Mode Detection in Switched Pursuit Tracking Tasks: Hybrid Estimation to Measure Performance in Parkinson's Disease

Meeko M. K. Oishi, Ahmad Ashoori, and Martin J. McKeown

Abstract— **Parkinson's disease (PD) is a neurodegenerative disorder that impairs motor skills, speech, and other voluntary movement, and may be associated with cognitive inflexibility. Fourteen PD subjects (both on and off medication) and 10 normal subjects performed a manual pursuit tracking task, in which the dynamics of the task suddenly change without explicit enunciation. The task dynamics have three modes, in which the error (the difference between the target and the user's cursor) is attenuated, exaggerated, or unchanged – hence we model the subject performing the tracking task as a hybrid system with arbitrary switching. Second-order stochastic LTI models of tracking performance in each mode are first obtained through system identification. We then use a multiple model adaptive estimation (MMAE) algorithm to determine a) whether each subject successfully adapted to the sudden change in tracking dynamics, and if so, b) the delay in switching to the new mode. These parameters were analyzed for all subjects, and found to be statistically significant across groups. While normal subjects consistently detected the change in task dynamics, PD subjects show considerably more difficulty in detecting the switch (especially off medication), and did not switch into the new mode as quickly as normal subjects. Our results suggest that PD subjects have considerable impairment in adapting to changing motor environments.**

Keywords: hybrid systems, mode detection, MMAE, Kalman filter, Parkinson's disease, LTI systems, second-order systems, system identification

I. Introduction

Parkinson's disease (PD) is a common neuro-degenerative disorder of the central nervous system, and is characterized pathologically by premature loss of cells that produce the chemical dopamine. Clinically, tremor, rigidity, bradykinesia (slowness of movement) and postural instability, can all be seen. While early descriptions empasized that the mind was spared, as people are living longer with the disease, it is becoming increasingly recognized that cognitive aspects may be affected. For example, cognitive "inflexibility" whereby subjects have difficulty flexibly changing strategies during performance of a task. Early in the disease, motor symptoms can be markedly improved by L-dopa medication, and while this may improve cognitive inflexbility, it may result in enhanced impulsivity [1].

The motor cortex of the brain, which ultimately drives the muscles via the spinal cord and peripheral nerves, is modualted by two major systems: the basal ganglia and cerebellum [2]. While Parkinson's disease has been considered a classic basal ganglia disease, connectivity between these two systems imply that both systems are affected. These two systems are critical in feedforward and feedback processes that enable dextrous motor control, adaptation to changing environments, and effective executive function. Optimal control and state estimation via a Kalman filter have been proposed to model the feedforward and feedback processes in the brain, such that discrepancies between actual and expected sensory consequences of motor actions are used to improve motor performance [3], [4], [5], [6]. While common measures of motor performance (e.g., average speed, maximum speed, root mean square error, delay in tracking tasks [7], [8], [9], [10], [11]) and of executive function (e.g., Wisconsin Card Sorting Task [12], [13]) often show poorer performance in Parkinson's disease, the exact mechanisms responsible are not well known.

Our work focuses on the use of control theoretic measures to help elucidate mechanisms in the brain in Parkinson's disease. Our approach builds on an input-output view of tracking tasks to create dynamical models in which the human is essentially a black-box [14], [15], [4], [16]. In our previous work [17], alternative measures of motor performance (based on second-order linear dynamical models of manual pursuit tracking) provided some insight into compensatory mechanisms used in PD to overcome the faulty feedback paths through the basal ganglia and the cerebellum. Gross measures such as root mean square error were not sensitive enough to distinguish overall performance across groups. While RMS tracking error in PD was similar to that of normal subjects, *how* the two groups accomplished tracking was quite different. We found that PD subjects' tracking error was significantly overdamped as compared to normal subjects, and that the effect of L-dopa was to decrease the damping ratio. In this paper, we build upon our prior work, but focus on the problem of detection of a sudden change in the tracking task.

Researchers have investigated a variety of methods to quantify behavioral performance in Parkinson's disease. Some recent efforts have focused on identifying large-scale effects, such as gait [18], [19], joint force [20], or limb movement [21], while other efforts have focused on small-scale effects, such as fine motor control [22], [23], [24].

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Some researchers have focused on modeling and analysis at the neuronal level [3], [5], [25]. Beyond mere analysis, the main goal of work at each of these physical scales is often to identify biomarkers or improve measures of disease severity for use by clinicians [23].

The issue of flexibility, or adaptation to sudden change, has been investigated in a variety of experiments [26], including the widely-used Wisconsin Card Sorting Task (WCST). In the WCST, the player receives a reward for correctly sorting cards according to criterion that can be learned as the game is played. However, at some point during the task, the rules for sorting change (without enunciation to the subject), and the player must learn the new rules in order to maximize their reward. PD subjects often have difficulty re-learning the rules, and continue to play by the old rules even though they may incur a loss for doing so. Similar ideas have been explored in other discrete task experiments [27], [28] through the use of cues that prime the subject to evaluate the relative merit of internally generated information as opposed to observed information. In continuous tracking tasks, visual or proprioceptive feedback is skewed for an entire task from what would normally be expected [9], [11].

We explore the idea of sudden change in manual pursuit tracking because continuous-time models can provide insight into compensatory mechanisms in PD. There are three possible "modes" of pursuit tracking in our experiment, based on whether the visual feedback of the actual tracking error is attenuated, exaggerated, or unchanged. Each mode is modeled as a second-order continuous system with white noise. Hence with sudden mode changes, the problem of pursuit tracking can be modeled as one of hybrid estimation and control. The human must regulate an error trajectory whose dynamics may suddenly change. Since the mode change is not explicitly announced to the subject, the subject must also estimate the current mode in order to maximize tracking performance. We draw upon multiple model adaptive estimation (MMAE) [29], [30], [31] to identify a) whether the subject can adapt to the sudden change in dynamics, and b) the time it takes to do so – that is, to identify a) the current mode, and b) the delay in detecting the correct mode after a mode change. The perceived unpredictability of the continuous-time tracking target is handled through the use of a Kalman filter, as in [32], [4], [6].

The novelty of our work relates to a) the application of hybrid estimation techniques to a manual pursuit tracking task with multiple modes, and b) a statistically significant assessment of the presence and delay of mode detection in PD compared to normal subjects. In Section II the experiment is described in detail. Three second-order LTI models are numerically identified using Matlab's System Identification Toolbox [33] for each subject, one for each of the three tasks. Section III describes the creation of Kalman filters and implementation of the MMAE algorithm for each subject. Results of this analysis are presented in Section IV along with discussion of their biological significance. Lastly, Section V provides conclusions and directions for future work.

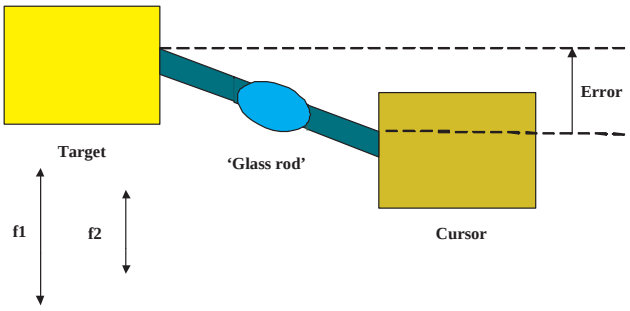

Fig. 1. Experimental setup. The target trajectory is $u(t) = \sin(f_1 t) + \sin(f_2 t)$. Users are instructed to keep the cursor $y(t)$ level with the target. The error $u(t) - y(t)$ is scaled by 0.3 in 'Better' mode, by 2.0 in 'Worse' mode, and unchanged in 'Normal' mode.

II. Experimental Setup and Model Creation

A. Experiment Description

Fourteen PD subjects (on and off L-dopa medication) with clinically diagnosed, mild to moderate PD and ten healthy, age-matched subjects without active neurological disorders were recruited for this study at the Pacific Parkinson's Research Centre at the University of British Columbia at Vancouver, Canada, after first providing informed consent (a full description of the experimental setup can be found in [17]). Subjects were asked to perform a tracking task by using a joystick in response to visual stimuli displayed on a computer screen, as shown in Figure 1. A horizontal "glass rod" connecting two boxes (each 60mm $\times$ 45mm) was shown on the display, where the box on the left (Target) oscillated in the vertical direction at a linear combination of two frequencies (f_1 and f_2), thus giving it a smooth but fairly complex appearing motion. Subjects were instructed to move the box on the right (Cursor) by using the joystick so that the glass rod remained horizontal at all times. All subjects practiced for 5 - 10 minutes, during which time f_1 and f_2 were determined for each subject's hand to maintain an error rate between 60 – 70% of the time. The individually determined frequencies, f_1 and f_2, were then held constant throughout the rest of the study.

PD subjects performed the task once after an overnight withdrawal (minimum of 12 hours since their last dose of L-dopa, minimum of 18 hours since the last dose of dopamine agonists) of their anti-Parkinson drugs and again one hour after admission of L-dopa.

Part 1: Over a single 90-second interval, a sequence of three separate tracking tasks was performed, with a short delay (5-10 seconds) between each task to mark its end. In each task, the visual feedback of the actual tracking errors was either amplified, attenuated or unaltered (but did not switch between the three options). In the 'Normal' task, the vertical distance between the target and cursor displayed on the monitor reflected the true error generated by the subject. In the 'Better' task, this distance was artificially reduced on the computer screen to 30% of the true error. In practice, the attenuation essentially made the tracking error better than expected. Finally, in the 'Worse' task, the the distance

between the target and the cursor was artificially doubled, making the tracking error worse than expected. Subjects performed eight sets of the 90-second intervals (e.g., a total of 8×3 tasks). Our previous work [17] focused solely on this part of the experiment.

Part 2: The same sequences of three different tasks was again performed over a single 90-second interval, but *without* a delay between tasks. The subject was not provided with any additional signal that might indicate that the task had changed. In effect, two unenunciated mode switches occurred in every 90-second interval. With a 10-second pause at the start of each interval, the first task lasted 20 seconds, and the remaining two tasks each lasted 30 seconds. This pattern was repeated eight times, resulting in a total of 8×2 mode switches. A total of 4 sequences were each tested twice: 'Normal-Better-Worse', 'Worse-Normal-Better', 'Better-Worse-Normal', and 'Better-Normal-Worse'.

B. Model Creation

Consider a discrete-time Markov jump linear system with three modes: 'Better', 'Normal', 'Worse'. In mode q the dynamics are given by

$$\begin{array}{rcl} x[k+1] & = & A_q x[k] + B_q u[k] + w[k] \\ y[k] & = & Cx[k] + D_q u[k] + v[k] \end{array} \tag{1}$$

with state $x \in \mathbb{R}^2$, input $u \in \mathbb{R}$, output $y \in \mathbb{R}$, and zero mean white Gaussian noise processes $w \in \mathbb{R}^2, v \in \mathbb{R}$ with covariances Q_q, R_q, respectively. We model the subject as a second-order LTI system in observer canonical form, consistent with previous work in manual pursuit tracking [15], [16].

Deterministic ARX models calculated in [17] via black-box LTI system identification were used to first estimate the measurement noise variance R_q, by computing the difference between predicted and actual outputs. Then grey-box identification was used to estimate constant matrices A_q, B_q, D_q and process noise covariance Q_q from the experimental data for each mode [33], [34] ($C = \begin{bmatrix} 1 & 0 \end{bmatrix}$). Each model was generated from Part 1 of the experiment, in which each task is completed separately. Four sets of input-output data were used to create the model, then the remaining four sets of input-output data were used to validate the model [35]. For all subjects, the average model accuracy (evaluated via Matlab's `compare` function [35]) was 80.91% $\pm$ 9.54%. Numerical issues due to the limited spectrum of the input signal are mitigated by the amount of data gathered.

A number of modeling frameworks were considered, however the chosen formulation has several advantages. 1) Previous work has established the Kalman filter as a reasonable model of the cerebellum [5]. 2) Previous work has established second-order linear systems as reasonable models of manual pursuit tracking [15], [16], [36]. 3) Using higher-order LTI systems or nonlinear systems with extended Kalman filters improves model accuracy only marginally. 4) The computational cost of switching becomes much higher with higher-order LTI systems or nonlinear systems.

III. Detecting Mode Changes

We reverse engineer the "best" switching sequence between modes, that most accurately reconstructs the experimental data. That is, *given the hybrid system with dynamics (1), and a known input/output sequence, we wish to determine the switching sequence which maximizes the likelihood that the estimated mode is the actual mode.*

A. MMAE Algorithm

Many variations of the multiple model adaptive estimation (MMAE) algorithm exist [29], [37], [38]; we apply one whose detection best matches reasonable switching times in normal subjects [39]. Residuals in each mode q

$$r_q[k] = y[k] - \hat{y}_q[k], \quad S_q[k] = E\{r_q[k] r_q^T[k]\} \tag{2}$$

are weighted adaptively to create a mode-dependent likelihood function. The predicted output

$$\hat{y}_q[k] = C\hat{x}_q[k] + D_q u[k] \tag{3}$$

is calculated according to a standard time-varying Kalman filter in mode q, with prediction and update components.

$$\begin{array}{rcl} \text{Prediction:} & & \\ \hat{x}_q[k|k-1] & = & A_q \hat{x}_q[k-1] + B_q u[k-1] \\ P_q[k|k-1] & = & A_q P_q[k-1] A_q^T + Q_q[k-1] \\ \text{Update:} & & \\ S_q[k] & = & CP_q[k|k-1]C^T + R_q[k] \\ K_q[k] & = & P_q[k|k-1]C^T S_q^{-1}[k] \\ \hat{x}_q[k] & = & \hat{x}_q[k|k-1] + K_q[k] r_q[k] \\ P_q[k] & = & (I - K_q[k]C)\, P_q[k|k-1] \end{array} \tag{4}$$

A posterior probability evaluator (PPE) generates a likelihood function

$$\Lambda_q[k] = \frac{1}{\sqrt{2\pi S_q[k]}} \cdot \exp\left\{ -\frac{r_q^2[k]}{2S_q[k]} \right\} \tag{5}$$

which is used to determine the probability $V_q[k]$ that mode q is the true mode $\mu[k]$. Since the true mode does not change very frequently, we choose

$$\begin{array}{rcl} V_q[k] & = & W_q[k-1] \\ W_q[k] & = & \frac{\Lambda_q[k] V_q[k]}{\sum_{j=1}^{3} \Lambda_j[k] V_j[k]} \end{array} \tag{6}$$

with final probability $W_q[k]$ of mode q, where $W_q[0] = \frac{1}{3}$ (since there are 3 modes of equal likelihood). Finally, the mode estimate

$$\hat{\mu}[k] = \arg\max_q W_q[k] \tag{7}$$

is the mode with the highest likelihood $W_q[k]$.

B. Implementing the MMAE

For each subject, three discrete modes correspond to the three sets of dynamics identified in Section II-B. The above MMAE algorithm was applied separately to each of the eight input/output sequences, resulting in 8 switching sequences $\hat{\mu}[k]$ for each subject.

One potential issue arose in considering the noise covariance of $w[k]$ and $v[k]$ in the Kalman filter (4). Normally,

the noise covariance is determined by heuristic, ad hoc approaches, which leads to the classical "tuning of the filter" problem. In the identifying system matrices (1), reproducing the output with a measurement noise of known covariance tends to create a large process noise. This in turn creates residuals (2) that are incapable of representing how well each model represents the actual output. Hence, we initialized the Kalman filters with noise variances approximately five orders of magnitude lower than originally calculated, in order to accommodate higher magnitude measurement noise. This significantly improved detection of the mode changes.

Finally, we note that since all dynamics were represented in observer canonical form, without process and measurement noise, it would be impossible to uniquely reconstruct the state trajectory for the autonomous system. Since for all mode pairs $p, q \in Q$, $\mathrm{rank}([\mathcal{O}_p\ \mathcal{O}_q]) \leq 2n$, any two modes may not be distinguishable [40]. However, as the discrete state jumps from mode p to mode q, the variance of the state will change, and hence so will the variance of the output.

IV. Results and Discussion

Amongst the four switching sequences ('Normal-Better-Worse', 'Worse-Normal-Better', 'Better-Worse-Normal', 'Better-Normal-Worse'), switching between 'Better' and 'Worse' tasks was the most obvious across all subjects. While switching between 'Better' and 'Normal' or between 'Normal' and 'Worse' was also evident in some subjects, we focus on switching that occurred from 'Better' to 'Worse' modes. Typical tracking performance for the 'Normal-Better-Worse' sequence is shown for a normal subject in Figure 2, for a PD subject off medication in Figure 3, and for a PD subject on medication in Figure 4. The top part of each of these figures shows the target position and cursor position (e.g., input u and output y), and the bottom part of each of these figures shows the switching sequence estimated according to (7). When switching occurs, large tracking errors immediately result in the new mode. This can be seen at $t = 20$ and $t = 50$ in Figures 2, 3, and 4. System matrices used to estimate the current mode for the particular subjects in these figures are listed in Table I.

A. Switching detection

The switch between 'Better' and 'Worse' tasks in the 'Normal-Better-Worse' sequence was detected by 10 out of 10 normal subjects (Table II). However, only 5 out of 14 PD subjects off medication detected the switch, while 7 out of same 14 PD subjects on medication detected the switch. As expected, proportionally far fewer PD subjects detected the switch than did normal subjects. Further, the effect of L-dopa is to increase the ability subjects to detect a switch, and hence make their performance slightly more similar to that of normal subjects.

Again, in 'Better-Worse-Normal' sequence, 10 out of 10 normal subjects detected the switch. Similarly, the 'Better' to 'Worse' mode transition was detected by only 4 out of 14 PD subjects off medication, and 9 PD subjects on medication. The variation in PD subjects who detected the switch pre- and post-medication may be due in part to fatigue [41], since more subjects improve after medication in the sequence in which the 'Better' to 'Worse' transition occurs earlier in the switching sequence.

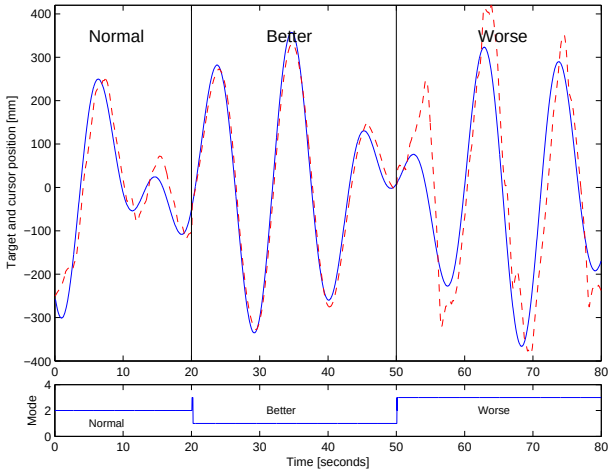

Fig. 2. Typical normal subject; the solid blue line represents target position, and the dashed red line represents cursor position. The switching delay is very small in comparison to the time scale of the entire 'Normal-Better-Worse' sequence. The subject detects both mode changes.

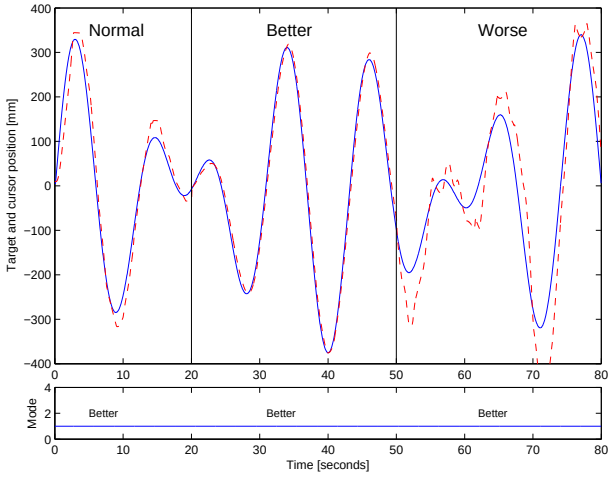

Fig. 3. Typical PD subject off medication; the solid blue line represents target position, and the dashed red line represents cursor position. The subject is unable to detect either mode change.

These results are consistent with the previous studies [12], [13], [9], [11] that demonstrate difficulty for PD subjects in adapting to sudden change. This difficulty may be attributed to the basal ganglia, which plays a significant role in switching from one task to another [12]. We also note many normal and PD subjects failed to detect mode changes between 'Normal' and 'Better' and between 'Normal' and 'Worse' tasks, perhaps because these tasks are more similar than 'Better' is to 'Worse'.

To determine whether the failure to detect switching was due to biological phenomenon or merely to poorly tuned switching algorithms, we implemented higher-order (and hence higher accuracy) models for subjects that did not detect

TABLE I

DYNAMICS FOR SUBJECTS SHOWN IN FIGURES 2, 3, AND 4, WITH $Q_q = \text{DIAG}(\alpha_q, 0)$.

Subject type	Fig	Matrices for mode prediction
Normal	2	$A_{\text{Better}} = \begin{bmatrix} 1.998 & 1 \\ -0.999 & 0 \end{bmatrix}, B_{\text{Better}} = \begin{bmatrix} 0.002 \\ -0.003 \end{bmatrix}, D_{\text{Better}} = 0.006, \alpha_{\text{Better}} = 2.2e7, R_{\text{Better}} = 1.1e4$
		$A_{\text{Normal}} = \begin{bmatrix} 1.994 & 1 \\ -0.994 & 0 \end{bmatrix}, B_{\text{Normal}} = \begin{bmatrix} 0.005 \\ -0.006 \end{bmatrix}, D_{\text{Normal}} = 0.004, \alpha_{\text{Normal}} = 7.1e7, R_{\text{Normal}} = 2.8e3$
		$A_{\text{Worse}} = \begin{bmatrix} 1.917 & 1 \\ -0.925 & 0 \end{bmatrix}, B_{\text{Worse}} = \begin{bmatrix} 0.039 \\ -0.031 \end{bmatrix}, D_{\text{Worse}} = 0.035, \alpha_{\text{Worse}} = 1.5e9, R_{\text{Worse}} = 3.9e3$
PD off med	3	$A_{\text{Better}} = \begin{bmatrix} 1.959 & 1 \\ -0.960 & 0 \end{bmatrix}, B_{\text{Better}} = \begin{bmatrix} 0.018 \\ -0.017 \end{bmatrix} D_{\text{Better}} = 0.018, \alpha_{\text{Better}} = 2.8e8, R_{\text{Better}} = 3.7e3$
		$A_{\text{Normal}} = \begin{bmatrix} 1.967 & 1 \\ -0.969 & 0 \end{bmatrix}, B_{\text{Normal}} = \begin{bmatrix} -0.044 \\ 0.046 \end{bmatrix}, D_{\text{Normal}} = 0.060, \alpha_{\text{Normal}} = 1.2e6, R_{\text{Normal}} = 2.6e3$
		$A_{\text{Worse}} = \begin{bmatrix} 1.874 & 1 \\ -0.874 & 0 \end{bmatrix}, B_{\text{Worse}} = \begin{bmatrix} 0.115 \\ -0.114 \end{bmatrix}, D_{\text{Worse}} = 0.134, \alpha_{\text{Worse}} = 4.3e7, R_{\text{Worse}} = 1.9e3$
PD on med	4	$A_{\text{Better}} = \begin{bmatrix} 1.991 & 1 \\ -0.993 & 0 \end{bmatrix} B_{\text{Better}} = \begin{bmatrix} -0.015 \\ 0.017 \end{bmatrix} D_{\text{Better}} = -0.001, \alpha_{\text{Better}} = 7.1e7, R_{\text{Better}} = 5.2e3$
		$A_{\text{Normal}} = \begin{bmatrix} 1.991 & 1 \\ -0.992 & 0 \end{bmatrix}, B_{\text{Normal}} = \begin{bmatrix} -0.004 \\ 0.005 \end{bmatrix}, D_{\text{Normal}} = -0.005, \alpha_{\text{Normal}} = 2.9e8, R_{\text{Normal}} = 1.8e3$
		$A_{\text{Worse}} = \begin{bmatrix} 1.907 & 1 \\ -0.914 & 0 \end{bmatrix}, B_{\text{Worse}} = \begin{bmatrix} 0.056 \\ -0.050 \end{bmatrix}, D_{\text{Worse}} = 0.055, \alpha_{\text{Worse}} = 2.2e8, R_{\text{Worse}} = 1.4e3$

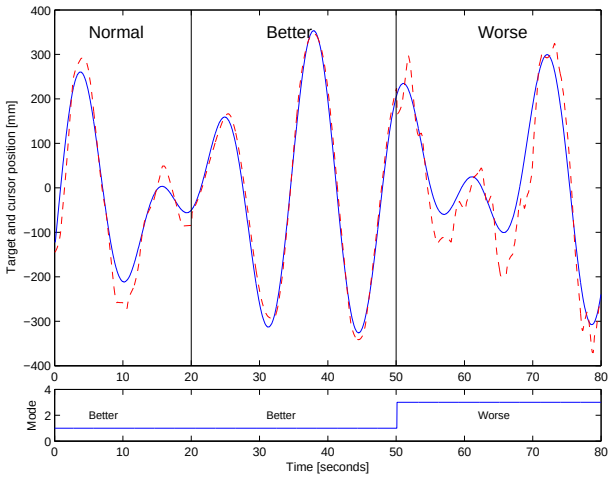

Fig. 4. Typical PD subject on medication; the solid blue line represents target position, and the dashed red line represents cursor position. This subject does detect the switch at $t = 50$s from 'Better' to 'Worse'.

switching. However, although the MMAE algorithm should be more likely to differentiate between these models, no additional switches were detected. Hence we conclude that the failure to detect switching is as legitimate portrayal of biological phenomenon.

B. Delay in switching detection

Latencies for the subjects capable of detecting switching between 'Better' and 'Worse' in the 'Normal-Better-Worse' sequence approached statistical significant across three groups (p = 0.07, ANOVA) as shown in Figure 5. For normal subjects, the delay had a mean value of 3.77 ± 0.83 time steps, while for PD subjects on medication the mean delay was 4.14 ± 0.89 time steps, and for PD subjects off medication the mean delay was 5.0 ± 0.70 time steps (see Table II). As expected, L-dopa decreased the amount of time required for PD patients to detect a mode change.

In the 'Better-Worse-Normal' sequence a similar pattern

TABLE II

SWITCHING DETECTION AND DELAYS BETWEEN 'BETTER' AND 'WORSE' MODES IN THE 'NORMAL-BETTER-WORSE' SEQUENCE.

	Normal	PD off med	PD on med	Statistical Significance
Number of subjects who detected the mode change	10/10	5/14	7/14	
Mean delay in switching detection [sampling units]	3.77	5	4.14	$p = 0.07$, ANOVA
Variance of delay in switching detection	0.83	0.70	0.89	$p = 0.88$, VARTEST

appeared, although not statistically significant (p = 0.3351, ANOVA). For normal subjects, the mean delay was 4.0 ± 1.41 time steps, while for PD subjects on medication the mean delay was 4.14 ± 1.34 time steps, and for PD subjects off medication the mean delay was 6.5 ± 3.69 time steps. So, the delays have been more homogenized (having less variance) after medication. The delay variance was significant across groups in this sequence (p = 0.05, variance test), which could be related to the homogenizing effect of the medication.

In [42], [43], reaction time was shown to be delayed in PD subjects during switching experiments. One interpretation is that PD subjects have a deficit in the ability to manipulate motor responses. Our results are consistent with this finding, for those subjects to detect switching at all – however, a key result of this work is that mode switching simply does not occur for many PD subjects, especially those off medication.

V. CONCLUSION

This paper described the application of second-order stochastic LTI models and an MMAE algorithm to the problem of mode estimation in a hybrid tracking task. A total of 24 subjects performed the task (10 normals, and 14 subjects with Parkinson's disease, both on and off medication). To the best of our knowledge, this is the first application of hybrid estimation techniques to characterize tracking performance

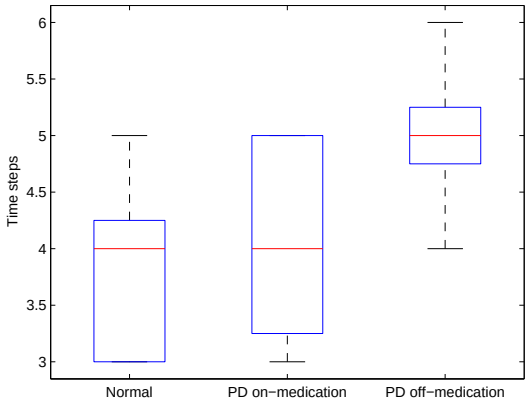

Fig. 5. The delay in switching detection for different groups in 'Normal-Better-Worse' block. Each time step is 0.03 seconds.

in PD. We found statistical significance across groups in mode detection between 'Better' and 'Worse' tasks, and found significantly fewer mode detections in PD subjects off medication than the PD subjects on medication, and near perfect detection in normal subjects. In addition, for subjects that did detect switching, PD subjects were slower to detect a mode change than normal subjects. Our results are consistent with related experiments in pursuit tracking and in flexibility. We believe that mode detection and switching delay provide more precise ways to quantify performance differences between PD and normals, with potential use as a marker for biological interpretation of mechanisms in the basal ganglia and cerebellum responsible for feedforward and feedback in motor control.

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