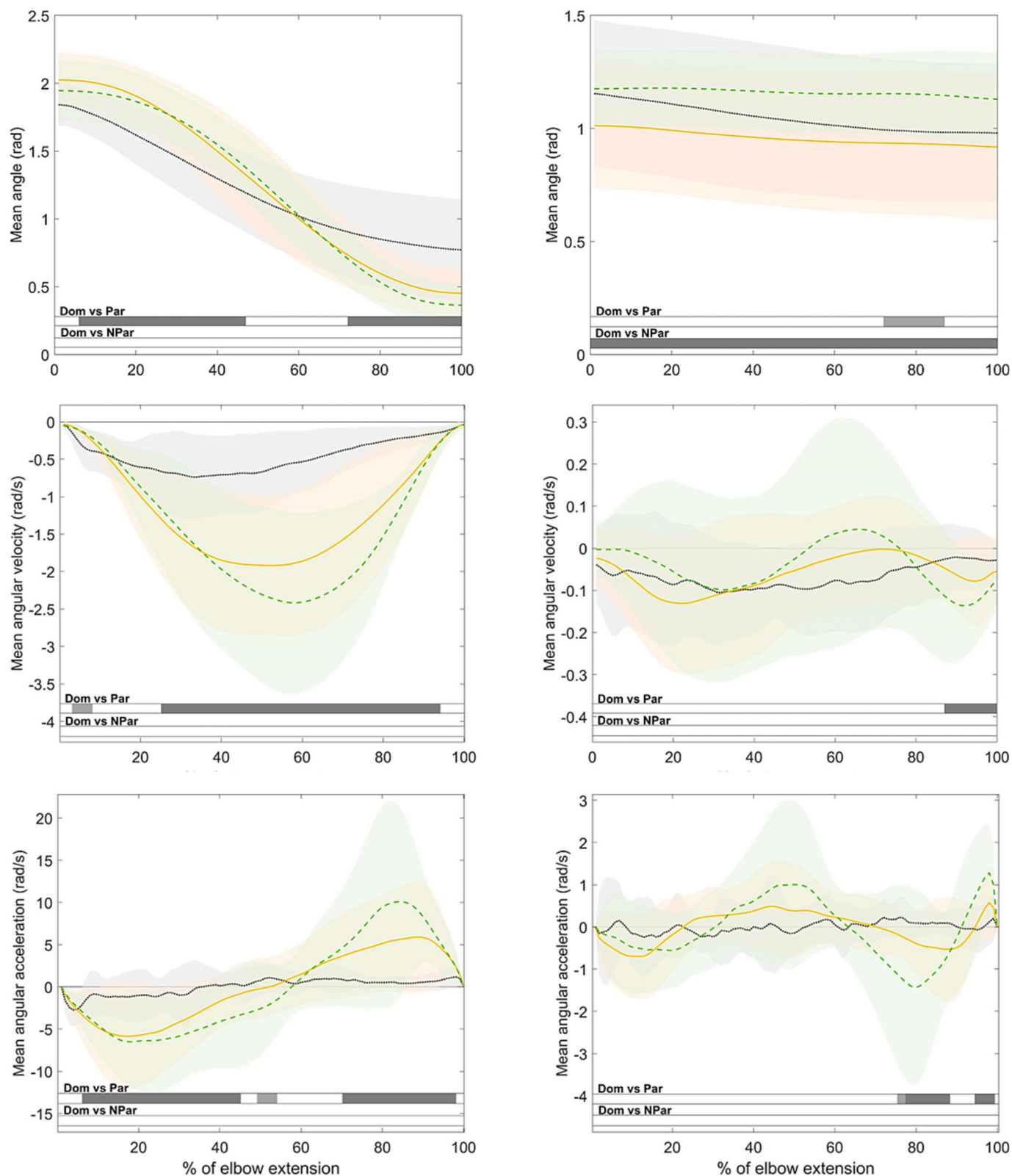


Fig. 2. Kinematic of parietic and non-parietic patients' upper limbs and dominant healthy subjects' upper limb. Mean and standard deviation of angle (first row), angular velocity (second row) and angular acceleration (third row) at the elbow (right column) and at the shoulder (left column) for parietic upper limb (in grey dotted line) and non-parietic upper limb (in orange solid line) of brain injured patients and for dominant upper limb (in green dashed line) of healthy subjects. Significant cluster p -value of two-tailed nonparametric independent t -test between both patients' upper limbs and healthy subjects' dominant upper limb are represented by grey bars on the bottom of each figure. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 2

Results of OD statistical analysis. Student t-test (t) or Mann Whitney's test (U) compared kinematic and kinetic data of PAR and N-PAR of patients with DOM of healthy subjects.

Variables	Comparisons	Values	ES	P-value	B-H critical P-value
Maximal angular velocity (rad.s ⁻¹)	Par vs Dom elbow	-1.45 vs -2.99	0.82* [0.69:0.90]	<0.001* (U)	0.005
	Par vs Dom shoulder	-0.18 vs -0.23	0.19 [-0.14:0.47]	0.27 (U)	0.043
	N-Par vs Dom elbow	-2.64 vs -2.99	0.19 [-0.13:0.48]	0.25 (U)	0.041
	N-Par vs Dom shoulder	-0.17 vs -0.23	0.25 [-0.07:0.52]	0.13 (U)	0.034
Time to peak velocity (% of extension)	Par vs Dom elbow	30.29 vs 56.20	1.95* [1.17:2.71]	<0.001* (t)	0.005
	Par vs Dom shoulder	36.60 vs 52.54	1.03* [0.40:1.65]	<0.001* (t)	0.005
	N-Par vs Dom elbow	47.71 vs 56.20	0.82* [0.23:1.39]	0.005* (t)	0.014
	N-Par vs Dom shoulder	45.98 vs 52.54	0.40 [-0.17:0.95]	0.16 (t)	0.035
Maximal negative angular acceleration (rad.s ⁻²)	Par vs Dom elbow	-9.16 vs -12.35	0.42* [0.13:0.65]	0.01* (U)	0.016
	Par vs Dom shoulder	-3.16 vs -3.83	0.16 [-0.16:0.45]	0.35 (U)	0.044
	N-Par vs Dom elbow	-10.49 vs -12.35	0.26 [-0.05:0.53]	0.11 (U)	0.030
	N-Par vs Dom shoulder	-2.80 vs -3.83	0.26 [-0.06:0.53]	0.12 (U)	0.032
Movement smoothness	Par vs Dom elbow	0.96 vs 0.10	-0.90* [-0.95: -0.82]	<0.001* (U)	0.005
	Par vs Dom shoulder	13.90 vs 5.14	-0.45* [-0.67: -0.16]	0.006* (U)	0.015
	N-Par vs Dom elbow	0.11 vs 0.10	-0.11 [-0.41:0.21]	0.53 (U)	0.048
	N-Par vs Dom shoulder	7.12 vs 5.14	-0.21 [-0.49:0.11]	0.20 (U)	0.036
Maximal positive angular acceleration (rad.s ⁻²)	Par vs Dom elbow	8.24 vs 16.83	0.61* [0.36:0.77]	<0.001* (U)	0.005
	Par vs Dom shoulder	3.13 vs 3.39	0.14 [-0.18:0.43]	0.40 (U)	0.046
	N-Par vs Dom elbow	12.18 vs 16.83	0.31* [0.002:0.57]	0.06 (U)	0.024
	N-Par vs Dom shoulder	2.35 vs 3.39	0.38* [0.07:0.62]	0.02 (U)	0.018
Maximal negative net joint torque (Nm.kg ⁻¹)	Par vs Dom elbow	-0.012 vs -0.014	0.34* [0.03:0.59]	0.04 (U)	0.021
	Par vs Dom shoulder	-0.017 vs -0.016	0.21 [-0.11:0.49]	0.21 (U)	0.038
	N-Par vs Dom elbow	-0.013 vs -0.014	0.21 [-0.11:0.49]	0.21 (U)	0.038
	N-Par vs Dom shoulder	-0.014 vs -0.016	0.28 [-0.04:0.54]	0.10 (U)	0.028
Maximal positive net joint torque (Nm.kg ⁻¹)	Par vs Dom elbow	0.011 vs 0.015	0.29 [-0.03:0.55]	0.09 (U)	0.026
	Par vs Dom shoulder	0.017 vs 0.027	0.49* [0.21:0.70]	0.003* (U)	0.013
	N-Par vs Dom elbow	0.012 vs 0.015	0.28 [-0.04:0.54]	0.10 (U)	0.028
	N-Par vs Dom shoulder	0.020 vs 0.027	0.35* [0.04:0.59]	0.04 (U)	0.021
Maximal negative net joint power (W.kg ⁻¹)	Par vs Dom elbow	-0.01 vs -0.03	0.53* [0.26:0.72]	0.001* (U)	0.01
	Par vs Dom shoulder	-0.006 vs -0.008	0.31 [-0.002:0.57]	0.06 (U)	0.024
	N-Par vs Dom elbow	-0.02 vs -0.03	0.19 [-0.13:0.48]	0.25 (U)	0.041
	N-Par vs Dom shoulder	-0.004 vs -0.008	0.37* [0.06:0.61]	0.03 (U)	0.019
Maximal positive joint power (W.kg ⁻¹)	Par vs Dom elbow	0.02 vs 0.03	0.51* [0.24:0.71]	0.002* (U)	0.011
	Par vs Dom shoulder	0.004 vs 0.004	0.26 [-0.06:0.53]	0.12 (U)	0.032
	N-Par vs Dom elbow	0.02 vs 0.03	0.15 [-0.17:0.45]	0.36 (U)	0.045
	N-Par vs Dom shoulder	0.004 vs 0.004	0.31 [0.002:0.57]	0.06 (U)	0.024
Movement fragmentation	Par vs Dom elbow	18.58 vs 5.80	-0.89* [-0.94: -0.81]	<0.001* (U)	0.005
	Par vs Dom shoulder	22.11 vs 8.12	-0.89* [-0.94:0.80]	<0.001* (U)	0.005
	N-Par vs Dom elbow	5.69 vs 5.80	-0.05 [-0.36:0.26]	0.75 (U)	0.05
	N-Par vs Dom shoulder	8.00 vs 8.12	-0.08 [-0.38:0.24]	0.65 (U)	0.049

* Represents a significant difference.

Table 3

Correlation coefficients. Correlation coefficients between the elbow and shoulder angles and between the times to reach the peak of velocity for these same joints. The coefficients are followed by their 95% confidence interval between square brackets.

	Correlations between joint angles (Kendall's Tau)	Correlations between the times to peak velocity (Pearson's r)
PAR	1	0.438 [0.061: 0.706]
N-PAR	1	0.565 [0.227: 0.781]
DOM	0.858 [0.778: 0.920]	0.348 [-0.064: 0.659]

effort due to muscle weakness (Murphy and Häger, 2015). Li and Tong (2016) found that the triceps brachii may show a decrease in force production capacity after brain injury. This weaker force production by agonist muscle during elbow extension can be accompanied by a stronger contraction of antagonist muscles which also limits the capacity to carry out a complete extension (el-Abd et al., 1993). These deficits are also found in PAR shoulder, but to a lesser extent due to a less ample displacement of the shoulder in the task studied. Noteworthy is that the angular position at the shoulder on N-PAR side differed from DOM throughout the duration of the movement. In agreement with the literature, which shows changes in sitting posture after brain injury (Boukadida et al., 2015; Taylor et al., 1994), this result reflects a different initial position while the form of the angular trajectory remains similar between N-PAR and DOM shoulders.

4.2. Stiffening of upper limbs

Further, we observed a very strong correlation between the angle at the elbow and the angle at the shoulder for PAR. This result is in line with many studies that have shown a deficit in inter-articular coordination strategies after brain injury (Cirstea et al., 2003a; Levin, 1996; Schwarz et al., 2021; Subramanian et al., 2020; Tomita et al., 2017). Together with the fact that this correlation was weaker for DOM, this finding provides strong evidence that brain-injured patients are no longer able to spatially control each joint individually (Zackowski, 2004). Furthermore, the time to reach angular peak velocity at the elbow and at the shoulder were significantly correlated for PAR but not for DOM. Such results indicate that patients control their paretic upper limb as a rigid system both spatially and temporally (Forestier and Nougier, 1998; Levin, 1996; Tomita et al., 2017), even when the shoulder is lightly loaded. The simultaneous control of elbow and shoulder in this rigid system can be explained by the fact that the

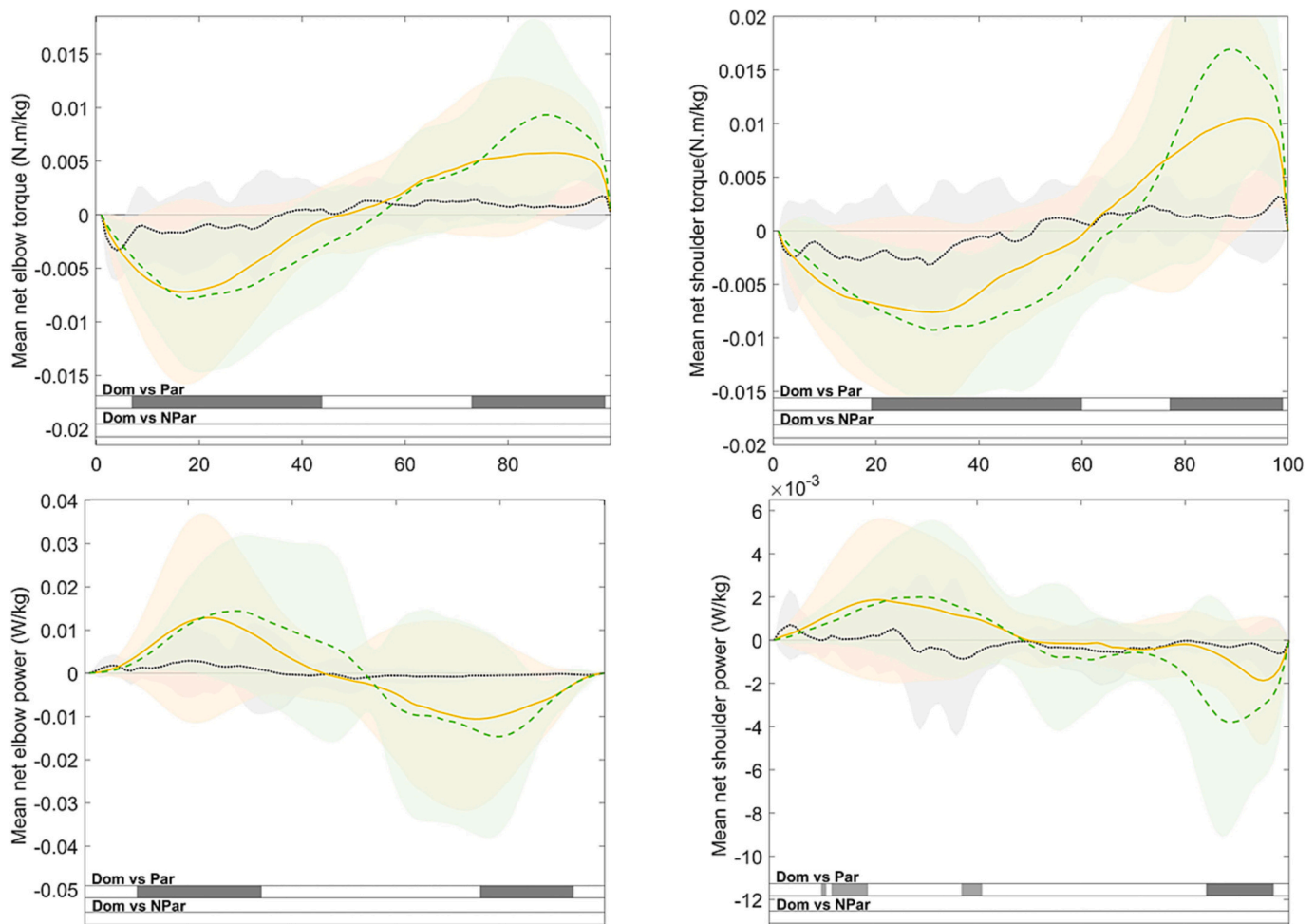


Fig. 3. Kinetic of paretic and non-paretic patients' upper limbs and dominant healthy subjects' upper limb. Mean and standard deviation of net joint torque and net joint power at the elbow (right column) and at the shoulder (left column) for paretic upper limb (in grey dotted line) and non-paretic upper limb (in orange solid line) of brain injured patients and for dominant upper limb (in green dashed line) of healthy subjects. Significant cluster p-value of two-tailed nonparametric independent t-test between both patients' upper limbs and healthy subjects' dominant upper limb are represented by grey bars on the bottom of each figure. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

nervous system controls the limb as a whole (McPherson and Dewald, 2019).

The conclusions for PAR apply to a lesser extent for N-PAR, where the correlation between the elbow angle and the shoulder angle, and the correlation between the time to reach angular peak velocity at the elbow and shoulder were significant. In line with Bustrén et al. (2017), these results suggest that, even if the movement is generally well executed by N-PAR, the motor strategies are altered compared to DOM after brain injury. The patients also present deficits for the N-PAR limb resulting in freezing degrees of freedom. De Paiva Silva et al. (2018) showed that the levels of deficit of PAR and N-PAR are linked, indicating a bilaterally impaired control. The same cortical regions would be at the origin of the development of motor strategies for movements on the ipsilateral and contralateral sides of the lesion. Furthermore, these deficits in N-PAR could also be linked to the spread of nervous drive over the corticospinal tract fibers descending on the ipsilateral side (De Paiva Silva et al., 2018). Noteworthy is that the observed impaired interarticular coordination for N-PAR is concomitant with the lack of significant correlation between spasticity, as assessed using the Tardieu scale, and movement fragmentation for PAR (see Supplementary Materials, suppl. Table 1). Even if we cannot exclude that stiffening of PAR could be attributed to significant spasticity in brain-injured patients, these findings strongly suggest that spasticity alone does not explain this deficit.

4.3. Fragmentation strategy after brain injury

In healthy subjects, elbow joint torque and power profiles depicted a biphasic form with well-marked acceleration and deceleration phases. The SnPM analysis showed significantly different clusters between PAR and DOM at the start and at the end of the movement: this finding highlights that the acceleration and deceleration phases are fragmented for PAR, leading to increase the number of alternations between concentric and eccentric power phases. Hence, the biphasic profile is absent for the paretic limb, and the movement performed is less efficient. The following potential mechanisms may explain the observed fragmentation.

At the kinetic level, movement fragmentation can reflect the motor deficits caused by the brain injury. McCrea and Eng (2005) suggested that impaired nervous drive could lead to errors in trajectory planning and that rectification would be required during the execution as in a closed-loop control. With regards to this hypothesis, the movement fragmentation would be associated with the kinematic deficits. However, Rohrer et al. (2002) showed that there was no link between improvement in movement smoothness and changes in peak velocity magnitude or in the Fugl-Meyer score. Moreover, movement smoothness is a learned skill and can be improved in brain-injured patients through short training (Cirstea et al., 2003b; Rohrer et al., 2002). Although movement fragmentation could partly be attributed to alterations in the

corticospinal pathway (McCrea and Eng, 2005), our results highlight that brain-injured patients also use compensatory strategies to successfully achieve elbow extension.

Nonetheless, brain injury is known to reduce the ability to perform complex movements by freezing the number of degrees of freedom under voluntarily control (Bernstein, 1967; Cirstea and Levin, 2000). Considering movement fragmentation, lower peaks in joint torque and power during the acceleration phase for PAR elbow, our results could indicate an inability to produce the desired muscle forces. Those limitations would require patients to implement compensatory strategies (Cirstea and Levin, 2000) and the movement fragmentation could be considered as a strategy to compensate limb stiffening and to allow patients to perform the extension movement despite a loss of energy efficiency.

Therefore, our results show a stiffening of PAR leading to a fragmentation of movement and a loss of energy efficiency. We suggest that this deficit takes part in patient's inability to manage the muscle contraction levels necessary to achieve the desired movement. The assessment of inter-articular coordination and movement smoothness yields crucial insights into the motor deficits of brain injured patients (Murphy and Häger, 2015; Rohrer et al., 2002). Furthermore, the exploration of kinetics offers additional insights into the mechanisms responsible for the smoothness alteration. These biomarkers of movement quality could facilitate longitudinal monitoring of motor recovery, the preparation of clinical interventions or the assessment of rehabilitation effects, ensuring optimal adaptation to patients' deficits. The inclusion, in this study, of brain-injured subjects with significant variability in their characteristics in terms of time after brain injury as well as in the type of brain injury requires to exercise caution when attempting to extrapolate the findings discussed above.

5. Conclusion

Even if participating patients included in this study present a great diversity in the location and type of brain lesion, our results show that brain-injured patients use a different and partly symmetrical control strategy constrained by motor deficits. The brain damage limits the ability to manage musculoskeletal redundancy and leads to stiffening of the upper limbs. Our results show that both PAR and N-PAR present temporal and spatial alterations of interarticular coordination during active elbow extension when shoulder is lightly loaded. Considering these results, future research should investigate the alteration of muscle efforts using musculoskeletal models considering patients' altered nervous strategies.

The movement fragmentation in response to constraints allows patients to perform the extension movement despite a decrease in its energy efficiency. In addition to interarticular coordination assessment, which appears as a useful tool to evaluate the ability to exploit motor abundance in brain injured patients, motor rehabilitation should improve the management of this fragmentation strategy with the primary objective to improve both the performance and the efficiency of active movement for patients. Clinicians could then ensure that the number of transitions between concentric and eccentric phases is reduced during the rehabilitation process.

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CRediT authorship contribution statement

Emilie Mathieu: Data curation, Writing – original draft, Writing – review & editing. **David Gasq:** Investigation, Project administration, Writing – review & editing. **Sylvain Crémoux:** Writing – review & editing. **Célia Delcamp:** Data curation, Investigation. **Camille Cormier:**

Data curation, Investigation. **Philippe Pudlo:** Writing – review & editing. **David Amarantini:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.clinbiomech.2024.106221>.

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