Encoding of Movement Dynamics by Purkinje Cell Simple Spike Activity During Fast Arm Movements Under Resistive and Assistive Force Fields

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Yamamoto K, Kawato M, Kotosaka S, Kitazawa S. Encoding of movement dynamics by Purkinje cell simple spike activity during fast arm movements under resistive and assistive force fields. J Neurophysiol 97: 1588–1599, 2007. First published November 1, 2006; doi:10.1152/jn.00206.2006. It is controversial whether simple-spke activity of cerebellar Purkinje cells during arm movements encodes movement kinematics like velocity or dynamics like muscle activities. To examine this issue, we trained monkeys to flex or extend the elbow by 45° in 400 ms under resistive and assistive force fields but without altering kinematics. During the task movements after training, simple-spike discharges were recorded in the intermediate part of the cerebellum in lobules V–VI, and electromyographic activity was recorded from arm muscles. Velocity profiles (kinematics) in the two force fields were almost identical to each other, whereas not only the electromyographic activities (dynamics) but also simple-spike activities in many Purkinje cells differed distinctly depending on the type of force field. Simple-spike activities encoded much larger mutual differences in the height of peak velocity. The difference in simple-spike activities averaged over the recorded Purkinje-cells increased ~40 ms before the appearance of the difference in electromyographic activities between the two force fields, suggesting that the difference of simple-spike activities could be the origin of the difference of muscle activities. Simple-spike activity of many Purkinje cells correlated with electromyographic activity with a lead of ~80 ms, and these neurons had little overlap with another group of neurons the simple-spike activity of which correlated with velocity profiles. These results show that simple-spike activity of at least a group of Purkinje cells in the intermediate part of cerebellar lobules V–VI encodes movement dynamics.

INTRODUCTION

Simple-spike (SS) activity of Purkinje cells, especially those in the cerebellar hemisphere of lobules IV–VI, has been reported to be modulated during reaching arm movements (Coltz et al. 1999; Fortier et al. 1989; Fu et al. 1997; Liu et al. 2003) and wrist movements (Gilbert and Thach 1977; Mano and Yamamoto 1980; Smith and Bourbonnais 1981; Thach 1968, 1970). However, it is controversial whether the SSs encode movement kinematics, such as the position, direction, and the velocity of movements, or movement dynamics like muscle activities and joint forces. During wrist movements, for example, Gilbert and Thach (1977) suggested that SS activity encodes movement dynamics, but Mano and Yamamoto (1980) reported that the SS activity correlated with the movement speed. Smith and Bourbonnais (1981) suggested that SS activity encodes movement dynamics during precision grips.

In more recent studies, however, no study has supported cerebellar SS coding of movement dynamics in arm reaching movements. Fortier et al. (1989) reported that the SS activity in a population of Purkinje cells collectively encoded movement direction during eight directional two joint arm movements. Ebner and his colleagues (Coltz et al. 1999, 2000; Fu et al. 1997) showed in eight directional visuomotor arm tracking movements that the SS discharge encodes movement velocity in addition to the hand position. The lack of evidence for movement dynamics in arm reaching movements is remarkable because it is often hypothesized in theoretical studies that the SS activity in the cerebellum represents inverse dynamics of arm movements (Kawato 1999; Kawato and Gomi 1992; Wolpert and Kawato 1998), and the SS activity in the cerebellum represents dynamic components of motor commands in ocular following eye movements (Gomi et al. 1998; Kawano 1999; Shidara et al. 1993; Yamamoto et al. 2002c).

Why is there no report about the association between cerebellar activity and movement dynamics during arm movements? A possible reason is that reaching movements involved movements around proximal joints of the elbow and the shoulder, which have not been examined in the previous studies that reported encoding of movement dynamics by SS activity (Gilbert and Thach 1977; Smith and Bourbonnais 1981). Another more plausible reason is that many studies during arm movements were not free from the correlation between movement kinematics and dynamics. Without removing this correlation, the relationship of neural activity with movement dynamics cannot be distinguished from its relationship with movement kinematics and vice versa (Todorov 2000). To see whether the movement dynamics in arm movements is represented in the cerebellum, it is essential to remove the correlation between movement kinematics and dynamics.

For this purpose, we trained monkeys to flex or extend their elbows by 45° in 400 ms under distinctly different force fields but without altering the movement kinematics. We used a resistive viscous force field and another negative viscous force field that assisted movements in proportion to the angular velocity of the elbow joint (Krouchev and Kalaska 2003; Wada et al. 2003; Yamamoto et al. 2000). After the animals were trained to achieve almost identical velocity profiles under the two force fields, we recorded SS activities from Purkinje cells...
in the intermediate part of lobules IV–VI. We focused on this part of the cerebellum because the area reportedly has connections with the primary motor cortex (Kelly and Strick 2003). Some neurons of primary motor cortex represent the dynamics of arm movements (Evarts 1968; Kakei et al. 1999; Kalaska et al. 1989; Scott and Kalaska 1997; Sergio and Kalaska 1997, 1998).

We here show that the disparity in SS activity of many Purkinje cells between the two force fields correlated with the difference in dynamics but not with the residual small differences in movement kinematics. We also show that the disparity in SS activity between two force fields appeared before the appearance of the difference in electromyographic (EMG) activities. From results of linear regression analysis, we further suggest that there are two groups of Purkinje cells, one representing movement dynamics and the other representing movement kinematics.

Some of the results reported here have been presented previously in abstract form (Yamamoto et al. 2000, 2002a,b).

METHODS

Subjects

Two male monkeys (Macaca fuscata, 10-11 kg, monkeys M and P) were used. The experiments were approved by the institutional committees for animal experimentation (National Institute of Advanced Industrial Science and Technology) and followed the Guiding Principles for the Care and Use of Animals approved by the Council of the Physiological Society of Japan.

Apparatus and task procedures

Each monkey was seated in a primate chair and placed the right arm on a single-joint manipulandum with a torque motor (Fig. 1). The manipulandum was developed in the Kawato Dynamic Brain Project (ERATO, JST). The same one was used by Krouchev and Kalaska (2003) and a similar one was used by Wada et al. (2003). The forearm and the wrist were fixed in a brace so that the monkey could make elbow flexion and extension movements without moving the wrist. A green cursor (hand position, 0.96° in diameter) and a white circle (target, 2.87°) were displayed on a CRT monitor that was 40 cm in front of the monkey and displayed a green cursor (hand position) and a white circle (target). The monkeys were required to move the cursor from the neutral position to the target by flexing or extending the elbow by 45°. During the movement, the torque motor generated a viscous force (τ) that was in proportion to the angular velocity of the elbow joint. The force was defined as

\[ \tau = -Wq \]

in which \( q \) denote the angle of the elbow joint (Fig. 1) and \( W \) denotes the coefficient of viscosity. In the experiments, we used two coefficients. One positive coefficient (0.25 Nms/rad) generated resistive, and the other negative coefficient (−0.25 Nms/rad) generated assistive force during the movements.

The two force fields were alternated every 10 trials. The number of flexions and extensions in each block of 10 trials fluctuated due to the completely random assignment of the movement direction. The background color on the CRT screen was switched according to the type of force field: it was black when the field was resistive and red when the field was assistive (Fig. 1) so that the monkey could tell which type of force field would be applied in the upcoming movement. Intertrial intervals were randomly variable (1–1.5 s). The animals carried out ~500 trials per day on average.

Training

The animals were initially trained for 30–50 wk without any force field with the black background color on the CRT. After the animals became able to carry out the task movements under the null field, the animals were trained under the assistive force field with the red background for 4–19 wk. Then we started training them under the null (black background) and the assistive (red background) force fields. The two fields were alternated every 12 trials at the beginning and every 10 trials thereafter. The null field was gradually replaced with the resistive force field by increasing the viscous coefficient day by day in 4–6 wk until the monkeys became able to switch between the two force fields.

Surgery

After training, animals were anesthetized with pentobarbital sodium, and a chronic recording chamber and head holder were implanted on the skull. The chamber was placed above lobule V of the cerebellum ipsilateral to the trained arm (right). Coronal and sagittal images of the head and the brain were taken beforehand with a 3T magnetic resonance scanner (GE-Sigma, Milwaukee, WI) to determine the position of implantation for each monkey. EMG electrodes were implanted subcutaneously on the long head of the biceps muscle of monkey M and on the long head of the biceps and the middle head of the triceps muscles of monkey P. Leads were passed through subcutaneous tunnels from each muscle to the site of a connector implanted on the monkey’s back (Miller et al. 1993).

Electrophysiology

The SS and complex spike discharges of cerebellar Purkinje cells were recorded extracellularly using high-impedance, glass-coated tungsten microelectrodes. Signals were amplified, digitized at 30 kHz, wave-form discriminated with a PC-based spike discriminator that applies a principal-component analysis to each spike form and converted to pulses before storage to computer at 2 kHz. Amplified signals were continuously recorded with a bandwidth of 10 kHz in a digital data recorder (TEAC, RD-125T) as a backup. Signals were continuously monitored through headphones and on an oscilloscope, and each discriminated spike form
was monitored on the PC display and two additional oscilloscopes. Purkinje cells were identified by the presence of spontaneous complex-spike discharge (Fig. 2B). The activities of 93 and 98 Purkinje cells were recorded from monkeys M and P, respectively. Of them, 62 and 34 yielded enough data for further analysis in that their SS discharges were recorded for >18 trials for both flexion and extension under each force field. To further confirm that the discharges were recorded from Purkinje cells, we calculated coefficients of variation of interspike intervals during a premovement period (−350 to −200 ms). Coefficients of variation ranged from 0.58 to 1.14 (mean, 0.80) in monkey M and 0.58 to 1.03 (mean, 0.78) in monkey P. These values were larger than those reported for granular layer units including presumed Golgi cells (0.04 to 0.37) (Miles et al. 1980). Figure 2B, C, and D exemplifies raw spike data recorded from a Purkinje cell (monkey M) and raster plots of SS and complex-spike discharges of the same Purkinje cell. We recorded from all Purkinje cells that were encountered, irrespective of their discharge characteristics.

The EMG signals were recorded from the biceps and triceps muscles at 2 kHz simultaneously at the time of neural recording. The EMG of triceps of monkey M was recorded with a surface EMG electrode.

Histology

At the conclusion of recordings in the monkeys, electrolytic lesions were made by passing DC (10 μA for 40 s) through the tip of the recording microelectrode. The animals were killed with pentobarbital and perfused through the heart with saline followed by 10% Formalin. The animal’s brain was removed, and frozen sections were cut at 50 μm in the sagittal plane, mounted on microscope slides, and stained with cresyl violet for cell bodies.

Using the lesions as a reference, recording sites were reconstructed from the serial sections. The recording areas are shown in Fig. 2A relative to a dorsal view of the cerebellar cortex. In both animals, the loci of the recordings were either anterior to the primary fissure in lobule V or posterior in lobule VI. In animal M (closed circles), the recordings were centered ∼7.5 mm to the right of the midline, and in animal P (open circles), they were centered 11 mm to the right of the midline. These recording sites overlapped with the area that was reported to be interconnected with the arm area in the primary motor cortex (Kelly and Strick 2003).

Data analysis

Stored data were analyzed off-line using MATLAB. The position (angle) signal of the manipulandum, measured by a high-resolution
encoder and recorded at 2 kHz simultaneously at the time of neural recording, was digitally differentiated to obtain velocity and acceleration that were filtered with a two-pole, 100-Hz low-pass Butterworth filter. EMG data were full-wave rectified and were filtered with a two-pole, 100-Hz high-pass Butterworth filter. The neural activities, movement velocities, and EMGs were aligned to the onset of arm movement that was defined as the time when both the movement speed and the acceleration exceeded thresholds of 6°/s and 200°/s².

Information analysis

To quantify the differences in velocity, EMG activity, and SSs under the assistive and resistive force fields, we calculated the mutual information between the type of force field and each parameter (velocity, EMG and SS). We calculated the information values in a given 50-ms time window (e.g., shaded area in Fig. 5A) and repeated the calculation as we moved the time window along the entire time course of elbow flexion and extension in steps of 0.5 ms. In each time window, we assigned four parameters to every trial: the angular velocity, the EMG signals for the biceps and triceps, and the count of SS within the 50-ms time window. To calculate the mutual information between one of the parameters and the type of force field, we divided all trials during recordings from each cell into a 2 × 2 contingency table according to whether the parameter was greater than or equal to the median or smaller than the median and whether the type of force field was resistive or assistive (Table 1). If the ith row and jth column of the 2 × 2 cell counts are nij, the mutual information I was calculated as

\[ I = \sum_{i,j} (n_{ij} - n_i n_j/N)(H_i - H_j) \]

where \( H_i \) and \( H_j \) are the marginal distributions, and \( N \) is the total number of trials.

### Comparison of latencies in SS and EMG modulations

To examine whether the onset of modulation of SS activities led or lagged that of the EMG modulations on average, we rectified the modulation of SS activity from the baseline firing rate in the following manner

\[ g(t) = |f(t) - f_{\text{control}}| \]

where \( f(t) \) is the temporal firing rate of each Purkinje cell at time \( t \) and \( f_{\text{control}} \) is the average of \( f(t) \) over a premovement period from 200 to 100 ms before the movement onset. \( f(t) \) was estimated for each Purkinje cell by smoothing the raw spike count histogram (0.5-ms bin) with a Gaussian filter (SD = 25 ms). The rectified modulation of SSs \( g(t) \) was calculated for each trial and averaged across all trials and then over all Purkinje cells in the two monkeys \( G(t) \). The onset of modulation was defined as the time when \( G(t) \) increased by 2 SD and reached the level during the premovement control period.

### SS activity in the first trials

We further examined whether the SS activity switches instantaneously in the first trial when the force field was switched from one to the other. For this purpose, we chose two (monkey M) and one (monkey P) Purkinje cells that showed distinct SS activity under the two force fields. These cells had mean mutual information (SS and force field) >0.25 bits from ~200 to 700 ms and had more than six first trials for both types of switching (resistive to assistive and vice versa). The mean SS activity in the first trials was compared with the SS activity averaged over all trials under the same force field (all trials) and that under the different (preceding) force field, by calculating correlation coefficients.

### Linear regression of EMG and velocity by SS activity

Temporal patterns of EMG activities and velocity profiles were reconstructed from single-cell SS activity to see whether the SS activity of each Purkinje cell resembled temporal patterns of the dynamics or those of kinematics of the movements. We reconstructed the temporal patterns (between −200 ms and 600 ms from the movement onset) from the SSs recorded from each Purkinje cells as follows
\[ m(t) = w \cdot f(t + d) + \text{bias} \]
\[ v(t) = w \cdot f(t + d) + \text{bias} \]
in which \( m(t) \), \( v(t) \), \( f(t) \), \( w_{ik} \), \( p \), \( d \), and \( d \) denote the EMG activity of the \( k \)th muscle at time \( t \), velocity at time \( t \), firing frequency of the \( i \)th cell at time \( t \), weighting coefficients of the \( i \)th cell on the \( k \)th muscle, weighting coefficients of the \( i \)th cell on velocity, the time lag from muscle activity to SS activity and the time lag from velocity to SS activity, respectively. Note that a negative time lag indicates that SS activity led muscle activity or velocity in time. Linear regressions were repeated with different time lags \( d \) that were changed from –200 to 200 ms with a step of 10 ms. The time lag that yielded the largest determination coefficient \( r^2 \) was chosen as an estimation. A single combination of \( w_{ik}, d \), and bias was estimated for each muscle to reconstruct all EMG activities in both flexion and extension under both force fields. Likewise, a single combination of \( w, d \), and bias was estimated to reconstruct all velocity profiles in both flexion and extension under both force fields.

**RESULTS**

**Velocity and EMG profiles after training**

After 4–6 mo of extensive training, the animals were able to flex and extend their elbows under the two distinct force fields without much altering the velocity profiles (kinematics). Figure 3A (top) shows, for example, that the mean velocity profile under the resistive force field (black curves) was almost identical to that in the assistive force field (red curves) in both flexion (left) and extension (right).

Figure 3B (biceps) and C (triceps) show the temporal profiles of EMG averaged over trials after extensive training (monkey M). It is evident that the EMG profiles of the agonists, the biceps in flexion (Fig. 3B, top left) and the triceps in extension (Fig. 3C, top right), were distinctly larger under the resistive force field (black traces) than under the assistive force field (red traces). This difference contrasts clearly with the average velocity profiles that were almost identical under the resistive (black curves in Fig. 3A) and assistive (red curves) force fields. Similar results were obtained from monkey P (not shown).

It is worth noting that there was little overshoot or undershoot in the peak velocity even in the very first trial when the force field was switched from the resistive to the assistive (red circles in top panels of Fig. 4) or from the assistive to the resistive (black circles). Whereas the peak velocity (kinematics) was maintained around a constant level between 200 and 300°/s in both force fields (Fig. 4, top), the mean EMG activity that represented movement dynamics alternated between larger and smaller values according to the type of force field. This change occurred at the first trial after the force fields were switched (Fig. 4, bottom, left: EMG of the biceps; right: EMG of the triceps). That is, the animals were able to switch between the two motor strategies, just by relying on the background color of the monitor (Fig. 1).

**SS activity under the two force fields**

Figure 5A (top) exemplifies the mean discharge rate of SSs that were recorded from a Purkinje cell in lobules V–VI (monkey M). In flexion (left), the mean discharge rate under the resistive force field (37 trials, black trace) was more than twice as large as that under the assistive force field (34 trials, red trace) in the first half of the movement (100–250 ms after the movement onset), showing a distinct difference. To quantify the difference, we calculated the mutual information between the type of force field and velocity (A, bottom) and between the type of force field and EMG activities (B and C, bottom). Traces are aligned at the onset of the movement (time 0).
In the case, we divided them into trials with SSs equal to or larger than 3 (spikes/50 ms) and those with SSs equal to or <2 (spikes/50 ms). Thus, we categorized the 71 trials in a $2 \times 2$ factorial manner, as shown in a contingency table (Table 1). The number of trials in each of the four cells [(16, 21; 31, 3)] was significantly different from the counts [(24, 13; 23, 11)] expected from the marginal distributions ($\chi^2$ test, $P < 0.0001$, df = 1, $\chi^2 = 16.2$). To describe more precisely, the two factors were not independent of each other, showing that we can to some extent predict the number of SSs from the type of force field or predict the type of force field from the number of SSs during this 50-ms period. The mutual information thus obtained was 0.20 bits (4.1 bits/s, see METHODS for calculation).

The largest possible value of mutual information was 1 bit (1 bit/50 ms). The information encoded by SS activity, it decreased as shown in Fig. 8 and it was not derived from the small residual difference of movement kinematics.

We also compared the onset of the disparity in SS activities (Fig. 5, top) however small they might seem. To test this possibility, we evaluated the difference in the velocity curve according to the type of force field using the same information measure. Significant differences in velocity profiles were detected as shown in the information curves (Fig. 3A, green traces in the bottom panels) that had peaks at $\sim150$ ms from the movement onset. It thus remains possible that the disparity in SS discharge reflected the small difference in kinematics.

To test whether the information measure in SS activity paralleled that in the velocity or that in EMG activity, we divided all trials under both force fields into two groups not by the force field but by the peak velocity (high- and low-velocity groups, Fig. 7A, top) and calculated the mutual information between the peak velocity and each parameter. This time, the difference in velocity profiles became larger (Fig. 7A, top), but the difference in EMG activities became much smaller (Fig. 7, B and C, top) than when the trials were divided according to the type of the force field (Fig. 3). Correspondingly, the information encoded by the velocity profiles became larger ($\sim12$ bit/s; Fig. 7A, bottom), but the information in EMG activities became smaller (Fig. 7, B and C, bottom) as compared with their counterparts in Fig. 3. As for the information encoded by SS activity, it decreased as shown in Fig. 8 and it correlated with the information encoded by EMG but not with the information encoded by the velocity.

The result shows that the information in SS shown in Fig. 6 was not derived from the small residual difference of movement kinematics but from the large difference of movement dynamics. Although we have shown that the SS activity reflected movement dynamics, we still do not know whether the SS activity led or lagged the EMG activity. To clarify this point further, we examined whether the onset of modulation in SS activity led or lagged that of EMG activity (Fig. 9, A and C). The modulation of SS activity crossed the 2 SD level at $64 \text{ ms}$. The results clearly show that the onset of SS modulation led the onset of the increase in muscle activity by $\sim40$ ms.

We also compared the onset of the disparity in SS activities with the onset of the disparity in EMG activities due to the difference in the force fields (Fig. 9, B and D). SS activity in the resistive force field started to differ from that in the assistive force field $79 \text{ ms}$ before the movement onset ($\sim79$...
ms, Fig. 9B), whereas EMG activities started to differ at ~55 ms. Again, the onset of changes in SS activities led the onset of changes in EMG activities. These temporal relationships suggest that the SS activities could be the origin of EMG activities at least in their initial part.

**SS activity in the first trials**

We further examined whether the SS activity switches instantaneously in the first trial when the force field was switched from one to the other. Panels in Fig. 10A show example data recorded from a Purkinje cell. Under the resistive force field (top right), SS activity showed a gradual decrease before the movement onset, and a further drop and succeeding quick recovery within ~100 ms after the movement onset. Under the assistive force field, on the other hand, the neuron showed a gradual increase before the movement onset, and an abrupt drop and gradual recovery in ~500 ms after the movement onset (bottom right). In the first trials after the force field was switched from assistive to resistive force field (top left), the time course of SS activity apparently resembled more the activity in the mean resistive force field (top left, $r = 0.73$, $P < 0.0001$) than the mean activity in the preceding assistive force field (bottom right, $r = 0.17$, $P = 0.43$). Likewise, SS activity in the first assistive trials (bottom left) showed a larger correlation with the mean SS activity in the assistive field (bottom right, $r = 0.81$, $P < 0.0001$) than with that in the resistive force field (top right, $r = 0.35$, $P = 0.10$). In three Purkinje cells that we examined (Fig. 10B), SS activity in the
first trials showed significantly greater correlation with the mean SS activity in the same force field (0.69 ± 0.19, mean ± SE) than with the mean SS activity in the preceding force field (0.18 ± 0.21; P < 0.01, paired t-test). The results suggest that SS activity switches instantaneously from the first trial.

Linear reconstruction of EMGs and velocity profiles from SSs

The results so far suggest that SS activity of at least a group of Purkinje cells in the recorded area encodes movement dynamics. However, it is still possible that SSs of others encode movement kinematics as reported previously (Coltz et al. 1999, 2000; Fortier et al. 1989; Fu et al. 1997). To examine this possibility, we further determined whether SS activity of each neuron had correlation to EMGs or velocity profiles.

First, EMG activities of each muscle were approximated by the SS firing rate recorded from each single Purkinje cell. A single pair of coefficients (w, d, and bias in Eq. 4) for each muscle was estimated to explain all EMG activities in both flexion and extension under both resistive and assistive force fields. Figure 11A shows the distribution of the estimated time lag (d) with the mode of ~80 ms. Estimated time lags were negative in the majority of Purkinje cells (61 and 70% for biceps and triceps, respectively, muscles in monkey M; 75 and 67% for biceps and triceps muscles, respectively, in monkey P), suggesting that the modulation in SS activities led the modulation in the EMG profile. The linear regression was
significant in all combinations of muscles and Purkinje cells ($P < 0.0001$) although the determination coefficient ($r^2$) was 0.17 on average and 0.52 at the largest (Fig. 11B).

When the velocity profiles were reconstructed from SS activity of single Purkinje-cells, the distribution of the estimated time lag distributed with the mode of $\pm 30$ and 0 ms (Fig. 11C). Estimated time lags were negative in around half of Purkinje cells (60% in monkey M, 48% in monkey P). The determination coefficient was 0.18 on average and 0.71 at the largest.

The results show that SS activity of individual Purkinje cell could explain a part of movement kinematics as well as a part of movement dynamics. To see whether Purkinje cells with SS activity that led movement kinematics were different from those that led movement dynamics, we thresholded the determination coefficients at 0.1 ($P < 10^{-15}$, uncorrected). Of the 60 Purkinje cells from the two monkeys, 49 (82%) led EMG activity of either biceps or triceps muscle (dynamics group) and 27 (45%) led velocity profiles (kinematics group) with an overlap of 16 (27%) between the two groups (Fig. 12A). The two groups of neurons seemed to overlap in the recorded area (Fig. 12B).

**DISCUSSION**

In the present study, we trained monkeys to carry out arm movements with almost identical velocity profiles (kinematics), but with distinct muscle activities (dynamics), by applying two distinctively different force fields. We showed that SS activity recorded from Purkinje cells in the intermediate part of the cerebellum (lobules V–VI) differed depending on the type of force field. We confirmed this quantitatively by showing that SS activities encoded much larger mutual information with the type of force field than that with the residual difference in the velocity profiles. The disparity in SS activities due to the difference in force fields appeared $\sim 40$ ms prior to the appearance of the disparity in EMG activities. We further found that SS activity of many Purkinje cells correlated with EMG activity with a lead of $\sim 80$ ms. These results suggest that SS activity of at least a group of Purkinje cells in the intermediate part of cerebellar lobules V–VI encodes movement dynamics that might be transformed into EMG activities with a latency of 40–80 ms.

**Dynamics or kinematics?**

Several previous studies have reported that SS activity of Purkinje cells in and around cerebellar lobule V correlated with movement kinematics (Coltz et al. 1999, 2000; Fortier et al. 1989; Fu et al. 1997; Mano and Yamamoto 1980). These studies do not necessarily contradict our finding even if we assume that SSs encode movement dynamics because movement kinematics are often correlated with movement dynamics.
(Todorov 2000). To conclude whether SSs are encoding movement kinematics or dynamics, it is critically important to remove the correlation between these parameters. We removed the correlation by training animals to carry out arm movements with almost identical kinematics, but with distinct dynamics, and showed that SS activity of some Purkinje cells in the intermediate part of cerebellar lobules V–VI encodes movement dynamics. However, this does not preclude the possibility that some Purkinje cells in the same area encode movement kinematics. By applying linear regression analysis, we indeed found that SS activity of some Purkinje cells led movement kinematics, whereas others led movement dynamics. This may suggest that both dynamics and kinematics are encoded by SS activity of Purkinje cells in the intermediate part of cerebellar lobules V–VI and that there are two groups of neurons representing dynamics or kinematics but with some overlap between the two. Outputs from the two groups of neurons may project to corresponding groups of neurons in the primary motor cortex (Kelly and Strick 2003), one encoding movement dynamics and the other encoding movement kinematics (Evarts 1968; Kakei et al. 1999; Kalaska et al. 1989; Scott and Kalaska 1997; Sergio and Kalaska 1997, 1998). The best way to confirm the existence of Purkinje cells that encode movement kinematics, however, is to carry out complementary experiments in which animals perform arm movements with almost identical dynamics but with distinct kinematics.

Coltz et al. (1999) reported that SSs in the intermediate and lateral regions of lobules V–VI correlated with the direction and speed of slow arm movements. They further emphasized that in most EMG recordings, EMGs in the slow arm movements did not correlate with the mean movement speed. From the results, they suggested that SSs encoded movement kinematics but not dynamics. However, 5 of 12 muscles (acromiodeltoid, biceps, flexor carpi radialis, spinodeltoid, and triceps) in their study did show speed-related modulation (Coltz et al. 1999). In addition, 45% (61 or 135) of EMG recordings were significantly fitted by a model that incorporated both movement speed and direction (Coltz et al. 1999). The percentage (45%) may be “far smaller” than the percentage of significant fit to velocity in SS discharge of Purkinje cells (70%, 92 of 132) but may not be so small as to exclude any possibility that the EMG activity of some critical muscles correlated with movement kinematics. It is worth noting again that encoding of kinematics would be verified by carrying out experiments in which animals perform task movements with identical dynamics but with distinct kinematics.

Recently Liu et al. (2003) dissociated a correlation between cursor and arm movements using a task in which a cursor on a monitor moved in the same or opposite direction to that of arm movements. They found two different groups of neurons in the lateral regions in and around cerebellar lobule V. The activities of neurons in one group correlated with the movement direction of the visual cursor movements (cursor-related neurons) and the activities of another group of neurons correlated with the direction of arm movements (task-related neurons). It is possible that task-related neurons encoded movement dynamics. Cursor-related neurons, on the other hand, might have encoded movement kinematics rather than dynamics. However, Liu et al. (2003) did not find significant trial-by-trial correlation between peak movement velocity and neuronal modulation levels for all cursor-related cells. Thus these cursor-related neurons may consist of a group of neurons that are different from those encoding arm movements per se. It is worth noting that most of these cursor-related neurons were found in the lateral part of the cerebellum (Liu et al. 2003) from which we did not record many neurons.

Mano and Yamamoto (1980) examined SS activity of Purkinje cells in the intermediate and lateral parts of lobules IV–VI during wrist flexion and extension. They reported that SS activity was often nonreciprocal in that the activity was increased (or decreased) in both directions in marked contrast to EMG activities of wrist-related muscles that were modulated reciprocally. The results of Mano and Yamamoto (1980) agree with those of the present study. For example, a Purkinje cell in Fig. 5C showed a nonreciprocal decrease during both flexion and extension under the resistive force field (black curves). However, the same neuron showed different patterns of activity under the assistive force field (red curves). Nonreciprocal modulation in one condition does not preclude the possibility that the neuron encodes movement dynamics.

Our finding that SS activity encodes movement dynamics during elbow movements agrees with previous findings in ocular and wrist movements (Gilbert and Thach 1977; Gomi et al. 1998; Shidara et al. 1993). Shidara et al. (1993) and Gomi et al. (1998) showed that SS activity recorded from Purkinje cells in the ventral paraflocculus can be reconstructed by an inverse dynamics representation of the eye movement. They concluded that these Purkinje cells primarily encode dynamic
components of the motor command. Gilbert and Thach (1977) examined SS activity during a wrist-movement task in which monkeys were required to keep a neutral position of a manipulandum by flexing or extending the wrist against a load perturbation. When the magnitude of the load was altered, the monkeys took 12–100 trials with the novel load before performing as regularly as previously. After the adaptation to the new load, wrist movements became similar to those in the preadaptation period, but SS discharge was altered. The results suggest that SS activity reflects load (dynamics) rather than the kinematics of the wrist movement though the kinematics of movements were not examined in detail. We verified SS encoding of dynamics in elbow joint movements by assuring that the velocity profiles were almost identical under distinctively different force fields.

It is worth noting in addition that it took 12–100 trials for the monkeys to adapt to the new load in Gilbert and Thach (1977), and it took as many trials for the SSs to achieve a postadaptation firing level. On the other hand, in the present study, the monkeys were able to switch between the two motor skills from the first trial after the force field was switched from one to the other. Accordingly SS activity was also switched from the first trial (Fig. 10). The difference might be due to the presence of the color cue in the present study and the absence of such a salient cue in Gilbert and Thach (1977). Contextual cues like distinct background colors may be essential or at least beneficial for the acquisition and switching of two independent motor skills under distinct force fields (Wada et al. 2003).

Transformation from SS to EMG

It has been reported that SS activity of many Purkinje cells in lobules V–VI led movement onsets of the arm, wrist, and fingers (Coltz et al. 1999; Fortier et al. 1989; Fu et al. 1997; Liu et al. 2003; Mano and Yamamoto 1980; Smith and Bourbonnais 1981; Thach 1968, 1970). When the onset of SS activity was compared with the onset of EMG activity, the lead time was 20–80 ms on average (Smith and Bourbonnais 1981; Thach 1970) among those neurons that led EMG onsets.

In the present study, the onset of modulation in SS activities led the onset of EMG modulation by 20–40 ms (Fig. 9). Reconstruction of the EMG activities from SS activities in single Purkinje cells had a delay of 80 ms on average (Fig. 11). These values (20–80 ms) were in good agreement with the mean leading time of SS activity in the previous studies (Smith and Bourbonnais 1981; Thach 1970).

Miller and his colleagues suggested that activity of multiple neurons in the primary motor cortex (Morrow and Miller 2003) and the red nucleus (Miller and Sinkjaer 1998) are summed into the muscle activity in the spinal cord. Because Purkinje cells are located upstream of these structures, it is not surprising that SS activity of a single Purkinje cell was not sufficient to reconstruct EMG activity (Fig. 11B). From the results of the present study and those of Miller and colleagues (Miller and Sinkjaer 1998; Morrow and Miller 2003), we suggest the possibility that activities of a group of Purkinje cells in the intermediate part of the cerebellum are transformed almost linearly into activities in the red nucleus and those in the primary motor cortex then summed into the muscle activity in the spinal cord.

Cerebellum and multiple motor skills

Our animals were able to switch their muscle activities and produce almost identical velocity patterns from the first trial after the force field was switched from one to the other (Fig. 4). This instantaneous switching was in agreement with previous studies in humans (Wada et al. 2003) and a monkey (Krouchev and Kalaska 2003) that used the same apparatus as in the present study. This type of instantaneous switching of motor skills under different force fields has been reported in experiments with salient (large field) color cues (Krouchev and Kalaska 2003; Osu et al. 2004; Wada et al. 2003) but not in studies without salient cues (Li et al. 2001; Padoa-Schioppa et al. 2002). Presentation of salient color cues (black and red) and extensive training in the present study must have contributed to the acquisition of instantaneous switching between two motor skills.

In explaining such ability to switch between multiple motor skills, it is hypothesized that the CNS learns and maintains multiple internal models and switches between them (Haruno et al. 2001; Kawato 1999; Osu et al. 2004; Wolpert and Kawato 1998). The present results agree with the theoretical framework that instantaneous switching is achieved by a responsibility predictor, which computes the responsibility signal, the gating signal, from only contextual information and can predictively switch between different internal models (Kawato 1999; Wolpert and Kawato 1998).

Imaging studies have suggested that the cerebellum is a candidate that acquires, maintains, and switches among multiple internal models (Imamizu et al. 2000, 2003, 2004). In theory, an inverse dynamics model that provides movement dynamics and also a forward kinematics model that outputs movement kinematics are acquired in pairs for controlling each motor skill (Haruno et al. 2001; Imamizu et al. 2004; Wolpert and Kawato 1998). Existence of two groups of neurons, representing dynamics or kinematics, suggests that SS activities in the intermediate part of cerebellar lobule V–VI constitute outputs of multiple internal models that provide different movement dynamics as well as kinematics. We also found that there are single Purkinje cells the SS activity of which fairly well explained the variance of EMG activity under the two force fields. The results suggest that some Purkinje cells represent correct motor commands that are utilized for movement control under different force fields. This agrees with one of two possibilities proposed in a previous imaging study (Fig. 8B of Imamizu et al. 2004); that is, the weighting from many
inverse-model outputs for computing the final motor command is carried out already in the cerebellum rather than outside the cerebellum.

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