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Submovements in visually-guided and memory-guided reaching tasks: changes in Parkinson's disease

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Abstract— **Movements in people with Parkinson's disease are often hypometric, although we have found that this was not the case in an experimental visually-guided reaching task. We wished to explore our hypotheses that (1) people with Parkinson's disease produce hypometric primary submovements but (2) are able to use visual feedback to accurately reach the target in a single overall movement, and (3) this effect may be greater in memory-guided tasks in which an internal representation of the target location is used instead of a fixation-centered representation of the target.**

Visually- and memory-guided reaching movements were examined in 22 people with mild to moderate severity Parkinson's disease on medication, along with age-matched and sex-matched controls. Primary submovements were extracted from 5149 movements using a method based upon zero crossings of jerk (3rd derivative of position), with several additional criteria to minimize the detection of submovements due to noise or tremor.

There was no difference in the end position of the overall reaching movement between the two groups, although the movement was smaller in the memory-guided task. In contrast, the gain of the primary submovement was smaller in the Parkinson's disease group than the control group, with this difference being greater on the memory-guided task. There was no task effect on the primary submovement gain in the control group.

Our results show that the underlying primary submovement in visually-guided movements in people with Parkinson's disease is hypometric, and that the degree of hypometria is even greater in memory-guided movements.

I. INTRODUCTION

Movements in people with Parkinson's disease are often hypometric, especially in length of steps in walking [1], the size of handwriting [2], and when performing repetitive movements such as finger and thumb tapping in the Unified Parkinson's Disease Rating Scale. However, in visually-guided reaching movements, in which there is an explicit target, and feedback of hand position is provided, this hypometria is much less evident [3], [4]. However, these latter movements may also be intrinsically hypometric but visual feedback may be used to get accurately to the target in a single, albeit complex, movement [5], [6]. This hypometria may be more pronounced in memory-guided movements that involve the basal ganglia to a greater degree [7], [8]. Decomposing movements into individual components should reveal the strategy that is used.

A fast movement to a fixed target can be thought of as being composed of one or more submovements, each of which is pre-programmed and generated by an inverse internal model [9], [10]. The first of these is called the initial, or primary, submovement. Then, based upon the output of a forward model and/or visual feedback and accuracy requirements, the primary submovement may be modified slightly near the end (allowing for the delay in the motor control system), corrective submovements may be made before the end of the primary submovement (concurrent/overlapping submovements), or after the primary submovement (discrete/non-overlapping submovements). These corrective submovements cause a deviation from the typical bell-shaped velocity profile of the end effector, which should be optimal when signal-dependent noise of motor units is present [11].

Several groups have developed methods to extract submovements from movement traces [12]–[14]. In a series of experiments looking at a knob-turning task in non-human primates and humans, objective methods have been developed to decompose overall movements into their submovements [15], [16]. However, there has been no published work applying these submovement decomposition methods to the abnormal movements seen in people with Parkinson's disease. This study aimed to apply these submovement decomposition methods to visually-guided and memory-guided reaching movements from people with Parkinson's disease.

II. METHOD

A calibrated, low-latency, near-field 3D virtual environment with an electromagnetic tracker was used to run the experiment and record the data [17]. A blue sphere in the virtual environment corresponded to the position of an electromagnetic sensor attached to the subject's fingertip.

The experiment involved 22 people with Parkinson's disease and 22 age-matched controls. All subjects performed the sessions in the morning, and the people with Parkinson's disease had taken their morning medication as normal. Each subject came in for two sessions, one for a visually-guided

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task and the other for a memory-guided task, with the order randomized.

In the visually-guided task, subjects started at a central position and were instructed to move as quickly and accurately as possible to a 5-mm radius target that appeared for 400 ms, concurrently with a beep, randomly at one of 15 equally-spaced locations on an arc $\pm 40^{\circ}$ of straight ahead, 150 mm from the home target. By having the target only flash briefly, quick movements were encouraged that relied on the gaze location to guide the movement to the target location. The target reappeared 1900 ms after the beep to allow the subject to receive visual feedback of performance.

The memory-guided task was similar to the visually-guided task except that the beep sounded 0.8 to 2.5 s after the target had appeared for 400 ms, and the subject was instructed not to look or move to the target until the beep. Hence fixation could not be used to guide the end effector to the target, thus forcing the use of spatial memory, with this monitored via a camera that was focused on the eye. The target reappeared 1500 ms after the beep to allow the subject to receive visual feedback of performance.

The method used to identify and extract submovements is based upon [15], [16]. As the positional data generated from our experimental system is 6D, reduction to a single dimension was achieved by projecting the movement path onto an axis defined with an origin at the home position and in direct line with the target, and ignoring the orientation data. The present results report only the analysis of the component of the movement towards the target.

Let t_{peak} and v_{peak} denote the time and magnitude of the peak velocity attained during the entire movement, and t_{start} denote the starting time of the movement, defined by finding the first t such that $|v(t)| > 0.05\ |v_{peak}|$ and then going back until $|v(t)| < 0.005$ ms^{-1}. The end time of the movement t_{end} is defined by the first t_{end} such that $|v(t)| < 0.05\ |v_{peak}|$ on the interval $t = [t_{end}, t_{end}+150$ ms). The movement final amplitude is defined to be

$$m_{fa} = x(t_{end}) - x(t_{start}), \tag{1}$$

where $x(t)$ is the position of the finger tip at time t. The final movement gain is then equal to

$$m_{fg} = m_{fa}/(150\text{ mm} - x(t_{start})). \tag{2}$$

Submovements are characterized by inflections in the acceleration trace (i.e., points where the acceleration decreases, increases, then decreases again), corresponding to a force pulse from the muscles. This is detected by a pair of zero crossings in the jerk trace (derivative of acceleration). To eliminate submovements being detected due to noise or a tremor, a zero crossing of the jerk trace is determined to be significant if the distance between the peaks either side of the zero crossing is greater than the maximum amplitude range of jerk during the rest period before that movement started.

To start extraction of the primary submovement, the peak velocity of the submovement v_{psp} is found by searching from the detected start time for the first acceleration zero crossing flanked by significant jerk zero crossings. Additionally, there is a requirement that $|v_{psp}| > 0.1\ |v_{peak}|$.

To complete the extraction of the primary submovement an assumption of a symmetric velocity profile is made, and is based upon several points. Firstly, in normal healthy subjects the brain optimizes the end effector to follow a straight path in visual coordinates [18] and produces symmetric velocity profiles [19]. Secondly, to improve accuracy and speed the motor system aims to minimize signal-dependent noise [11], which results in velocity profiles that are bell-shaped. Thus, the estimated velocity profile of the primary submovement for each case is generated by mirroring the time from the start of the movement to the peak velocity. By integrating this velocity profile, an estimated amplitude of the primary submovement, m_{psa}, is generated. From this the gain of the primary submovement is equal to

$$m_{psg} = x(m_{psa})/(150\text{ mm} - x(t_{start})). \tag{3}$$

A Python application was developed to iterate over the recorded data, analyse each movement, generate a PDF plot of each movement, and save results into a comma separated values (csv) file for importing into the statistical environment R [20]. A linear mixed-effects model [21] was used for the multiple dependent variables, with fixed effects of Group and Task, and a random effect of subject.

III. RESULTS

Each subject performed 60 movements in each session, leading to 5149 valid trials. Movements had either a single primary submovement (Fig. 1, left-hand side), a large single primary submovement with a single secondary corrective submovement (Fig. 1, centre), a small single primary submovement with larger secondary submovements (Fig. 1, right-hand side).

Looking at the final gain of the movement to the target (Fig. 2), there was no Group effect [F(1,42) = 0.834, p = 0.37] but there was a Task effect, with the memory-guided movements shorter than the visually-guided movements [mean gain 0.95 vs 0.92, F(1,42) = 4.0, $p < 0.05$]. Also, the variability of the movement gain was greater in the memory-guided task due to not being able to use fixation to guide the end-effector to where the target had flashed. There was no interaction between Group and Task [F(1,42) = 0.569, p = 0.45].

Looking at the primary submovement gain (Fig. 3), there was a Group effect with the Parkinson's disease group having a smaller gain for the primary submovement in the visually-guided movement [mean 0.73 vs 0.85, F(1,42) = 7.6, $p < 0.01$]. There was a Task effect [F(1,42) = 17, $p < 0.0001$], and Group and Task interaction [F(1,42) = 9.1, $p < 0.01$]. The effect of Task and was due to the Parkinson's disease group gain being smaller on the memory-guided task [mean 0.68 vs 0.73, $p < 0.0001$] but no change in the control group [mean 0.84 vs 0.85, p = 0.47]. In the Parkinson's disease group a large number of movements consisted of a series of velocity pulses, with a low peak velocity overall,

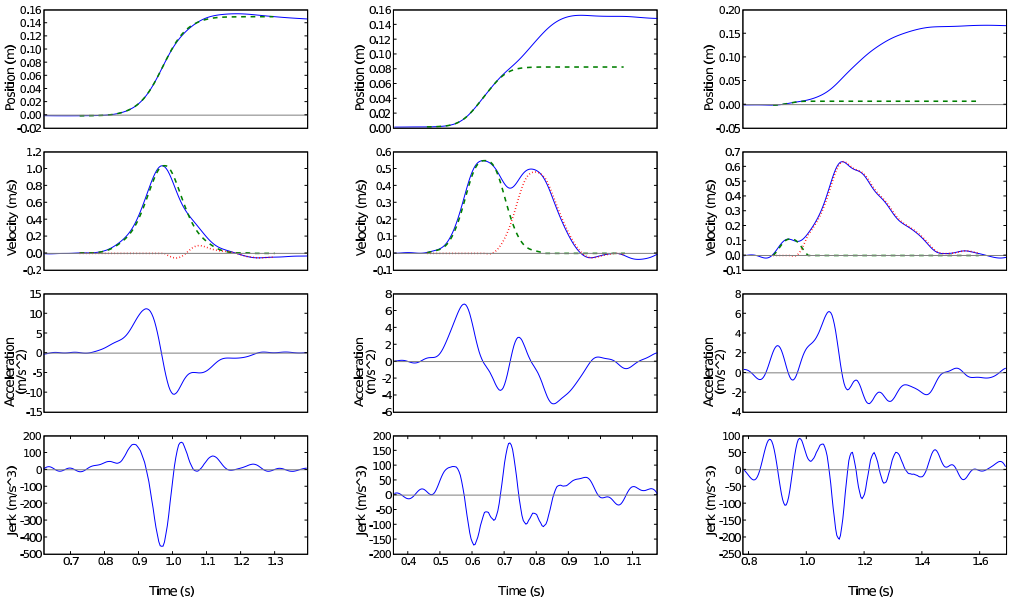

Fig. 1. Example movements and their decompositions. The four panels correspond to position, velocity, acceleration, and jerk (the derivative of acceleration). The dashed line indicates the estimated end position (in the position trace) and velocity profile (in the velocity trace) of the primary submovement. The dotted line in the velocity trace is the residue velocity left after subtracting the primary submovement. Left: A movement from a control subject with a single bell-shaped velocity profile. Centre: A movement from a person with Parkinson's disease with a single overlapping submovement. Right: a small single primary submovement with several larger secondary submovements from a person with Parkinson's disease (not decomposed).

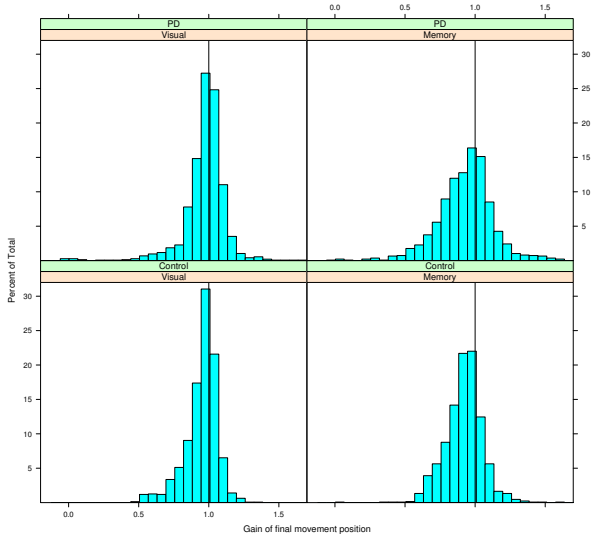

Fig. 2. Distribution of final movement gain for factors of Group and Task. The top row is of movements from the Parkinson's disease group and the bottom row movements from the control group. The left-hand column is the visually-guided task and the right-hand column is the memory-guided task. The vertical line at $x = 1.0$ represents the desired gain. There is a significant effect of Task and a significant interaction between Group and Task.

or a movement had a small primary submovement with larger secondary submovements, contributing to the bimodal distribution of primary submovement gain (Fig. 3, top row).

IV. DISCUSSION

This is the first study to quantify the the size of the primary submovement in visually- and memory-guided reaching tasks in Parkinson's disease. The primary hypothesis was that although the movement endpoint in people with Parkinson's disease is not hypometric in an experimental visually-guided task, the primary submovement is hypometric compared to that of control subjects. A second hypothesis was that there is a greater effect in people with Parkinson's disease when the task is memory-guided and relies on an internal representation of the target rather than a visual fixation of the target location.

The final gain in both tasks was not different between groups, although there was a task effect. On the memory-guided task, overall gains were smaller, related to spatial memory underestimating the distance to the target. Additionally, there was far greater variability in the memory-guided task, highlighting the greater uncertainty in spatial memory compared to a vision-fixated target location.

In comparison to the final gain, the gain of the primary submovement was found to be substantially smaller in the Parkinson's disease group compared to controls in both tasks. Also, while the gain of the primary submovement was equal in both tasks for the control group (even though the final gain of the memory-guided movement was smaller), in the

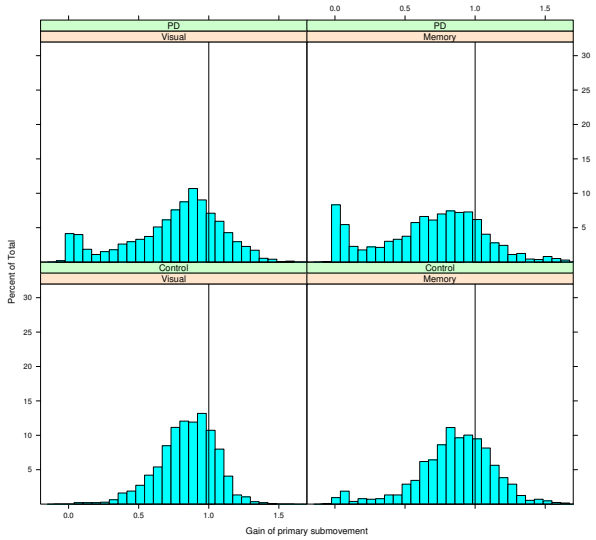

Fig. 3. Distribution of primary submovement gain based upon a significant zero crossing of acceleration for factors of Group and Task. The top row is of movements from the control group and the bottom row movements from the Parkinson's disease group. The left-hand column is the memory-guided task and the right-hand column is the visually-guided task. The vertical line at $x = 1.0$ represents the desired gain. There is a significant effect of Group, and an interaction between Task and Group.

Parkinson's disease group there was a smaller gain in the memory-guided task compared to the visually-guided task (even though the final gain in the memory-guided task was not different to that of controls). This highlights the extra difficulty people with Parkinson's disease have in making a movement of the correct size [22], especially when the target is represented in spatial memory or is internally generated.

In older people and in pathological conditions there are several other contenders for submovements beside a primary submovement and corrective submovement. Firstly, if the multiple limb segments are not coordinated perfectly this can also lead to irregularities in the movement traces, not directly attributable to a separately generated submovement. Additionally, multiple forms of tremor (e.g., rest, action, and essential) all add irregularities to the movement trace and are a form of overlaid unintentional submovements. However, use of criteria that a detection has to be significant in comparison to jitter at rest, which in many cases causes the algorithm to treat v_{peak} as the peak velocity of the submovement, means that in many cases the algorithm is likely over-estimating the size of the primary submovement in subjects with tremor.

More sophisticated submovement decomposition methods [23], [24], in combination with additional sensors attached to all upper-limb segments in the analysis, may be able to tease out the contributions of the different joints and the components of the movement that are due to a form of tremor. This will offer greater insight into how the production and size of submovements change in people with Parkinson's disease, and may also offer an objective measure to quantify motor improvements due to treatment.

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