Facial and upper-limb movement abnormalities in individuals with psychotic-like experiences: A motion analysis study

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Abstract

Slow movements and irregular muscle contraction have been reported separately in different studies targeting individuals with psychotic-like experiences (PLEs). To date, it remains unknown whether these two movement abnormalities, possibly associated with hypo- and hyper-dopaminergia respectively, co-existed in one sample with PLEs and interrelated in the early stage of psychotic progression. Therefore, this study was to examine if facial and upper-limb slow movements and irregular muscle contraction co-existed in individuals with PLEs, interrelated, and were associated with PLEs. A total of 26 individuals with PLEs, who were identified using the 16-item Prodromal Questionnaire, and 26 age- and gender-matched healthy controls received the facial and upper-limb movement measurement. A motion capture system was used to record the movement procedure and thus calculate kinematic variables that represented severity of slow movements and irregular muscle contraction. Results showed that facial and upper-limb slow movements and facial irregular muscle contraction existed in individuals with PLEs. For the total sample, slower facial movements were associated with less regular facial muscle contraction; slower upper-limb movements were associated with less regular upper-limb muscle contraction. Slower and less regular facial and upper-limb movements were associated with more severe PLEs. Compensatory changes in dopaminergic neural pathways in response to elevated dopamine might explain connection between slow movements and irregular muscle contraction. Because of the ability to detect facial and upper-limb movement abnormalities objectively and sensitively, motion analysis has great applicability to sensorimotor studies for people in the psychosis continuum.

Keywords: Bradykinesia; Dyskinesia; Prodromal questionnaire; Dopamine; Motion analysis

Introduction

Movement abnormalities have been indicated to be a main manifestation across different stages of psychotic progression [1-3]. First-episode drug-naïve psychotic patients have been found to present parkinsonism (e.g., one major type is bradykinesia, meaning slow movements) and dyskinesia (i.e., irregular and involuntary muscle contraction, such as tics and jerks) [2]. The findings have been strong evidence that bradykinesia and dyskinesia are intrinsic to psychotic illnesses [2]. In medicated patients with psychosis [4,5], the movement deficits are associated with both the illness itself and movement side effects of antipsychotics. In individuals with clinical high risk of the psychotic onset, who have obvious distress and the high rate of conversion to full-blown psychotic diseases [6,7], earlier studies [8-12] have indicated existence of upper-limb bradykinesia and facial and upper-limb dyskinesia in this clinical-high-risk population. Moreover, facial and upper-limb dyskinesia was found to be associated with more severe psychotic symptoms [13,14] and even be predictive of subsequent conversion to full-blown psychosis in the clinical-high-risk population [13]. It is noteworthy that the general population who has experienced subclinical psychosis, or called psychotic-like experiences (PLEs) [15], has also been found to exhibit slow movements and irregular muscle contraction in the upper limb [8,11,16,17]. Earlier research [18,19] has showed that children and youth who will or are at risk of developing psychotic illnesses display movement deficits.

One of the possible neural explanations of the link between movement abnormalities and psychotic symptoms is that neural deficits in basal ganglia, which are known to play a pivotal role in the etiology of psychotic symptoms [20,21], also cause movement abnormalities [2,14,22-25]. Investigating aberrant movements in psychotic disorders has drawn increasing research attention because movement abnormalities have been present even before the onset of psychotic

illnesses, can be measured objectively, and serve as external markers of underlying neural aberrance and vulnerability [26,27]. It is worthy of examining movement abnormalities particularly in individuals with PLEs, whose movements are free from influences of antipsychotics, to help gain understanding of pathoetiology of psychotic illnesses.

Bradykinesia and dyskinesia in the psychosis continuum may involve disparate dopaminergic mechanisms [2,13,20]. Specifically, bradykinesia is a hypokinetic movement that is thought to result from reduced dopamine in basal ganglia and overinhibition of neural impulses to the cortex [2,20]. By contrast, dyskinesia is a hyperkinetic movement that is thought to reflect overactivation of dopaminergic neurons [2,13]. To date, although slow movements and irregular muscle contraction have been reported separately in different studies on individuals with PLEs [8,11,16,17], it remains unclear whether these two movement disorders coexist in one sample of individuals with PLEs and whether they interrelate. In addition, scarce studies have examined both facial and upper-limb movement abnormalities in individuals with PLEs, which is worth investigation given the clear link of facial and upper-limb dyskinesia to the psychotic onset [13]. Bridging these knowledge gaps might contribute to understanding if hypo- and hyper-dopaminergia coexist in the early stage of psychotic progression. In addition, if association between the two movement abnormalities exists, this result might show connections between hypo- and hyper-dopaminergia and problematic modulation of dopaminergic homeostasis [9].

The challenge of studying movement abnormalities in individuals with PLEs is associated with the subtlety of presentation of movement abnormalities. Because individuals with PLEs have not yet exhibited obvious and serious symptoms, their movement alterations are easily missed by the naked eye. Indeed, earlier studies [28,29] have recommended the use of mechanical instruments to measure the initial and subtle movement alterations in individuals

who have not had the psychotic onset. This study addressed this measurement challenge by using motion analysis, which has proved sensitive to detecting facial and upper-limb movement problems [30-32]. Another advantage of motion analysis was provision of continuous scale data so as to facilitate investigation of variable association.

To sum up, this study was to examine (1) if slow movements and irregular muscle contraction coexisted in individuals with PLEs; (2) if slower movements was associated with less regular muscle contraction; and (3) if facial and upper-limb slow movements and irregular muscle contraction were associated with PLEs. We hypothesized that slow movements and irregular muscle contraction coexisted in individuals with PLEs, interrelated, and were associated with more severe PLEs. The results presented a big picture of links among different movement abnormalities and PLEs in the early stage of the illness continuum. This investigation provided an arena for elucidating possible dopaminergic mechanisms in the period before the onset of psychotic illnesses.

Methods

Participants

Individuals with PLEs and controls were recruited from secondary schools and universities in Hong Kong. Inclusion criteria for individuals with PLEs were: a score of or above nine in the 16-item Prodromal Questionnaire (PQ-16) [33] to show that individuals had subclinical psychotic symptoms (i.e., PLEs), a score of or above 22 in the Montreal Cognitive Assessment [34] to represent normal general cognition and thus comprehension of experimental instructions, and a score above 60 in the Edinburgh Handedness inventory [35] to indicate right-handedness. Exclusion criteria were presence of psychiatric diagnoses, taking psychotropic medication, and

neurological or musculoskeletal diseases or conditions that may affect facial or upper-limb movements. Controls had a score below nine in PQ-16 and satisfied the above-mentioned inclusion criteria regarding the Montreal Cognitive Assessment and the Edinburgh Handedness inventory. Exclusion criteria for controls were the aforementioned ones plus presence of first-degree relatives with diagnoses of psychiatric diseases. Exclusion criteria were checked using participants' self-report. This study was approved by the institutional ethics review board (reference number: HSEARS20180201007-01). The informed consent from participants was obtained before the study procedure.

Procedure

The PQ-16 was distributed to the community. Respondents who got a PQ-16 score of nine or more were invited to the lab in the university to receive further screening. Individuals with PLEs who satisfied all of the criteria proceeded to receive the facial and upper-limb movement measurement. Although PLEs in participants were assessed using PQ-16, researchers also conducted the interview of the Positive and Negative Syndrome Scale (PANSS) for collecting extra information. For controls, respondents who had a PQ-16 score below nine and met all criteria received the movement measurement and the interview of PANSS as well.

Assessments of psychotic symptoms

The PQ-16 is a self-report measure used to screen for subclinical psychotic symptoms [33,36]. This study adopted the Chinese version of PQ-16 [33]. It contains 16 true/false items regarding presence of subclinical psychotic symptoms over the past month, in which nine items are for hallucinations, five items for delusional ideas, and two items for negative symptoms. If the answer is true for an item, the respondent needs to further rate the distress level via a four-point Likert scale (0: no distress; 3: severe distress). The sum of distress scores is used. The

suggested cutoff score of nine or more yields a sensitivity of 85% and a specificity of 87% in distinguishing non-help-seeking individuals with PLEs from those without PLEs [33]. The PQ-16 shows good test-retest reliability (the intraclass correlation coefficient = .88), internal consistency (Cronbach's alpha = .72), and concurrent validity [33].

The PANSS [37] is a commonly-used interview measure developed to assess severity of psychotic symptoms in psychotic patients over the past week. Although PANSS is not designed to assess subclinical psychotic symptoms and thus may not be sensitive for individuals with PLEs, this study conducted PANSS for collecting extra information. The PANSS includes three domains: positive symptoms (seven items), negative symptoms (seven items), and general psychopathology (16 items). The score for each item ranges from one (absent) to seven (extreme). The PANSS has high internal consistency for each domain (Cronbach's α = .73 - .83), satisfactory test-retest reliability (r = .80 and .68 for positive and negative symptom domains), and strong construct validity. The research personnel administering PANSS in this study received intensive training through video-watching and actual practice. The interrater reliability of the research personnel and a senior psychiatrist was calculated (the intraclass correlation coefficient: .85 for the positive symptom domain, .80 for the negative symptom domain, and .83 for the general psychopathology domain).

Facial and upper-limb movement measurement

A motion capture system (VICON T160; Oxford Metrics Inc., Oxford, UK) was used to measure facial and upper-limb movement abnormalities. The system contained eight optical cameras, a connected computer, and small reflective markers, which adhered to the right thumb nail, the right ulnar styloid process (representing the wrist), and the right lip corner of the participant. Additional three markers were attached to the nose tip and the zygomatic process of

the temporal bone (both sides) to control confounding influences of head movements [32] when movements of the right lip corner were analyzed. Facial markers were 4 mm in diameter; hand markers were 6.4 mm in diameter. Optical cameras were able to record three-dimensional movements of markers, and thus the thumb, the wrist, and the lip corner, when the participant executed tasks. The capture rate of cameras was 120 Hz. Recorded data of markers were used to calculate kinematic variables, which represented severity of movement abnormalities. The calculation was conducted using the Matlab software (Mathworks, Natick, Massachusetts, USA). Because participants were right-handed, we only analyzed right-side data to prevent multiple testing and inflated type I errors.

For the facial task, the participant was seated and showed blank facial expression at the beginning. The participant was required to make the happy facial expression via grinning without a jaw drop to the maximal level as quickly as possible once hearing the starting beep sound, and keep the highest expressiveness for one second. The research personnel checked accuracy of facial expression of the participant via visual inspection. After one practice trial, three trials used for data analysis were recorded by the motion capture system.

For the upper-limb task, the participant sat in front of the table with the trunk being harnessed to the chair to prevent possible trunk movements during the task. The table height was adjusted to parallel the elbow height of the participant. Both hands were placed at the starting position at the edge of the table in front of shoulders (Fig. 1). Two hollow objects (outer diameter: 6 cm; inner diameter: 4.4 cm; 1.5 cm high) were placed in front of the starting positions at a distance of 70% of the participant's arm length, which was from the axilla to the distal wrist crease [38,39]. The end target (pin) was placed in front of the participant's midline at a distance of 21% of the arm length. Once hearing the starting signal, the participant was

required to use thumbs and index fingers of both hands simultaneously to reach for and grasp the corresponding objects, move the left object to the end target, and then move the right object to the end target (Fig. 2). The entire movement procedure was asked to be executed as quickly as possible. After one practice trial, three trials used for data analysis were recorded by the motion capture system. The reach-to-grasp movement of the right hand was analyzed (Fig. 2).

The normalized movement time (nMT) was calculated to reflect severity of movement slowness. Movement time was the interval between the movement onset timing and end timing, defined as the timing when the velocity of the lip or wrist marker reached 5 % of the peak velocity and that when the velocity of the lip marker dropped to 5 % of the peak velocity or when the thumb velocity dropped to 0 mm/second respectively [30,31,38,40-42]. Because the displacement of the lip movement and the reaching distance of the upper-limb movement varied among participants, facial and upper-limb movement time needed to be normalized through being divided by the lip displacement and by the arm length respectively [38,39,42]. Taken together, facial nMT and upper-limb nMT were calculated. Larger nMT meant slower movements.

The normalized number of movement units (nNMU) was calculated to reflect severity of irregular muscle contraction. The velocity profile of a typical grinning or reaching movement included an acceleration phase and a deceleration phase, both of which contributed to a peak (or a so-called movement unit) in the profile. More movement units appearing in a velocity profile represented that the movement showed more involuntary muscle contractions and tics and was less smooth. Similar to movement time, the number of movement units was normalized through being divided by the lip displacement for the facial movement and by the arm length for the upper-limb movement [38,39,42]. Taken together, facial nNMU and upper-limb nNMU were

calculated. Larger nNMU meant less smooth and less regular muscle contraction. The use of nMT and nNMU to reflect severity of movement abnormalities in psychotic patients has been examined in earlier research [42].

Statistical analysis

Possible differences between individuals with PLEs and controls in demographic and symptom data were tested using independent sample t tests and chi-square tests. In addition, independent sample t tests were used to compare facial and upper-limb nMT and nNMU between groups. Relationships among four movement variables (facial and upper-limb nMT and nNMU) and psychotic symptoms in the total sample were tested calculating Pearson's correlation coefficients. The IBM SPSS Statistics software was used. The alpha level (two-tailed) was set at 5 %.

Results

Characteristics of participants

A total of 26 individuals with PLEs and 26 controls were recruited in this study (Table 1). Both groups matched in terms of age, the gender ratio, and education. Individuals with PLEs had lower scores of the Montreal Cognitive Assessment and higher scores of PQ-16 as well as positive, negative, and general symptoms of PANSS than did controls.

Differences in movements between individuals with PLEs and controls

Differences between individuals with PLEs and controls were found in facial and upper-limb nMT and facial nNMU (Table 2). Individuals with PLEs had larger facial and upper-limb nMT and facial nNMU than did controls, which meant presence of facial and upper-limb slow movements as well as facial irregular muscle contraction in individuals with PLEs.

Correlations among movement abnormalities and psychotic symptoms

In all participants (Table 3), correlations were found between facial slow movements and facial irregular muscle contraction (p <.001), and between upper-limb slow movements and upper-limb irregular muscle contraction (p = .011). The score of PQ-16 was correlated with facial slow movements (p = .005), upper-limb slow movements (p = .041), facial irregular muscle contraction (p = .009), and upper-limb irregular muscle contraction (p = .036). For PANSS scores, positive symptoms were correlated with facial slow movements (p = .011) and facial irregular muscle contraction (p = .014). Negative symptoms were correlated with upper-limb slow movements (p = .012).

Discussion

The three research hypotheses were supported. Facial and upper-limb slow movements and facial irregular muscle contraction coexisted in individuals with PLEs. Slower facial movements was associated with less regular facial muscle contraction; slower upper-limb movements was associated with less regular upper-limb muscle contraction. Slower and less regular facial and upper-limb movements were associated with more severe PLEs.

Existence of slow movements and irregular muscle contraction in individuals with PLEs shown in this study is consistent with earlier research findings [8,11,16,17]. This study extends earlier findings by showing that individuals with PLEs not only had upper-limb movement abnormalities, but also had facial movement abnormalities. Moreover, this study further indicated that slow movements and irregular muscle contraction coexisted in one sample with PLEs. In addition, this study measured both facial and upper-limb slow movements and irregular muscle contraction and reported association between these movement abnormalities and PLEs.

Concurrence of slow movements and irregular muscle contraction and their connection with PLEs may be explained by basal ganglia alterations in individuals with PLEs [43-45]. The basal ganglia alterations in individuals with PLEs, compared with ones without PLEs, include different white matter microstructure in striatal regions, increased or decreased volumes of the caudate, or decreased volumes of the putamen and accumbens [43-45]. Basal ganglia mediate sensorimotor, perceptual, and cognitive inputs from the cortex and then transmit refined outputs back to cortical regions through the thalamus [46]. The refined motor signals through this neural circuitry affect movements of the limb and face through the pyramidal tracts projecting from the cortex to the spinal cord and cranial nerves, including the facial nerve [47]. Once basal ganglia become abnormal, disorganized outputs from basal ganglia may overinhibit or overstimulate cortical regions, leading to both erroneous perception/information processing (resulting in hallucination and delusion) and motor problems (such as slow movements and irregular muscle contraction) [20,48].

To our best knowledge, this is the first study to show association between slow movements and irregular muscle contraction in the early stage of psychotic progression. This link between slow movements and irregular muscle contraction was free from antipsychotic influences because none of our participants took antipsychotics. Mechanisms behind this link remain unclear for individuals with PLEs. However, existing research on individuals at clinical high risk of the psychotic onset may provide evidence in the period that is before the psychotic onset for discussing underlying mechanisms of the association between slow movements and irregular muscle contraction. Recent research [49,50] has indicated that individuals with clinical high risk of the psychotic onset have abnormal elevation of dopamine synthesis capacity in the dorsal striatum, including the sensorimotor striatum, which is closely involved in human movement

control. This evidence echoes the existence of irregular muscle contraction, which is thought to correlate with overactive dopamine neuronal activity [2,13]. It is noteworthy that earlier studies on drug-naïve psychotic patients [2,51] have introduced a notion that occurrence of slow movements, which is thought to correlate with reduced dopamine [2,20], may serve as a compensatory response to the hyper-dopaminergic state. It has been proposed that neural pathways of basal ganglia generate adaptive changes against dopamine overstimulation in drug-naïve psychotic patients [2,51]. This notion may explain that slower movements were associated with less regular muscle contraction. Current evidence has indicated that individuals with PLEs have basal ganglia alterations [43-45]. Future research on striatal dopamine function in individuals with PLEs is warranted.

The motion analysis technique records the procedure of movements at specific body landmarks or segments of interests (such as lip corners and wrists). Researchers are able to use the recorded movement trajectory data to calculate variables (such as nMT and nNMU), which directly, objectively, and sensitively reflect movement speed and quality of the entire movement procedure. Because of these advantages, motion analysis has been extensively applied to detecting movement problems (such as slowness and lack of smoothness) in patients with different neurological diseases, such as impaired basal ganglia [32,41,52,53] and brain damage [38,39,54]. Given that the sensorimotor domain of psychotic diseases has called for investigation [55,56], the motion analysis technique will benefit understanding of characteristics of movement deficits in patients with psychosis. In addition, earlier research [42] has reported that motion analysis is more sensitive to detecting facial and upper-limb movement abnormalities in psychotic patients than clinical rating scales. Due to the subclinical degree of movement abnormalities in individuals with PLEs and individuals with clinical high risk of the psychotic

onset, motion analysis has greatly potential application to sensorimotor studies for people in the psychosis continuum [30,31,40,42,57,58].

This study had several limitations. First, the sample size was relatively small, which may lead to insufficient statistical power of detecting existence of upper-limb irregular muscle contraction in our sample. Indeed, earlier research [59] has indicated that irregular muscle contraction is more evident in orofacial regions than limbs in drug-naïve psychotic patients, which may account for the result that facial irregular muscle contraction, but not upper-limb irregular muscle contraction, was found under the current sample size. It is needed for future research to examine concurrence of facial and upper-limb irregular muscle contraction and concurrence of slow movements and irregular muscle contraction at upper limbs in individuals with PLEs by adopting a large sample size. Second, this study recruited individuals with PLEs from the community, whose movement manifestations might differ from individuals with clinical high risk of the psychotic onset. Future research is worth validating the findings of this study in clinical-high-risk individuals. Third, this study only provided behavioral evidence that slow movements was linked to irregular muscle contraction in the early stage of psychotic progression and lacked examination of dopamine function. Future research is warranted to examine if hypo-dopaminergia and hyper-dopaminergia coexist in individuals with PLEs. Fourth, this study did not collect data of the daily activity level, which may be an index of movement performance in individuals with PLEs and is worth investigation in future research. Fifth, this study only administered PQ-16, which is a screening measure, to obtain data of PLEs. Future research should consider further adopting Structured Interview for Psychosis-Risk Syndrome [60] or Comprehensive Assessment of At-Risk Mental States [61], both of which are sensitive tools for assessing severity of attenuated psychotic symptoms and identifying clinical-high-risk

individuals. Sixth, it has been indicated [62] that asymmetric drug-induced movement deficits, which may reflect brain asymmetry, are associated with severity of positive symptoms in chronic psychiatric patients. Considering prevention of multiple testing and inflated type I errors, this study focused on measurement of right-side movements in right-handed individuals with PLEs. Future research is suggested to measure both-side movement abnormalities by using both motion analysis and observation-based movement scales and to investigate if movement asymmetry exists and is associated with psychotic progression in individuals with PLEs.

Conclusions

To our best knowledge, through the use of the motion analysis technique, this study was the first to demonstrate that slow movements and irregular muscle contraction at the face co-existed in individuals with PLEs and correlated. This study showed that facial and upper-limb movement abnormalities predated the onset of full-blown psychosis and were not simply by-products of antipsychotic treatment. This study also showed association of facial and upper-limb slow movements and irregular muscle contraction to PLEs. Future research with a large sample size needs to examine movements and dopamine function simultaneously and validate the findings of this study in the clinical-high-risk population.

Conflict of interest

None

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Author contributions

SMW contributed to conceptualization, methodology, data analysis, and writing of the first draft. BYHL contributed to methodology. LCK and HMH contributed to the task designs of motion analysis, writing of the Matlab program, and data processing. WCO contributed to methodology. All authors contributed to manuscript reviewing.

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Table 1 Characteristics of participants

	Individuals with PLEs (*n*=26)	Controls (*n*=26)	Group differences
	Mean (SD)	Mean (SD)	*p*
Age (years)	19.09 (3.68)	19.51 (3.00)	.650
Education (years)	12.31 (3.08)	13.12 (2.50)	.305
MoCA scores	27.31 (1.38)	28.15 (1.43)	**.035**
EHI scores	92.50 (9.93)	88.46 (11.20)	.175
PQ-16	13.04 (4.35)	1.50 (1.36)	**<.001**
PANSS-Positive symptoms	10.62 (2.28)	8.35 (1.60)	**<.001**
PANSS-Negative symptoms	11.15 (3.02)	9.19 (1.63)	**.006**
PANSS-General psychopathology	25.85 (4.65)	21.31 (3.17)	**<.001**
PANSS-Total	47.62 (7.16)	38.85 (4.76)	**<.001**
	% (*n*)	% (*n*)	*p*-value
Female	46.15 (12)	38.46 (10)	.575

Note: PLEs, psychotic-like experiences; MoCA, the Montreal Cognitive Assessment; EHI, the Edinburgh Handedness Inventory; PQ-16, the 16-item Prodromal Questionnaire; PANSS, the Positive and Negative Syndrome Scale.

Table 2 Differences in facial and upper-limb movements between individuals with PLEs and controls

	Individuals with PLEs (*n*=26)	Controls (*n*=26)	Test statistics	
	Mean (SD)	Mean (SD)	*t*	*p*
nMT (Slow movements)				
Face	.1378 (.1280)	.0728 (.0392)	-2.48	**.019**
Upper-limb	.0016 (.0003)	.0014 (.0002)	-2.28	**.027**
nNMU (Irregular muscle contraction)				
Face	.5709 (.5573)	.3195 (.1928)	-2.17	**.038**
Upper-limb	.0036 (.0008)	.0033 (.0005)	-1.45	.154

Note: PLEs, psychotic-like experiences; nMT, the normalized movement time; nNMU, the normalized number of movement units.

Table 3 Correlation coefficients among movement abnormalities and psychotic symptoms in participants (N=52)

	Movement abnormalities				PQ-16	PANSS			
	nMT (Slow movements)		nNMU (Irregular muscle contraction)						
	Face	UL	Face	UL		Positive	Negative	General[a]	Total
nMT (Slow movements)									
Face	NA	-.11	**.98*** **	-.14	**.38** **	**.35***	-.08	.07	.12
UL	NA	NA	-.13	**.35***	**.28***	.07	**.35***	.27	**.30***
nNMU (Irregular muscle contraction)									
Face	NA	NA	NA	-.15	**.36** **	**.34***	-.07	.06	.12
UL	NA	NA	NA	NA	**.29***	.04	.17	.14	.16

Note: PQ-16, the 16-item Prodromal Questionnaire; PANSS, the Positive and Negative Syndrome Scale; nMT, the normalized movement time; nNMU, the normalized number of movement units; UL, the upper limb; NA, not applicable.

[a]For additional analysis, in our participants (N=52), correlation coefficients between the motor retardation item of general psychopathology and each movement abnormality were: face nMT/Slow movements (r = -.11, p = .443); UL nMT/Slow movements (r

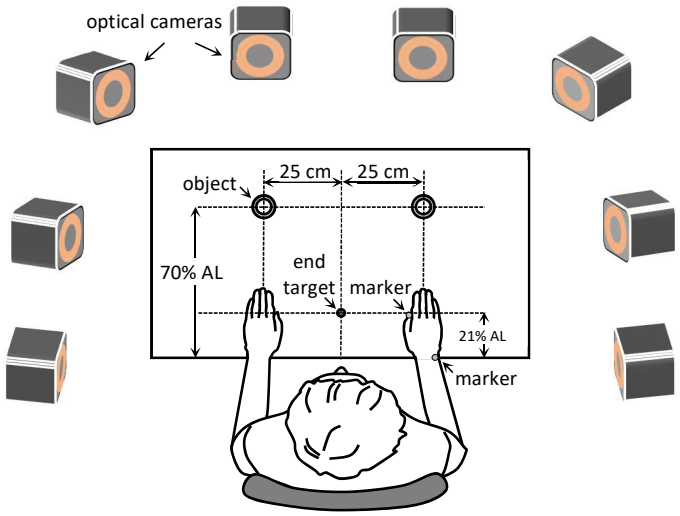
= .32, p = .019); face nNMU/Irregular muscle contraction (r = -.10, p = .498); UL nNMU/Irregular muscle contraction (r = .09, p = .526).

*p < .05; **p < .01; ***p < .001.

Figure captions

Fig. 1 The setup of the upper-limb movement measurement and the starting position of the participant. *AL* the arm length

Fig. 2 The procedure of the upper-limb movement task. (A) When the participant heard the starting beep sound, both hands were required to move from the starting position in order to reach for and grasp corresponding objects, which was the reach-to-grasp movement. (B) Subsequently, the left hand moved the left object to the end target, and the right hand moved the right object to the end target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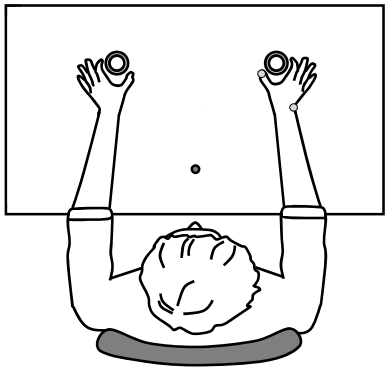
(A)



(B)