

1 **The cerebellum does more than sensory-prediction-error-based learning in**
2 **sensorimotor adaptation tasks**

3 *Peter A. Butcher^{1,2}, Richard B. Ivry^{3,4}, Sheng-Han Kuo⁵, David Rydz⁵, John W. Krakauer⁶ and*
4 *Jordan A. Taylor^{1,2}*

5
6 ¹Department of Psychology and ²Princeton Neuroscience Institute, Princeton University; ³Department of
7 Psychology and ⁴Helen Wills Neuroscience Institute, University of California, Berkeley; ⁵Department of
8 Neurology, Columbia Medical Center, Columbia University; ⁶Department of Neurology, Johns Hopkins
9 University School of Medicine

10
11 Correspondence should be directed to:

12 Peter Butcher
13 Department of Psychology
14 428 Peretsman Scully Hall
15 Princeton University
16 Princeton, NJ 08544
17 Phone: 609-258-4432
18 Email: pbutcher@princeton.edu

19
20 Running head: Cerebellum and aiming in visuomotor rotations

21 Keywords: visuomotor rotation, cerebellum, implicit learning, explicit learning, aiming, motor learning

22 Figures: 5

23 Abstract: 250 words

24 Introduction: 565 words

25 Discussion: 1960

26
27
28
29 **Acknowledgements**

30 We thank Sam McDougle for feedback on the manuscript. P.A.B and J.A.T. were supported by
31 R01NS084948 from the National Institute of Neurological Disorders and Stroke and the Princeton
32 Neuroscience Institute's Innovation Fund. R.B.I. was supported by National Institutes of Health grants
33 NS074917 and NS092079. The authors declare no competing financial interests.

35 **Abstract**

36 Individuals with damage to the cerebellum perform poorly in sensorimotor
37 adaptation paradigms. This deficit has been attributed to impairment in sensory-
38 prediction-error-based updating of an internal forward model, a form of implicit learning.
39 These individuals can, however, successfully counter a perturbation when instructed
40 with an explicit aiming strategy. This successful use of an instructed aiming strategy
41 presents a paradox: In adaptation tasks, why don't individuals with cerebellar damage
42 come up with an aiming solution on their own to compensate for their implicit learning
43 deficit? To explore this question, we employed a variant of a visuomotor rotation task in
44 which, prior to executing a movement on each trial, the participants verbally reported
45 their intended aiming location. Compared to healthy controls, participants with
46 spinocerebellar ataxia (SCA) displayed impairments in both implicit learning and aiming.
47 This was observed when the visuomotor rotation was introduced abruptly (Exp. 1) or
48 gradually (Exp. 2). This dual deficit does not appear to be related to the increased
49 movement variance associated with ataxia: Healthy undergraduates showed little
50 change in implicit learning or aiming when their movement feedback was artificially
51 manipulated to produce similar levels of variability (Exp. 3). Taken together the results
52 indicate that a consequence of cerebellar dysfunction is not only impaired sensory-
53 prediction-error-based learning, but also a difficulty in developing and/or maintaining an
54 aiming solution in response to a visuomotor perturbation. We suggest that this dual
55 deficit can be explained by the cerebellum forming part of a network that learns and
56 maintains action-outcome associations across trials.

57

58 **New and noteworthy**

59 Individuals with cerebellar pathology are impaired in sensorimotor adaptation.
60 This deficit has been attributed to an impairment in error-based learning, specifically,
61 from a deficit in using sensory prediction errors to update an internal model. Here, we
62 show that these individuals also have difficulty in discovering an aiming solution to
63 overcome their adaptation deficit, suggesting a new role for the cerebellum in
64 sensorimotor adaptation tasks.

65 **Introduction**

66 Visuomotor rotation tasks, which induce a discrepancy between movements of
67 the limb and visual feedback, are a powerful tool for elucidating principles of motor
68 learning (for a review see Krakauer, 2009). While learning in these tasks has typically
69 been thought to be implicit, reflecting the training of an internal forward model via
70 sensory prediction errors (Mazzoni & Krakauer 2006; Tseng et al., 2007; Schlerf et al.,
71 2012), it has become clear that multiple processes can contribute to performance.
72 These include reinforcement learning (Huang et al., 2011; Nikooyan and Ahmed, 2015;
73 Galea et al., 2011), use-dependent plasticity (Diedrichsen et al., 2010; Verstynen and
74 Sabes, 2011; Huang et al., 2011), and instructed aiming strategies (Mazzoni and
75 Krakauer, 2006; Benson et al., 2011; Taylor and Ivry, 2011).

76 Most relevant to the current study is our recent work dissociating changes in
77 performance that arise from modifications in aiming, which can be explicitly reported,
78 and learning processes that are implicit, such as sensory-prediction-error-based
79 learning (Taylor et al., 2014; Bond & Taylor, 2015; McDougle et al., 2015). The latter
80 has been associated with the cerebellum, with compelling evidence coming from studies
81 showing that individuals with cerebellar pathology display significant impairments in a
82 range of sensorimotor adaptation tasks (Martin et al., 1996; Weiner et al., 1983; Rabe et
83 al., 2009; Schlerf et al., 2013; Smith & Shadmehr, 2005). For example, in visuomotor
84 rotation tasks, people with spinocerebellar ataxia (SCA) exhibit a reduced ability to
85 counter experimentally-imposed perturbations. Importantly, they show attenuated
86 aftereffects when the perturbation is removed (Schlerf et al., 2013; Werner et al., 2009).
87 This is consistent with the idea that an intact cerebellum is required to compute sensory
88 prediction errors via a forward model (Izawa et al., 2012; Miall & Wolpert, 1996; Haruno
89 et al., 2001). This hypothesis is further supported by neuroimaging in humans (Schlerf
90 et al., 2012) and neurophysiology in non-human species (Brooks et al., 2015) showing
91 cerebellar activity that is correlated with sensory prediction errors.

92 In the context of a visuomotor rotation, we define aiming as choosing to move the
93 hand towards a location other than the target, with the goal of having the cursor land on
94 the target. *A priori*, there is no reason to believe that aiming is cerebellar dependent.
95 Indeed, when individuals with cerebellar degeneration are provided with an explicit

96 aiming strategy and visual cues to support the implementation of that strategy, they
97 show near perfect performance in a visuomotor adaptation task (Taylor et al., 2010).

98 Given their ability to use an instructed strategy, it is puzzling that individuals with
99 cerebellar damage are impaired in visuomotor rotation tasks when they are not provided
100 with an explicit strategy. That is, why do they fail on their own to come up with an aiming
101 solution to compensate for their impaired implicit learning, especially given that the
102 perturbation results in a salient error that remains for many trials? This paradox
103 suggests that the cerebellum may be necessary, not only for implicit learning, but also
104 for discovering, implementing, and/or adjusting an appropriate aiming solution when it is
105 not provided directly through instruction (see also, Vaca-Palomares et al., 2013).

106 We have developed a simple method to assess trial-by-trial fluctuations in aiming
107 during visuomotor rotation tasks (Taylor et al., 2014) that allows for continuous
108 assessment of aiming behavior and implicit learning. In the current set of experiments,
109 we employed this method to determine the source(s) of impairment in individuals with
110 cerebellar damage.

111

112 **Materials and Methods**

113 *Participants*

114 Experiment 1: Ten individuals with spinocerebellar ataxia (SCA, average age =
115 53.7, SD = 12.6 years; 2 female; 5 right-handed) were recruited for the study at the
116 2012 National Ataxia Foundation Annual Meeting (San Antonio, TX) and from the
117 Berkeley, California community. SCA participants were only included if they did not
118 exhibit clinical signs of cerebellar-type multiple system atrophy or evidence of moderate
119 cognitive impairment. Four of the SCA group had a confirmed genetic subtype; for the
120 others, the diagnosis was sporadic adult onset ataxia of unknown etiology (Table 1).
121 The severity of ataxia was assessed with the International Cooperative Ataxia Rating
122 Scale (ICARS, Trouillas et al., 1997). The SCA participants had an average ICARS
123 score of 26.5 (SD = 14.7) out of a maximum score of 100. The participants were also
124 screened for cognitive impairment with the Montreal Cognitive Assessment (MOCA;
125 average = 28.1, SD = 1.5; all scored within normal range of 26-30) (Nasreddine et al.,
126 2005).

127 Twelve age-matched control participants were recruited from the Berkeley or
128 Princeton, New Jersey communities. These participants, based on self-reports, had no
129 known neurological conditions. The data from two of the controls were not included in
130 the final analysis: One consistently moved too slowly and the other failed to report the
131 aiming locations on many trials (see below). Thus, the control group consisted of ten
132 participants (average age = 59.7, SD = 14.7; SD = 1.4; 4 female; 10 right-handed). The
133 control participants were also administered the MOCA (average = 27.6, SD = 2.2), with
134 two scoring just below the normal range cutoff of 26 (23 and 25). They were included in
135 the analyses given that their performance was similar to the other control participants on
136 the experimental task.

137 Experiment 2: Twelve individuals with SCA were recruited from the Princeton
138 community and from the Columbia University Medical Center (CUMC). These
139 participants were selected after a clinical assessment determined that they did not
140 exhibit symptoms of extra-cerebellar pathology (Parkinson's disease, cerebellar-type
141 multiple system atrophy). One individual was excluded from the analysis after failing to
142 provide aiming reports, and a second was unable to complete the task in the time
143 allotted, resulting in a final dataset of ten participants (average age = 48.1, SD = 16.2
144 years; average years of education = 17, SD = 1.6; 4 female; 8 right-handed; 6 confirmed
145 genetic subtype; Table 1). The severity of ataxia symptoms was evaluated with the
146 Scale for the Assessment and Rating of Ataxia Severity (Schmitz-Hubsch et al., 2006).
147 The SCA participants had an average SARA score of 10.6 (SD = 6.2) out of a maximum
148 score of 40. The SARA scale was used in experiment 2 reflected the preference of the
149 CUMC neurologists. Due to time constraints, the MOCA was not administered to one of
150 the SCA participants. Surprisingly, eight of the nine remaining SCA participants scored
151 below the normal range on the MOCA (average score: 22.7, SD 4.8). In part, this may
152 reflect the fact that, for four of these participants, English was a second language. It is
153 also possible that this reflects a more severely compromised sample (although this is
154 not supported by their ataxia scores), or more stringent scoring criteria. Ten age-
155 matched control participants were recruited from the Princeton community (average age
156 = 53.3, SD = 10.5 years; 5 female; 10 right-handed). The control participants were

157 administered the MOCA (average = 25.5, SD = 1.1); five participants scored just below
158 the normal range (three 25 and two 24).

159 Experiment 3: Twenty young adults were recruited from the research participation
160 pool of the Department of Psychology at Princeton University (average age = 19.7, SD
161 = 1.5; 9 female; 20 right-handed).

162 The experimental protocols were approved by the Institutional Review Boards at
163 the University of California, Berkeley (experiment 1), CUMC (experiment 2), and
164 Princeton University (experiments 2 and 3). All participants provided written informed
165 consent. Participants in experiments 1 and 2 were paid an honorarium of \$20 per hour,
166 while participants in experiment 3 were compensated with class credit or \$12.

167

168 *Experimental Apparatus*

169 In all three experiments, participants made 7 cm horizontal reaching movements to
170 visual targets. The targets were displayed on a 15-inch (Exp. 1) or 17-inch (Exps. 2 and
171 3) LCD monitor. The monitor was mounted horizontally, positioned approximately 25 cm
172 above a digitizing tablet (Intuous Pro Large, Wacom). Given the position of the monitor,
173 vision of the limb was occluded. All participants, regardless of handedness, were tested
174 with their right hand. A digitizing pen was held in the right hand - regardless of
175 handedness preference - and the movement required sliding the pen across the surface
176 of the tablet. Feedback of hand position, when provided, was displayed in the form of a
177 circular cursor displayed on the monitor.

178

179 *Procedure*

180 We employed a variant of a visuomotor rotation task which requires participants to
181 verbally report their aiming location on each trial. This procedure has been described in
182 detail in a previous report (Taylor et al., 2014). At the start of each trial, a white ring was
183 presented, indicating the distance of the participant's hand from a start position (5 mm
184 circle at center of screen). By continually making the ring smaller the participant could
185 guide his or her hand to the start position. When the hand was within 1 cm of the center
186 of the start position, the ring was replaced by a cursor, allowing the participant to
187 precisely position the hand inside the start circle (Figure 1A).

188 After maintaining the start position for 1s, a green target circle (7 mm diameter)
189 was presented. The target appeared at one of eight locations, separated by 45° along
190 an invisible ring (radial distance from start circle of 7 cm). Each of the eight locations
191 was presented within a block of eight trials and, within a block, the locations were
192 randomly selected.

193 Participants were instructed to make a ballistic reaching movement, with the goal
194 of getting the feedback cursor to appear at the target location. Participants were
195 encouraged to reach beyond the target location, effectively shooting through the target.
196 We chose to have the participants reach past the target to minimize the impact of
197 intention tremor on accuracy, given that this symptom can become pronounced at the
198 end of a rapid movement. Feedback was presented as an endpoint location in
199 experiments 1 and 3. In these experiments, the cursor disappeared when the movement
200 amplitude exceeded the 5 mm start circle and did not reappear until the amplitude
201 reached 7 cm. Endpoint feedback was presented for 1.5 s at this location (subject to the
202 visual perturbation— see below) in the form of a red cursor (3.5 mm diameter). In
203 experiment 2, the cursor was visible during the outbound portion of the reach (online
204 feedback). Once the amplitude reached 7 cm, the cursor position was frozen for 1.5 s.
205 In all experiments, a pleasant “ding” sound was played if the feedback cursor
206 intersected the target region; otherwise a mildly aversive “buzz” sound was played.

207 To encourage participants to make fast movements, a digital vocal sample saying
208 “too slow” was played if the movement time was more than 400 ms. The movement
209 protocol, involving an emphasis on fast movements with limited feedback (Exps. 1 and
210 3), was adopted to minimize feedback corrections, a problem for individuals with SCA
211 (Tseng et al., 2007).

212 The visual workspace included a ring of numbered “landmarks”, spaced at regular
213 intervals of 5.6° (Figure 1A). The numbers increased and decreased in the clockwise
214 and counterclockwise directions, respectively, from the target. As such, the order of the
215 landmarks rotated with the target. Prior to each movement on aiming report trials, the
216 participant verbally reported the landmark they planned to reach towards. These verbal
217 reports were recorded by the experimenter. Trials in which the participants failed to
218 report their aim were excluded from the analysis. In experiment 1, the SCA participants

219 failed to provide a report on 15.0% of the report trials, while control participants failed to
220 provide a report on 1.8% of the trials. In experiment 2, the percentage of failed reports
221 dropped to 1.6% and 0.4% for the SCA and control participants, respectively. In
222 experiment 3, the college-age controls failed to provide reports on 1.1% and 1.0% of the
223 trials for the No-Variance and Variance-Added groups, respectively. Refinements in the
224 clarity of task instructions are likely responsible for the higher response rate in
225 experiments 2 and 3.

226 The experiment was divided into five blocks: baseline, baseline-report, rotation,
227 washout no-feedback, and washout with feedback. The participants first completed a
228 baseline block of 48 trials with veridical feedback (Figure 1B). The report task was then
229 described and participants completed eight trials, verbally reporting the aiming landmark
230 prior to each reach. Feedback was veridical on these trials (and participants almost
231 always reported the aiming location as “zero”).

232 The visual rotation was introduced in the rotation block. In experiment 1, this was a
233 45° counterclockwise rotation, imposed for 128 trials. In experiment 2, the rotation was
234 introduced gradually over 320 trials, with 0.144° added on each trial until the full 45°
235 counterclockwise rotation was achieved. For experiment 3, a counterclockwise rotation
236 was present for 128 trials. For one group of participants (No-Variance), the size of the
237 rotation was constant at 45°; for a second group of participants, the size of the rotation
238 on a given trial was drawn from a Gaussian distribution with a mean of 45° and a
239 standard deviation of 11° (Variance-Added). In all three experiments, participants
240 reported the aiming location prior to making their reach.

241 Following the rotation block, participants made an additional 40 reaches, but the
242 rotation, cursor feedback, and the aiming landmarks were removed (washout no-
243 feedback). For this block, participants were explicitly instructed to aim directly to the
244 green target. Neutral auditory feedback, in the form of a ‘knock’ sound, indicated when
245 the reach amplitude exceeded 7 cm, but otherwise provided no information related to
246 target accuracy. Veridical feedback was restored for a final 40-trial block (washout with
247 feedback).

248

249 *Movement analysis.*

250 All initial data analyses were performed using Matlab (MathWorks) and statistical
251 analyses were performed in SPSS (IBM, 2011). Task performance was assessed by
252 calculating the angular difference between the target and the initial heading angle of the
253 hand. A participants' hand location could drift within the start circle during the report
254 period of the trial, therefore we computed heading angle relative to the location where
255 their hand left the start circle. This was done by fitting a straight line between samples
256 taken at 1 and 3 cm from the start position (Taylor et al., 2014). We used initial heading
257 angle rather than endpoint angle to allow for a visual comparison between the online
258 and endpoint feedback conditions. The initial heading angle is also less susceptible to
259 noise that might come about from the SCA participants' ataxia.

260 For averaging across trials, movement trajectories were rotated to a common axis
261 (e.g., as though the target was always located at 0°). With this convention, a positive
262 angle indicates a deviation in the clockwise direction and a negative angle indicates a
263 deviation in the counterclockwise direction. Note that the heading angles are reported in
264 hand space rather than as target error. With this convention, hand heading angle will
265 change in the opposite direction of the rotation as performance improves.

266 The mean hand heading angle was calculated on an individual basis for four
267 different epochs: 1) The last eight trials of the baseline block, 2) the first and 3) last
268 eight trials of the rotation block (early and late rotation), and 4) the first eight trials of the
269 no-feedback washout block (washout). Trials were binned into groups of eight trials, we
270 report the mean and standard error of the mean for each bin. Since the eight targets
271 were presented in a random order within each cycle, this ensured that all targets were
272 equally represented in each bin. In experiment 1, one participant with SCA failed to
273 complete the last eight trials of the rotation block so the mean of their second to last bin
274 of eight trials was used. We did not fit an exponential function to the time series of hand
275 heading angles during the rotation block given the non-monotonic nature of the aim
276 report data (Taylor et al., 2014).

277 To obtain an estimate of implicit learning, the reported aiming angle was
278 subtracted from the measured hand heading angle on each trial. We refer to this
279 measure as implicit learning, since it could contain a number of processes in addition to
280 error-based updating of a forward model, such as use-dependent learning and

281 reinforcement learning. The mean aiming angle and implicit learning was calculated for
282 three different epochs: 1) The last eight trials of the baseline block, 2) the first and 3)
283 last eight trials of the rotation block (early and late rotation).

284 In terms of kinematics, we measured velocity and movement time. Velocity was
285 computed by submitting the hand position data to a fourth-order Savitzky-Golay filter
286 (Savitzky & Golay, 1964; Smith et al., 2000). Movement onset could not always be
287 based on the time at which the hand left the start circle because the hand occasionally
288 wandered from this position during the report phase. As such, movement onset was
289 estimated by a two-part procedure. We first identified the point in the time series where
290 the movement amplitude reached 2 cm from the start position. From this point, the time
291 series was searched backwards to find the time point when the participant either left the
292 start circle, or when the movement started from outside the start position, the time point
293 with the minimum radial distance from the start position.

294 We also excluded trials from the rotation block if the implicit learning estimate for
295 that trial was more than 3 standard deviations outside the median implicit learning
296 estimate for that participant. We used this criterion as a proxy to identify trials in which
297 the participant may not have provided an accurate report of their aiming location, or that
298 the movement itself was highly discrepant. Using this criterion, less than 1% of the trials
299 were excluded for the SCA participants over experiments 1 and 2 and control
300 participants in experiment 2. In experiment 1, control participants had 2.1% of trials
301 excluded. For experiment 3, 1.5% and 1.6% of the trials were excluded for the
302 Variance-Added and No-Variance groups, respectively.

303

304 *Power Analysis*

305 We performed a power analysis to estimate the minimum sample size required to
306 obtain an expected effect size, using the dataset from Taylor et al. (2010). Specifically,
307 we focused on two “pure” measures of implicit learning, the extent of hand angle drift
308 when participants were provided with an aiming strategy, and the magnitude of the
309 aftereffect, comparing these measures between SCA and control participants. Power
310 was estimated for an independent samples *t*-test, using a two-tailed α of 0.05 and power
311 of 0.95. Based on the group means and standard deviations from the measure of

312 implicit learning (i.e., drift) in Taylor et al. (2010), the effect size is $d = 2.63$ ($\mu_{\text{Control}} =$
313 11.3° , $\sigma_{\text{Control}} = 2.2^\circ$, $\mu_{\text{SCA}} = 5.9$, $\sigma_{\text{SCA}} = 1.9^\circ$). From this value, a minimum sample size
314 of five participants is required in each group. A similar estimate of sample size is
315 obtained using the aftereffect values ($\mu_{\text{Control}} = 6.2^\circ$, $\sigma_{\text{Control}} = 2.4^\circ$, $\mu_{\text{SCA}} = 0.3$, $\sigma_{\text{SCA}} =$
316 1.6° , $d = 2.89$). To be conservative, we doubled this number and recruited a minimum of
317 10 participants for each group for all of the experiments (Button et al., 2013).

318

319 **Results**

320 ***Experiment 1: Cerebellar damage impairs both aiming and adaptation to an*** 321 ***abrupt rotation***

322 Following a baseline block with veridical endpoint feedback, we introduced the
323 aiming report task, requiring participants to indicate the number corresponding to the
324 aiming location prior to each movement (Figure 1A). For the first eight trials in which
325 feedback remained veridical (baseline-report block), participants in both groups tended
326 to report aiming directly at the target (Control: $-0.5 \pm 0.3^\circ$; SCA: $-0.3 \pm 0.4^\circ$; group
327 comparison: $t_{18} = 0.5$, $p = 0.65$). Consistent with their aiming reports, the heading angles
328 of the reaches were directed towards the target with a small clockwise bias (Control: 2.9
329 $\pm 0.9^\circ$; SCA: $2.0 \pm 1.8^\circ$; group comparison: $t_{18} = 0.5$, $p = 0.65$). In sum, the behavior
330 during the baseline phase was similar between the two groups.

331 The introduction of the perturbation (rotation block) induced changes in heading
332 angle from baseline for both groups (Figure 2A). To examine the initial phase of
333 learning, we focused on the first eight trials. Participants with SCA displayed smaller
334 heading angles ($5.0 \pm 3.8^\circ$) over these trials compared to the control participants ($20.7 \pm$
335 3.5° ; Figure 2D). The difference in performance was even more pronounced at the end
336 of the rotation block. Over the final eight trials, the controls had almost completely
337 countered the perturbation ($41.3 \pm 5.0^\circ$); in contrast, SCAs were only partially
338 countering the perturbation ($16.5 \pm 7.9^\circ$). To determine whether there were any
339 differences in performance over the course of the rotation block, we performed a mixed
340 factorial repeated measures ANOVA with factors of Group (Control and SCA) and Time
341 (Early Rot and Late Rot). A main effect of time is expected, since participants should

342 compensate for more of the perturbation at the end of the rotation block than in the
343 beginning. There are two, non-mutually exclusive, ways in which SCA participants'
344 performance could differ from controls. SCA participants might be generally impaired in
345 compensating for the perturbation, relative to controls, in which case a main effect of
346 group would be expected. Additionally, SCA participants could be slower to respond to
347 the perturbation, in which case an interaction would be expected. The ANOVA revealed
348 a main effect of group ($F_{(1,18)} = 12.6$, $p = 0.002$) and a main effect of time ($F_{(1,18)} = 10.4$, $p =$
349 0.005). No Group X Time interaction was present ($F_{(1,18)} = 0.8$, $p = 0.37$). Thus, the
350 performance of both groups improved (cursor terminated closer to the target) over the
351 course of the rotation block, and the performance improvement was considerably
352 greater for the controls compared to the SCAs.

353 To measure the size of the aftereffect, participants completed a no-feedback block
354 in which they were instructed to aim directly to the target. Feedback in this block was
355 limited to an auditory tone, indicating when the movement amplitude had exceeded 7
356 cm. A comparison of the first eight trials of this block to the 8 baseline trials revealed a
357 reliable aftereffect for both controls ($12.2 \pm 1.3^\circ$; $t_9 = 5.5$, $p = 0.0004$) and SCAs ($7.1 \pm$
358 2.2° ; $t_9 = 3.0$, $p = 0.02$; Figures 2A, 2D). However, when comparing the two groups, the
359 magnitude of the aftereffect was significantly larger in the control group compared to the
360 SCA group ($t_{18} = 2.0$, $p = 0.03$). Additionally, our measure of implicit learning provides
361 complementary evidence that this process is impaired in the SCA group (see below).

362

363 *Verbal Reports*

364 The time series of the aiming reports revealed that a large portion of the
365 performance changes for the controls was due to a change in their reported aiming
366 location (Figure 2B). For control participants, the mean aiming location was shifted from
367 $13.3 \pm 3.3^\circ$ over the first eight trials to $22.1 \pm 5.9^\circ$ by the last eight trials of the rotation
368 block. On average, these values were attenuated in the SCA participants (Figure 2E).
369 Here, the early and late aiming angles were shifted from the target location by $1.6 \pm 2.8^\circ$
370 and $13.3 \pm 7.0^\circ$, respectively. As with hand heading angle, to determine whether there
371 were any differences in the verbal aiming reports over the rotation block, the aiming
372 reports for early and late in the rotation block were submitted to a mixed factorial

373 ANOVA with the same factors of Group (Control and SCA) and Time (Early Rot and
374 Late Rot). The ANOVA revealed an effect of Time ($F_{(1,18)} = 6.3$, $p = 0.03$), suggesting
375 aiming angles increased over the rotation block, but only a trend for an effect of Group
376 ($F_{(1,18)} = 3.2$, $p = 0.09$). No Group X Time interaction ($F_{(1,18)} = 0.1$, $p = 0.74$) was present.
377 From visual inspection of the aiming time series, control participants appeared to have
378 an initial large shift in aiming angle, which then began to decrease slowly over the
379 course of the rotation block. Given this non-monotonic nature of the aiming report time
380 series, as well as the high variance in the initial trials, we performed an additional *post-*
381 *hoc* analysis comparing the aim reports for the two groups across the whole rotation
382 phase. On this composite measure, the aiming reports indicated larger shifts in aiming
383 location for control ($25.7 \pm 3.8^\circ$) compared to SCA participants ($11.2 \pm 5.1^\circ$; $t_{18} = 2.3$, $p =$
384 0.035).

385 To estimate implicit learning, we subtracted the reported aiming angle from the
386 hand heading angle for each trial (Figure 2C). We again focused on the mean for the
387 first and last eight trials. For control participants, the estimate of implicit learning
388 increased from $2.8 \pm 1.4^\circ$ early in the rotation block to $14.6 \pm 2.3^\circ$ by the end. By
389 comparison, SCA participants had an estimate of no implicit learning (0.6 ± 2.1) early in
390 the rotation block, which only barely increased to $2.9 \pm 3.4^\circ$ by the end of the block. As
391 was done for hand heading angle and the verbal aim reports, to compare changes in
392 implicit learning over the rotation block, these values were submitted to an ANOVA with
393 factors of Group and Time. The ANOVA revealed significant main effects of Group ($F_{(1,18)}$
394 $= 5.6$, $p = 0.03$), resulting from generally impaired implicit learning for SCA participants,
395 and Time ($F_{(1,18)} = 17.7$, $p = 0.001$), due to implicit learning estimates increasing from
396 early to late in the rotation block. A Group x Time interaction ($F_{(1,18)} = 8.1$, $p = 0.01$) was
397 also present, suggesting the impaired implicit learning in SCA participants differed
398 relative to control participants between early and late in the rotation. As can be seen in
399 Figure 2F, the estimate of implicit learning was markedly lower for the SCAs, an effect
400 that was especially pronounced late in the rotation block. Thus, using both the estimate
401 of implicit learning during the rotation block and the aftereffect measure, adaptation was
402 impaired in SCA participants compared to controls.

403 In summary, the individuals with spinocerebellar ataxia exhibited a performance
404 impairment when presented with a 45° visuomotor rotation, similar to that observed in
405 previous studies of sensorimotor adaptation (e.g., Martin et al., 1996; Schlerf et al.,
406 2013). By obtaining verbal reports, we were able to dissociate adjustments in aiming
407 from implicit learning. The results indicate a dual-impairment: Not only was implicit
408 learning attenuated in the SCA participants, but they also tended to aim to locations
409 closer to the target during the early stages of the rotation block compared to controls.
410 While SCA participants did modify their aim, these adjustments failed to effectively
411 counter the rotation such that their overall performance only compensated for about half
412 of the perturbation by the end of training. Thus SCA participants were impaired in self-
413 discovery of an aiming strategy, which stands in contrast to our previous finding that
414 SCA participants are quite competent in carrying out strategy if it is provided by
415 instruction (Taylor et al 2010).

416

417 ***Experiment 2: Gradually introducing a rotation fails to alleviate the impairment in***
418 ***aiming and adaptation as a result of cerebellar damage***

419 With an abruptly introduced rotation, there appears to be two stages of aiming: an
420 initial large change in aim to compensate for the salient error induced by the
421 perturbation, followed by small trial-to-trial adjustments to maintain accurate
422 performance in the presence of small errors. It is likely that this first initial stage is the
423 selection and implementation of an explicit general aiming strategy, however, the extent
424 to which this second smaller adjustment phase is explicitly generated is less clear. It
425 may be that the small trial-to-trial adjustments are achieved by something like implicit
426 aiming, where an implicit mechanism is used to generate the adjustment to which
427 explicit access is gained afterwards. Providing an instructed aiming strategy would
428 direct the initial large change in aim, however, as adaptation increases and
429 compensates for more of the perturbation, to maintain accurate performance and offset
430 adaptation, small adjustments in aim may be necessary from trial-to-trial. Given that
431 when SCA individuals are provided with an instructed aiming strategy they can counter
432 an abrupt rotation, and maintain performance, they may not be impaired when only
433 small trial-to-trial changes in aim are necessary. To investigate this, in Exp. 2, we

434 introduced the perturbation gradually over the rotation block. Note, we extended the
435 rotation block from 128 to 320 trials and provided online cursor feedback as the
436 participants reached towards the target to increase the contribution of implicit learning.

437 For the first eight trials of aiming under veridical feedback (baseline-report block),
438 participants in both groups tended to report aiming towards the target (Control: $-2.4 \pm$
439 2.2° ; SCA: $-0.9 \pm 1.2^\circ$; group comparison: $t_{18} = 0.6$, $p = 0.56$). Consistent with their aim
440 reports, the heading angles for these first eight baseline trials were directed towards the
441 target with a small clockwise bias (Control: $1.4 \pm 0.6^\circ$; SCA: $3.9 \pm 1.6^\circ$; group
442 comparison: $t_{18} = 1.5$, $p = 0.15$). As in experiment 1, the emphasis on participants
443 making quick slicing movements towards the target resulted in relatively similar
444 behavior for both groups of participants.

445 Following the baseline block, a 45° counterclockwise visuomotor rotation was
446 gradually introduced over 320 trials. The small perturbation (only 1.15° on the 8th trial)
447 did not induce reliable changes from baseline in hand heading angle over the first eight
448 trials of the rotation block for either the controls ($-0.7 \pm 2.8^\circ$) or the SCA participants (1.6
449 $\pm 1.2^\circ$; Figure 3D). By the end of the rotation block, when the full 45° rotation was
450 present, both groups had adjusted their hand heading angles in response to the
451 perturbation (Figure 3A). This change, averaged over the last eight trials, was
452 substantially larger in the controls ($35.8 \pm 1.3^\circ$) compared to the SCA participants (18.2
453 $\pm 4.2^\circ$). As in experiment 1, to compare performance over the rotation block these
454 values were submitted to a mixed factorial ANOVA. The ANOVA revealed a main effect
455 of Group $F_{(1,18)} = 8.8$, $p = 0.008$), a main effect of Time ($F_{(1,18)} = 93.9$, $p < 0.0001$) and a
456 Time X Group interaction ($F_{(1,18)} = 13.3$, $p = 0.002$). Thus, as in experiment 1, the main
457 effect of Group reveals that SCA participants compensated less for the perturbation
458 than did control participants, showing that their performance impairment is observed
459 with both abrupt and gradual perturbations (see Schlerf et al., 2013). Additionally, the
460 presence of an interaction suggests that the relative impairment of SCA participants to
461 controls differed between early and late in the rotation. Visual inspection suggests the
462 difference in performance was larger late in the rotation block, which is expected given
463 that the gradual introduction of the perturbation resulted in only a $\sim 1^\circ$ perturbation
464 during this early phase.

465 On the no-feedback washout block, cursor feedback was withheld and the
466 participants were instructed to aim directly for the target. Comparing the first eight trials
467 of this block to the eight baseline trials revealed a reliable aftereffect for the controls
468 ($26.0 \pm 1.5^\circ$; $t_9 = 15.8$, $p < 0.0001$) and SCAs ($16.6 \pm 1.8^\circ$; $t_9 = 5.2$, $p = 0.0005$).
469 However, the magnitude of the aftereffect was smaller in SCA compared to control
470 participants ($t_{18} = 4.0$, $p = 0.0004$; Figures 3A, 3D). Thus, on this measure of implicit
471 learning, the SCA participants were impaired relative to the control group.
472

473 *Verbal Reports*

474 In contrast to experiment 1, the time series of the aiming reports revealed that only
475 a small portion of the change in hand heading angle for the controls was due to a
476 change in their aiming location (Figure 3B). Over the first eight trials of the rotation
477 block, their mean aiming location was $-2.4 \pm 2.3^\circ$. Note that this shift is in the
478 counterclockwise direction and would effectively increase the perturbation; we assume
479 this reflects noise or an attempt to negate the effects of intrinsic bias (Gibo et al., 2013;
480 Vindras et al., 1998 & 2005; Ghilardi et al., 1995). By the last eight trials, the aim was
481 shifted by $5.3 \pm 3.1^\circ$ in the clockwise direction, effectively helping to counter the rotation.
482 In contrast, the SCA participants did not consistently shift their aiming location over the
483 course of the rotation block (Figure 3E). Compared to baseline, they showed a shift of $-$
484 $1.7 \pm 0.8^\circ$ over the first eight trials and a shift of $-0.6 \pm 0.4^\circ$ over the last eight trials.
485 When comparing the verbal aiming reports with an ANOVA, the effect of Time was
486 significant ($F_{(1,18)} = 6.1$, $p = 0.024$), but the effect of Group was not ($F_{(1,18)} = 1.5$, $p = 0.24$).
487 There was a trend towards a Time X Group interaction ($F_{(1,18)} = 3.3$, $p = 0.085$).

488 We performed two additional post-hoc comparisons to quantify the extent of
489 aiming in the control and SCA participants. First, to determine whether aiming direction
490 changed over the course of the rotation block, a within subject *t*-test was conducted,
491 comparing aiming during the last 8 baseline trials and the last 8 rotation trials. By this
492 measure, the control participants adjusted their aim by the end of the rotation block ($t_9 =$
493 2.2 , $p = 0.051$), although this was only marginally significant. In contrast, the SCA
494 participants did not exhibit a reliable shift in aim ($t_9 = 0.3$, $p = 0.79$). Second, to compare
495 overall aiming between the two groups, a *t*-test was performed comparing the aim

496 reports averaged across the entire rotation phase. Using this measure, we observed a
497 reduced shift in aiming direction for the SCA group compared to the controls ($t_{18} = -2.1$, p
498 = 0.047). Thus, despite the relatively small changes in aiming observed for the control
499 participants, these comparisons suggest larger changes in aim for the control
500 participants compared to SCA participants. Indeed, the SCA participants failed to adjust
501 their aim in a consistent manner during the rotation block, despite the fact that the target
502 error became quite pronounced by the end of the block.

503 We employed the subtractive procedure to estimate implicit learning (Figure 3C),
504 and focused on the first and last eight trials during the rotation block for our statistical
505 analysis (Figure 3F). For control participants, the estimate of implicit learning increased
506 from $3.8 \pm 2.1^\circ$ early in the rotation block to $33.1 \pm 4.5^\circ$ by the end. By comparison, SCA
507 participants had an estimate of no implicit learning (-2.3 ± 3.1) early in the rotation
508 block, which increased to $13.2 \pm 5.5^\circ$ by the end of the block. An ANOVA on these
509 values revealed main effects of both Group ($F_{(1,18)} = 7.0$, $p = 0.016$) and Time ($F_{(1,18)} =$
510 59.7 , $p < 0.0001$). The main effect of group results from smaller implicit learning
511 estimates overall for SCA participants compared to controls. In addition, the Group X
512 Time interaction was significant ($F_{(1,18)} = 5.6$, $p = 0.030$), reflecting the fact that the control
513 participants had a larger increase in implicit learning over the course of the rotation
514 block compared to the SCA participants. This result converged with that observed in the
515 measure of the aftereffect, replicating the impaired adaptation for SCA participants
516 shown in experiment 1.

517 While our results are comparable across experiments 1 and 2, we caution against
518 drawing any strong inferences from any differences in results between the experiments.
519 First, there is the problematic nature of null results (the lack of a difference between
520 controls and SCA participants on the aftereffect data in experiment 1). Second, there
521 are substantial differences between the two tasks. In experiment 2, participants
522 completed more than twice as many rotation trials as in experiment 1, and received
523 online cursor feedback. We would expect both factors to enhance implicit learning (in
524 controls), offering greater sensitivity when comparing their performance to the SCA
525 participants.

526 The results of experiment 2 demonstrate that the SCA participants were impaired
527 in responding to a 45° gradual visuomotor rotation. As in experiment 1, their deficit
528 appears to be manifest in measures of both implicit learning and aiming. The aiming
529 deficit was apparent in experiment 2, even though only small adjustments in aiming
530 location are necessary from trial-to-trial to maintain performance. We note that, for both
531 groups, the amount of aiming was markedly attenuated in this experiment, and this may
532 have contributed to the fact that the target error remained substantial at the end of the
533 experiment. For controls, the final error was around 9°; for the SCA group, the final error
534 was around 27°. Despite this large error, the SCA participants failed to alter their aiming
535 locations; they were unable to compensate for their impairment in implicit learning by
536 deploying an aiming solution.

537 Given the variable performance of SCA participants in experiments 1 and 2, and
538 that SCA leads to highly heterogeneous damage to the cerebellum, it may be tempting
539 to map behavior to damage in cerebellar subregions or with specific subtypes of SCA.
540 However, given this variability, a larger sample size than we have here would be
541 necessary (see Kansal et al., 2016). With the current dataset, any conclusions involving
542 SCA subtypes, or more specific regions of the cerebellum, would likely be driven by
543 lesions and performance in only a few participants, where lesion reconstruction is
544 unlikely to yield reliable results (Rorden et al., 2007; Kimberg et al., 2007). Additionally,
545 correlating behavioral deficits to damage in cerebellar subregions is likely to be best
546 tested in individuals with focal lesions, where the pathology is more localized than the
547 broad deterioration in SCA.

548

549 ***Experiment 3: Higher motor variability does not account for the aiming and***
550 ***adaptation deficits due to cerebellar damage***

551 A feature of spinocerebellar ataxia is the presence of increased movement
552 variability. Indeed, even though we focused on the initial heading angle in experiments 1
553 and 2, the reaching movements for the SCA participants were more variable than the
554 controls. For example, limiting the analysis to the initial baseline block of experiment 1
555 (before the aiming task was introduced), the standard deviation of the heading angles
556 for the SCA and control groups were 11° and 5°, respectively. This increase in

557 movement variability may make it difficult for individuals with SCA to develop a reliable
558 aiming solution because they cannot converge on a consistent direction. To make this
559 concrete, consider the situation where an individual, after encountering a 45° clockwise
560 perturbation as in experiment 1, opts to aim to a location that is 30° in the
561 counterclockwise direction. Evaluating the utility of this aiming solution will be hampered
562 if the reach deviates widely from the selected trajectory (independent of adjustments
563 induced by implicit learning). Similarly, increased motor variability in experiment 2 might
564 make it difficult for the SCA participants to gauge the effects of the increasing
565 perturbation.

566 The impact of motor variance on learning has been explored in previous studies of
567 visuomotor adaptation (Wu et al., 2014; Therrien et al., 2015, He et al., 2016). In terms
568 of the effects of cerebellar pathology, Schlerf et al. (2013) found that individuals with
569 SCA exhibited impaired implicit learning, even when one considers how increased
570 motor variability might impinge upon learning and performance. Here we ask how an
571 increase in motor variability might influence the discovery of an aiming solution. Rather
572 than create conditions in which we directly manipulate motor variability, we added noise
573 to the movement feedback presented to adults and examined the effect this had as they
574 learned to respond to a 45° perturbation.

575 College-aged adults were randomly assigned to one of two groups in experiment
576 3. In the No-Variance group, the procedure was identical to experiment 1 with the
577 participants exposed to a constant 45° counterclockwise perturbation during the rotation
578 block. In the Variance-Added group, we (crudely) simulated the effects of ataxia by
579 pseudo-randomly varying the size of the rotation on each trial during the rotation block
580 (Figure 4). To this end, a noise term was added to the 45° perturbation. The size of the
581 rotation on each trial was based on a random sample from a Gaussian distribution with
582 a mean of 45° and standard deviation of 11°. The value of 11° was chosen because it is
583 the mean of the individual standard deviations of the hand heading angle for the SCA
584 participants in Experiment 1. Given that feedback is limited to the reach endpoint (at 7
585 cm), the participants in the Variance-Added group would experience a noisy 45°
586 perturbation.

587 During the initial baseline block (prior to reporting aim) the two groups had similar
588 standard deviations of their heading angles (No-Variance: $6.3 \pm 0.4^\circ$; Variance-Added:
589 $6.4 \pm 0.9^\circ$; $t_{18} = 0.04$, $p = 0.97$), suggesting there were no baseline differences in
590 movement variance between the two groups. For the first eight trials of aiming with
591 veridical feedback (baseline-report block), the participants in both groups tended to
592 report aiming towards the target (No-Variance: $-1.1 \pm 0.6^\circ$; Variance-Added: $0.1 \pm 0.2^\circ$;
593 group comparison: $t_{18} = 0.6$, $p = 0.56$). The hand heading angles were in the direction of
594 the target, although the Variance-Added group exhibited a significant, albeit small
595 clockwise bias ($2.7 \pm 0.7^\circ$) that was not observed in the No-Variance group ($0.0 \pm 0.5^\circ$,
596 $t_{18} = 3.1$, $p = 0.01$). Given that the two groups received identical (veridical) feedback in
597 these first two blocks, this difference likely represents chance variation in the
598 participants' baseline reaching bias (Ghilardi et al., 1995).

599 The 45° perturbation was introduced in the rotation block, along with the increase
600 in endpoint variance for the Variance-Added group. Both groups displayed adjustments
601 in heading angle in response to the perturbation (Figure 5A). These adjustments were
602 similar for the No-Variance and Variance-Added participants, both early (No-Variance
603 mean: $12.6 \pm 4.5^\circ$; Variance mean: $11.7 \pm 6.2^\circ$) and late in the rotation block (No-
604 Variance mean: $44.2 \pm 1.2^\circ$; Variance-Added mean: $41.8 \pm 6.6^\circ$; Figure 5D). As in
605 previous experiments, a mixed factorial ANOVA was used to compare learning over the
606 rotation blocks, although in this case the factor of Group is comparing Variance-Added
607 and No-Variance groups. There was a main effect of Time ($F_{(1,18)} = 76.1$, $p < 0.0001$), but
608 no effect of Group ($F_{(1,18)} = 0.07$, $p = 0.80$) nor a Group X Time interaction ($F_{(1,18)} = 0.04$, p
609 = 0.84), thus no differences in performance were present between the groups. The
610 groups also exhibited similar aftereffects in the no-feedback block in which they were
611 instructed to aim directly for the target. Both groups exhibited a significant aftereffect
612 (No-Variance: $6.3 \pm 0.9^\circ$; $t_9 = 4.8$, $p = 0.0009$; Variance-Added: $7.9 \pm 0.8^\circ$; $t_9 = 5.2$, $p =$
613 0.0006), and the magnitude of the aftereffect was similar for the two groups ($t_{18} = 1.4$, p
614 = 0.90). These results suggest that the imposition of added endpoint variance did not
615 impact overall performance in response to a visuomotor perturbation.

616 Per experimenter instructions, participants in both groups moved rapidly, and there
617 was no difference in movement duration between the groups during the rotation block
618 (No-Variance: 274 ± 57 ms; Variance-Added: 214 ± 14 ms; $t_{18} = 1.02$, $p = 0.32$).
619

620 *Verbal Reports*

621 The aiming report data reveals that a large portion of learning was due to an
622 adjustment in aiming location (Figure 5B), and that this effect was comparable for the
623 two groups. For the No-Variance group, the mean aiming location was shifted $7.3 \pm 5.0^\circ$
624 over the first eight trials, and increased to $34.8 \pm 1.4^\circ$ by the last eight trials of the
625 rotation block. Participants in the Variance-Added group had an initial shift of $12.3 \pm$
626 5.0° , which increased to $30.8 \pm 5.8^\circ$ by the last eight trials of the rotation block. A mixed
627 factorial ANOVA comparing aiming over the rotation block revealed the main effect of
628 Time was significant ($F_{(1,18)} = 33.3$, $p < 0.0001$), with participants increasing the angle of
629 their aiming over the course of the rotation block (Figure 5E). However, there was no
630 main effect of Group ($F_{(1,18)} = 0.01$, $p = 0.92$) nor a Group X Time interaction ($F_{(1,18)} = 1.3$, p
631 = 0.27). While the overall pattern suggests that the aiming shift over the entire rotation
632 block was larger for the No-Variance group ($33.7 \pm 2.3^\circ$) compared to the Variance-
633 Added ($29.0 \pm 3.9^\circ$) group, this difference was not significant (comparison of means
634 taken over the whole rotation block: $t_{18} = 1.0$, $p = 0.31$). Indeed, the group difference
635 reflects the performance of one participant in the Variance-Added group who only
636 reported small aiming angles.

637 Implicit learning was estimated for each trial by subtracting the aiming angle from
638 the hand heading angle (Figures 5C, 5F). Participants in the No-Variance group had an
639 estimate of implicit learning that increased from $5.3 \pm 1.5^\circ$ early in the rotation block to
640 $9.4 \pm 1.3^\circ$ by the end. In the Variance-Added group participants had an estimate implicit
641 learning that increased from 0.0 ± 1.6 early in the rotation block to $9.4 \pm 1.4^\circ$ by the end
642 of the block. Implicit learning was compared with an ANOVA based on the first and last
643 eight trials of the rotation block, revealing a main effect of Time ($F_{(1,18)} = 26.0$, $p =$
644 0.0001), but no main effect of Group ($F_{(1,18)} = 2.9$, $p = 0.11$) and only a trend for an
645 interaction between these factors ($F_{(1,18)} = 4.2$, $p = 0.055$). While the group effect and
646 interaction were not significant, a post-hoc comparison of the groups early ($t_{18} = 2.5$, $p =$

647 0.02) and late ($t_{18} = 0.0$, $p = 0.99$) in the rotation block, suggests that while participants in
648 the Variance-Added group may have initially had less implicit learning early during the
649 rotation block, their implicit learning caught up to the No-Variance group by the end of
650 the rotation. When a further post-hoc comparison is made of implicit learning estimates
651 averaged across the entire rotation block, no difference was present between the No-
652 Variance group (7.2 ± 0.9) and the Variance-Added group (6.5 ± 1.2 ; $t_{18} = 0.5$, $p = 0.62$),
653 confirming no difference in overall implicit learning was present.

654 In summary, the results of experiment 3 show that the injection of random endpoint
655 noise had only a modest effect on performance. Interestingly, the effect of noise was, at
656 least in terms of overall performance, limited to a possible slight reduction in the
657 magnitude of implicit learning as estimated early during the rotation block, but no
658 differences were present late in the rotation block, nor in aftereffect. There were also
659 differences between the groups in how they modified their aim on a trial-by-trial basis,
660 with the Added-Variance group continually modifying their aiming location in response
661 to the added noise. Nonetheless, these participants were able to learn the mean of the
662 45° rotation and they changed their mean aiming angle to a similar degree as
663 participants who experienced a constant perturbation. Taken together, these results
664 suggest that an increase in endpoint variability does not result in profound deficits in
665 implicit and aiming components of visuomotor learning. Thus, the difficulty exhibited by
666 individuals with SCA in adopting an appropriate aiming solution is likely unrelated to
667 their increased movement variability.

668

669 **Discussion**

670 Previous work has repeatedly demonstrated that individuals with damage to the
671 cerebellum are impaired in sensorimotor learning. The emphasis in this literature has
672 focused on implicit deficits in error-based learning. However, here we have shown that
673 SCA also leads to an impairment in the ability to discover and maintain an aiming
674 solution to offset a visuomotor perturbation.

675

676 Impaired aiming in SCA

677 To study the use of aiming behavior, participants reported their aiming location
678 prior to each reach (Taylor et al., 2014). This task provides a direct probe on the use
679 and evolution of an aiming solution and, by subtractive logic, a continuous estimate of
680 implicit learning. We observed a dual-deficit in participants with SCA when presented
681 with a visuomotor rotation. Not only did the SCA group exhibit impaired implicit learning,
682 they also showed a failure to aim to counter the observed target error. They adjusted
683 their aim across the perturbation block, choosing locations that tended to be in the
684 appropriate direction to counter the rotation, but failed to fully compensate for the
685 perturbation.

686 This aiming deficit helps answer the question posed in the Introduction: If people
687 can compensate for a rotation through a multiplicity of processes, why do individuals
688 with SCA not compensate for an implicit learning deficit through an increased reliance
689 on aiming? The current results indicate that the ability to discover an aiming solution is
690 also compromised.

691 This dual-deficit was not only observed when a 45° perturbation was introduced
692 abruptly (Exp 1), but also when the 45° perturbation was introduced gradually (Exp 2). It
693 has been assumed that aiming makes a minimal contribution to performance when the
694 perturbation is introduced in a gradual manner. However, this assumes that implicit
695 learning – the form arising from sensory-prediction-error-based learning – will be
696 sufficient to compensate for the perturbation. The current data, as well as a recent
697 report (Bond and Taylor, 2015), suggest that implicit learning is insufficient to achieve
698 good performance when the perturbation is large (for controls, as well as SCA
699 participants), and thus, the endpoint error will grow over the course of the rotation block.
700 In experiment 2, the control participants began to adjust their aim, with deviations in the
701 aiming location becoming consistent after approximately 150 trials into the rotation
702 block. In contrast, the SCA group failed to invoke compensatory aiming even when the
703 error became quite large. A priori, we might have expected the SCA participants to have
704 larger aiming angles than the controls in order to compensate for their impaired implicit
705 learning.

706 The inclusion of participants with SCA types 1 and 2 in our sample may be a point
707 of concern given previous reports indicating greater extra-cerebellar involvement in

708 these groups compared to other subtypes of SCA (Burk et al., 2003). In particular, might
709 the aiming deficit in the SCA group analyses arise from pathology in these extra-
710 cerebellar regions? The SCA2 participants in experiment 1 exhibited some of the
711 greater impairments in aiming. However, analyses conducted after removal of these
712 three participants in experiment 1, as well as the three SCA1 and one SCA2
713 participants in experiment 2, yields a similar trend as in the reported analyses, in terms
714 of overall performance, aberrant aiming and attenuated implicit adaptation. If aiming
715 processes remained intact we would expect to see comparable overall performance, as
716 SCA participants could use larger changes in aim to compensate for their impaired
717 implicit adaptation. This was not observed in any of the participants, nor in the literature
718 where cerebellar damage has consistently been linked with impairments in both overall
719 performance and aftereffects (Martin et al., 1996; Weiner et al., 1983; Rabe et al., 2009;
720 Schlerf et al., 2013; Smith & Shadmehr, 2005), suggesting they are not effectively using
721 aiming or other compensatory strategies to make up for their impaired implicit
722 adaptation. Most relevant to our experiments, to minimize the chance of extracerebellar
723 involvement in our sample, we only included SCA participants with pure cerebellar
724 symptoms, excluding any participants with resting tremor, rigidity, or bradykinesia,
725 which would suggest parkinsonism and basal ganglia involvement. Additionally, the
726 SCA participants in experiment 2 were examined in detail by a movement disorders
727 specialist (SHK).

728 It is also important to note that the SCA2 participants who showed aiming
729 impairments also tended to have more advanced ataxia as measured in the
730 neurological assessment (Table 1 ataxia scale). As such, we cannot dissociate disease
731 progression from SCA subtype. Moreover, the patterns of pathology show considerable
732 overlap at more advanced disease stages, independent of subtype, and the extra-
733 cerebellar pathology in SCA1 and SCA2 is not evident in areas associated with
734 executive function or strategy use (Seidel et al., 2012). When measured
735 macroscopically, the extra-cerebellar pathology is primarily in the brainstem, with
736 occasional extension to midbrain regions (Mascalchi et al., 2014; Nave et al., 2008;
737 Paulson, 2009; Seidel et al., 2012).

738 We are not making the claim that the aiming deficits reported here should be taken
739 to mean that aiming is solely dependent on cerebellar function. There is extensive
740 evidence that cortico-cerebellar networks encompass almost the entire neocortex, with
741 prominent projections to prefrontal cortex (Buckner et al., 2011; Bostan et al., 2013;
742 Caligiore et al., 2017; Kelly and Strick, 2003). Even if the pathology in our sample was
743 limited to just the cerebellum, behavioral changes likely reflect disruption in a network
744 that spans cerebellar and extra-cerebellar regions. Indeed, this point is made clear in
745 studies of diaschisis showing hypometabolism of frontal cortex in patients with
746 cerebellar degeneration (Meyer et al., 1993).

747 One could take the position that the deficits in planning, including something like
748 the implementation of an aiming strategy, in individuals with SCA, should be attributed
749 to brain areas such as prefrontal cortex known to be essential for these aspects of
750 cognition. This inference (Klinke et al., 2010) however, seems questionable when we
751 consider that complex processes such as those subsumed by the term “executive
752 function” surely require a network perspective. It is likely that the neuropsychological
753 impairments observed in individuals with SCA reflect a disruption of cerebello-cortical
754 networks. More generally, concerns with using lesion methods to make direct structure-
755 function inferences are relevant for all neuropsychological research in humans and non-
756 human species. Although it is important to keep these issues in mind, it is also clear that
757 there is some degree of commonality in patients with cerebellar degeneration,
758 supporting the idea that despite their heterogeneity, we can learn something about
759 cerebellar function through the study of this group.

760 Taken together, experiments 1 and 2 demonstrate that SCA participants have
761 difficulty adjusting their aim to counter a visuomotor rotation. Thus, in addition to an
762 impairment in sensory-prediction-error-based learning, these individuals have difficulty
763 using the feedback to develop and implement an appropriate aiming solution. Below, we
764 consider possible explanations for this impairment in aiming.
765

766 Increased movement variability does not account for impairments in aiming

767 In experiment 3, the addition of trial-by-trial noise to a constant perturbation was
768 used to test the hypothesis that the observed aiming impairment is due to ataxia-related

769 movement variability. This manipulation, however, only had a modest effect on the
770 performance of the young healthy adults. Most relevant to the current study, the
771 additional variability did not produce a significant effect on either overall performance or
772 on aiming behavior. The Variance-Added group altered their aiming direction shortly
773 after the onset of the (noisy) perturbation. While they continued to make aiming
774 adjustments across the rotation block, the asymptotic size of the aiming shift was
775 statistically indistinguishable from that observed in the No-Variance group.

776 We recognize that our noise manipulation is, at best, a weak approximation of the
777 consequences of ataxia. We assume the college students attribute the increased
778 variability to the environment, and not their motor system. Moreover, the increased
779 variance is transient, unlike the chronic nature of ataxia. Nonetheless, the results of
780 experiment 3 suggest that an inability to develop an appropriate aiming solution is not
781 solely due to the motor variability associated with ataxia.

782 The Variance-Added group did exhibit a small, reduction in the estimate of implicit
783 learning early the rotation block. However, there was no difference in the implicit
784 learning estimate by the end of the rotation block, nor when compared across the entire
785 rotation block. However, this delayed onset of implicit learning is likely distinct from the
786 implicit learning deficit observed in SCA. Under conditions of high external noise, the
787 learning rate should be reduced (Kalman, 1960; He et al., 2016). Moreover, using a
788 computational model, Schlerf and colleagues (2013) reported that the slower learning
789 rates in SCA are unlikely to be the result of increased motor noise. Indeed, when
790 directly examined, minimal correlation is found between impairments in visuomotor
791 learning and the severity of ataxia (Martin et al., 1996; Schlerf et al., 2013).

792

793 Flexibility in aiming is not directly driven by sensory prediction errors

794 A second hypothesis is that sensory prediction errors are necessary to form an
795 effective aiming solution. While some form of performance error would be needed to
796 adjust aiming, it is unlikely that a sensory prediction error is the primary signal. A
797 sensory prediction error occurs whenever there is a mismatch between the expected
798 and actual consequences of a movement. This sensory prediction error is used to
799 update a forward model to improve prediction. In a standard visuomotor rotation task,

800 this error is defined as the difference between the cursor position and target (when
801 taken as a proxy of the location for expected feedback). However, when participants
802 use an instructed aiming strategy to counter the rotation, their hand slowly drifts away
803 from the aiming location across trials (Mazzoni and Krakauer 2006). This occurs
804 because a persistent sensory prediction error, the mismatch between the cursor
805 position and aiming location in this context, continues to result in implicit updating of a
806 forward model. Eventually, this implicit learning leads to poor performance (i.e., large
807 target errors) and participants have to change their aim to offset continued implicit
808 learning (Taylor and Ivry 2011). Models of performance in this aiming task suggest that
809 changes in aim are driven by performance error, the difference between the target and
810 cursor feedback locations, rather than sensory prediction error (Taylor and Ivry 2011;
811 Taylor et al 2014). Sensory prediction errors, on the other hand, appear to lead to
812 implicit learning even when irrelevant to the task goal (Mazzoni and Krakauer 2006;
813 Schaefer and Thoroughman 2011; Morehead et al 2014).

814 Another problem with the hypothesis that the aiming deficit is related to an
815 impairment in learning from sensory prediction errors is that the time course of aim
816 reports looks quite different than that observed for implicit learning. First, with large
817 perturbations (e.g., experiment 1), the aim reports are non-monotonic, with an initial
818 large increase and then a gradual reduction over the course of the rotation block.
819 Second, while the average data might look smooth, the aiming reports for individuals
820 can change abruptly and in either direction, behavior that is reminiscent of exploration
821 (see Taylor et al., 2014). This stands in contrast, to the slow, monotonic updating of an
822 internal model that is considered to be the hallmark of motor adaptation (Huberdeau et
823 al., 2015). Thus, it seems unlikely that the output of a forward model is the driving force
824 for finding an aiming solution.

825 826 An action-outcome maintenance account of impaired aiming in SCA

827 A third hypothesis considers less direct ways in which the cerebellum might
828 support learning, and by extension, the cognitive capacity required for discovering an
829 aiming solution. The cerebellum is known to be highly connected with much of the
830 cerebral cortex, including prefrontal cortex (Buckner et al., 2011), and damage to the
831 cerebellum can produce a constellation of neuropsychological impairments similar,

albeit in milder form, to that observed in patients with lesions of prefrontal cortex (Buckner et al., 2013; Bodranghien et al., 2016). Drawing on ideas developed in perceptual domains (Cohen et al., 1997; Prabhakaran et al., 2000; Gazzaley et al., 2005), we have proposed that a network including prefrontal cortex and cerebellum forms something akin to a motor working memory system, one essential in the action domain (Ivry & Fiez, 2000; Spencer and Ivry, 2009). By this idea, the cerebellum helps represent and/or maintain task-relevant stimulus-response associations across trials. Theories of prefrontal cortex function have suggested a role in implementing and maintaining task sets across trials (Dosenbach et al., 2006 and 2007), possibly in a hierarchical manner where abstraction increases rostrally, allowing for the simultaneous search for action rules at multiple levels of abstraction (Badre and D'Esposito, 2009; Badre et al., 2010). By extension, the task set implemented by the prefrontal cortex would include the task relevant action-outcome associations, a network involving the cerebellum. Consistent with this theory, Spencer and Ivry (2009) showed that the impact of SCA on sequence learning was considerable when the task involved indirect, arbitrary S-R associations, but that SCA participants performed as well as controls in sequence learning when the S-R associations were direct.

An extension of this hypothesis may account for the aiming deficit observed in the current study. Converging on an appropriate aiming solution entails a cyclic process of hypothesis testing, generating possible solutions and then evaluating their efficacy. This process would require the maintenance of stimulus-response associations or, perhaps in the case of visuomotor rotations, action-outcome associations.

Moreover, the memory demands are likely greatly increased when there are eight target locations, especially with a visuomotor rotation (Krakauer et al., 2000) where implicit generalization appears quite narrow (Heuer and Hegele, 2011). Here an aiming hypothesis generated in response to an action at one target, may appear appropriate when applied to neighboring targets, but fail miserably when applied to distant targets. For example, aiming above the target location would be effective in countering a clockwise rotation for targets presented on the right side of the display but would be counterproductive if applied to targets on the left side of the display. Thus, the participant would need to maintain action-outcome associations at multiple target

863 locations to eventually learn that the solution to the perturbation is common to all target
864 locations in a polar coordinate reference frame. This idea would suggest that if the SCA
865 participants had a compromised ability to maintain action-outcome association, then
866 they would naturally have an impaired ability to counter a visuomotor rotation across the
867 workspace.

868 Our action-outcome maintenance hypothesis makes three predictions. First, we
869 would expect that the ability to employ an aiming solution might be related to the
870 individual's cognitive status. Second, we would expect that individuals with SCA would
871 be able to develop an appropriate aiming solution if the perturbation was simpler. For
872 example, these individuals might be able to use aiming to compensate for a
873 translational perturbation. Third, we would predict that individuals with ataxia would
874 disproportionately benefit if the demands on working memory were reduced, say by
875 displaying the previous aim choice for each target, or by the use of only a single target
876 location. These last two predictions can motivate future work on the multi-faceted
877 contribution of the cerebellum to sensorimotor learning. At present, the results
878 presented here underscore a role for the cerebellum, not only in implicit aspects of
879 motor performance, but also when those tasks require a more explicit association to link
880 a stimulus with an appropriate response to meet task goals.

881 Here we observed a dual-deficit in sensory-prediction-error-based learning (e.g., a
882 forward model) and aiming. While at first glance the latter may seem odd, it perhaps
883 shouldn't be all that surprising given the cerebellum's involvement in learning for many
884 different types of tasks, from eye-blink conditioning to sequence learning. Indeed, the
885 nearly uniform circuitry of the cerebellum, along with its connections to many areas of
886 the rest of the brain (Buckner et al., 2011), suggests that it may be contributing to
887 learning processes in a generalized manner that remains to be determined.

888

889

890

891 **Bibliography**

- 892 Badre, D., & D'Esposito, M. (2009). Is the rostro-caudal axis of the frontal lobe
893 hierarchical? *Nature reviews. Neuroscience*, 10(9), 659–669.
- 894 Badre, D., Kayser, A. S., & D'Esposito, M. (2010). Frontal cortex and the discovery of
895 abstract action rules. *Neuron*, 66(2), 315–326.
- 896 Benson, B. L., Anguera, J. A., & Seidler, R. D. (2011). A spatial explicit strategy reduces
897 error but interferes with sensorimotor adaptation. *Journal of Neurophysiology*,
898 105(6), 2843–2851.
- 899 Bodranghien, F., Bastian, A., Casali, C., Hallett, M., Louis, E. D., Manto, M., Mariën, P.,
900 et al. (2016). Consensus Paper: Revisiting the Symptoms and Signs of Cerebellar
901 Syndrome. *Cerebellum*, 15(3), 369–391.
- 902 Bond, K. M., & Taylor, J. A. (2015). Flexible explicit but rigid implicit learning in a
903 visuomotor adaptation task. *Journal of Neurophysiology*, 113(10), 3836–49.
- 904 Bostan, A. C., Dum, R. P., & Strick, P. L. (2013). Cerebellar networks with the cerebral
905 cortex and basal ganglia. *Trends in Cognitive Sciences*, 17(5), 241–54.
- 906 Brooks, J. X., Carriot, J., & Cullen, K. E. (2015). Learning to expect the unexpected:
907 rapid updating in primate cerebellum during voluntary self-motion. *Nature
908 Neuroscience*, 18(9), 1310–1317.
- 909 Buckner, R. L. (2013). The Cerebellum and Cognitive Function: 25 Years of Insight from
910 Anatomy and Neuroimaging. *Neuron*, 80(3), 807–815.
- 911 Buckner, R. L., Krienen, F. M., Castellanos, A., Diaz, J. C., and Yeo, B. T. T. (2011).
912 The organization of the human cerebellum estimated by intrinsic functional
913 connectivity. *Journal of Neurophysiology*, 106(5), 2322–45.
- 914 Bürk, K., Globas, C., Bösch, S., Klockgether, T., Zühlke, C., Daum, I., & Dichgans, J.
915 (2003). Cognitive deficits in spinocerebellar ataxia type 1, 2, and 3. *Journal of
916 Neurology*, 250(2), 207–211.

917

- 918 Button, K. S., Ioannidis, J. P. A., Mokrysz, C., Nosek, B. A., Flint, J., Robinson, E. S. J.,
919 and Munafo, M. R. (2013). Power failure: why small sample size undermines the
920 reliability of neuroscience. *Nature Reviews Neuroscience*, 14(May), 365–376.
- 921 Caligiore, D., Pezzulo, G., Baldassarre, G., Bostan, A. C., Strick, P. L., Doya, K.,
922 Herreros, I. (2017). Consensus Paper: Towards a Systems-Level View of Cerebellar
923 Function: the Interplay Between Cerebellum, Basal Ganglia, and Cortex.
924 Cerebellum.
- 925 Christou, A. I., Miall, R. C., McNab, F., Galea, J. M., Baddeley, A., Jonides, J., Anguera,
926 J. A., et al. (2016). Individual differences in explicit and implicit visuomotor learning
927 and working memory capacity. *Scientific Reports*, 6, 36633.
- 928 Cohen, J. D., Perlstein, W. M., Braver, T. S., Nystrom, L. E., Noll, D. C., Jonides, J., &
929 Smith, E. E. (1997). Temporal dynamics of brain activation during a working memory
930 task. *Nature*, 386(6625), 604–608.
- 931 Diedrichsen, J., White, O., Newman, D., & Lally, N. (2010). Use-dependent and error-
932 based learning of motor behaviors. *Journal of Neuroscience*, 30(15), 5159–66.
- 933 Dosenbach, N. U. F., Visscher, K. M., Palmer, E. D., Miezin, F. M., Wenger, K. K.,
934 Kang, H. C., Burgund, E. D., Grimes, A. L., Schlagger, B. L., & Petersen, S. E.
935 (2006). A Core System for the Implementation of Task Sets. *Neuron*, 50(5), 799–
936 812.
- 937 Dosenbach, N. U. F., Fair, D. A., Miezin, F. M., Cohen, A. L., Wenger, K. K., Dosenbach,
938 R. A. T., Fox, M. D., Snyder, A. Z., Vincent, J. L., Raichle, M. E., Schlagger, B. L., &
939 Petersen, S. E. (2007). Distinct brain networks for adaptive and stable task control in
940 humans. *Proceedings of the National Academy of Sciences of the United States of
941 America*, 104(26), 11073–8.
- 942 Galea, J. M., Vazquez, A., Pasricha, N., Xivry, J.-j. O. D., and Celnik, P. (2011).
943 Dissociating the roles of the cerebellum and motor cortex during adaptive learning:
944 the motor cortex retains what the cerebellum learns. *Cerebral Cortex (New York,*
945 *N.Y. : 1991)*, 21(8), 1761–70.

- 946 Gazzaley, A., Cooney, J. W., McEvoy, K., Knight, R. T., & D'Esposito, M. (2005). Top-
947 down enhancement and suppression of the magnitude and speed of neural activity.
948 *Journal of Cognitive Neuroscience*, 17(3), 507–17.
- 949 Ghilardi, M. F., Gordon, J., & Ghez, C. (1995). Learning a visuomotor transformation in
950 a local area of work space produces directional biases in other areas. *Journal of*
951 *Neurophysiology*, 73(6), 2535–2539.
- 952 Gibo, T. L., Criscimagna-Hemminger, S. E., Okamura, A. M., and Bastian, A. J. (2013).
953 Cerebellar motor learning: are environment dynamics more important than error
954 size? *Journal of Neurophysiology*, 110(2), 322–33.
- 955 Haruno, M., Wolpert, D. M., & Kawato, M. (2001). Mosaic model for sensorimotor
956 learning and control. *Neural Computation*, 13(10), 2201–2220.
- 957 He, K., Liang, Y., Abdollahi, F., Fisher Bittmann, M., Kording, K., & Wei, K. (2016). The
958 Statistical Determinants of the Speed of Motor Learning. *PLOS Computational*
959 *Biology*, 12(9), e1005023.
- 960 Heuer, H., & Hegele, M. (2011). Generalization of implicit and explicit adjustments to
961 visuomotor rotations across the workspace in younger and older adults. *Journal of*
962 *Neurophysiology*, 106(4), 2078–2085.
- 963 Huang, V. S., Haith, A., Mazzoni, P., & Krakauer, J. W. (2011). Rethinking motor
964 learning and savings in adaptation paradigms: model-free memory for successful
965 actions combines with internal models. *Neuron*, 70(4), 787–801.
- 966 Huberdeau, D. M., Krakauer, J. W., & Haith, A. M. (2015). Dual-process decomposition
967 in human sensorimotor adaptation. *Current Opinion in Neurobiology*, 33, 71–77.
- 968 IBM (2011). IBM SPSS Statistics 20 Core System User's Guide.
- 969 Ivry, R. B., & Fiez, J. A. (2000). Cerebellar contributions to cognition and imagery. The
970 new cognitive neurosciences, 2, 999-1011.
- 971 Izawa, J., Criscimagna-hemminger, S. E., and Shadmehr, R. (2012). Cerebellar
972 contributions to reach adaptation and learning sensory consequences of action.
973 *Journal of Neuroscience*, 32(12), 4230–4239.

- 974 Kagerer, F. A., Contreras-Vidal, J. L., and Stelmach, G. E. (1997). Adaptation to gradual
975 as compared with sudden visuo-motor distortions. *Experimental Brain Research*,
976 115(3), 557–561.
- 977 Kalman, R. E. R. (1960). A New Approach to Linear Filtering and Prediction Problems.
978 Journal of Basic Engineering, 82(1), 35-45.
- 979 Kansal, K., Yang, Z., Fishman, A. M., Sair, H. I., Ying, S. H., Jedynak, B. M., ... Onyike,
980 C. U. (2017). Structural cerebellar correlates of cognitive and motor dysfunctions in
981 cerebellar degeneration. *Brain*, 140(3), 707–720.
982 <https://doi.org/10.1093/brain/aww327>
- 983 Kelly, R. M., & Strick, P. L. (2003). Cerebellar loops with motor cortex and prefrontal
984 cortex of a nonhuman primate. *The Journal of Neuroscience : The Official Journal of*
985 *the Society for Neuroscience*, 23(23), 8432–8444.
- 986 Kimberg, D. Y., Coslett, H. B., & Schwartz, M. F. (2007). Power in Voxel-based Lesion –
987 Symptom Mapping. *Journal of Cognitive Neuroscience*, 19, 1067–1080.
- 988 Klinke, I., Minnerop, M., Schmitz-Hübsch, T., Hendriks, M., Klockgether, T., Wüllner, U.,
989 & Helmstaedter, C. (2010). Neuropsychological Features of Patients with
990 Spinocerebellar Ataxia (SCA) Types 1, 2, 3, and 6. *The Cerebellum*, 9(3), 433–442.
- 991 Krakauer, J. W., Pine, Z. M., Ghilardi, M.-F., & Ghez, C. (2000). Learning of Visuomotor
992 Transformations for Vectorial Planning of Reaching Trajectories. *J. Neurosci.*,
993 20(23), 8916–8924.
- 994 Krakauer, J. W. (2009). Motor learning and consolidation: the case of visuomotor
995 rotation. *Advances in Experimental Medicine and Biology*, 629, 405–21.
- 996 Küper, M., Wünnemann, M. J. S., Thürling, M., Stefanescu, R. M., Maderwald, S., Elles,
997 H. G., Görcke, S., et al. (2014). Activation of the cerebellar cortex and the dentate
998 nucleus in a prism adaptation fMRI study. *Human Brain Mapping*, 35(4), 1574–1586.
- 999 Martin, T. A., Keating, J. G., Goodkin, H. P., Bastian, A. J., and Thach, W. T. (1996).
1000 Throwing while looking through prisms. I. Focal olivocerebellar lesions impair
1001 adaptation. *Brain*, 119(4) 1183–98.

- 1002 Mascalchi, M., Diciotti, S., Giannelli, M., Ginestroni, A., Soricelli, A., Nicolai, E., Toschi,
1003 N. (2014). Progression of Brain Atrophy in Spinocerebellar Ataxia Type 2: A
1004 Longitudinal Tensor-Based Morphometry Study. *PLoS ONE*, 9(2), e89410.
- 1005 Mazzoni, P. and Krakauer, J. W. (2006). An Implicit Plan Overrides an Explicit Strategy
1006 during Visuomotor Adaptation. *Journal of Neurophysiology*, 26(14), 3642–3645.
- 1007 McDougle, S. D., Bond, K. M., & Taylor, J. A. (2015). Explicit and Implicit Processes
1008 Constitute the Fast and Slow Processes of Sensorimotor Learning. *Journal of*
1009 *Neuroscience*, 35(26), 9568–9579.
- 1010 Meyer, J. S., Obara, K., & Muramatsu, K. (1993). Diaschisis. *Neurological Research*,
1011 15(6), 362–366.
- 1012 Miall, R. and Wolpert, D. (1996). Forward models for physiological motor control. *Neural*
1013 *Networks*, 9(8), 1265–1279.
- 1014 Morehead, J. R., Taylor, J. A., Parvin, D., Marrone, E., & Ivry, R. B. (2014). Implicit
1015 Adaptation via Visual Error Clamp. *Translational and computational motor control*
1016 *meeting. Washington, DC*.
- 1017 Medina, J. F., Nores, W. L., Ohyama, T., & Mauk, M. D. (2000). Mechanisms of
1018 cerebellar learning suggested by eyelid conditioning. *Current Opinion in*
1019 *Neurobiology*, 10(6), 717–724.
- 1020 Nasreddine, Z. S., Phillips, N. A., Bedirian, V., Charbonneau, S., Whitehead, V., Collin,
1021 I., Cummings, J. L., and Chertkow, H. (2005). The Montreal Cognitive Assessment,
1022 MoCA: a brief screening tool for mild cognitive impairment. *Journal of the American*
1023 *Geriatric Society*, 53(4), 695–99.
- 1024 Nave, R. Della, Ginestroni, A., Tessa, C., Salvatore, E., De Grandis, D., Plasmati, R.,
1025 Mascalchi, M. (2008). Brain white matter damage in SCA1 and SCA2. An in vivo
1026 study using voxel-based morphometry, histogram analysis of mean diffusivity and
1027 tract-based spatial statistics. *NeuroImage*, 43(1), 10–19.
- 1028 Nikooyan, A. A., & Ahmed, A. A. (2015). Reward feedback accelerates motor learning
1029 Reward feedback accelerates motor learning. *Journal of Neurophysiology*, 113,
1030 633–646.

- 1031 Paulson, H. L. (2009). The spinocerebellar ataxias. *Journal of Neuro-Ophthalmology*:
1032 *The Official Journal of the North American Neuro-Ophthalmology Society*, 29(3),
1033 227–37.
- 1034 Prabhakaran, V., Narayanan, K., Zhao, Z., & Gabrieli, J. D. (2000). Integration of
1035 diverse information in working memory within the frontal lobe. *Nature Neuroscience*,
1036 3(1), 85–90.
- 1037 Rabe, K., Livne, O., Gizewski, E. R. R., Aurich, V., Beck, A., Timmann, D., and Donchin,
1038 O. (2009). Adaptation to visuomotor rotation and force field perturbation is correlated
1039 to different brain areas in patients with cerebellar degeneration. *Journal of*
1040 *Neurophysiology*, 101(4), 1961.
- 1041 Rorden, C., Karnath, H.-O., & Bonilha, L. (2007). Improving lesion-symptom mapping.
1042 *Journal of cognitive neuroscience*, 19(7), 1081–1088.
- 1043 Savitzky, A. and Golay, M. J. E. (1964). Smoothing and Differentiation of Data by
1044 Simplified Least Squares Procedures. *Analytical Chemistry*, 36(8), 1627–1639.
- 1045 Schaefer, S. Y., Shelly, I. L., & Thoroughman, K. A. (2012). Beside the point: motor
1046 adaptation without feedback-based error correction in task-irrelevant conditions.
1047 *Journal of Neurophysiology*, 107(4), 1247–1256.
- 1048 Schlerf, J. E., Ivry, R. B., and Diedrichsen, J. (2012). Encoding of Sensory-prediction
1049 Errors in the Human Cerebellum. *Journal of Neuroscience*, 32(14), 4913–4922.
- 1050 Schlerf, J., Xu, J., Klemfuss, N. M., Griffiths, T. L., and Ivry, R. B. (2013). Individuals
1051 with cerebellar degeneration show similar adaptation deficits with large and small
1052 visuomotor errors. *Journal of Neurophysiology*, 109, 1164–1173.
- 1053 Schmitz-Hubsch, T., Du Montcel, S. T., Baliko, L., Berciano, J., Boesch, S., Depondt,
1054 C., Giunti, P., Globas, C., Infante, J., Kang, J. S., Kremer, B., Mariotti, C., Meleggi,
1055 B., Pandolfo, M., Rakowicz, M., Ribai, P., Rola, R., Schols, L., Szymanski, S., Van
1056 De Warrenburg, B. P., Durr, A., and Klockgether, T. (2006). Scale for the
1057 assessment and rating of ataxia. *Neurology*, 66, 1717–1720.

- 1058 Seidel, K., Siswanto, S., Brunt, E. R. P., den Dunnen, W., Korf, H.-W., & Rüb, U. (2012).
1059 Brain pathology of spinocerebellar ataxias. *Acta Neuropathologica*, 124(1), 1–21.
- 1060 Smith, M. A., Brandt, J., and Shadmehr, R. (2000). Motor disorder in Huntington's
1061 disease begins as a dysfunction in error feedback control. *Nature*, 403(February),
1062 544–549.
- 1063 Smith, M. A. and Shadmehr, R. (2005). Intact ability to learn internal models of arm
1064 dynamics in Huntington's disease but not cerebellar degeneration. *Journal of*
1065 *Neurophysiology*, 93(5), 2809–21.
- 1066 Spencer, R. M. C., & Ivry, R. B. (2009). Sequence learning is preserved in individuals
1067 with cerebellar degeneration when the movements are directly cued. *Journal of*
1068 *Cognitive Neuroscience*, 21(7), 1302–1310.
- 1069 Taylor, J. A., Klemfuss, N. M., and Ivry, R. B. (2010). An explicit strategy prevails when
1070 the cerebellum fails to compute movement errors. *Cerebellum*, 9(4), 580–6.
- 1071 Taylor, J. A. and Ivry, R. B. (2011). Flexible cognitive strategies during motor learning.
1072 *PLoS Computational Biology*, 7(3), e1001096.
- 1073 Taylor, J. A, Hieber, L. L., & Ivry, R. B. (2013). Feedback-dependent generalization.
1074 *Journal of Neurophysiology*, 109(1), 202–15.
- 1075 Taylor, J. A., Krakauer, J. W., and Ivry, R. B. (2014). Explicit and implicit contributions to
1076 learning in a sensorimotor adaptation task. *Journal of Neuroscience*, 34(8), 3023–
1077 32.
- 1078 Therrien, A. S., Wolpert, D. M., & Bastian, A. J. (2016). Effective reinforcement learning
1079 following cerebellar damage requires a balance between exploration and motor
1080 noise. *Brain*, 139(1), 101–114.
- 1081 Tseng, Y.-W., Diedrichsen, J., Krakauer, J. W., Shadmehr, R., and Bastian, A. J.
1082 (2007). Sensory prediction errors drive cerebellum-dependent adaptation of
1083 reaching. *Journal of Neurophysiology*, 98(1), 54–62.
- 1084 Vaca-Palomares, I., Diaz, R., Rodriguez-Labrada, R., Medrano-Montero, J., Vazquez-
1085 Mojena, Y., Velazquez-Perez, L., and Fernandez-Ruiz, J. (2013). Spinocerebellar

- 1086 ataxia type 2 neurodegeneration differentially affects error-based and strategic-
1087 based visuomotor learning. *Cerebellum*, 12, 848–855.
- 1088 Verstynen, T., & Sabes, P. N. (2011). How each movement changes the next: an
1089 experimental and theoretical study of fast adaptive priors in reaching. *Journal of*
1090 *Neuroscience*, 31(27), 10050–9.
- 1091 Vindras, P., Desmurget, M., Prablanc, C., & Viviani, P. (1998). Pointing Errors Reflect
1092 Biases in the Perception of the Initial Hand Position. *Journal of Neurophysiology*,
1093 79(6), 3290–3294.
- 1094 Vindras, P., Desmurget, M., & Viviani, P. (2005). Error Parsing in Visuomotor Pointing
1095 Reveals Independent Processing of Amplitude and Direction. *Journal of*
1096 *Neurophysiology*, 94(2), 1212–1224.
- 1097 Weiner, M. J., Hallett, M., and Funkenstein, H. H. (1983). Adaptation to lateral
1098 displacement of vision in patients with lesions of the central nervous system.
1099 *Neurology*, 33(6), 766–772.
- 1100 Werner, S., Bock, O., and Timmann, D. (2009). The effect of cerebellar cortical
1101 degeneration on adaptive plasticity and movement control. *Experimental Brain*
1102 *Research*, 193, 189–196.
- 1103 Werner, S., Schorn, C. F., Bock, O., Theysohn, N., & Timmann, D. (2014). Neural
1104 correlates of adaptation to gradual and to sudden visuomotor distortions in humans.
1105 *Experimental Brain Research*, 232(4), 1145–1156.
- 1106 Wu, H. G., Miyamoto, Y. R., Castro, L. N. G., Olveczky, B. P., & Smith, M. A. (2014).
1107 Temporal structure of motor variability is dynamically regulated and predicts motor
1108 learning ability. *Nature Neuroscience*, 17(2), 312–321.
- 1109

1110 **Figure legends**

1111

1112 Figure 1 - General task outline

1113 A) On each trial a single target appeared at one of eight possible locations arranged
1114 equally around the start position. After the target appeared, but before moving,
1115 participants were asked to verbally report which number they were aiming towards for
1116 the current trial. B) In all three experiments, participants first completed 56 baseline
1117 trials with veridical feedback. This was followed by a rotation block in which the visual
1118 feedback was rotated about the start position. Finally, participants completed a no
1119 feedback washout block, where both visual and auditory feedback were withheld. In
1120 experiments 1 and 3 participants completed 128 rotation trials for a total of 264 trials,
1121 while in experiment 2 participants completed 220 rotation trials for a total of 456 trials.

1122

1123 Figure 2 - Experiment 1: Abrupt Rotation performance metrics.

1124

1125 The top row depicts group averaged data for A) hand heading angle, B) aim reports,
1126 and C) estimates of implicit learning (Hand heading angle - Aim). The data are based on
1127 averages taken over bins of 8 trials for each participant, and then averaged across
1128 participants for each group. Shaded lines represent confidence intervals around the
1129 mean. Bins are marked with the trial number of the last trial of that bin. The bottom row
1130 shows individual participant data (dots) and group means (horizontal bar) for D) hand
1131 heading angle, E) aim report, and F) implicit learning estimate, with the data averaged
1132 over the first 8 trials of the rotation block, the last 8 trials of the rotation block, or the first
1133 8 trials of no-feedback washout block.

1134

1135 Figure 3 - Experiment 2: Gradual Rotation performance metrics.

1136

1137 The top row depicts group averaged data for A) hand heading angle, B) aim reports,
1138 and C) estimates of implicit learning (Hand heading angle - Aim). The data are based on
1139 averages taken over bins of 8 trials for each participant, and then averaged across
1140 participants for each group. Shaded lines represent confidence intervals around the
1141 mean. Bins are marked with the trial number of the last trial of that bin. The bottom row
1142 shows individual participant data (dots) and group means (horizontal bar) for D) hand
1143 heading angle, E) aim report, and F) implicit learning estimate, with the data averaged
1144 over the first 8 trials of the rotation block, the last 8 trials of the rotation block, or the first
1145 8 trials of no-feedback washout block.

1146

1147

1148 Figure 4 – Sample perturbation schedule for Variance-Added group in experiment 3.

1149

1150 The visuomotor rotation for each trial in the rotation block was drawn from a
1151 gaussian distribution with a mean of 45° and standard deviation of 11°. The variance of
1152 the gaussian was based on the mean variance in hand heading angle seen in
1153 participants with ataxia in experiment 1. Participants in the No-Variance *control* group
1154 experienced a constant 45° rotation on all trials in the rotation block (not shown).

1155

1156 Figure 5 - Experiment 3: Abrupt Rotation with variable rotation performance metrics

1157

1158 The top row depicts group averaged data for A) hand heading angle, B) aim reports,
1159 and C) estimates of implicit learning (Hand heading angle - Aim). The data are based on
1160 averages taken over bins of 8 trials for each participant, and then averaged across
1161 participants for each group. Shaded lines represent confidence intervals around the
1162 mean. Bins are marked with the trial number of the last trial of that bin. The bottom row
1163 shows individual participant data (dots) and group means (horizontal bar) for D) hand
1164 heading angle, E) aim report, and F) implicit learning estimate, with the data averaged
1165 over the first 8 trials of the rotation block, the last 8 trials of the rotation block, or the first

1166 8 trials of no-feedback washout block.

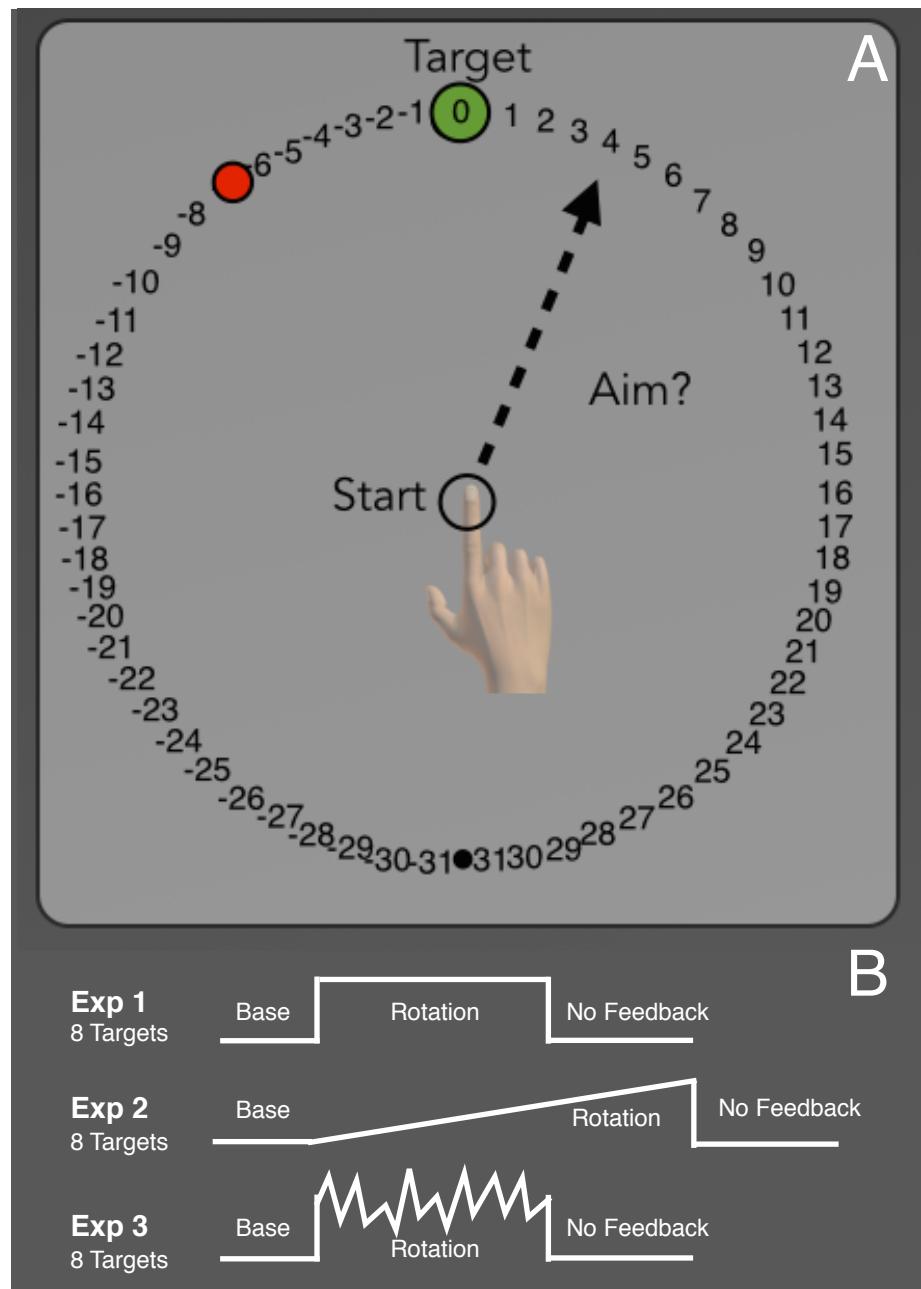


Figure 1 - General task outline

A) On each trial a single target appeared at one of eight possible locations arranged equally around the start position. After the target appeared, but before moving, participants were asked to verbally report which number they were aiming towards for the current trial. B) In all three experiments, participants first completed 56 baseline trials with veridical feedback. This was followed by a rotation block in which the visual feedback was rotated about the start position. Finally, participants completed a no feedback washout block, where both visual and auditory feedback were withheld. In experiments 1 and 3 participants completed 128 rotation trials for a total of 264 trials, while in experiment 2 participants completed 220 rotation trials for a total of 456 trials.

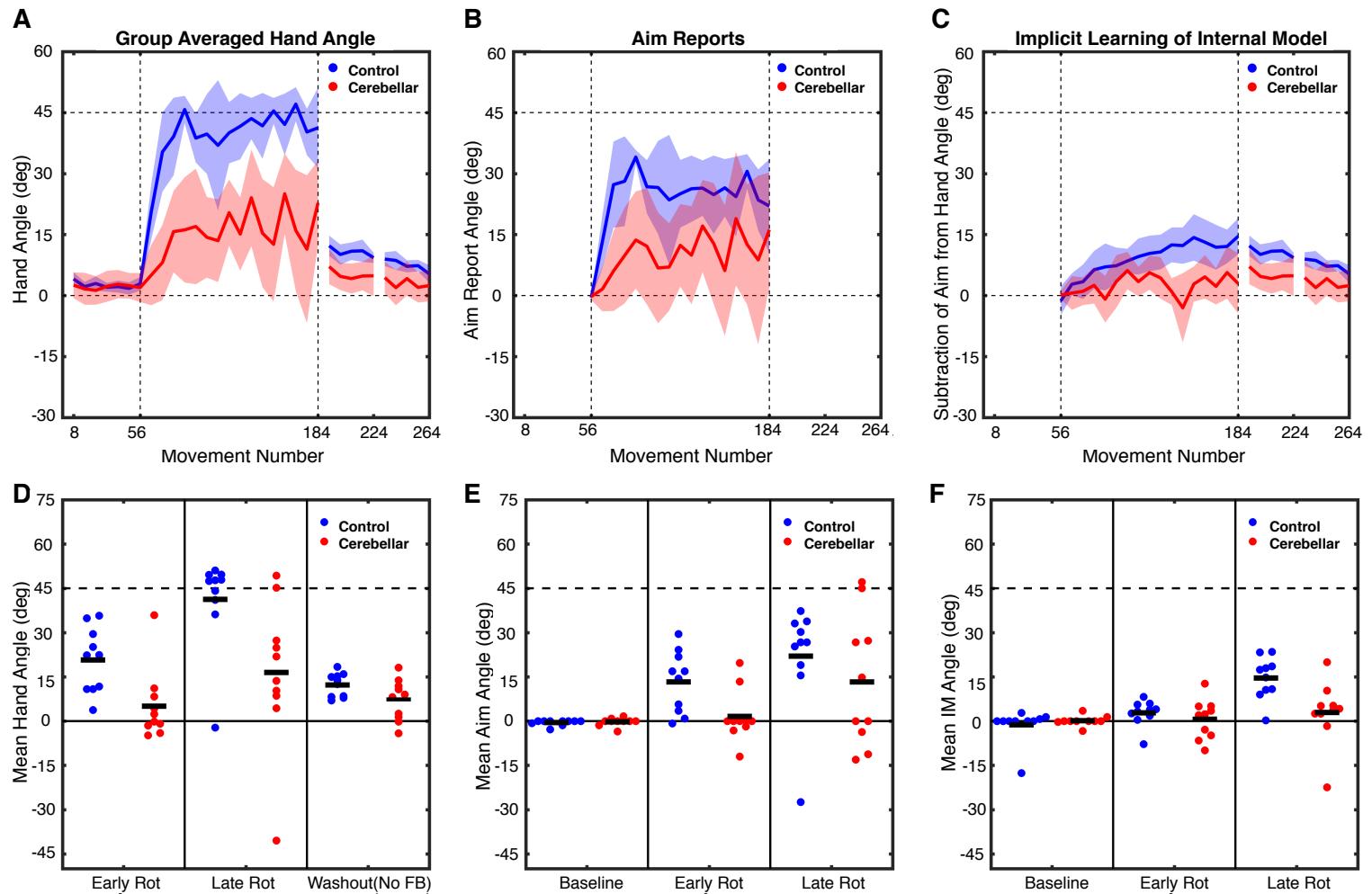


Figure 2 - Experiment 1: Abrupt Rotation performance metrics.

The top row depicts group averaged data for A) hand heading angle, B) aim reports, and C) estimates of implicit learning (Hand heading angle - Aim). The data are based on averages taken over bins of 8 trials for each participant, and then averaged across participants for each group. Shaded lines represent confidence intervals around the mean. Bins are marked with the trial number of the last trial of that bin. The bottom row shows individual participant data (dots) and group means (horizontal bar) for D) hand heading angle, E) aim report, and F) implicit learning estimate, with the data averaged over the first 8 trials of the rotation block, the last 8 trials of the rotation block, or the first 8 trials of no-feedback washout block.

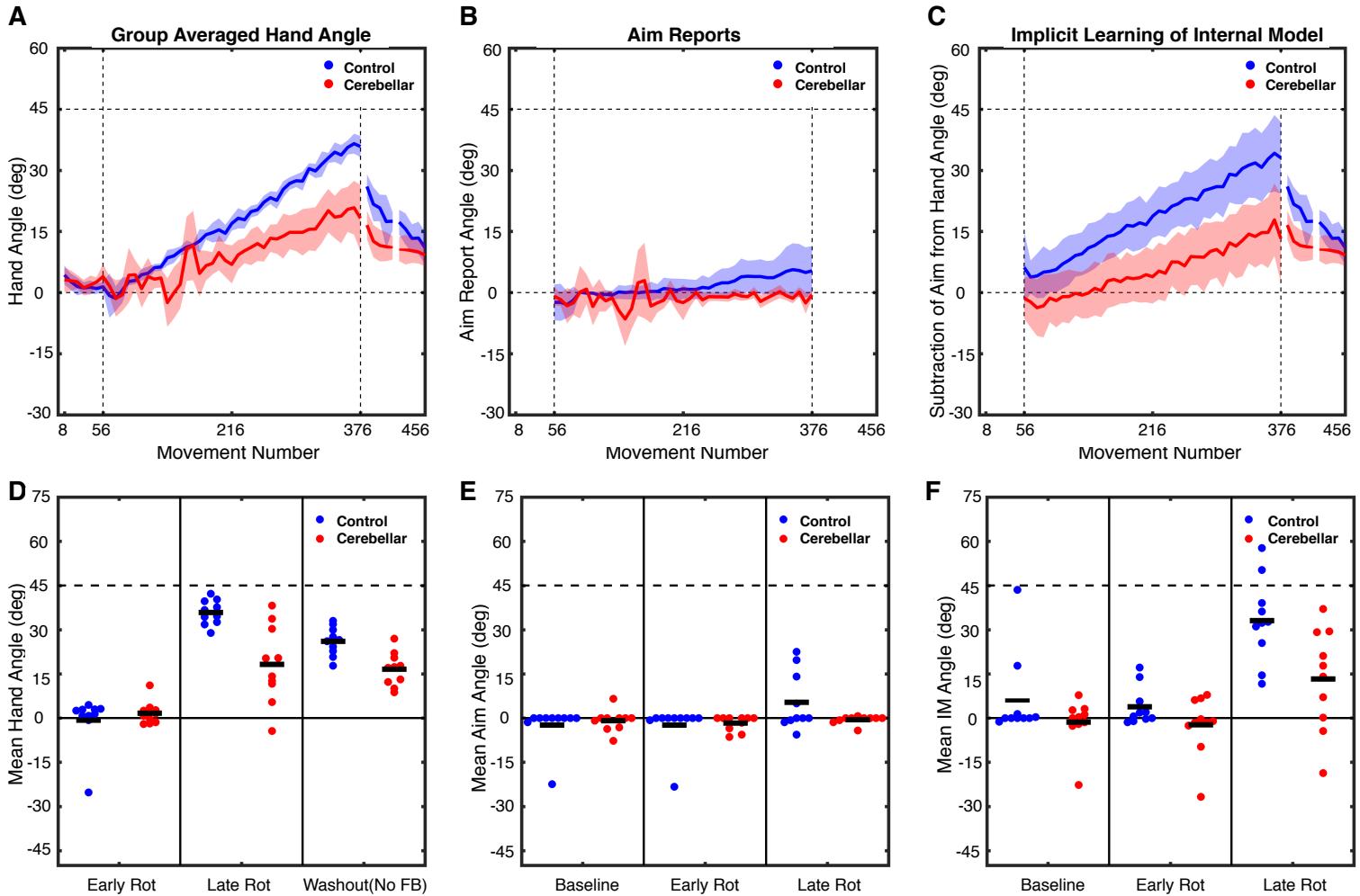


Figure 3 - Experiment 2: Gradual Rotation performance metrics.

The top row depicts group averaged data for A) hand heading angle, B) aim reports, and C) estimates of implicit learning (Hand heading angle - Aim). The data are based on averages taken over bins of 8 trials for each participant, and then averaged across participants for each group. Shaded lines represent confidence intervals around the mean. Bins are marked with the trial number of the last trial of that bin. The bottom row shows individual participant data (dots) and group means (horizontal bar) for D) hand heading angle, E) aim report, and F) implicit learning estimate, with the data averaged over the first 8 trials of the rotation block, the last 8 trials of the rotation block, or the first 8 trials of no-feedback washout block.

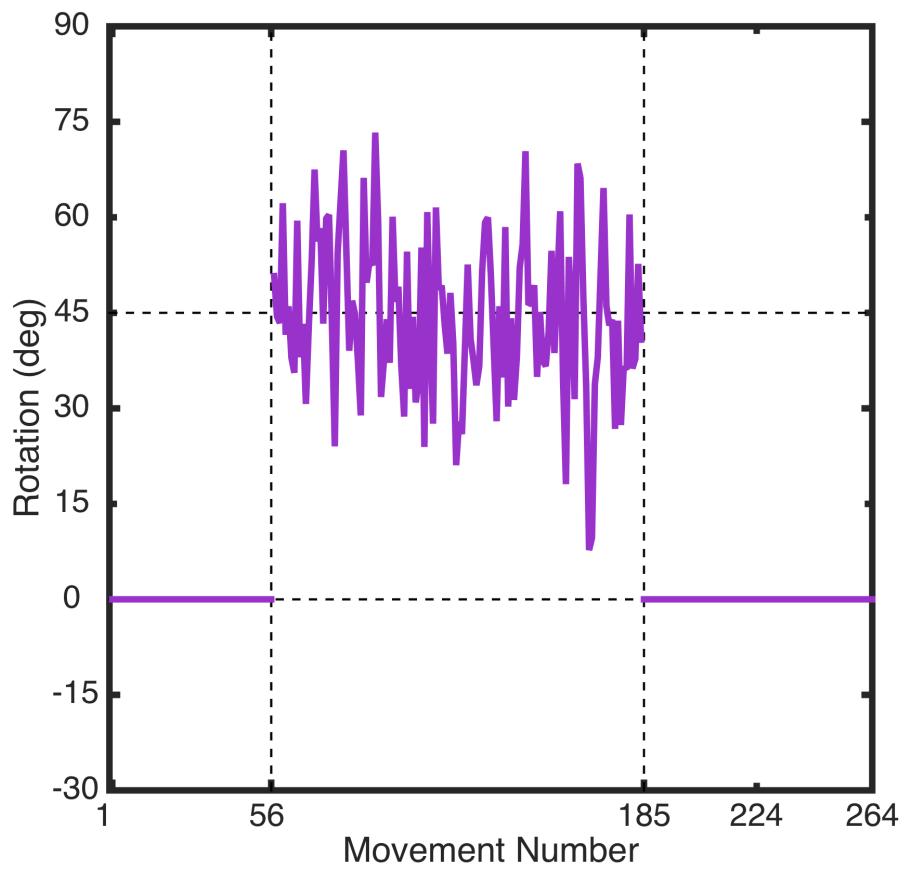


Figure 4 - Sample perturbation schedule for Variance-Added group in experiment 3.

The visuomotor rotation for each trial in the rotation block was drawn from a gaussian distribution with a mean of 45° and standard deviation of 11° . The variance of the gaussian was based on the mean variance in hand heading angle seen in participants with ataxia in experiment 1. Participants in the No-Variance control group experienced a constant 45° rotation on all trials in the rotation block (not shown).

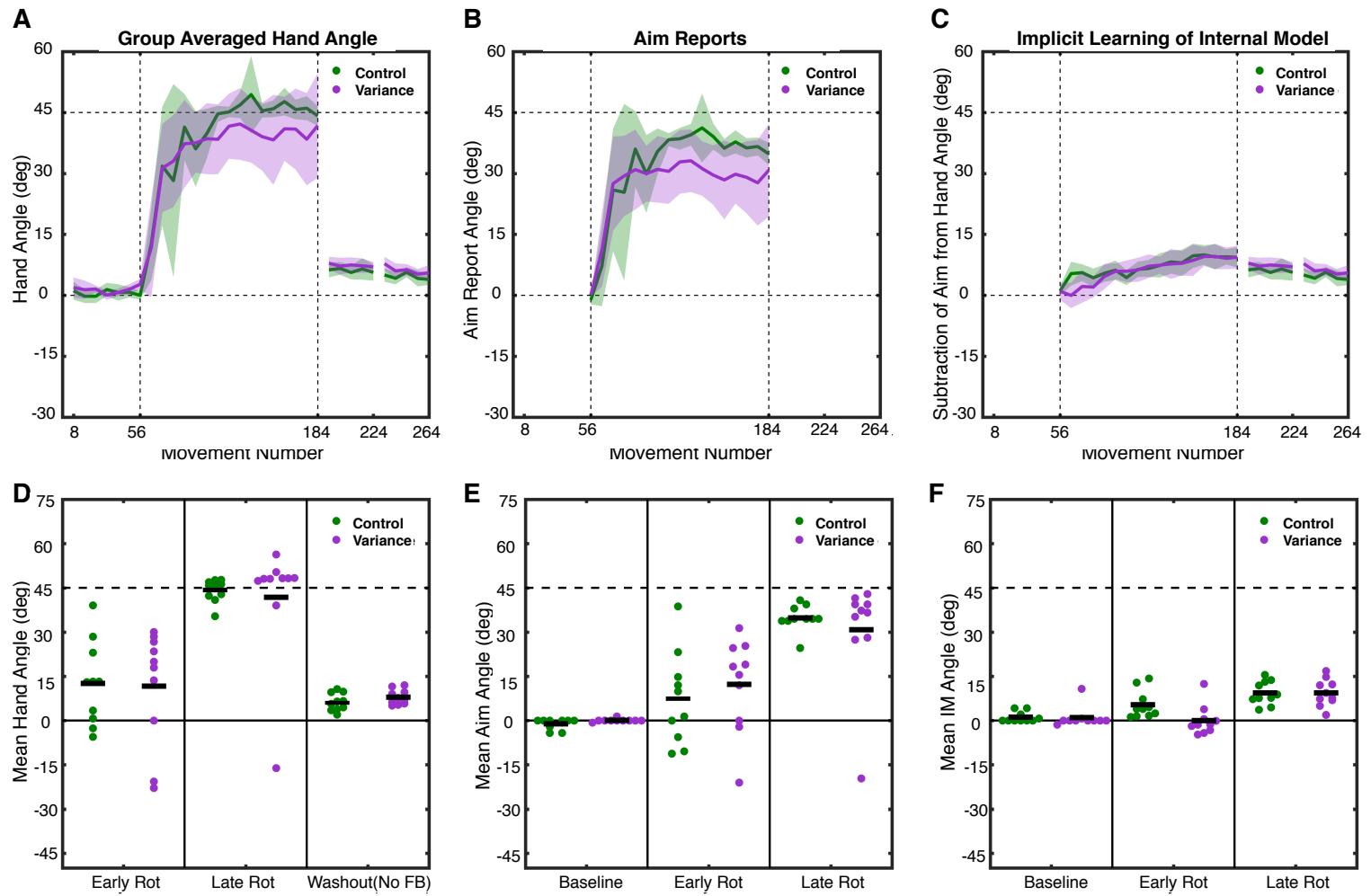


Figure 5 - Experiment 3: Abrupt Rotation with variable rotation performance metrics

The top row depicts group averaged data for A) hand heading angle, B) aim reports, and C) estimates of implicit learning (Hand heading angle - Aim). The data are based on averages taken over bins of 8 trials for each participant, and then averaged across participants for each group. Shaded lines represent confidence intervals around the mean. Bins are marked with the trial number of the last trial of that bin. The bottom row shows individual participant data (dots) and group means (horizontal bar) for D) hand heading angle, E) aim report, and F) implicit learning estimate, with the data averaged over the first 8 trials of the rotation block, the last 8 trials of the rotation block, or the first 8 trials of no-feedback washout block.

| Participant | Gender | Age | Ed (years) | Handedness | Type | Diag (years) | Ataxia scale | MOCA |
|----------------|--------|-----|------------|------------|------------------|--------------|--------------|------|
| Abrupt | | | | | | | | |
| ARSCA1 | Female | 63 | 16 | Left | SCA8 | 11 | ---- | 27 |
| ARSCA2 | Male | 73 | 20 | Left | SAOA | 4 | 20.5 | 28 |
| ARSCA3 | Male | 41 | 16 | left | SAOA | 19 | 27 | 29 |
| ARSCA4 | Female | 55 | 14 | Right | SCA2 | 13 | 48 | 26 |
| ARSCA5 | Male | 47 | 16 | Left | SAOA | 19 | 22 | 29 |
| ARSCA6 | Male | 42 | 16 | Right | SCA2 | 2 | 41 | 28 |
| ARSCA7 | Male | 62 | 19 | Right | SAOA/MSA | 4 | 12 | 30 |
| ARSCA8 | Male | 70 | 16 | Right | SAOA/Family Hist | 9 | 11 | 27.5 |
| ARSCA9 | Male | 44 | 22 | Left | SAOA/MSA | 2 | 12 | 30 |
| ARSCA10 | Male | 40 | 16 | Right | SCA2 | 11 | 45 | 26 |
| Gradual | | | | | | | | |
| GRSCA1 | Female | 35 | 17 | Right | SCA1 | 4 | 6* | 24 |
| GRSCA2 | Female | 30 | 17 | Left | SCA2 | 3 | 17* | ---- |
| GRSCA3 | Male | 44 | 17 | Right | SCA1 | 1 | 9* | 19 |
| GRSCA4 | Male | 79 | 13 | Right | SAOA | 6 | 11* | 14 |
| GRSCA5 | Male | 46 | 17 | Right | ADCA | 1 | 7* | 20 |
| GRSCA6 | Male | 41 | 17 | Right | SAOA | ---- | 23.5* | 23 |
| GRSCA7 | Male | 49 | 17 | Right | SAOA | 3 | 8* | 22 |
| GRSCA8 | Male | 36 | 17 | Right | SCA1 | 3 | 10.5* | 25 |
| GRSCA9 | Female | 69 | 19 | Left | SAOA | 8 | 3* | 30 |
| GRSCA10 | Female | 52 | 22 | Right | SCA6 | 14 | 1 | 28 |

* SARA, otherwise ICARS

Table 1: SCA neuropsychology and demographic information