Intact Mirror-Tracing and Impaired Rotary-Pursuit Skill Learning in Patients With Huntington’s Disease: Evidence for Dissociable Memory Systems in Skill Learning

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Skill learning in early-stage Huntington’s disease (HD) patients was compared with that of normal controls on 2 perceptual-motor tasks, rotary pursuit and mirror tracing. HD patients demonstrated a dissociation between impaired rotary-pursuit and intact mirror-tracing skill learning. These results suggest that different forms of perceptual-motor skill learning are mediated by separable neural circuits. A striatal memory system may be essential for sequence or open-loop skill learning but not for skills that involve the closed-loop learning of novel visual-response mappings. It is hypothesized that working memory deficits in HD resulting from frontostriatal damage may account broadly for intact and impaired long-term learning and memory in HD patients.

Dissociations in patients with brain injuries between intact and impaired forms of learning provide evidence for the existence of memory systems, particular neural networks that mediate specific mnemonic processes. Patients with global amnesia have bilateral lesions in medial-temporal or diencephalic brain regions, and they exhibit a relatively circumscribed deficit in the learning and remembering of new events and facts, a form of memory that has been termed declarative (Cohen & Squire, 1980). The deficit is evident in explicit tests of recall and recognition for recent episodes or materials encountered in those episodes (Graf & Schacter, 1985). Amnesic patients, however, have shown intact memory performance on two broad classes of tasks where memory is measured implicitly or indirectly by a change in performance and without explicit reference to any prior study or training episode: skill-learning tasks and repetition-priming tasks.

In skill-learning tasks, memory is measured as improve-

ment with practice in perceptual-motor, perceptual, or cognitive performance. Amnesic patients have shown intact skill learning on a number of tasks, including mirror tracing (Gabrieli, Corkin, Mickel, & Crowdon, 1993; Milner, 1962), rotary pursuit (Cermak, Lewis, Butters, & Goodglass, 1973; Corkin, 1968; Heindel, Butters, & Salmon, 1988), serial reaction time (Nissen & Bulieler, 1987; Nissen, Willingham, & Hartman, 1989), and reading mirror-reversed text (Cohen & Squire, 1980; Martone, Butters, Payne, Becker, & Sax, 1984). In repetition-priming tasks, memory is measured as a change in the speed or accuracy of processing a stimulus, usually a word or picture, due to prior exposure to that stimulus. Amnesic patients have shown intact repetition priming on a number of tasks involving words (e.g., Graf, Shimamura, & Squire, 1985; Graf, Squire, & Mandler, 1984; Warrington & Weiskrantz, 1970), representational pictures (Cave & Squire, 1992), and novel, nonrepresentational pictures (Gabrieli, Milberg, Keane, & Corkin, 1990; Musen & Squire, 1992; Schacter, Cooper, Tharan, & Rubens, 1991).

The sparing of skill learning and repetition priming in amnesia indicates that those forms of memory do not depend upon the medial-temporal and diencephalic brain structures that are vital for declarative memory, but it does not indicate how many different forms of memory are involved in either skill learning or repetition priming or what neural networks subserve those forms of memory. Studies of patients with neurodegenerative diseases, especially Huntington’s disease (HD) and Alzheimer’s disease (AD), have provided evidence about the functional neural architecture of skill learning and repetition priming. HD patients have shown impaired skill learning on tasks of rotary pursuit (Heindel et al., 1988; Heindel, Salmon, Shults, Walicke, & Butters, 1989), reading mirror-reversed text (Martone et al., 1984), and, more variably, serial reaction time (Knopman & Nissen,
patients have shown the reverse dissociation, exhibiting intact skill learning on tasks of rotary pursuit (Eslinger & Damasio, 1986; Heindel et al., 1988, 1989), mirror tracing (Gabrieli et al., 1993), and serial reaction time (Knopman, 1991; Knopman & Nissen, 1987) and impaired repetition priming (e.g., Gabrieli et al., 1994; Heindel et al., 1989; Keane, Gabrieli, Fennema, Growdon, & Corkin, 1991; Monti et al., 1996; Salmon et al., 1988; Shimamura, Salmon, Squire, & Butters, 1987).

The double dissociation between skill learning and repetition priming in HD and AD indicates that at least some forms of skill learning and repetition priming are mediated by different memory systems. Further, knowledge of the prominent pathology in the early stages of the two diseases (in later stages of both diseases patients are untestable) provides evidence about the neural networks that mediate skill learning and repetition priming. Postmortem studies (Vonsattel et al., 1985), in vivo structural imaging (Starkstein et al., 1989), and in vivo functional imaging (Kuhl et al., 1982; Young et al., 1986) indicate that the caudate and putamen, structures that are part of the basal ganglia or striatum, are especially affected in HD. Conversely, the striatum is relatively spared in AD (Arnold, Hyman, Flory, Damasio, & Van Hoesen, 1991; Brun & Englund, 1981). Thus, it appears that the basal ganglia are part of a memory system that mediates various forms of skill learning but not repetition priming.

AD patients have marked hippocampal lesions (Arnold et al., 1991; Brun & Englund, 1981; Hyman, Van Hoesen, Damasio, & Barnes, 1984), but it is unlikely that those lesions account for their repetition-priming deficits because amnesic patients with hippocampal lesions exhibit preserved repetition priming. AD patients, unlike most amnesic patients, have marked pathology in frontal, temporal, and parietal association areas, with relative sparing of primary sensory, cerebellar, and striatal regions. Therefore, cortical areas damaged in AD, but not in HD or amnesia, may constitute memory systems that mediate repetition priming.

Repetition priming, however, can be further decomposed into perceptual priming, reflecting implicit memory for stimulus form, and conceptual priming, reflecting implicit memory for stimulus meaning (e.g., Blaxton, 1989; Roediger, Weldon, & Challis, 1989). Both perceptual and conceptual priming are spared in amnesia, but the two forms of priming dissociate in AD patients who are often intact on perceptual priming tasks (e.g., Gabrieli et al., 1994; Keane et al., 1991) but are impaired on conceptual priming tasks (e.g., Monti et al., 1996; Salmon et al., 1988). Therefore, perceptual forms of priming may be mediated by modality-specific perceptual cortices, which are relatively spared in AD, and conceptual forms of priming may be mediated by association cortices, which are severely compromised in AD. This interpretation is supported by evidence from lesion and imaging studies indicating that visual perceptual priming is mediated by occipital cortex and that conceptual priming is mediated by left frontal and temporal-parietal cortices (Blaxton et al., 1996; Buckner et al., 1995; Demb et al., 1995; Fleischman et al., 1995; Gabrieli, Fleischman, Keane, Reminger, & Morrell, 1995; Gabrieli, Singh, Stebbins, & Goetz, 1996; Keane, Gabrieli, Mapstone, Johnson, & Corkin, 1995; Raichle et al., 1994; Squire et al., 1992; Swick & Knight, 1996).

In the present study, we aimed to examine whether two forms of perceptual-motor skill learning that are spared in amnesia are further dissociable. Such a dissociation would indicate that various forms of skill learning, like various forms of repetition priming, are mediated by separable memory systems. Therefore, we examined the skill learning of one group of early-stage HD patients on both rotary-pursuit and mirror-tracing tasks. We hypothesized that HD patients would show impaired learning of the rotary-pursuit skill as other HD patients have done before (Heindel et al., 1988, 1989). The performance of HD patients on mirror tracing has not been examined previously. Cerebellar lesions impair mirror-tracing skill learning (Sanes, Dimitrov, & Hallett, 1990), but it is not known whether the striatum is essential for such skill learning.

One difficulty in assessing motor skill learning in HD is the inherent confound between baseline performance ability and learning in a group of patients with a motor disease. If patients are tested under conditions identical to those for normal control participants, then any deficit could reflect initial performance difficulties, or learning difficulties, or both. This is not just a nuisance issue in measurement, because it is plausible that changes in the same neural network that mediates initial, unskilled performance constitute the basis of later, skilled performance. To minimize the confounding effects of initial performance and skill learning, some investigators have equated initial performance between HD and control groups by testing the HD patients under easier conditions (Heindel et al., 1988, 1989). We chose the same approach in our study and also explored the implications of that approach. For rotary pursuit, HD and controls were tested at speeds of rotation that resulted in equivalent initial performance. In addition, 3 other HD patients were tested under conditions that resulted in superior initial performance, so that we could examine what consequences such manipulations of initial performance have for rotary-pursuit skill learning. For mirror tracing, HD and controls were tested with one of three patterns of descending difficulty (star, diamond, square).

**Method**

There were 21 participants: 12 HD patients (5 male, 8 female) and 9 (3 male, 6 female) healthy normal control participants (NC; see Table 1). The diagnosis of HD was made by a board-certified neurologist based on a positive family history of HD and the presence of choreatic movements. Severity of HD was assessed with the Huntington’s Disease Functional Capacity Scale (Shoulson & Fahn, 1979), which stages disease severity from 1 (minimal disability) to 5 (complete disability). The HD patients were all in the early stages of the disease: 4 patients were rated at Stage 2, 6
Table 1

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Note. MMSE = Mini-Mental State Examination; Ham-D = Hamilton Depression Rating Scale. RP = rotary pursuit; MT = mirror tracing; RP+ = rotary pursuit with 10-s baseline; HDFCS = Huntington’s Disease Functional Disability Scale; HD = Huntington’s disease; NC = normal control.

In years. bDuration in HD in years.

Results

Rotary Pursuit

The skill-learning measure was time-on-target per trial (maximum of 20 s). The HD and NC groups were tested at mean rotations per minute, respectively, of 28.3 s (SD = 9.3) and 44.4 s (SD = 9.8); this difference was significant, $t(12) = 3.10, p < .01$. The two groups were well matched on initial time-on-target performance with the HD and NC.
groups scoring, respectively, means of 5.6 s and 6.3 s (p > .70). Time-on-target scores were analyzed in a repeated measures analysis of variance (ANOVA) with variables of group (HD, NC), block (Block 1, Block 2, Block 3), and trial (eight trials per block); trials were nested within blocks. Participants showed skill learning by increasing time-on-target across blocks, F(2, 32) = 54.55, MSE = 3.04, p < .001, and trials, F(7, 112) = 4.55, MSE = 1.54, p < .01. The HD group, however, showed less skill learning than the NC group as evidenced by a significant effect of group, F(1, 16) = 16.87, MSE = 63.46, p < .001, and a significant interaction between group and block, F(2, 32) = 38.31, p < .001. A separate analysis of the data from the 6 HD and 8 NC participants who also performed mirror tracing showed a reliable skill-learning deficit in the HD patients, with a significant effect of group, F(1, 12) = 12.96, MSE = 62.45, p < .01, and a significant interaction between group and block, F(7, 84) = 3.14, MSE = 3.08, p < .01 (see Figure 1).

The HD patients demonstrated less skill learning than the NC group, but the question remained as to whether they showed any skill learning at all. A repeated measures ANOVA on the mean time-on-target per block for the HD patients did not reveal a reliable change across blocks (p > .26). The HD patients who performed the task at rotation rates that yielded an initial time-on-target of about 10 s (superior to that of the NC and other HD group) also showed no rotary-pursuit skill learning (see Figure 2).

**Mirror Tracing**

In the HD group, 4 participants traced the star, 1 participant traced the diamond, and 1 participant traced the square. In the NC group, 7 participants traced the star, and 1 participant traced the square. The skill-learning measures were completion time (number of seconds taken to trace a pattern) and errors (number of contacts with the metal plate signifying departures from the pattern; see Figure 3). Mean performance on Trial 1 was not matched as closely as in the rotary-pursuit task. On Trial 1, the HD and NC groups did not differ reliably for completion time (HD: M = 93.8, SD = 69.6; NC: M = 60.6, SD = 32.1), t(12) = 1.2, p =
variables of group (HD, NC), block (Block 1, Block 2), and trial (five trials per block); trials were nested within blocks. Participants showed skill learning by tracing the patterns progressively faster across blocks, F(1, 12) = 15.44, MSE = 1125.31, p < .01, and trials, F(4, 48) = 15.70, MSE = 129.12, p < .001, and by making fewer errors across blocks, F(1, 12) = 8.86, MSE = 107.80, p < .05, and trials, F(4, 48) = 8.85, MSE = 22.30, p < .001. For both skill-learning measures, there were Block X Trial interactions, indicating that within-block improvement across trials decreased across blocks: for completion time, F(4, 48) = 4.57, p < .01; for errors, F(4, 48) = 3.38, p < .05. There was no main effect of group on either skill-learning measure and, most important, no interaction between group and either block or trial, indicating that the HD and NC groups improved similarly on mirror tracing.

Discussion

This study examined whether HD patients would show a skill-learning deficit on mirror tracing, as they have on skill-learning tasks of rotary pursuit, serial reaction time, and mirror reading. The HD patients in the present study were impaired relative to control participants on rotary-pursuit skill learning. In fact, HD patients showed no reliable rotary-pursuit skill learning whether their initial level of performance was equal to or superior to that of control participants. From the first to the last trial, HD and NC groups improved their rotary-pursuit performance by 16% and 48%, respectively. The HD impairment is consistent with prior findings (Heindel et al., 1988, 1989). In stark contrast, the same HD patients showed mirror-tracing skill learning that was statistically indistinguishable from that of control participants. HD mirror-tracing skill learning was robust. From the first to the last trial, HD and NC groups improved the accuracies of their performances by 70% and 48%, respectively, and increased the speed of their perfor-
mances by 56% and 67%, respectively. Thus, there was a clear-cut dissociation in a group of HD patients between impaired rotary-pursuit skill learning and intact mirror-tracing skill learning.

The preservation of mirror-tracing skill learning in HD must be considered with some caution for several reasons. First, initial HD performance was not equated fully with that of the NC group. On the first trial, HD patients were not significantly slower than NC participants, but they were significantly less accurate. The finding, however, that HD patients had intact mirror-tracing skill learning on the speed measure where there was no reliable initial difference favors the interpretation that the HD group showed normal learning. Second, the interpretation of preserved mirror-tracing skill learning rests on the absence of any statistically reliable interaction between groups and learning measures, and this absence occurred with small groups of participants. The study, however, had sufficient statistical power to detect the rotary-pursuit deficit. Further, inspection of the HD learning curves on the mirror-tracing task shows that their skill learning closely paralleled that of the NC group. Thus, there is no apparent evidence that HD patients had any deficit that would become significant in a larger sample. It will be assumed, therefore, for purposes of the remaining discussion, that HD patients had intact mirror-tracing skill learning that was complicated by their motor and cognitive disease.

There are no published studies of mirror tracing in HD, but there is a study of prism adaptation in HD (Paulsen, Butters, Salmon, Heindel, & Swenson, 1993). In that study, participants pointed toward a target while wearing distorting prisms that shifted objects 20° laterally. HD patients, relative to control and AD participants, failed in adapting to the prisms after visuomotor feedback. The prism and mirror-tracing tasks share some apparent features: Both tasks involve visually guided motor learning in an environment with transposed visuomotor relations. Paulsen et al., however, found that there was a correlation between dementia severity and impaired prism adaptation in the HD patients (but not in the AD patients): Less demented HD patients were unimpaired. The dementia severity of HD patients in the present study resembles that of the less demented HD group in the Paulsen study. The two studies are actually in accord, therefore, in finding that mildly demented HD patients retain the ability to acquire motor skills in altered visuomotor environments.

Other studies with HD patients provide further evidence that dementia severity or other patient variables can have major consequences on HD patients' skill-learning abilities. Heindel et al. found correlations between dementia severity and both rotary pursuit learning (1988, 1989) and weight biasing (Heindel, Salmon, & Butters, 1991). More demented HD patients have shown impaired cognitive skill learning on the Tower-of-Hanoi and Tower-of-Toronto puzzles, but HD patients with less dementia have shown intact cognitive skill learning on those tasks (although this latter finding was more variable for the Tower-of-Toronto study; Butters, Wolfe, Martone, Granholm, & Cermak, 1985; Saint-Cyr, Taylor, & Lang, 1988). Dementia severity appears to be an important variable in determining HD patients' skill-learning perfor-

There are several ways to consider the dissociation between HD performance on two skill-learning tasks. One constraint is that it is single, not a double, dissociation. Thus, it could be that learning on both tasks involves the same memory system, but that rotary pursuit has greater demands upon that system than does mirror tracing. It was, however, more difficult to equate HD patients on the mirror-tracing than the rotary-pursuit task, and more HD patients could perform the rotary-pursuit than the mirror-tracing task. These findings do not favor the notion that mirror tracing is less demanding of a common memory system than is rotary pursuit. Alternatively, skill learning on the two tasks may be mediated by memory systems that differ only by a single component that is essential for rotary pursuit but not mirror tracing. In either case, no double dissociation would be expected.

A third possibility is that the two skill-learning tasks depend upon separable brain regions. The integrity of the striatum appears essential for rotary-pursuit skill learning, because such learning is consistently impaired in HD patients and also in patients with Gilles de la Tourette's syndrome who have abnormal striatal anatomy (Stebbins, Singh, Weiner, Goetz, & Gabrieli, 1995). Conversely, the cerebellum may be essential for mirror-tracing skill learning. Patients with lesions in some, but not all, locations in the cerebellum have been reported to show impaired mirror-tracing skill learning (Sanes et al., 1990). Cerebellar patients have also been reported to show impaired motor adaptation in other altered visual environments (Gauthier, Hofferer, Hoyt, & Stark, 1979; Weiner, Hallett, & Funkenstein, 1983) and impaired visually guided tracking (Beppu, Suda, & Tanaka, 1984; Beppu, Nagooka, & Tanaka, 1987). Therefore, it is possible that a double dissociation between the two kinds of learning could occur in patients with striatal or cerebellar lesions. The performance, however, of cerebellar patients on rotary-pursuit skill learning remains to be determined.

Rotary pursuit and mirror tracing differ in many ways, and it is unknown as to what critical difference between the tasks renders skill learning impaired or intact in HD. Two studies have reported single dissociations in HD on skill-learning tasks that were more matched in perceptual and motor demands. In a serial reaction time task, HD participants were impaired in learning a sequence-specific skill for pressing four keys in response to targets appearing in four locations in a recurring sequence (Willingham & Koroshetz, 1993). HD patients had an impairment in using the recurring sequence to predict where the next target would appear. The same HD patients showed intact skill learning when the task was to press a key one position to the right of the target. In this task, there was no sequence to be learned. Rather, participants learned a new mapping between visual stimuli (targets in a particular location) and motor responses (pressing the key one position to the right). In a task where participants tracked a cursor with a joystick, HD patients showed a normal pattern of skill learning when the cursor moved
randomly but impaired learning when the cursor moved in a repeating pattern (Willingham, Korosheiz, & Peterson, 1996).

Willingham proposed that the two within-task dissociations could be explained by a distinction between the learning of repetitive motor sequences, which depends upon the striatum, and the learning of new mappings between visual cues and motor responses, which does not depend upon the striatum. Thus, HD patients were impaired for learning repeated sequences of movements in both studies but were intact for learning new, constant mappings between visual cues and motor responses. The same distinction could apply to the present study. Skill learning on rotary pursuit may involve the learning of a pattern-specific series of repetitive movements. Indeed, the rotary-pursuit task in the present study and the tracking of a repeating sequence (Willingham et al., 1996) are quite similar in this regard. Rotary-pursuit skill learning does not require any new mapping between visual cues and motor responses. Conversely, skill learning on mirror tracing appears straightforwardly to reflect the learning of a new mapping between reversed visual inputs and motor outputs. Further, mirror tracing may not involve sequence-specific learning because both control and AD groups demonstrated nearly perfect responses to a recurrent sequence, involves responding to future target locations that are sequentially predicted upon the basis of currently perceived target locations. Further, knowledge of results is delayed in rotary pursuit. Participants receive visual feedback that their hand has left the target after a faulty prediction has been followed. Thus, neither error correction nor successful motor programming can be driven by present perceptual information. Rather, participants must construct a model that uses present perceptual feedback as a predictor for motor programming and update that model after an error has occurred.

The idea that the striatum has a specific role in open-loop learning has both supportive and contradictory evidence. Studies with Parkinson’s disease (PD) patients have indicated that the striatum may be critical for open-loop motor movements that are guided by prediction rather than by response to current sensory information (Cooke, Brown, & Brooks, 1978; Flowers, 1978; Stern, Mayeux, Rosen, & Ilson, 1983; but see, Hockerman & Aharon-Perez, 1994). When performing tracking tasks, PD patients were abnormally dependent upon visual feedback and did not show the anticipatory motor planning that normal participants did. Another study, however, reported a normal advantage for PD patients when they tracked a target moving in a repeating versus a random pattern (Bloxham, Mindel, & Frith, 1984). PD patients without dementia, however, exhibit intact rotary-pursuit learning (Heindel et al., 1989), so it appears that motor and skill-learning deficits vary considerably among PD, as well as HD, patients. The sequencing—mapping and open—closed loop distinctions need not be contradictory. For example, animal studies indicate that striatal cells code open-loop movements only during the performance of sequences of movements (Kimura, Aosaki, Hu, Ishida, & Watanabe, 1992). Thus, under many circumstances, the learning of sequences may involve open-loop learning, whereas the learning of mappings may involve closed-loop learning.

Finally, it is of interest to consider whether there are similarities between the perceptual-motor skill-learning deficits seen in HD and more nonmotor learning and memory deficits also seen in early HD. HD patients are impaired often on Tower cognitive skill-learning tasks (Butters et al., 1983; Saint-Cyr et al., 1988). These cognitive tasks are open-loop in nature because they require the selection of movements in response to a mental model of a problem solution; there is no perceptual information that provides definitive feedback to guide performance (except for the final step of the problem). The patients in the present study also exhibited a characteristic HD pattern of verbal memory deficits comprised of relatively intact recognition performance and impaired performance on tests of free recall, self-ordering pointing, and recency judgments (Singh, Gabrieli, Stebbins, & Goetz, 1994). Gabrieli et al. (1996) proposed that this pattern of intact and impaired verbal memory performance reflects the severe impairment of working memory in HD, PD, and Tourette’s syndrome. Verbal memory tasks are intact or impaired to the extent they make demands upon the reduced working memory capacities exhibited by HD, PD, and Tourette’s patients (Gabrieli et al., 1996; Singh et al., 1994; Stebbins et al., 1995). Open-loop skill learning that involves the planning of sequences of movements may also have much higher demands on working memory than closed-loop skill learning that uses visual cues to guide. Indeed, Goldman-Rakic (1987) noted that a fundamental feature of working memory is that it guides behavior on the basis of internal, symbolic models as opposed to external, perceptual feedback.

Thus, it is plausible that damage to frontostriatal working memory systems in HD, PD, and Tourette’s syndrome diminishes long-term learning and memory on tasks that require internally guided, open-loop thought, including
certain perceptual-motor skills (rotary pursuit, serial response time with sequences), cognitive skills (Tower problems), and strategic verbal memory (recall, recency judgments). Other learning and memory tasks that are less dependent on working memory capacity may be relatively or fully intact in HD, PD, and Tourette’s, including perceptual-motor skills (mirror tracing) and nonstrategic verbal memory tasks (recognition). It is likely that the diverse forms of long-term learning and memory impaired in striatal patients do not depend on a single frontostratial system but rather on multiple, anatomically distinct frontostratial loops (Alexander, DeLong, & Strick, 1986). The loops mediate working memory processes in different domains (Wilson, O’Scalaidhe, & Goldman-Rakic, 1993), but they may mediate the same kinds of working memory contributions in each of those domains. This hypothesis, although speculative, would provide an integrated account for the various forms of learning and memory that do and do not depend upon the integrity of the basal ganglia.

References

domain of preserved function in amnesia. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 11, 386–396.


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