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Visual control improves the accuracy of hand positioning in Huntington's disease

Kontrola wzrokowa zwiększa precyzję ułożenia dłoni w chorobie Huntingtona

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Abstract

Background: The study aimed at demonstrating dependence of visual feedback during hand and finger positioning task performance among Huntington's disease patients in comparison to patients with Parkinson's disease and cervical dystonia. **Material and methods:** Eighty-nine patients participated in the study (23 with Huntington's disease, 25 with Parkinson's disease with dyskinesias, 21 with Parkinson's disease without dyskinesias, and 20 with cervical dystonia), scoring ≥20 points on Mini-Mental State Examination in order to assure comprehension of task instructions. Neurological examination comprised of the motor section from the Unified Huntington's Disease Rating Scale for Huntington's disease, the Unified Parkinson's Disease Rating Scale Part II–IV for Parkinson's disease and the Toronto Western Spasmodic Torticollis Rating Scale for cervical dystonia. In order to compare hand position accuracy under visually controlled and blindfolded conditions, the patient imitated each of the 10 examiner's hand postures twice, once under the visual control condition and once with no visual feedback provided. **Results:** Huntington's disease patients imitated examiner's hand positions less accurately under blindfolded condition in comparison to Parkinson's disease without dyskinesias and cervical dystonia participants. Under visually controlled condition there were no significant inter-group differences. **Conclusions:** Huntington's disease patients exhibit higher dependence on visual feedback while performing motor tasks than Parkinson's disease and cervical dystonia patients. Possible improvement of movement precision in Huntington's disease with the use of visual cues could be potentially useful in the patients' rehabilitation.

Keywords: movement disorders, Huntington's disease, Parkinson's disease, cervical dystonia, visual feedback

Streszczenie

Wprowadzenie: Badanie miało na celu ukazanie wpływu wzrokowego sprzężenia zwrotnego na poziom wykonania prób ułożenia dłoni oraz palców wśród pacjentów z chorobą Huntingtona w porównaniu z pacjentami z chorobą Parkinsona i dystonią szyjną. **Materiał i metody:** W badaniu wzięło udział 89 pacjentów (23 z chorobą Huntingtona, 25 z chorobą Parkinsona i dyskinezami, 21 z chorobą Parkinsona bez dyskinez i 20 z dystonią szyjną), z wynikiem ≥20 punktów w Mini-Mental State Examination, co zapewniało rozumienie instrukcji testowych. Badanie neurologiczne obejmowało podskale ruchowe z Ujednoliconej Skali Oceny Choroby Huntingtona, Ujednoliconej Skali Oceny Choroby Parkinsona – części II–IV oraz Skalę Oceny Dystonii Szyjnej z Toronto. W celu porównania precyzji ułożenia ręki w warunkach pod kontrolą wzrokową oraz bez niej pacjent odtwarzał dwukrotnie 10 pozycji prezentowanych przez badającego – jeden raz w każdym z powyższych warunków. **Wyniki:** Pacjenci z chorobą Huntingtona wykazali się mniejszą precyzją w próbach naśladowania ułożenia ręki badającego w warunkach bez kontroli wzrokowej w porównaniu z uczestnikami z chorobą Parkinsona bez dyskinez i dystonią szyjną. W przypadku prób z kontrolą wzrokową nie wystąpiły istotne różnice między grupami. **Wnioski:** Pacjenci z chorobą Huntingtona wykazują większą zależność od wzrokowego sprzężenia zwrotnego przy wykonywaniu zadań ruchowych niż pacjenci z chorobą Parkinsona i dystonią szyjną. Możliwość uzyskania poprawy precyzji ruchów po dostarczeniu wskazówek wzrokowych u pacjentów z chorobą Huntingtona można wykorzystać w rehabilitacji tej grupy chorych.

Słowa kluczowe: zaburzenia ruchowe, choroba Huntingtona, choroba Parkinsona, dystonia szyjna, wzrokowe sprzężenia zwrotne

INTRODUCTION

Huntington's disease (HD) is a neurodegenerative disorder characterised by motor, psychiatric and cognitive dysfunctions. Motor symptoms encompass both involuntary movements (mainly choreic, but later in the disease course also dystonic) and voluntary movements impairment, mainly bradykinesia (Ross et al., 2014; Sławek et al., 2013). In HD patients, both the preparation and execution phases of movement are impaired (Bilney et al., 2003a; García Ruiz et al., 2002), with the terminal phase of movement being particularly prone to distortions (Lemay et al., 2008). Defective control of motor programmes in HD may be attributed to the striatal atrophy itself, but also to the disrupted frontostriatal connections, especially those with the supplementary motor area, as well as to cortical atrophy. In HD, errors in feedback control of voluntary movement (Smith et al., 2000) and timing-dependent deficits (Rao et al., 2014) may be observable even in the preclinical stage of the disease. Of note, patients with HD are much more dependent on visual cues when performing tracing movements. The accuracy of movement under blindfolded condition deteriorates to a greater extent in HD than in healthy subjects. Carella et al. (2003) revealed that HD patients displayed greater variation of errors while performing the task under blindfolded condition than healthy controls. The observed effect was not associated with the severity of involuntary movements. The ability to correct movement trajectory with visual feedback may be more impaired if time-constraints are present (Lemay et al., 2005).

Our study aimed at verifying whether the same effect of high visual dependence is present when positioning one's hand according to the model. If it was the case, it would be informative in terms of potential compensatory strategies. What is more, we compared HD patients to individuals with other movement disorders (Parkinson's disease – PD and cervical dystonia – CD) to see if the visual dependence effect when planning hand positioning was a unique feature in HD, or if it was common also in other movement disorders. All clinical groups had involuntary movements, as the presence of involuntary movements may alter the reliance on proprioception cues when limb positioning. Also, severe involuntary movements may impair the precision of voluntary movements and the patients may be unaware of the involuntary movements and appreciate only their consequences while performing specific actions (Sitek et al., 2014). PD patients were chosen because of bradykinesia (as early and core feature) and the possible presence of choreic dyskinesias, similar to chorea in HD. Thus, PD group was divided into two subgroups: patients with choreic dyskinesias (PDdys) and without dyskinesias (PDndys). CD patients, in whom no abnormalities in upper limbs (apart from possible shoulder elevation) and execution of movement are expected (Anderson, 1995), were selected as a comparison group instead of healthy controls, as this cohort shares many emotional problems (e.g. depression and anxiety) and social stigma associated with visible neurological symptoms (Gündel et al., 2003). It was hypothesised that HD patients' performance will be significantly better in the condition with visual feedback and that this difference will not be so prominent in the other clinical groups.

MATERIAL AND METHODS

Procedure

Patients with clinical diagnosis of HD, PD (PDdys and PDndys) or CD were recruited from a specialty outpatient Movement Disorder Clinic and Department of Neurology in St. Adalbert Hospital in Gdansk. All patients underwent computed tomography or magnetic resonance imaging in order to rule out other causes of movement disorders. In all HD patients the clinical diagnosis was supported by genetic testing confirming trinucleotide CAG expansion in HTT exon-1. All HD patients had motor, cognitive and psychiatric manifestations of HD.

Participants were included in the study if their Mini-Mental State Examination (MMSE) (Folstein et al., 1975) score was ≥20 points in order to assure comprehension of task instructions. Patients with comorbid conditions that could affect cognition (e.g. other diseases affecting central nervous system, alcohol abuse) were excluded. All subjects volunteered for the study and gave their informed consent to the test procedure. The study procedure had previously been approved by the Bioethic Committee of the Medical University of Gdansk.

Patients

Eighty-nine patients participated in the study (23 with HD, 25 with PDdys, 21 with PDndys, and 20 with CD). Group demographics and disease characteristics are presented in Tab. 1. The groups were matched in terms of sex and years of education. HD and CD groups were matched in terms of age. PD group was significantly older, which is due to the typical later onset in PD. Disease duration was similar in HD and PDndys, but not PDdys, as choreic dyskinesias are a relatively late feature in PD course.

Measures

Neurological assessment

Apart from the assessment of general cognitive status with the use of MMSE, each group of patients underwent disease specific neurological assessment. Neurological examination comprised of the motor section from the Unified Huntington's Disease Rating Scale (UHDRS) (Huntington Study Group, 1996) for HD, the Unified Parkinson's Disease Rating Scale (UPDRS) Part II–IV (Paulson and

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	HD *N* = 23 [a][1]	PDdys *N* = 25 [b]	PDndys *N* = 21 [c]	CD *N* = 20 [d]	*F/H/t/U/*χ^2 tests[2]
Demographics					
Age	49.83 ± 11.12[3] [b,c]	65.68 ± 10.03 [a,d]	64.67 ± 7.59 [a,d]	51.75 ± 12.98 [b,c]	$F(3;85) = 14.11$; $p < 0.001$
Education (years)	12	12	13	12	$H(3, N = 89) = 1.99$; $p = 0.57$; s.i.
Male : female	14 : 9	12 : 13	15 : 6	8 : 12	$\chi^2 = 4.92$; $p = 0.18$; s.i.
Disease characteristics					
Duration of disease	5 [b]	12 [a,c]	4 [b]	8 [-]	$H(3, N = 89) = 28.76$; $p < 0.0001$
UPDRS III	NA	22.04 ± 9.14	18.29 ± 10.38	NA	$t(44) = 1.31$; $p = 0.20$; s.i.
UHDRS motor	38.09 (± 14.33)	NA	NA	NA	NA
TWSTRS severity	NA	NA	NA	15.55 ± 6.41	NA
MMSE	26[3]	27	28	28.50	$H(3, N = 89) = 14.48$; $p = 0.02$; s.i.

HD – Huntington's disease; **PDdys** – Parkinson's disease with dyskinesias; **PDndys** – Parkinson's disease without dyskinesias; **CD** – cervical dystonia; **s.i.** – statistically insignificant; **UHDRS** – Unified Huntington's Disease Rating Scale; **NA** – not assessed; **UPDRS** – Unified Parkinson's Disease Rating Scale; **TWSTRS** – Toronto Western Spasmodic Torticollis Rating Scale.
[1] Letters a–d denote significant intergroup differences as indicated in the first row of the table.
[2] The differences between the two groups were analysed either with *t*-unpaired test, *U* Mann–Whitney test, or chi-square test. The differences among the four groups were tested either with one-way analysis of variance test with Scheffe *post hoc* comparisons or with *H* Kruskal–Wallis test with *post hoc* Dunn's test.
[3] Mean ± standard deviation is reported in the case of normal data distribution.
[4] Median is reported in the case of non-normal data distribution.

Tab. 1. The comparison of four clinical groups' demographics and disease characteristics

Stern, 1997) for PD and the Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS) (Consky et al., 1990) for CD.

Assessment of hand position accuracy

In order to compare hand position accuracy under visually controlled and blindfolded conditions, the patient imitated each of the 10 examiner's hand postures twice, once under visual control condition and once with no visual feedback provided. Half of the trials were performed for the first time with visual control and half of the trials were administered behind a screen. The same number of trials was performed with right and left hand in order to exclude the possible impact of asymmetry. For trials without visual control the screen from Tactile Form Recognition Test from the Polish version of Halstead–Reitan Neuropsychological Test Battery was used (Kądzielawa et al., 1990), so that the patient could not see his/her own hand, while the examiner's hand position was visible for the patient throughout the task performance. The patient score could range from 0 to 10, where 10 corresponded to intact performance. Three scores were computed for each patient corresponding to performance under visual control, blindfolded performance and the difference score showing the degree of dependence on visual control.

The testing comprised of the following 20 trials (Fig. 1):

1. LEFT HAND **without** visual control: fist with thumb placed horizontally on the top;
2. RIGHT HAND **without** visual control: palm up, thumb bended;
3. LEFT HAND with visual control: clenched fist, thumb bended;
4. RIGHT HAND with visual control: clenched fist, thumb upward;
5. LEFT HAND **without** visual control: thumb touching ring finger;
6. RIGHT HAND **without** visual control: thumb between middle finger and ring finger;
7. LEFT HAND with visual control: thumb between index finger and middle finger;
8. RIGHT HAND with visual control: thumb touching middle finger;
9. LEFT HAND **without** visual control: thumb touching index finger;
10. RIGHT HAND **without** visual control: thumb touching little finger;
11. LEFT HAND with visual control: thumb bended;
12. RIGHT HAND with visual control: fist with thumb placed horizontally on the top;
13. LEFT HAND **without** visual control: clenched fist, thumb upward;
14. RIGHT HAND **without** visual control: clenched fist, thumb bended;
15. LEFT HAND with visual control: thumb between middle finger and ring finger;
16. RIGHT HAND with visual control: thumb touching ring finger;
17. LEFT HAND **without** visual control: thumb touching middle finger;
18. RIGHT HAND **without** visual control: thumb between index finger and middle finger;

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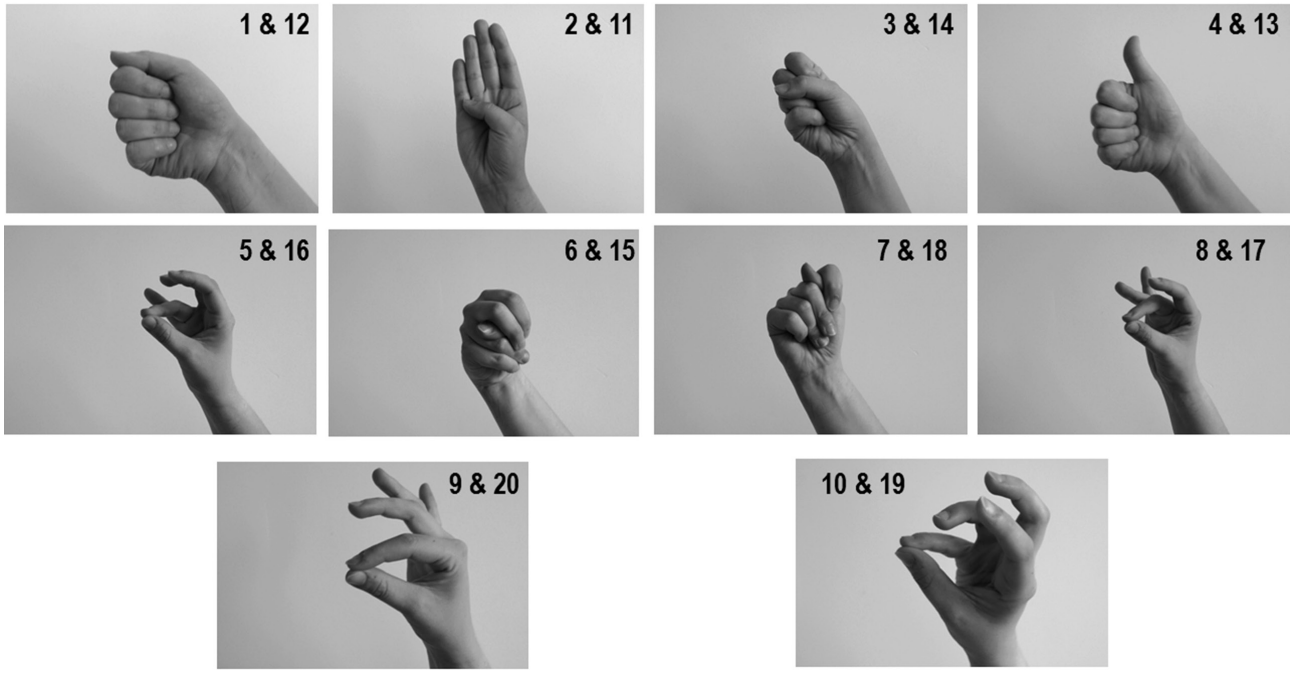

Ryc. 1. Tests used to evaluate hand position. The numbers of two test positions provided next to each photo (Photo Anna Cieślukowska)

19. LEFT HAND with visual control: thumb touching little finger;
20. RIGHT HAND with visual control: thumb touching index finger.

Data analysis

Inter-groups differences were analysed with *H* Kruskal–Wallis test with *post hoc* comparisons with Dunn's test, whenever applicable. Differences between two groups were tested either with Mann–Whitney *U* test, *t* unpaired test or χ^2 tests, as appropriate.

RESULTS

The performance of four groups was comparable under visually controlled condition. However, HD patients' performance was lower than in the case of PDndys and CD patients under blindfolded condition (Tab. 2). As the comparison of difference scores did not prove to be statistically significant at the stage of *post hoc* comparisons, subsequently, the performance of HD group was contrasted with the results obtained by other clinical groups as a whole. This comparison showed that HD group performed less

	HD ***N*** **= 22** **[a]**	**PDdys** ***N*** **= 23** **[b]**	**PDndys** ***N*** **= 21** **[c]**	**CD** ***N*** **= 20** **[d]**	***H*** **tests** **(3, *N* = 89)**
Performance with visual control (A) ***Me*** **Min. ÷ max.**	10.00 5 ÷ 10	10.00 7 ÷ 10	10.00 9 ÷ 10	10.00 8 ÷ 10	*H* = 5.53 *p* = 0.14; s.i.
Performance without visual control (B) ***Me*** **Min. ÷ max.**	8.50 4 ÷ 10 **[c,d]**	10.00 6 ÷ 10 **[-]**	10.00 8 ÷ 10 **[a]**	10.00 8 ÷ 10 **[a]**	*H* = 13.60 *p* = 0.004
Difference between A and B ***M/Me*** **(± *SD*)** **Min. ÷ max.**	1.39/1.00 (1.77) (−2) ÷ 5 **[-]**	0.44/0.00 (0.77) 0 ÷ 3 **[-]**	0.26/0.00 (0.72) (−1) ÷ 2 **[-]**	0.15/0 (0.88) (−2) ÷ 2 **[-]**	*H* = 9.98 *p* = 0.02

HD – Huntington's disease; **PDdys** – Parkinson's disease with dyskinesia; **PDndys** – Parkinson's disease without dyskinesia; **CD** – cervical dystonia; **s.i.** – statistically insignificant.
Inter-groups differences were analysed with *H* Kruskal–Wallis test with *post hoc* comparisons with Dunn's test.
Letters a–d denote significant inter-group differences as indicated in the first row of the table.

Tab. 2. Comparison of palm position accuracy under visually controlled and blindfolded conditions

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	HD ***N* = 23**	**PDdys, PDndys, CD** ***N* = 66**	***z/U/t* tests**
Performance with visual control (A) ***M/Me* (± *SD*)** **Min. ÷ max.**	9.13/10.00 (1.32) 5 ÷ 10	9.65/10.00 (0.69) 7 ÷ 10	$U = 600.00$ $z = -1.84$ $p = 0.07$
Performance without visual control (B) ***M/Me* (± *SD*)** **min. ÷ max.**	7.86/8.50 (2.03) 4 ÷ 10	9.35/10.00 (0.94) 6 ÷ 10	$U = 423.00$ $z = -3.41$ $p = 0.001$
Difference between A and B ***M/Me* (± *SD*)** **Min. ÷ max.**	1.39/1.00 (1.77) (−2) ÷ 5	0.30/0.00 (0.78) (−2) ÷ 3	$t(87) = 2.84$ $p = 0.009$

HD – Huntington's disease; **PDdys** – Parkinson's disease with dyskinesia; **PDndys** – Parkinson's disease without dyskinesia; **CD** – cervical dystonia.
The differences between the two groups were analysed either with *t*-unpaired test or *U* Mann–Whitney test. Mean ± standard deviation is reported in the case of normal data distribution. Median is reported in the case of non-normal data distribution.

Tab. 3. Comparison of palm position accuracy under visually controlled and blindfolded conditions in HD patients vs. other clinical groups

accurately than other groups under blindfolded condition and that the score difference between both trials was larger in HD than in other groups. Thus, the HD group was shown to be more visually dependent in hand positioning task than PDndys and CD patients (Tab. 3).

DISCUSSION

The present study was designed to demonstrate that the accuracy of hand positioning in HD patients depends on visual input to a higher extent than in other movement disorders, especially PD. In concordance with the previous study by Carella et al. (2003), we observed higher dependence of motor task performance on visual feedback in HD patients. Deterioration among HD participants was observed when visual cues were not provided, which suggests that proprioceptive signals are not sufficient to compensate movement jerkiness. There were no significant differences between HD patients and other groups in hand positioning accuracy under visual control. This means that reliance on visual cues may not only improve movement trajectory in HD, which was evidenced by Carella et al. (2003) using precise kinematic and error parameters, but it may also significantly affect the attainment of a target hand position, as shown in our study.

Movement corrections may rely on both automatic and voluntary processes. However, involuntary mechanisms are used much more often. Healthy subjects are unaware of the slight flexible movement corrections during movement trajectory (Gaveau et al., 2014). On the one hand, in HD such involuntary correction mechanisms may be ineffective due to both altered sensory processing and the interference of corrective movements with involuntary movements. On the other hand, as HD patients have impaired awareness of choreic movements (Sitek et al., 2011), they may fail to use voluntary processes to make movement corrections unless cued to do so.

As the basal ganglia regulate sensory processing, their atrophy may lead to altered proprioceptive sensations concerning limb position and movement, which in turn may entail excessive dependence on visual feedback (Seiss et al., 2003). Visuomotor integration deficits in HD (Say et al., 2011) may significantly impair movement correction. Smith et al. (2000) observed that movements in HD begin normally, but become irregular 200–300 ms into their course – when the corrective actions based on visual or proprioceptive information are required. Carella et al. (2003) suggest that if both sensory input to the cortex and striatal output to the thalamus (responsible for providing sensory information to the cortex) are altered, proprioceptive feedback control of movements seems to be the most likely the cause of increased dependence on visual cues. Defective error control may be connected with deficits in afferent signals which play an important role in this process. Abnormal somatosensory evoked potentials and long-loop transcortical reflexes, observed in HD patients, can underlie the inefficient movement correction under blindfolded condition (Boulet et al., 2005; Deuschl et al., 1989; Noth et al., 1985).

Contrary to these reports, Despard et al. (2015) noted in a computerised task greater movement variability in the presence of visual cues as compared to their absence. However, it is possible that in some computerised tasks with very precise measurements, oculomotor deficits may negatively influence the patient's performance under visually controlled conditions and somehow overshadow the advantage of visually controlled condition over the blindfolded condition. Also, as in visuomotor tasks executed by healthy subjects, occipital-prefrontal-motor functional network facilitates the modulation of instructed motor responses to visual cues (Papadelis et al., 2016), it could be argued that widespread neurodegeneration affecting connectivity early

in the disease course (Shaffer et al., 2017) may prevent HD patients from using the visual cues successfully.

Current therapeutic approaches in movement disorders are widely described in the literature mainly with reference to PD interventions (Bilney et al., 2003a, 2003b; Quinn et al., 2013a). Rehabilitation strategies for HD patients have also developed over recent years (Quinn and Busse, 2012), albeit symptomatic pharmacotherapy remains the main therapeutic option (Zielonka et al., 2015). In terms of specific training strategies, Ciancarelli et al. (2013) propose effective rehabilitation treatment in HD but emphasise that therapy must be multifunctional and continuous. Their intervention included exercises to strength, endurance, range of motion, gait abnormalities, sensory deficits and other disabilities diagnosed in each patient participating in this programme. Significant effects were also observed in home-based programmes (Quinn et al., 2013b). Also, rhythmic auditory cueing was described as potentially effective in the rehabilitation of patients with movement disorders, but the evidence base is limited (Schaefer, 2014; Wittwer et al., 2013).

Visual imagery training may improve graphomotor performance in HD (Yágüez et al., 1999). To our knowledge, the usefulness of visual cues in the comprehensive physiotherapy of HD patients was not systematically studied. In our study, the movement corrections made by the HD patients under visually controlled condition were successful as their performance did not differ from other groups under the condition of visual feedback available, which suggests the effectiveness of visual control. Obviously, visual feedback is potentially available to the patient in most rehabilitation techniques, as the exercises are not performed under blindfolded condition. However, the availability of visual feedback does not mean that each patient is likely to use it to the same extent. The failure to use visual feedback in HD in real-life situations may stem from the fact that the disease affects insight both in a global manner (so that the patients underestimate disability, motor, cognitive and behavioural impairment) and more specifically when the presence and severity of involuntary movements is concerned (Sitek et al., 2011, 2014). Our study procedure using alternating conditions (two trials with visual control and then two trials without visual control) was likely to focus the patient's attention on the target task and potentially encouraged the use of visual control to make hand positioning corrections, when the visual feedback was available. Under the condition without visual control, the patient could not compare his/her hand position with the target hand position visually, while in visually controlled condition such a comparison could trigger movement corrections.

Our procedure demonstrated clearly that the performance improves with visual feedback. However, the task procedure, used in our study, may seem ecologically invalid, as in real life, usually visual feedback is either available or not, and its availability does not change many times during a given task performance.

Our study highlights the need for the development of strategies relying on visual feedback to improve movement precision among HD patients. Initiating voluntary motor adjustments requires additional effort and may not happen spontaneously in an HD patient that is likely to suffer from apathy. HD patients could potentially benefit from being cued by the physiotherapist to use visual feedback and visual imagery during movement preparation and execution, as they may not use it spontaneously as often as needed due to the deficient awareness of the involuntary movements likely to alter the trajectory of voluntary movements.

The main limitations of our study were: small sample size, ceiling effect in many individuals under visually controlled conditions and low number of trials (narrow range of results). Due to the fact that the patients had clearly distinguishable motor symptoms, the rater was not blinded to the clinical diagnosis. Also, there may have been some rater bias as the patients' performance was not video-taped. Another caveat that may limit the generalisation of our study results to real-life conditions is that all movement plans were externally-driven in our procedure, while in real-life the use of internally-driven movement plans is frequently needed. Internally-driven movement plans are associated with a much wider brain activation both in the ipsilateral and contralateral cortex than externally-driven movement plans (Ariani et al., 2015). As in HD, callosal fibres are affected early in the disease course (Poudel et al., 2015), internally-driven movement plans may be potentially more impaired than externally-driven movement plans. The effectiveness of visual cueing may vary under externally- and internally-driven conditions. As most rehabilitation plans are highly structured, the use of visual cues in externally-driven conditions may not generalise to internally-driven actions, more characteristic of real life conditions.

While our study addressed the accuracy of hand positioning, most of the previous research in healthy individuals focused on reach-to-grasp movements and analysed the timing of movement. Of note, it was demonstrated that as far as reach-to-grasp movements are concerned, they are initiated faster if any cue (congruent or incongruent) is provided to the individual, both under visually guided and memory-guided condition (Seegelke et al., 2016). It can be hypothesised that visual condition encourages voluntary movement corrections that improve accuracy but also potentially delay the timing of the final hand position attainment. Unfortunately, in our study we did not measure time parameters, so it is still an open question.

Overall, the results of our study suggest the importance of visual feedback for correct hand positioning in HD and may be potentially useful for the development of compensatory strategies. Further studies are needed to compare the usefulness of visual cues with auditory cues, which are also frequently used in rehabilitation.

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CONCLUSIONS

Our data suggest the important role of visual cues in movement adjustment in HD. The potential suitability of compensatory strategies relying on visual feedback when executing hand movements requires further verification and comparison with other cues, e.g. auditory, under conditions allowing for the use of different sensory modalities.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations, which might negatively affect the content of this publication and/or claim authorship rights to this publication.

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References

Anderson TJ: Spasmodic torticollis. In: Moore P (ed.): Handbook of Botulinum Toxin Treatment. Blackwell Science, Oxford 1995: 103–130.

Ariani G, Wurm MF, Lingnau A: Decoding internally and externally driven movement plans. J Neurosci 2015; 35: 14160–14171.

Bilney B, Morris ME, Denisenko S: Physiotherapy for people with movement disorders arising from basal ganglia dysfunction. N Z J Physiother 2003a; 31: 94–100.

Bilney B, Morris ME, Perry A: Effectiveness of physiotherapy, occupational therapy, and speech pathology for people with Huntington's disease: a systematic review. Neurorehabil Neural Repair 2003b; 17: 12–24.

Boulet C, Lemay M, Bédard MA et al.: Early Huntington's disease affects movements in transformed sensorimotor mappings. Brain Cogn 2005; 57: 236–243.

Carella F, Bressanelli M, Piacentini S et al.: A study of arm movements in Huntington's disease under visually controlled and blindfolded conditions. Neurol Sci 2003; 23: 287–293.

Ciancarelli I, Tozzi Ciancarelli MG, Carolei A: Effectiveness of intensive neurorehabilitation in patients with Huntington's disease. Eur J Phys Rehabil Med 2013; 49: 189–195.

Consky ES, Basinski A, Belle L et al.: The Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS): assessment of validity and inter-rater reliability. Neurology 1990; 40 (Suppl 1): 445.

Despard J, Ternes AM, Dimech-Betancourt B et al.: Characterising upper limb movements in Huntington's disease and the Impact of restricted visual cues. PLoS One 2015; 10: e0133709.

Deuschl G, Lücking CH, Schenck E: Hand muscle reflexes following electrical stimulation in choreatic movement disorders. J Neurol Neurosurg Psychiatry 1989; 52: 755–762.

Folstein MF, Folstein SE, McHugh PR: "Mini-mental state." A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975; 12: 189–198.

García Ruiz PJ, Hernández J, Cantarero S et al.: Bradykinesia in Huntington's disease. A prospective, follow-up study. J Neurol 2002; 249: 437–440.

Gaveau V, Pisella L, Priot AE et al.: Automatic online control of motor adjustments in reaching and grasping. Neuropsychologia 2014; 55: 25–40.

Gündel H, Wolf A, Xidara V et al.: High psychiatric comorbidity in spasmodic torticollis: a controlled study. J Nerv Ment Dis 2003; 191: 465–473.

Huntington Study Group: Unified Huntington's Disease Rating Scale: reliability and consistency. Mov Disord 1996; 11: 136–142.

Kądzielawa D, Mroziak J, Bolewska A et al. (eds.): Podręcznik do Baterii Testów Neuropsychologicznych Halsteada-Reitana [The Halstead-Reitan Test Battery Manual]. Laboratorium Technik Diagnostycznych Wydziału Psychologii UW, Warszawa 1990.

Lemay M, Chouinard S, Richer F et al.: Huntington's disease affects movement termination. Behav Brain Res 2008; 187: 153–158.

Lemay M, Fimbel E, Beuter A et al.: Sensorimotor mapping affects movement correction deficits in early Huntington's disease. Exp Brain Res 2005; 165: 454–460.

Noth J, Podoll K, Friedemann HH: Long-loop reflexes in small hand muscles studied in normal subjects and in patients with Huntington's disease. Brain 1985; 108: 65–80.

Papadelis C, Arfeller C, Erla S et al.: Inferior frontal gyrus links visual and motor cortices during a visuomotor precision grip force task. Brain Res 2016; 1650: 252–266.

Paulson HL, Stern MB: Clinical manifestations of Parkinson's disease. In: Watts LR, Koller WC (eds.): Movement Disorders: Neurologic Principles and Practice. McGraw-Hill, New York 1997: 183–199.

Poudel GR, Stout JC, Domínguez D JF et al.: Longitudinal change in white matter microstructure in Huntington's disease: the IMAGE-HD study. Neurobiol Dis 2015; 74: 406–412.

Quinn L, Busse M: Physiotherapy clinical guidelines for Huntington's disease. Neurodegener Dis Manag 2012; 2: 21–31.

Quinn L, Busse M, Dal Bello-Haas V: Management of upper extremity dysfunction in people with Parkinson disease and Huntington disease: facilitating outcomes across the disease lifespan. J Hand Ther 2013a; 26: 148–154.

Quinn L, Rosser A, Busse M: Critical features in the development of exercise-based interventions for people with Huntington's disease. Eur Neurol Rev 2013b; 8: 10–13.

Rao AK, Marder KS, Uddin J et al.: Variability in interval production is due to timing-dependent deficits in Huntington's disease. Mov Disord 2014; 29: 1516–1522.

Ross CA, Aylward EH, Wild EJ et al.: Huntington disease: natural history, biomarkers and prospects for therapeutics. Nat Rev Neurol 2014; 10: 204–2016.

Say MJ, Jones R, Scahill RI et al.: Visuomotor integration deficits precede clinical onset in Huntington's disease. Neuropsychologia 2011; 49: 264–270.

Schaefer RS: Auditory rhythmic cueing in movement rehabilitation: findings and possible mechanisms. Philos Trans R Soc Lond B Biol Sci 2014; 369: 20130402.

Seegelke C, Güldenpenning I, Dettling J et al.: Visuomotor priming of action preparation and motor programming is similar in visually guided and memory-guided actions. Neuropsychologia 2016; 91: 1–8.

Seiss E, Praamstra P, Hesse CW et al.: Proprioceptive sensory function in Parkinson's disease and Huntington's disease: evidence from proprioception-related EEG potentials. Exp Brain Res 2003; 148: 308–319.

Shaffer JJ, Ghayoor A, Long JD et al.: Longitudinal diffusion changes in prodromal and early HD: Evidence of white-matter tract deterioration. Hum Brain Mapp 2017; 38: 1460–1477.

Sitek EJ, Sołtan W, Wieczorek D et al.: Self-awareness of motor dysfunction in patients with Huntington's disease in comparison to Parkinson's disease and cervical dystonia. J Int Neuropsychol Soc 2011; 17: 788–795.

Sitek EJ, Thompson JC, Craufurd D et al.: Unawareness of deficits in Huntington's disease. J Huntingtons Dis 2014; 3: 125–135.

Sławek J, Sołtan W, Sitek EJ: Choroba Huntingtona – w 20. rocznicę odkrycia genu *IT15*; patogeneza, diagnostyka i leczenie. Pol Przegl Neurol 2013; 9: 85–95.

Smith MA, Brandt J, Shadmehr R: Motor disorder in Huntington's disease begins as a dysfunction in error feedback control. Nature 2000; 403: 544–549.

Wittwer JE, Webster KE, Hill K: Rhythmic auditory cueing to improve walking in patients with neurological conditions other than Parkinson's disease – what is the evidence? Disabil Rehabil 2013; 35: 164–176.

Yágüez L, Canavan AG, Lange HW et al.: Motor learning by imagery is differentially affected in Parkinson's and Huntington's diseases. Behav Brain Res 1999; 102: 115–127.

Zielonka D, Mielcarek M, Landwehrmeyer GB: Update on Huntington's disease: advances in care and emerging therapeutic options. Parkinsonism Relat Disord 2015; 21: 169–178.