Cerebellar Involvement in the Explicit Representation of Temporal Information[a]

RICHARD IVRY
Department of Psychology
University of California
Berkeley, California 94720

INTRODUCTION

In considering the neural bases of temporal information processing, two distinct questions should be kept in mind. First, it is important to distinguish between those tasks in which timing is explicitly represented and those tasks in which the temporal properties are an implicit, emergent property. For example, a baseball pitcher must rotate his shoulder forward before his hand releases the ball. While the time at which each of these events should occur may be explicitly represented, it is also possible that this temporal information is implicit: the actions needed to release the ball may be initiated once the forward action of the arm reaches a particular position or velocity. Even in repetitive movements, it may not be necessary to postulate an explicit timing mechanism. For example, when people repetitively swing their arm in a pendular motion, the preferred rate of oscillation can be predicted simply by consideration of biomechanical factors such as the length and mass of the arm.[1]

Nonetheless, it seems reasonable to assume that there are motor tasks in which timing is explicitly controlled. For example, while biomechanical analyses can predict the preferred rate of oscillation, people are capable of adjusting that rate. Indeed, an impressive feature of the literature on repetitive movements is that people are able to produce such movements over a wide range of frequencies.[2–4] Models of this phenomenon generally postulate an explicit timing mechanism with an adjustable rate parameter.[5] Explicit timing would also seem necessary for certain perceptual tasks, such as when people are asked to judge which of two tones is longer in duration. A variety of mechanisms have been proposed to account for performance on these tasks, all sharing the feature of an explicit timing device.[6]

Assuming that there are tasks in which timing is explicitly controlled, we can then turn to the second question. Specifically, does this set of tasks involve the operation of a common timing mechanism, or are there multiple timing mechanisms in the brain? Neuropsychological research has tended to favor the latter answer, with researchers pointing to the fact that damage to a wide range of neural systems can disrupt performance on tasks that involve temporal information processing.[7] These results have led Richelle *et al.*[8] to conclude that we should "admit that there are as many clocks as there are behaviors exhibiting timing properties" (p. 90). However, conclusions such as this may be ill-advised. As noted previously, behaviors that exhibit temporal regularities need not require the operation of an internal clock.

---

[a] The author was supported in part by National Institute of Neurological Disorders and Stroke Grant NS30256 and in part by a fellowship from the Sloan Foundation.

Moreover, the plethora of neural disabilities associated with impaired performance on timing tasks may simply reflect the fact that these tasks require the integration of a number of cognitive operations, only one of which may be devoted to the representation of temporal information.

These two questions have shaped the framework for our research strategy on timing. We have chosen tasks that not only appear to require explicit timing, but are also as simple as possible in order to reduce the importance of nontemporal processes. Our initial research with healthy young adults led to the hypothesis that these tasks involve the operation of a common internal clock, and our subsequent patient research indicated that the cerebellum is the critical neural structure associated with this timing mechanism. More recently, we have shifted our focus to investigate the generality of the cerebellar timing hypothesis. This issue can be approached on two fronts. First, we can use a new set of tasks to assess whether the integrity of the cerebellum is necessary for successful performance, thus indicating the role of timing in these tasks. Second, we can reexamine tasks that have been associated with the cerebellum to see whether the timing hypothesis provides a parsimonious interpretation of the data.

THE CEREBELLUM AS AN INTERNAL CLOCK

Two tasks have been central to our research. The first, a production of time task, is a repetitive finger-tapping task. Each trial begins with the presentation of a series of computer tones, separated by isochronous intervals. The intertone interval in our research has varied from 325 ms to 550 ms. By pressing a response key, the subject attempts to synchronize his or her responses with the tones. After a series of paced responses, the tones cease and the subjects' task is to continue tapping at the target rate until they have produced 30 unpaced intertap intervals (ITIs). Our primary measure is the variability of the standard deviation of the ITIs. Given that the target ITI is arbitrarily defined, we assume that this task requires explicit control of the timing of the response.

The second task is a perception of time task. On each trial two intervals are presented, a standard and a comparison interval. The intervals can be unfilled, marked at the beginning and end by either 50-ms tones or light flashes, or filled, in which case the interval is represented by the duration of a continuous tone or visual stimulus. The subjects' task is to indicate whether the comparison interval is shorter or longer than the standard interval. The duration of the comparison interval is varied, and responses to the different values are collected. From the obtained psychophysical function, a difference threshold can be estimated to provide a measure of temporal acuity on this task. For example, if the standard interval is 400 ms, a subject with good temporal acuity might have a difference threshold (1 standard deviation) of 25 ms, whereas a subject with poor acuity might have a difference threshold of 35 ms.

A significant correlation between performance on the motor and perception timing tasks is found in healthy young adults.[9] People who were consistent on the repetitive-tapping task also had lower difference thresholds on the time-perception task. Significant correlations were also obtained when subjects performed the repetitive-tapping task with different effectors (hand, foot, forearm), whereas little correlation was observed when comparing performance on this task with that measured on other motor tasks that did not require precise timing.[10] These results suggest that there is a common timing mechanism that is utilized in both perception and action, a result that meshes with our intuition. The lack of correlation between the repetitive-tapping and the nontiming motor tasks indicates that the correlations between the timing tasks cannot be attributed to general factors such as motivation.

Evidence of an internal clock that is shared by both motor and perceptual systems raises the possibility that this function might be dependent on a restricted set of neural structures. That is, if a common internal clock is associated with a certain neural structure, then lesions of this structure should disrupt performance on both motor and perceptual tasks that require precise timing. Positive results, of course, would not only provide converging evidence for a common timing mechanism, but would also be an important first step in identifying the neural bases of this form of timing.

To explore this issue, we tested three different patient groups on the repetitive-tapping and auditory time-perception tasks.[11] The three groups were (1) patients with lesions that included premotor cortical areas ($n = 8$), (2) patients with Parkinson's disease ($n = 30$), and (3) patients with cerebellar lesions ($n = 30$). While all of these patients had suffered a loss of coordination, the control of timing is presumably only one component of coordination. We were interested in whether the tapping task would yield any dissociations in performance. In particular, normal performance on the tapping task for one of these groups would suggest that the affected neural structures for that group are not part of an internal timing mechanism.

Two of the patient groups, the cortical patients and the cerebellar patients, were impaired on the tapping task in comparison to age-matched control subjects (FIG. 1). The mean standard deviation for these groups was approximately 50 percent higher than that obtained from the control subjects. In contrast, the Parkinson patients performed comparably to the control subjects. A subset of the Parkinson patients were tested in a second session after having skipped their L-DOPA medication for about 16 hours. While these patients showed a severe exacerbation of Parkinson symptoms in the *off* medication state, their performance on the tapping task was unchanged (but was significantly altered on a second motor task measuring their ability to produce isometric force pulses).

The finding that two patient groups were impaired on the tapping task is problematic. There are at least two interpretations of these results. One possibility is that timing may be a distributed process, dependent on the normal functioning of both cortical and cerebellar structures. Alternatively, since the tapping task may be disrupted by other functional deficits that do not involve timing, only one (or neither) of these structures may be involved in timing. For example, the patient may be able to time the movements, but may have difficulty in executing the movements. Thus, the perception task is critical in determining if the deficits on the tapping task truly reflected a deficit in timing, since there were no motor requirements in this task.

The results of the perception task supported the hypothesis that timing was not a distributed process. Only the patients with cerebellar lesions were impaired on this task (FIG. 2). Whereas the difference threshold for control, Parkinson, and cortical patients was about 30 ms, the value for the cerebellar group was 46 ms. An important control task emphasized that this deficit was specific to timing. When judging the loudness of a variable pair of tones in comparison to the loudness of a standard tone pair, the cerebellar patients performed as well as the control subjects. An unexpected, but important finding was that the cortical patients were impaired on this task (perhaps because some of the lesion extended into primary or secondary auditory areas). Thus, the two perception tasks yielded a double dissociation. The cerebellar patients were selectively impaired on the time-perception task, while the cortical patients were selectively impaired on the loudness-perception task. In neuropsycho-

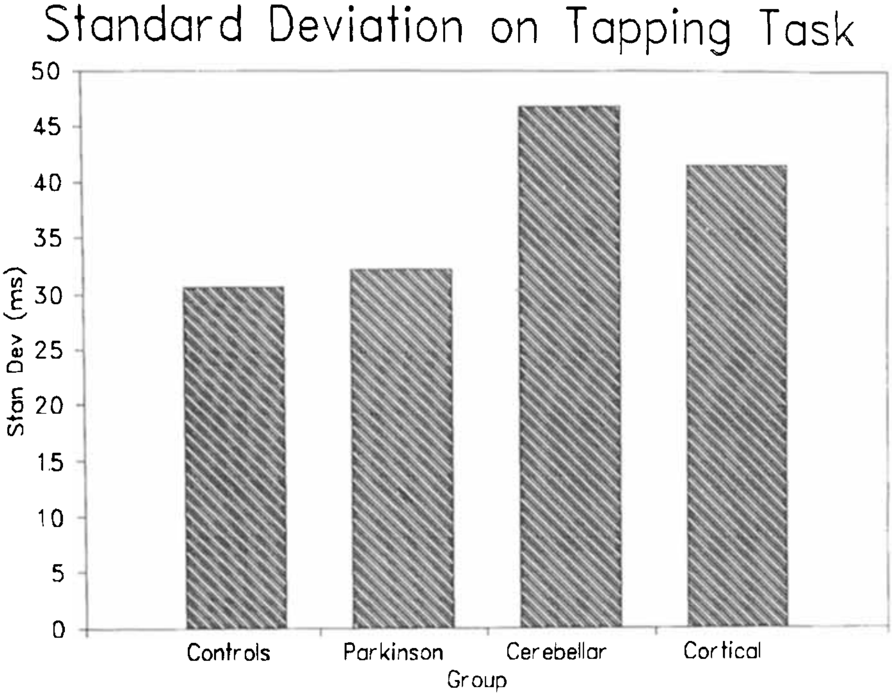


FIGURE 1. Mean standard deviation of intertap intervals on the repetitive tapping task for age-matched control subjects and three neurological groups with movement disorders. (From Ivry and Keele.[11] Reproduced by permission.)

logical research, double dissociations provide the strongest evidence that a particular mental operation (e.g., timing) is specifically associated with a neural system (e.g., the cerebellum).

Taken together, the impaired performance of the cerebellar patients on the repetitive-tapping and time-perception tasks provided evidence that the cerebellum could be characterized as an internal timing mechanism. A role for the cerebellum in temporally coordinating motoric events has been suggested by other researchers.[12,13] A timing hypothesis can also provide an account of many of the symptoms observed in cerebellar patients. For example, electromyography has indicated that intentional tremor and dysmetria result from abnormal timing of antagonist muscle activity to counteract the forces generated by agonist muscles.[14,15] The finding that cerebellar patients were impaired on a purely perceptual task was more surprising given the traditional association of this neural structure with motor control. Our hypothesis is that the prominent role of the cerebellum in motor control is because most coordinated actions require fine timing. More important, the computational capabilities of this structure are not restricted to the motor domain, but are also accessible to nonmotor tasks that are dependent on precise timing. In other words, the domain of cerebellar function is not defined in terms of tasks (e.g., motor control), but in terms of the computation performed by this structure (e.g., timing).

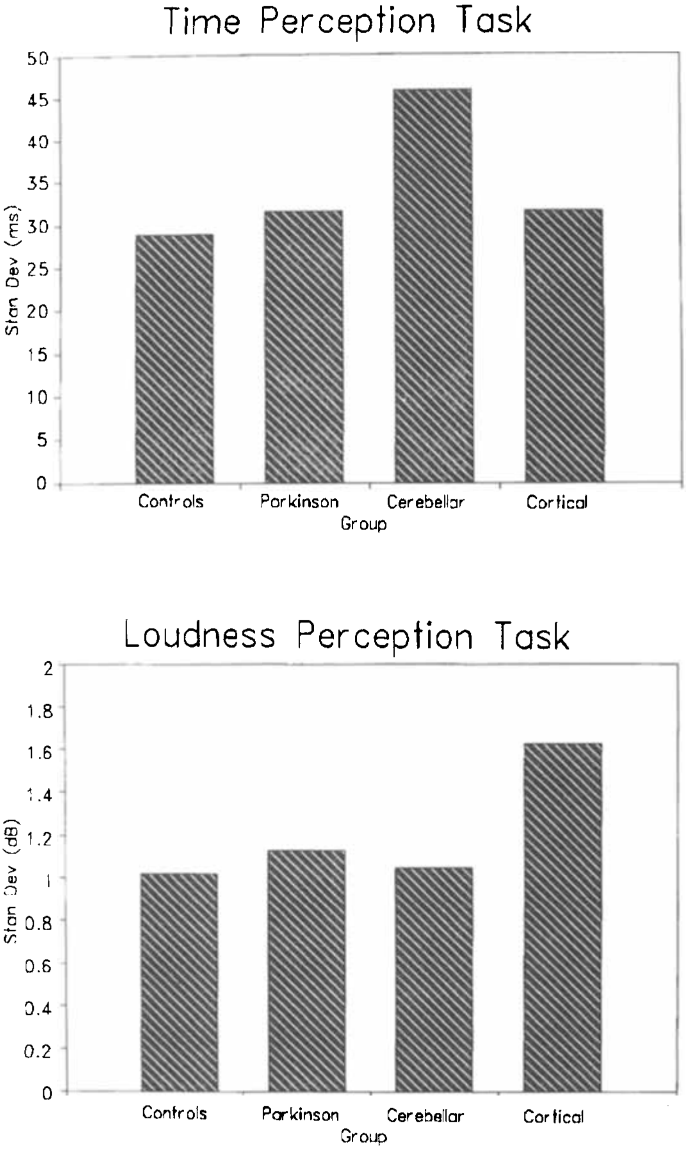


FIGURE 2. Mean standard deviation on a time perception (**top panel**) and a loudness perception (**bottom panel**) task. The scores were obtained using the parameter estimation by sequential testing procedure (PEST). (From Ivry and Keele.[11] Reproduced by permission.)

In subsequent research, we have sought to replicate these results, to identify those regions within the cerebellum that are most essential for timing, and to assess whether the cerebellum plays a role in other tasks that require timing. In one study, patients with focal cerebellar lesions, either in medial or lateral regions, were tested on the repetitive-tapping task.[16] Since the lesions were unilateral, each patient could serve as his or her own control by comparing performance when tapping with a finger on the impaired side with his or her performance when tapping with a finger on the unimpaired side. A mathematical model was used to determine whether the increased variability when tapping with the affected hand was due to a timing deficit or a problem in motor implementation.[4] A second double dissociation was found in this study. The model indicated that the source of the deficit for patients with medial lesions was due to problems in implementation, whereas the source of the deficit for patients with lateral lesions was due to a problem in timing. This result is consistent with the pattern of neural connectivity of the cerebellum.[17,18] Medial regions project primarily to spinal structures that are associated with motor execution. In contrast, lateral cerebellar regions project to regions of the cerebral cortex, including areas associated with motor planning.

In extensions of our perceptual results, patients with cerebellar lesions were tested on the time-perception task under various conditions (unpublished data). First, as in the original study, unfilled intervals were marked by 50-ms auditory clicks. In the second condition, the standard and comparison duration were continuous tones creating filled intervals. In the third condition, the temporal stimuli were signaled by the illumination of a light for a variable duration. The patients performed more poorly than control subjects in all three conditions. As before, no difference was found between the patients and control subjects on a nontemporal perception task, the loudness perception task.

A different population of subjects with coordination problems provided further evidence of the role of the cerebellum in timing. A significant proportion of children are diagnosed as developmentally clumsy. Clumsiness, however, can take many forms and there is some evidence that the symptoms may reflect subclinical neurological disorders. In our study, two groups of clumsy children were identified: those with soft neurological signs consistent with basal ganglia dysfunction and those with soft neurological signs of cerebellar dysfunction. Only the children with cerebellar symptoms were impaired on the repetitive-tapping and time-perception tasks.[19,20]

Given the inherent limitations in human neuropsychological research, we are currently developing an animal model to investigate the neural mechanisms of time perception. In our first experiment, twelve rats were trained on a time-discrimination task in which they were rewarded for pressing one lever following the presentation of a visual stimulus for 360 ms and a second lever following the presentation of a 670-ms visual stimulus. One of these two stimuli were presented on half of the trials. On the other trials, a range of probe durations were presented and responses to these stimuli were not rewarded. From the responses to the complete set of stimuli, a psychometric function can be plotted and used to estimate a point of subjective equality and a standard deviation for each rat. Following extensive training on this task, bilateral cerebellar lesions were made in eight of the animals. The lesions were targeted for the lateral cerebellar nuclei (dentate and interpositus) given that output from the neocerebellum must pass through these nuclei. Four other animals received sham lesions.

In this initial experiment, the lesioned animals were significantly impaired in comparison to either their presurgery performance or the postsurgery performance of the sham group (FIG. 3). As in our human studies, cerebellar lesions increased the variability of the rats' responses. In addition, the point of subjective equality, the 50 percent point in the psychometric functions, was shifted to an earlier point in time for seven of the eight rats with cerebellar lesions. For some of the animals, the deficits were relatively long lasting, extending throughout the two month postsurgery testing period. For others, recovery appeared to be complete by the end of the experiment.

In further studies, we plan to determine if the rate of recovery is correlated with the foci of the lesions. Furthermore, we also need to conduct some important control experiments, such as demonstrating that cerebellar lesions do not produce a general impairment in the rats' performance on psychometric testing. For example, if a stimulus parameter such as brightness was varied, we would not expect to observe any deficit following cerebellar lesions. Finally, we are attempting to identify the boundary conditions of the cerebellar timing system. Rats are being simultaneously trained on two duration discrimination tasks, a short-range timing task and a long-range timing task. In the short-range task, the durations are centered around 525 ms; for the long-range task, the durations are centered around 33 s. We predict that cerebellar lesions will selectively impair performance on the short-duration task and not the long-duration task. Such a result, coupled with previous findings implicating cholinergic systems in timing tasks on the order of 10–30 s[21,22] would begin to identify dissociable neural mechanisms of timing.

The tapping and time-perception results had led to the cerebellar timing hypothesis. Given this hypothesis, the role of the cerebellum in other tasks that require temporal information processing can be investigated. Ivry and Diener[23] found that patients with cerebellar lesions were impaired in judging the velocity of a moving

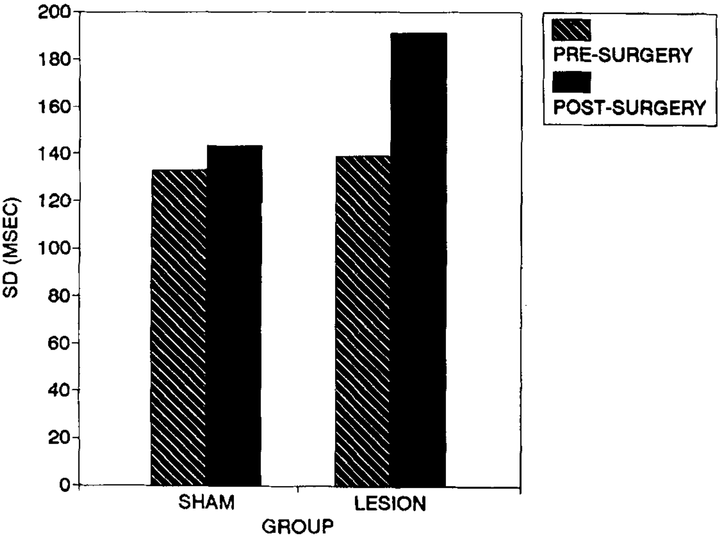


FIGURE 3. Mean standard deviation for experimental (n=8) and sham (n=4) rats on the time-perception task. The scores were obtained using the method of constant stimuli. (Unpublished data.)

visual stimulus (Fig. 4). In three experiments, subjects had to judge the relative speed of a display of moving dots. They were instructed to maintain fixation during the stimulus presentation to eliminate any effects that might be due to the aberrant eye movements observed in cerebellar patients. Performance on this task was similar for patients who were unable to maintain fixation. Moreover, errors on the perceptual task were not correlated with electroocular activity. These results suggest that the deficit in the velocity perception task are not the indirect consequence of a motor problem in controlling eye movements.

The velocity perception study provides an example of how the issue of implicit and explicit timing can be investigated. Velocity, by definition, requires an analysis of information over time. However, the temporal information need not be explicitly represented. Velocity detectors could be hardwired, with different activation thresholds being associated with inputs arising from neighboring spatial locations.

However, the timing capabilities of the cerebellum might be exploited for computing precise velocity information. The activity in some neocerebellar Purkinje cells is correlated with the speed of a moving visual stimulus, independent of the motion effects introduced by eye, head, and body motion.[24] These cells could thus be generating a viewer-independent representation of the stimulus motion that would be essential for generating predictive pursuit or saccadic eye movements, functions that are known to be impaired in cerebellar patients.[25-27] These findings suggest that problems in generating predictive eye movements following cerebellar lesions may arise from deficits in computing velocity information. If this hypothesis is correct, then on the basis of parsimony, we would infer that velocity perception requires the

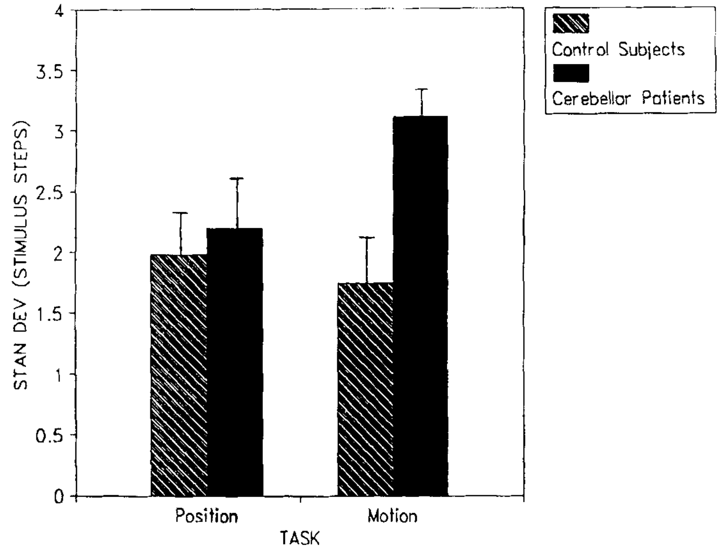


FIGURE 4. Mean standard deviation for age-matched control subjects and patients with cerebellar lesions on two visual-perception tasks. In the control task, the stimuli were similar to the motion task, but the position of the comparion stimulus was shifted relative to the position of the standard stimulus. Temporal information processing was assumed to only be required in the motion task. (From Ivry and Diener.[23] Reproduced by permission.)

explicit processing of temporal information and that because of this requirement, the cerebellum is an integral part of the process.

CEREBELLAR TIMING IN SPEECH PERCEPTION AND PRODUCTION

Speech perception and speech production are, of course, preeminent examples of tasks in which information must be processed over time. The logic that guided our research on velocity perception can also be applied to these task domains. We have investigated one speech phenomenon that might be expected to utilize an explicit timing mechanism: the perception and production of voicing in consonant–vowel syllables. In producing voiced phonemes such as /b/ and /d/, the vocal cords begin to vibrate at approximately the same time as the release of airflow. In contrast, the vocal-cord vibration is delayed in the production of unvoiced phonemes such as /p/ and /t/. The time between airflow release and vocal-cord vibration is referred to as *voice-onset time* (VOT). In English, the VOT for the syllable "ba" is approximately 0 ms, whereas the VOT for the syllable "pa" is about 40 ms.

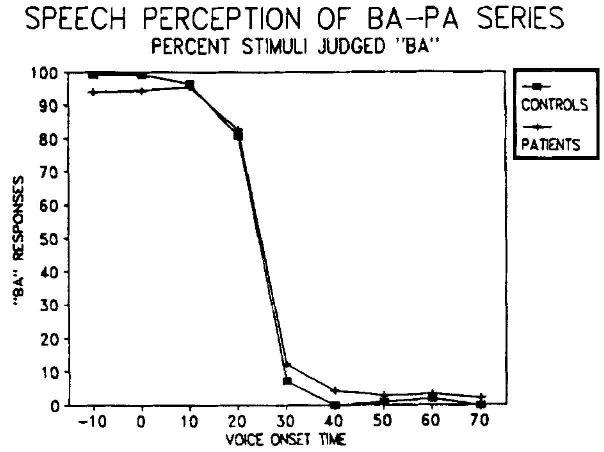
Using a speech synthesizer to generate the formants associated with "ba" and "pa," a continuum of speech sounds can be created by varying VOT (done by deleting information in the vicinity of the fundamental and first formant). A seminal result in speech perception is that such continua are perceived categorically.[28] Below a critical VOT value, subjects consistently perceive "ba"; above that value, the percept is consistently "pa," and the boundary from one percept to the other is much steeper than that observed with nonspeech stimuli containing similar frequency information.

To determine if the cerebellar timing mechanism was involved in the perception of VOT, cerebellar patients were tested on a "ba-pa" continuum.[29] For control purposes, the patients were also tested on a "ba-da" continuum. For these stimuli, the synthetic manipulation is of the second and third formant transitions, with the temporal events being constant. On both continua, the performance of the cerebellar patients was similar to that of age-matched control subjects (FIG. 5). This null result was replicated in a second group of patients (unpublished data).

Different interpretations of these null results are raised when considering the issue of implicit versus explicit timing. On the one hand, VOTs may be explicitly computed. If so, the lack of a deficit would indicate that the requisite processing falls outside the domain of the cerebellar timing system. It has been argued that speech perception involves dedicated processors.[30] Perhaps one of these processors may be specialized for computing temporal information in the speech signal.

On the other hand, the timing differences between voiced and voiceless phonemes may only be represented implicitly. These differences may be correlated with a different acoustic cue, one that is exploited by psychological processors involved in speech perception. For example, there are marked spectral differences between voiced and voiceless phonemes, with the former having much greater power in the lower frequency region.[31,32] A psychological operation that analyzed the distribution of energy in the frequency spectrum at signal onset should be able to determine if a sound is voiced or voiceless without needing any explicit representation of temporal information (other than be able to note the onset of a signal). If this were the case, then the null results for the cerebellar patients could simply reflect the fact that precise timing is not needed for this speech-perception task.[33]

While we have not identified any speech-perception deficits in cerebellar patients, the timing hypothesis does offer a parsimonious account of certain aspects of the



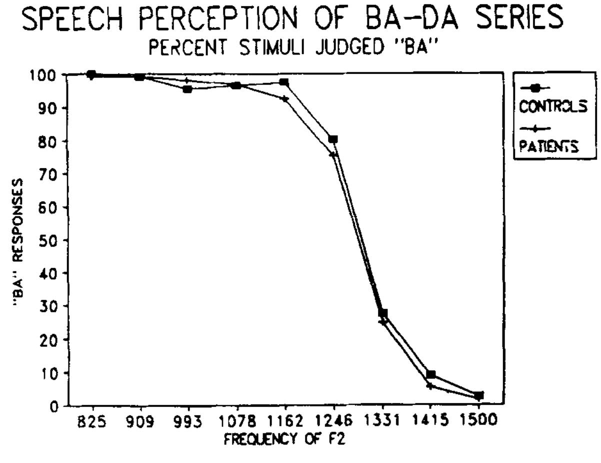


FIGURE 5. Identification functions for the speech-perception tasks for age-matched control subjects and patients with cerebellar lesions. **Top panel** is for the "ba-pa" continuum in which the stimuli vary in simulated VOT. **Bottom panel** is for the "ba-da" control continuum in which the stimuli vary in the trajectory of the simulated second and third formant transitons. (From Ivry and Gopal.[29] Reproduced by permission.)

speech disorders observed in these patients. Cerebellar dysarthria is characterized by inconsistency in the articulation of phonemes and heightened variability in the control of rate and stress.[34] We hypothesized that if cerebellar dysarthria were due to a selective deficit in temporally coordinating events across different articulators, then errors in articulation should conform to a predictable distribution. Specifically, errors should be distributed on the voicing dimension and not in terms of place of articulation ("ba" versus "da"). For example, if, due to poor temporal coupling, the initiation of vocal-cord vibration was delayed when attempting to say "ba," the percept might be "pa." A problem in temporally coordinating the actions underlying voicing and consonantal release would not lead a "ba" to be heard as a "da." The temporal structure of these two syllables is similar; they differ in terms of the configuration of the positioning of the lips and tongue.

These predictions were supported in two analyses.[29] When healthy subjects labeled the patients' productions, mispercepts were almost always along the VOT dimension and rarely along the control, spectral dimension (TABLE 1). For example, if the patient said "ba," the judge was much more likely to hear it as "pa" than "da." Of the 26 labeling errors in this task (13 percent of total productions), 88 percent were due to errors along the voicing dimension. Moreover, acoustic measurements of VOT in the productions of cerebellar patients were significantly more variable than those obtained from age-matched controls. Spectral events, measured during the steady-state vocalic portion of the syllables, revealed fewer differences between the patients and control subjects.

At first glance, the speech results might seem at odds with the claim that the cerebellum operates as an internal timing mechanism. The timing hypothesis was motivated by the finding that cerebellar patients show a dual deficit in both production and perception tasks that require precise timing. In the speech domain, the timing hypothesis can account for at least some aspects of the patients' production problems, but these same patients were unimpaired on the speech-perception task. While this discrepancy may indicate problems for the cerebellar timing hypothesis, it is also possible that the critical factor is that the cerebellum is only involved when the task re-

TABLE 1. Errors in Perceptual Classifications of Syllables Produced by Patients with Cerebellar Dysarthria

Stimulus	Response	Total
Controls		
Voiced	Voiceless	2
Voiceless	Voiced	0
Total Voicing Errors		2
Labial	Alveolar	3
Alveolar	Labial	1
Total Place Errors		4
Patients		
Voiced	Voiceless	12
Voiceless	Voiced	11
Total Voicing Errors		23
Labial	Alveolar	0
Alveolar	Labial	3
Total Place Errors		3

quires explicit timing. It seems reasonable to assume that this is the case in the repetitive-tapping task. As with other complex movements, the problems seen in the speech of cerebellar patients can be attributed to the need for precise temporal coordination between the activity of different muscle groups. However, even if timing is explicitly controlled in the production of actions such as speech, the perception of these actions need not involve the representation of temporal information.

CEREBELLAR TIMING AND SENSORIMOTOR LEARNING

The cerebellum has been assumed to play a major role in sensorimotor learning. While the first evidence came from studies examining adaptation of the vestibulo-ocular reflex,[35] recent work has involved a classical conditioning paradigm.[36,37] In this paradigm, a conditioned stimulus (CS), such as a tone, is repeatedly paired with an unconditioned stimulus (US), such as an airpuff, delivered to the cornea. The airpuff will trigger an unconditioned response (UR), the retraction of the nictitating membrane. Over trials, the tone will come to elicit a similar response (CR). Numerous studies have now demonstrated that acquisition and/or retention of the CR is severely disrupted or even abolished following cerebellar lesions. Since the UR remains intact, the effect is attributed to a learning deficit rather than a motor deficit. The learning hypothesis is further supported by studies indicating that the cerebellum is a plausible site for integrating information from pathways conveying signals associated with the US and CS.[38]

It is important to ask why the cerebellum is critical for the acquisition and retention of conditioned responses. A first proposal might be that the cerebellum is essential for learning the simple, reflexivelike responses elicited in conditioning paradigms. If this hypothesis were correct, then the cerebellum would be expected to play a role in all forms of classical conditioning. This hypothesis is refuted by the available evidence. In the eyeblink paradigm, the CS not only comes to elicit an eyeblink CR, but it also becomes associated with other fear-related CRs, such as a decrease in heart rate. Cerebellar lesions have no effect on the heart-rate response.[39]

The dissociation between the eyeblink and heart rate CRs indicate that only a subset of conditioned responses is dependent on the cerebellum. One hypothesis that would account for this dissociation would be that the cerebellar learning domain is restricted to responses dependent on the skeletal musculature. Autonomic responses (such as heart rate) are assumed to be mediated by other neural systems. An alternative hypothesis is that the cerebellum is essential when the task requires the animal to represent the temporal relations between different stimuli. That is, the cerebellum is critical for learning tasks that require explicit timing.

Consider the eyeblink paradigm. The conditioned eyeblink is only adaptive if it occurs prior to the airpuff, allowing the animal to reduce the negative effects of the aversive US. The animal must then learn the temporal relationship between the CS and US in order to be able to produce an anticipatory CR. That the animal is learning about the timing between the two stimuli rather than simply linking the CR to the first stimulus is shown by studies that manipulate the CS–US interval. Over a range of CS–US intervals, the peak amplitude of the CR occurs just prior to the onset of the US. Thus, the time at which the CR peaks is directly related to the CS–US interval.[40] In conditioning of autonomic responses, such as changes in heart rate, there is only a weak correlation between CR latency and the CS–US interval.

Thus, there are two different accounts of the cerebellar role in learning. One hypothesis focuses on the task domain, postulating that the cerebellum is essential for learning skeletal responses. The other hypothesis emphasizes a computational mechanism, postulating that the cerebellum will be required when the task requires the representation of precise temporal information. A recent unpublished study by Steinmetz provides an initial unconfounding of these two hypotheses.

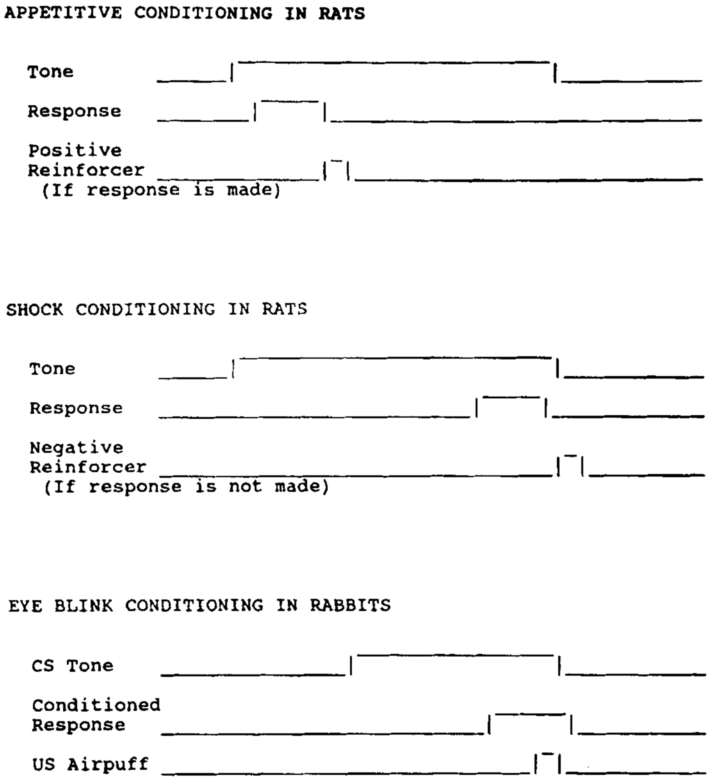
Rats were trained on one of two operant conditioning tasks.[41] The stimulus and (overt) response was the same in both tasks. A 1-s tone was presented and the animal had to press a lever during that 1-s interval in order to receive a reinforcement. What differed between the two groups was the nature of the reinforcer. For the positive condition, rats received food if they responded prior to the termination of the tone. For the negative condition, rats were shocked if they failed to respond before the end of the tone.

Separate groups of rats were trained to comparable levels of performance on the positive and negative tasks. The animals in both groups were then given bilateral cerebellar lesions. A striking dissociation was evident during postsurgery testing. Whereas the lesions produced a marked impairment in performance on the negative task, performance on the positive task was unaffected. This result is at odds with predictions derived from a hypothesis emphasizing the importance of the cerebellum for learned skeletal responses. In both conditions, the response is a single lever press. The lesions, however, only disrupted learning in one condition.

Examination of the response topography suggests that the dissociation observed by Steinmetz may be accounted for by the timing hypothesis (FIG. 6). While the response is always a lever press, the time at which the response occurs is different for the two groups. Rats trained in the positive condition, appear to respond as quickly as possible: their response latencies are skewed toward the initial half of the 1-s tone interval and appear to form a simple reaction time distribution. In contrast, the distribution of response latencies in the negative condition are skewed toward the end of the 1-s tone interval. In this condition, the rats appear to be delaying their responses for as long as possible. It is unclear why there is this difference in the distribution of the response latencies. For whatever reason, in the positive condition, the rats obtain the reward as quickly as possible, and in the negative condition, they respond at the last possible moment. (Perhaps evolutionary pressures have acted to create this difference. If food is available, one must act fast to beat competitors. If punishment is imminent, delay taking avoidance action in the hope that an external agent will intervene.) These distributions suggest that the animal requires explicit timing in only the negative condition. Thus, the selective effects of cerebellar lesions may be the result of the timing demands in the negative condition. The null results for the positive condition would reflect the fact that the critical associative process for this group is not dependent on the explicit representation of temporal information.

Steinmetz's experiment may have inadvertently unconfounded the skeletal musculature and timing hypotheses. However, it is important to note that a second confound remains. All of the cases in which the cerebellum is associated with learning not only require precise timing, but also involve situations where the animal must learn an avoidance response. That is, in all of these conditions, the animal is able to use an external error signal (airpuff, retinal slip, or shock) to modify its behavior. Perhaps the cerebellum is essential for learning avoidance responses involving the skeletal musculature.[37] The cerebellum was not involved in the condition where the rat presses the lever in order to obtain a food reward, but here there is really no error signal (other than internal states associated with hunger or frustration).

What is needed are experiments that unconfound timing and error correction. Together with Steinmetz, we are currently conducting such an experiment. In the positive-timed condition, we are setting up a situation where the rat must time a


APPETITIVE CONDITIONING IN RATS

SHOCK CONDITIONING IN RATS

EYE BLINK CONDITIONING IN RABBITS

FIGURE 6. Schematic diagram of the temporal relationship between the stimuli and response in three animal learning paradigms (see text). Note that the animals' responses are delayed until the end of the tone in the shock-conditioning and eye-blink-conditioning paradigms.

response in order to receive a food pellet. To do this, the stimulus is again a 1-s tone, but the rats are only reinforced when they respond during the last 300 ms of the stimulus. In a negative-untimed condition, the interval between the tone onset and shock is varied. We expect this variation will make the rats respond as quickly as possible, thus removing the need to do any timing. If timing is the critical factor, then lesioned animals should show a learning impairment in the positive-timed condition. If error correction is important, then the lesions should only disrupt performance in the negative-untimed condition.

CONCLUSIONS

The research reviewed in this chapter offers some resolutions to the two questions posed in the Introduction. Studies with healthy subjects indicated a correlation between performance on movement and perception tasks that required precise timing. This evidence bolstered our assumption that these tasks involve the explicit representation of temporal information. Neuropsychological studies indicated that the cerebellum was a critical neural structure for representing this information. Further patient and animal research has provided one means of assessing whether other tasks require the explicit representation of temporal information. These studies have demonstrated that the timing hypothesis can provide a parsimonious account of a number of seemingly disparate functions associated with the cerebellum.

Two points, however, should be kept in mind in considering the generality of the cerebellar timing hypothesis. First, it would be unreasonable to assume that the cerebellum is involved in all tasks that require the explicit representation of temporal information. This information occurs over a variety of scales, and the cerebellar timing system may be capable of representing this information over a limited temporal range. If the domain of the cerebellum was originally limited to the coordination of action, then its timing capabilities may have been constrained by evolutionary processes to a range relevant for motor control. Second, it cannot be inferred from the timing hypothesis that there is a single timing mechanism in the cerebellum. Rather, the hypothesis is offered as a general computational description of cerebellar function. It is reasonable to suppose that this computational capability is distributed throughout the cerebellum, with different regions representing the temporal information utilized in different tasks.

ACKNOWLEDGMENTS

Steven Keele, Eliot Hazeltine, and Art Shimamura provided useful comments in the preparation of this paper.

REFERENCES

1. Turvey, M. T., L. D. Rosenblum, P. N. Kugler & R C. Schmidt. 1986. Fluctuations and phase symmetry in coordinated rhythmic movements. J. Exp. Psychol.: Hum. Percept. Perform. **12:** 564–583.
2. Ivry, R. & D. Corcos. Slicing the variability pie: Component analysis of coordination and motor dysfunction. *In* Variability and Motor Control, K. Newell and D. Corcos, Eds. In press.
3. Kelso, J. A. S. 1984. Phase transitions and critical behavior in human bimanual coordination. Am. J. Physiol. **15:** R1000–R1004.
4. Wing, A. 1980. The long and short of timing in response sequences. *In* Tutorials in Motor Behavior, G. Stelmach and J. Requin, Eds. North-Holland. New York.
5. Haken, H., J. A. S. Kelso & H. Bunz. 1985. A theoretical model of phase transitions in human hand movements. Biol. Cybern. **51:** 347–356.
6. Ivry, R. & R. Hazeltine. 1992. Models of timing-with-a-timer. *In* Time, Action, and Cognition, F. Macar, V. Pouthas, and W. Freidman, Eds.: 183–189. Kluwer. Dordrecht, the Netherlands.
7. Pastor, M. A., J. Artieda, M. Jahanshahi & J. A. Obeso. 1992. Time estimation and reproduction is abnormal in Parkinson's disease. Brain **115:** 211–225.
8. Richelle, M., H. Lejeune, J. J. Perikel & P. Fery. 1985. From biotemporality to nontemporality: Toward an integrative and comparative view of time in behavior. *In* Time, Mind, and Behavior, J. A. Michon and J. L. Jackson, Eds.: 75–99. Springer-Verlag. Berlin/New York.

9. Keele, S., R. Pokorny, D. Corcos & R. Ivry. 1985. Do perception and motor production share common timing mechanisms? Acta Psychol. **60:** 173–193.
10. Keele, S., R. Ivry & R. Pokorny. 1987. Force control and its relation to timing. J. Motor Behav. **19:** 96–114.
11. Ivry, R. B. & S. W. Keele. 1989. Timing functions of the cerebellum. J. Cogn. Neurosci. **1:** 134–150.
12. Houk, J. C., S. P. Singh, C. Fisher & A. G. Barto. 1990. An adaptive sensorimotor network inspired by the anatomy and physiology of the cerebellum. *In* Neural Networks for Control, W. T. Miller, R. S. Sutton, and P. J. Werbos, Eds.: 301–348. MIT Press. Cambridge, Mass.
13. Pellionisz, A. & R. Llinas. 1982. Space-time representation in the brain: The cerebellum as a predictive space-time metric tensor. Neuroscience **7:** 2949–2970.
14. Hore, J., B. Wild & H. C. Diener. 1991. Cerebellar dysmetria at the elbow, wrist, and fingers. J. Neurophysiol. **65:** 563–571.
15. Vilis, T. & J. Hore. 1980. Central neural mechanisms contributing to cerebellar tremor produced by limb perturbations. J. Neurophysiol. **43:** 279–291.
16. Ivry, R., S. Keele & H. Diener. 1988. Dissociation of the lateral and medial cerebellum in movement timing and movement execution. Exp. Brain Res. **73:** 167–180.
17. Asanuma, C., W. Thach & E. Jones. 1983. Anatomical evidence for segregated focal groupings of efferent cells and their ramifications in the cerebellothalamic pathway of the monkey. Brain Res. Rev. **5:** 267–298.
18. ———. 1983. Brainstem and spinal projections of the deep cerebellar nuclei in the monkey with observations on the brainstem projections of the dorsal column nuclei. Brain Res. Rev. **5:** 299–322.
19. Lundy-Ekman, L., R. Ivry, S. W. Keele & M. Woollacott. 1991. Timing and force control deficits in clumsy children. J. Cogn. Neurosci. **3:** 370–377.
20. Williams, H. G., M. H. Woollacott & R. Ivry. 1992. Timing and motor control in clumsy children. J. Motor Behav. **24:** 165–172.
21. Meck, W. H. 1983. Selective adjustment of the speed of internal clock and memory processes. J. Exp. Psychol.: Anim. Behav. Processes **9:** 171–201.
22. Meck, W. H. & R. M. Church. 1987. Cholinergic modulation of the content of temporal memory. Behav. Neurosci. **101:** 457–464.
23. Ivry, R. B. & H. C. Diener. 1991. Impaired velocity perception in patients with lesions of the cerebellum. J. Cogn. Neurosci. **3:** 355–366.
24. Suzuki, D. & E. Keller. 1988. The role of the posterior vermis of monkey cerebellum in smooth-pursuit eye movement control. II. Target velocity-related Purkinje cell activity. J. Neurophysiol. **59:** 19–40.
25. Aschoff, J. & B. Cohen. 1971. Changes in saccadic eye movements produced by cerebellar cortical lesions. Exp. Neurol. **32:** 123–132.
26. Ritchie, L. 1976. Effects of cerebellar lesions on saccadic eye movements. J. Neurophysiol. **39:** 1246–1256.
27. Westheimer, G. & S. Blair. 1974. Functional organization of primate oculomotor system revealed by cerebellectomy. Exp. Brain Res. **21:** 463–472.
28. Abramson, A. & L. Lisker. 1968. Voice timing: Cross-language experiments in identification and discrimination. Paper presented at the meetings of the Acoustical Society of America, Ottawa, Ont., Canada.
29. Ivry, R. & H. S. Gopal. Speech production and perception in patients with cerebellar lesions. *In* Attention and Performance, Vol. XIV, D. Meyer and S. Kornblum, Eds. Lawrence Erlbaum. Hillsdale, N.J. In press.
30. Liberman, A. & I. Mattingly. 1985. The motor theory of speech perception revised. Cognition **21:** 1–36.
31. Lisker, L. 1975. Is it VOT or a first-formant transition detector? J. Acoust. Soc. Am. **56:** 1547–1551.
32. Stevens, K. N. & D. H. Klatt. 1974. Role of formant transitions in the voiced-voiceless distinction for stops. J. Acoust. Soc. Am. **55:** 653–659.
33. Ivry, R. & P. Lebby. Hemispheric differences in auditory perception are similar to those found in visual perception. Psychol. Sci. In press.

34. DARLEY, F., A. ARONSON & J. BROWN. 1969. Differential diagnostic patterns of dysarthria. J. Speech Hear. Res. **12:** 246–269.
35. ITO, M. 1984. The Cerebellum and Neural Control. Raven Press. New York.
36. YEO, C. H. 1991. Cerebellum and classical conditioning of motor responses. Ann. N.Y. Acad. Sci. **627:** 292–304.
37. THOMPSON, R. F. 1990. Neural mechanisms of classical conditioning in mammals. Philos. Trans. R. Soc. Ser. B **329:** 161–170.
38. STEINMETZ, J. E., D. G. LAVOND & R. F. THOMPSON. 1989. Classical conditioning in rabbits using pontine nucleus stimulation as a conditioned stimulus and inferior olive stimulation as an unconditioned stimulus. Synapse **3:** 225–233.
39. LAVOND, D. G., J. S. LINCOLN, D. A. MCCORMICK & R. F. THOMPSON. 1984. Effect of bilateral lesions of the dentate and interpositus nuclei on conditioning of heart-rate and nictitating membrane/eyelid responses in the rabbit. Brain Res. **305:** 323–330.
40. WICKENS, D. D., A. F. NIELD, D. S. TUBER & C. WICKENS. Strength, latency, and form of conditioned skeletal and automatic responses as functions of CS-UCS intervals. J. Exp. Psychol. **80:** 165–170.
41. LOGUE, S. F., P. D. MILLER & J. E. STEINMETZ. 1991. A signalled bar-pressing preparation for study of the neural bases of appetitive and aversive learning in the rat. Neurosci. Abstr. **17:** 660.