Chapter 15

Slicing the Variability Pie: Component Analysis of Coordination and Motor Dysfunction

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As emphasized by the chapters in this volume, the study of variability is essential for understanding coordination. We can choose to focus on the competence of the performer, identifying his or her capabilities and skills. But in doing so, we are acknowledging the limitations of this performer. An important source of constraint underlying these limitations is that the individual performances of a particular task are variable. Differences between individuals may result from differences in variability between individuals on a particular task. On the other hand, individual differences may exist in terms of general abilities required for coordinated movement.

TASK-SPECIFIC APPROACH TO VARIABILITY

One approach to studying variability is to examine constraints imposed by a particular task. For instance, Kelso (1984; Kelso & Ding, this volume, chap. 11) has identified conditions conducive to stable performance on repetitive bimanual rhythmic movements and conditions yielding unstable, variable performance on this same task. Although the same approach may be applied to other tasks, a basic goal is to describe the coordinative structures specific for that task. Research on individual differences has also emphasized the need for examining task-specific constraints. For example, from a large set of correlational studies, Fleishman (1966) concluded that, although there may be a number of basic component abilities shared across tasks, extended practice increased the importance of task-specific sources of variation.

COMPONENT-ANALYSIS APPROACH TO VARIABILITY

In our research, we have taken a different approach to the study of variability. Like Fleishman, we start with the premise that there are many sources of variability in

straints, may be idiosyncratic to that particular task. Others arise because of variability in the operation of component mental processes. Of course, these hypothetical mental processes cannot be directly observed; we must define an appropriate dependent variable that is observable or derived from an overt action—and in doing so, a new problem arises. Our observable measure will likely include many sources of variability, making it difficult to tease out the hypothetical basic component sources.

Our solution to this dilemma has been to devise model tasks that can provide experimental tests of putative mental operations involved in the production of coordinated actions. In designing these tasks, we attempt to isolate one operation in each task, or at least structure that task so that the operation under consideration would be expected to contribute greatly to the total variability. Two such tasks are a repetitive tapping task and a force control task (Keele, Ivry, & Pokorny, 1987). The former was chosen to measure variability of an internal timing system. In contrast, the latter task was chosen to measure variability of processes involved in the regulation of force output.

The tapping task is based on a paradigm introduced by Wing and Kristofferson (1973). On each trial, a computer presents a series of evenly spaced tones. In Keele et al. (1987), the tones were separated by an interval of 400 ms. When ready, the subject begins tapping on a response key, attempting to synchronize his or her responses with the pacing tones. After 12 paced responses, the tones cease, and the subject continues tapping for 30 more intervals. Subjects are generally able to respond at a mean rate close to the target interval. Our primary measure of the consistency of the internal timing system is the standard deviation of the intertap intervals during the unpaced portion of the trials.

Each trial in the force control task begins with the presentation of a horizontally oriented line on a computer monitor. The vertical placement of the line indicates the target force for that trial: The higher the line, the greater the target force. The computer then plays a tone, after which the subject makes an isometric force pulse on a strain gauge. Feedback is presented graphically to indicate if the produced force is greater or less than the target force. Six responses with feedback are made in this manner. Then the subject makes six more responses to the same target without feedback. Each response is initiated only after the computer plays a stimulus tone. The consistency of force control is assessed as the standard deviation of the forces produced without feedback.

Thus, in both the timing and the force control tasks, we primarily measure the consistency of the subjects' responses when external information about performance is absent. Although forces were measured during the tapping task, subjects were not given any explicit instructions about regulating force. Similarly, we attempted to minimize timing requirements in the force control task by randomly spacing the stimulus tones.

In separate blocks, the timing and force control tasks were performed with movements of either the index finger or the forearm (Keele et al., 1987). The correlation matrix of the standard deviation scores is shown in Table 15.1. When calculated by task, the correlations were high, reaching .90 on the timing task and .76 on the force task. Correlations by effector were much smaller, ranging from .18 to .34. These results suggest that the two tasks were dependent on different component operations. We attributed the timing correlation to the operation of an internal timing system (see also Keele, Pokorny, Corcos, & Ivry, 1985) and the force correlation to the operation of an internal process regulating force output or a variable correlated with force control. We argued that previous difficulties in accounting for individual differences in coordination may have arisen in part because the selected tasks involved substantial contributions from both the timing and the force control systems.

Table 15.1 Correlation Matrix Between Timing and Force Control

	Timing		Force
	Finger	Arm	Finger
Timing			
Arm	.90		
Force			
Finger	.30	.34	
Arm	.18	.21	.76

STUDIES OF TIMING VARIABILITY

In this section, we focus on timing variability. This work is presented in two parts. First, we review our studies of patients with neurological impairments. This work provides converging evidence supporting the hypothesis that timing can be considered one component of motor control. Moreover, the evidence implicates the cerebellum as playing a critical role in the operation of an internal timing system. Deficits in this neural system are associated with increases in timing variability in both movement and perception tasks. The latter finding is perhaps the strongest piece of evidence that a common timing module is exploited across multiple task domains.

Second, we will present some new data exploring an alternative way to assess timing variability. These data complement the correlational work with healthy subjects and the patient research in pointing toward an internal timing system that spans motor and perceptual domains. The method also has the potential for a logical extension of the component approach. Our earlier work has primarily focused on separating variance associated with timing from variance associated with other components of motor control such as force control. One goal of this new approach is to begin a component analysis of the clock itself. That is, in order to understand the operation of an internal clock at a mechanistic level, we believe it will be necessary to develop a model of the component processes that form the clock.

Timing Variability in Patients With Neurological Lesions

There recently has been a burgeoning interest among cognitive psychologists in the study of patients with neurological disorders. Although there are many reasons for

this, both theoretical and technological, three are of particular interest for our purposes. First, the logic underlying the study of patient populations is similar to that developed in the individual difference studies with healthy populations. The individual difference studies assume that there are consistent differences in the operation of a given process. A range in performance will be achieved because of this variation: For example, in some individuals, an internal timing process is more consistent than in other individuals. In the patient studies, the range of differences may be amplified as a consequence of the neurological impairment. If the internal timing process is dependent on a particular neural system, then lesions of this system are expected to produce increased timing variability. Note that for both methodologies, the evidence is essentially correlational. In the studies with healthy subjects, the correlations are based on individual variation found in a sample drawn from a homogeneous population. In the patient studies, the correlations are based on group differences that arise as a result of samples being drawn from heterogeneous populations. These populations are developed by categorizing patients according to the neural system(s) affected by their lesions.

Second, the study of neurological patients can provide converging evidence for the utility of a cognitive model. Based on studies with healthy subjects, we have argued that force and timing can be considered to be two relatively independent components of coordination. Although this model does not provide any a priori constraints concerning physiological mechanisms, a reasonable conjecture would be that different neural systems are involved in the operation of these two components. If this were so, then lesions of one neural system should produce a deficit in timing control, whereas lesions of a different neural system should produce a deficit in force control. Double dissociations of this sort are generally interpreted as strong evidence for the existence of two processes in neuropsychological research.

Third, linkage of a particular mental operation with a given neural system is of interest for localization theories of the brain. Many students of behavior have kept an eye on the relation of mind and matter and view neuropsychological research as a valuable tool for providing insight on this issue. Thus, the study of patients can prove useful for further developing our cognitive model as well as for identifying the crucial neural systems required for timing and force control.

Repetitive Tapping Task

Patients with cortical and subcortical motor disorders were tested on the repetitive tapping task (Ivry & Keele, 1989). Patients were assigned to one of three groups, depending on whether their lesions were centered in the cerebellum, basal ganglia, or frontal cerebral cortex. Classification criteria were based on a clinical examination and neuroradiographic data.

The cerebellar group ($n = 27$) included patients with either focal or diffuse lesions. The focal lesions ($n = 11$) were the result of tumor or stroke, and the resultant motor deficits were restricted to the hand ipsilateral to the lesion. The diffuse lesions ($n = 16$) were the result of atrophic processes. In some of the patients with a degenerative disorder, there was evidence of extracerebellar involvement, especially of the pons and olive.

For studying the effects of basal ganglia lesions, we recruited 28 patients with Parkinson's disease. All of these patients were taking L-dopa medication at the time of testing. This treatment ameliorates some Parkinson symptoms, but there were still obvious motor problems in these patients.

The third patient group ($n = 7$) had focal lesions from strokes, centered in the posterior region of the frontal cortex. All of these subjects presented some degree of hemiparesis in the hand contralateral to the lesion, indicating that the lesions included upper limb areas of motor cortex. However, we selected only subjects for whom the deficit was not so severe as to prevent performance of the tapping task.

A fourth group of elderly control subjects ($n = 21$) consisted of healthy people with no history of neurological disease or disturbance. The mean age of this group, 67 years old, was slightly older than the mean ages of the patient groups (range from 51 to 63 years old).

As described previously, each trial of the continuation tapping task required the production of 12 paced and 30 unpaced intervals. The target intertap interval was 550 ms in the patient study. This pace was chosen so that the subjects would not be performing near their maximal rate of tapping. The subjects completed at least 12 tapping trials, grouped into blocks of 6 trials each. As in our studies with healthy subjects (Keele et al., 1985, 1987), the primary measure of interest was the variability of the unpaced intertap intervals.

Figure 15.1 shows the mean standard deviations for the four groups. Two results stand out. First, there was no difference between the Parkinson patients and the control subjects. This result was striking, considering that these patients showed the usual array of Parkinson symptoms, including bradykinesia and rigidity. Despite these deficits, the Parkinson patients were as consistent as the age-matched control subjects. Second, the mean standard deviation for both the cerebellar and the cortical groups was significantly higher than for either the control subjects or the Parkinson patients. There was no difference between the mean standard deviations for the cerebellar and the cortical patients.

The data presented in Figure 15.1 look at overall variability on the tapping task. There are many reasons that a person could have trouble with this task. One reason may be because of inconsistency in an internal timing process. Alternatively, the clock may operate properly, indicating the appropriate time at which a response should be made, but the motor system may have difficulty in executing that response. That is, the clock may correctly determine when a series of responses should occur, but the motor apparatus may introduce variability in implementing those responses. We next turn to a finer grained analysis of the patients' variability on the continuation tapping task.

Analysis of Tapping Data With the Wing-Kristofferson Model

Wing and Kristofferson (1973) proposed a formal model for decomposing the total variability on the repetitive tapping task into two independent sources. The model is described in detail in their paper (see also Wing, 1980; Ivry, Keele, & Diener, 1988). Briefly, the key assumptions of the model are as follows. Each interval is assumed to be the sum of three events. Two of the events are attributed to the implementation system—namely, the time required to implement the key press that

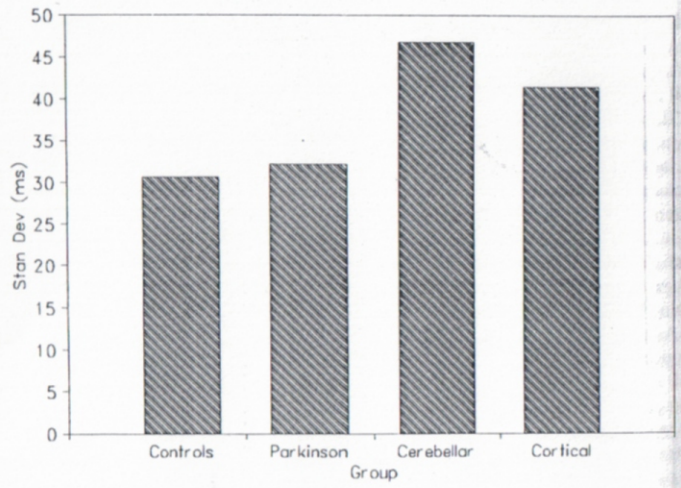

Figure 15.1 Mean standard deviations of the intertap intervals for the four groups on the tapping task.
Note. From "Timing Functions of the Cerebellum" by R. Ivry and S. Keele, 1989, *Journal of Cognitive Neuroscience*, **1**, pp. 136-152. Adapted by permission.

initiates the interval and the time required to implement the key press that terminates the interval. The third event is the interval metered out by a central clock. Wing and Kristofferson treat the clock and implementation processes as two random variables with normal variances. The mean of the clock is set (by the pacing signal) to the target interval, and the mean of the implementation durations, referred to as motor delays, is an unknown constant. Because the two processes are assumed to be independent, the total variability is simply the sum of the variances of the component parts. That is,

$$\sigma_I^2 = \sigma_C^2 + 2\sigma_{MD}^2 \tag{15.1}$$

where c and md stand for clock and motor delay (implementation), respectively.

A critical assumption of the Wing-Kristofferson model is that all of the component events occur independently. Each output from the clock process is assumed to be independent of preceding clock outputs, each motor delay is assumed to be independent of other motor delays, and, as stated previously, all of the clock outputs and motor delays are assumed to be independent of one another. In other words, the model assumes that the task is performed in an open-loop mode. From this assumption, Wing and Kristofferson have shown that an estimate of the variance associated with the implementation process is a function of the covariance between successive intervals, or

$$\sigma_{MD}^2 = -\text{autocov}(1)$$

A graphic depiction of this formalization is given in Ivry
The variance associated with the clock can now be estim
overall variance of the intertap intervals is obtained dire
estimate of motor delay variance is obtained from the co
tracting this value from the overall variance will yield an es
The Wing-Kristofferson model has received empirical
studies with healthy subjects (reviewed in Wing, 1980). A
model can be made using patients with peripheral neuropath
Keele & Ivry, 1987). Given that timing variability correlate
et al., 1985, 1987), we assume that the clock is a cent
accessible to all effectors. Thus, the model should attribute a
in peripheral patients to the implementation system. We v
of the peripheral nervous system to affect the timing proc
Four patients with peripheral neuropathies were tested
Their etiologies varied: Two had ulnar nerve damage, on
and one had suffered an entrapped nerve at the shoulder. T
not so much for a specific lesion, but rather because thei
were the result of a peripheral neuropathy.
One important feature in testing these patients is that thei
The patients were only impaired when using the hand ipsila
a within-subject design can be employed in which perfor
effector is compared to performance with an unimpaired
involved comparing tapping performance with the index f
right hand. In one case the comparison was between two f
Overall, the mean standard deviation when tapping with
was 28 ms. When tapping with the impaired effector, the m
a 20% increase. The clock and motor delay estimates ar
There is a negligible increase in the clock estimate for ta
hand. In contrast, the motor delay estimate is over 40%
hand. Although the percentage increase varied from 15%
the impaired hand yielded a higher motor delay estimat
block-by-block comparisons. Thus, as predicted, the mo
variability in patients with peripheral neuropathies to the
Fortified by this neuropsychological validation of the W
we then performed a series of within-subject comparisons u
of the central nervous system (Ivry & Keele, 1989; Ivry et al
patients were recruited to perform the tapping task under tw
In the *on* condition, the patients were tested while following
regimen. In the *off* condition, the patients skipped their mor
prior to testing. Clinically, this *on-off* manipulation produ
of the patients became much more rigid and bradykinetic
these changes, there was no effect in their performance
overall variability as well as the estimates of the clock and
nents were essentially identical under both conditions. As

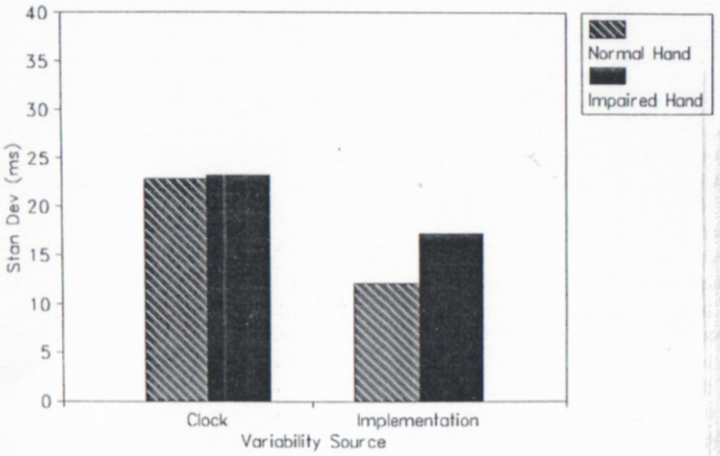

Figure 15.2 Clock and motor delay estimates from the Wing-Kristofferson model for the patients with peripheral neuropathies.
Note. From "Timing Functions of the Cerebellum" by R. Ivry and S. Keele, 1989, *Journal of Cognitive Neuroscience*, **1**, pp. 136-152. Adapted by permission.

the within-subject experiment confirmed that lesions of the basal ganglia do not affect tapping consistency or the operation of a central timing process.

Within-subject comparisons were made for seven patients with cortical lesions and eight patients with focal cerebellar lesions (Ivry & Keele, 1989). In all of these cases, the lesions were unilateral, allowing a comparison to be made between an impaired effector and unimpaired effector on the tapping task. The mean clock and motor delay estimates for these two groups are shown in Figure 15.3, a and b. Averaging across the patients within the two groups, the Wing-Kristofferson model attributed the increased variability to both the clock and the implementation components.

However, by averaging within each group, we may have obscured individual deficits that can be identified by the within-subject comparisons. We were unable to identify any such differences by further analysis of the tapping data for patients with cortical lesions. Moreover, the data for some of the cortical patients showed consistent violations of the Wing-Kristofferson model.

A clearer picture emerged in an extended analysis of a group of cerebellar patients with focal lesions (Ivry et al., 1988). Seven patients were tested. Each subject produced a minimum of eight six-trial blocks, four blocks with each hand. The patients were separated into two subgroups, based on neuroradiographic and clinical criterion. For four of the subjects, the lesion foci were lateral, encompassing portions of the cerebellar hemisphere on the impaired side. These patients all presented symptoms associated with hemispheric lesions, notably dysmetria in voluntary movements. The lesions were more medial for the other three patients, and their primary

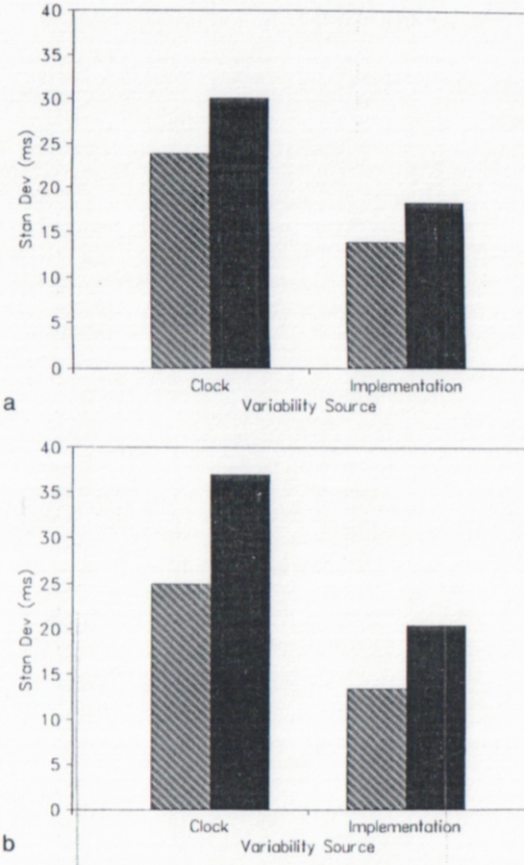

Figure 15.3 Clock and motor delay estimates from the Wing-Kris
tients with (a) unilateral cortical or (b) cerebellar lesions.
Note. From "Timing Functions of the Cerebellum" by R. Ivry and S. Keel
tive Neuroscience, **1**, pp. 136-152. Adapted by permission.

symptoms, disturbances of balance and gait, were more typi
lesions.

The results from the Wing-Kristofferson analysis are sho
and b. The increased tapping variability for the patients wi
attributed to the clock process. In contrast, the increased ta
attributed to the implementation process for the patients with
result was extremely consistent over tapping blocks: The sev

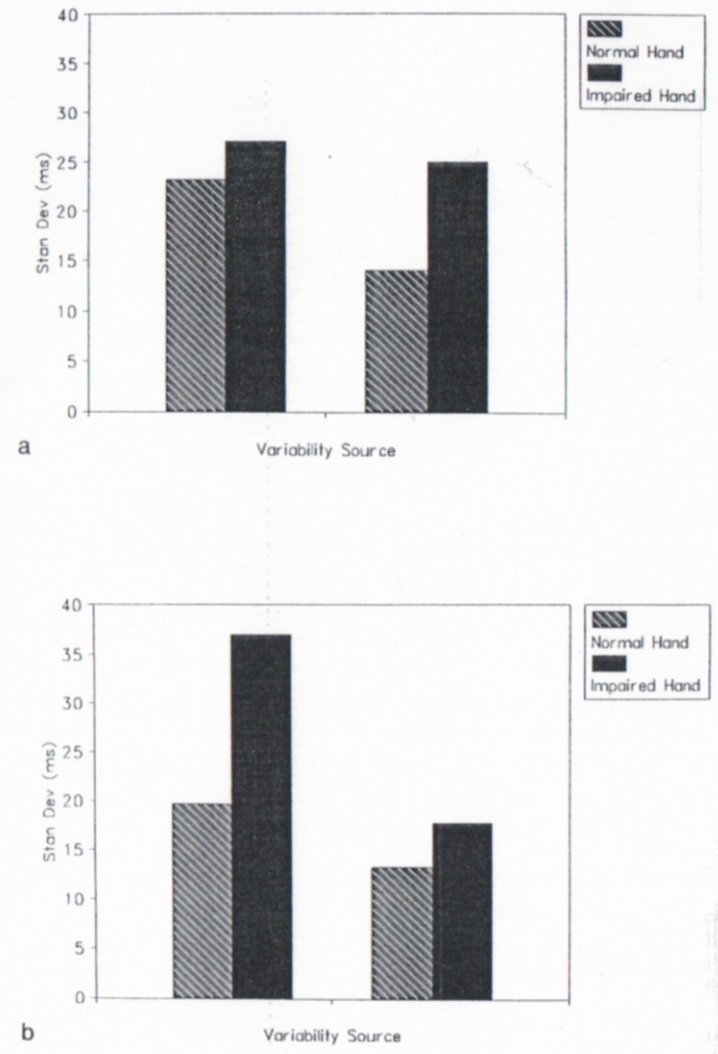

Figure 15.4 Clock and motor delay estimates from the Wing-Kristofferson model for patients with lesions centered in either (a) the medial cerebellum or (b) the lateral cerebellum.

Note. From "Dissociation of the Lateral and Medial Cerebellum in Movement Timing and Movement Execution" by R. Ivry, S. Keele, and H. Diener, 1988, *Experimental Brain Research*, **73**, pp. 167-180. Adapted by permission.

a total of 100 six-trial blocks, or 50 impaired-unimpaired co described above was reversed in only 1 of the 50 comparis

These data indicate that an internal timing system is disru of the lateral regions of the cerebellum. Medial cerebellar tapping variability, but this increase appears to be the res processes associated with implementing a response. This do accord with functional models of the cerebellum derived fro of neural connectivity of the lateral and medial regions (e.g 1974). Much of the output from the lateral regions projects cortical areas via the ventrolateral thalamus (Asanuma, Th Goldberg, 1985; Schell & Strick, 1984). Medial cerebellar r vate descending pathways in the brainstem and spinal cord Jones, 1983b; Wilson, Uchino, Maunz, Susswein, & Fukus distinction can be made between neural pathways that ascend The former could presumably contribute to motor planni whereas the latter would be expected to contribute to mov (Allen & Tsukahara, 1974). As argued previously, setting the of the movement may be one component of motor program where timing control is part of the explicit movement goal, the lateral cerebellum plays a critical role in this process.

Time Perception in Patients With Neurological Les

One problem with the tapping data is that the results were qu with cerebellar and cortical lesions. Both groups were more v subjects and the Wing-Kristofferson estimates were proble patients. It is possible that both the cortex and the cerebellum timing system. That is, timing may be a distributed process spanning a number of neural structures. On the other hand, o may be most critical for timing. Lesions of the other area n deficit because of limitations in the Wing-Kristofferson mod partitions total variability into two components, labeled the cl tation systems. The implementation estimate is theoretica component is subtracted out, the remaining variance is attribu nent. However, variability in other central (i.e., nonimpleme also be contained in the remainder (Ivry & Keele, 1989) Kristofferson referred to the two components as clock and i accurate dichotomy would be central and peripheral compo be just one part of the central component.

Given these limitations, it is important to consider other tas timing. One task we have employed involves the perception On each task, the subject hears four 50-ms tones, grouped tones each. The first two tones are separated by a fixed in experiments, this interval was set at 400 ms. Then, after a pair of tones is presented. The interval between this pair is v task is to judge whether the second, comparison interval is the first, standard interval. Based on the correctness of the

duration of the comparison interval is adjusted. After a number of trials, an estimate of perceptual acuity is obtained. This estimate is given as a standard deviation, corresponding to a threshold at which the subject's performance is approximately 75% correct (Pentland, 1980).

If there exists a task-independent internal timing system, then we would expect to find a positive correlation between performance on the motor tapping task and performance on the perception-of-duration task. Keele et al. (1985) obtained a significant correlation of .53 (.60 following a reliability correction) between these two tasks in a study with 32 healthy college students. This result, coupled with the cross-effector tapping correlations, formed the cornerstone for the hypothesis that one component of coordination was an internal timing process.

The perception task can also be used in the patient research. If a particular neural system is part of an internal timing system, then patients with lesions of this system should be more variable in making duration judgments. Indeed, the perception-of-duration task has a major advantage over the tapping task, in that there are no motor requirements. For the tapping task, we selected patients with disorders of movement, but we were constrained in that we could not test patients with the most severe problems, because they were unable to complete the task. The perception task is not similarly constrained; the only requirement is that the patients be able to understand the directions.

Eight patients with cortical lesions, 28 Parkinson patients, 27 patients with cerebellar lesions, and 21 elderly control subjects were recruited (Ivry & Keele, 1989). Most of the subjects had also been tested in the tapping study. The subjects were tested on two perception tasks: the perception-of-duration task and a control task in which they compared the loudness of auditory stimuli. As in the duration perception task, each trial for the control task consisted of two pairs of two tones each. The interval between both pairs was always 400 ms. The volume of the second pair was either more or less intense than the volume of the first pair. The same psychophysical procedure was used to obtain loudness thresholds. This task was included to ensure that any deficit obtained on the perception-of-duration task could not be attributed to a generalized problem with auditory tasks or psychophysical testing procedures.

The results for the two tasks are shown in Figure 15.5, a and b (Ivry & Keele, 1989). Statistical analyses revealed a second double dissociation implicating the cerebellum in timing control. The cerebellar patients were significantly more variable than the control subjects on the perception-of-duration task. The cortical patients performed approximately as well as the control subjects on this task. However, these subjects were significantly more variable on the perception-of-loudness task. Although it was not predicted, we believe this latter result arose because some of the cortical lesions extended into the auditory cortex. Nonetheless, this finding strengthens the perception-of-duration results in two ways. First, it demonstrates that both perception tasks were sensitive enough to identify potential deficits. Second, it emphasizes that the cerebellar deficit on the perception-of-duration task is specific and not the result of a generalized impairment.

Role of the Cerebellum in Other Tasks Requiring Timing

The finding of a deficit on a purely perceptual task following lesions of the cerebellum was exciting. This neural system has traditionally been associated with motor

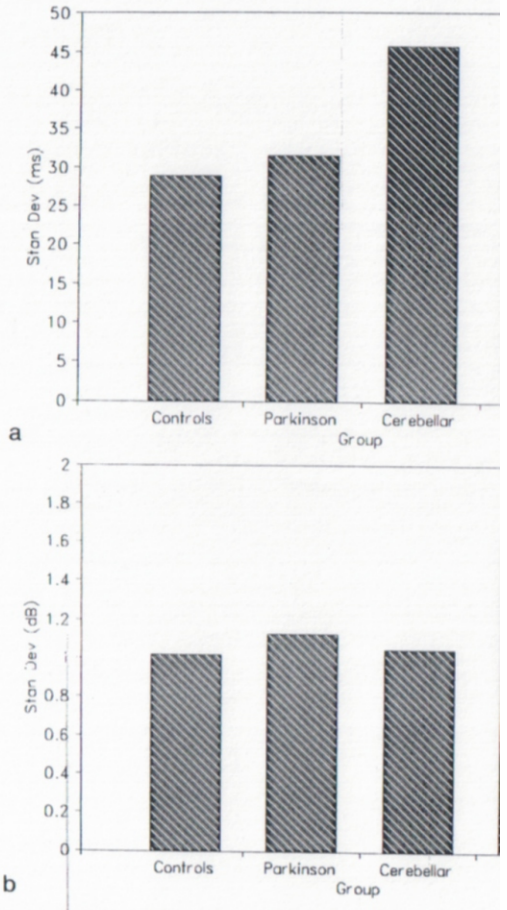

Figure 15.5 Mean standard deviation on (a) the perception of dur
ception of loudness tasks.
Note. From "Timing Functions of the Cerebellum" by R. Ivry and S. Keel
tive Neuroscience, **1**, pp. 136-152. Adapted by permission.

functions. Our results do not dispute this belief: They simply p
hypothesis regarding one computational role of the cerebellu
especially skilled movements, require precise coordination of b
ral events. We have hypothesized that the cerebellum can be
timing system. At least one function of this neural structu

component operations needed to produce the temporal aspects of coordinated movements. Moreover, we believe this computational capability extends beyond the motor domain. The results on the perception-of-duration task indicate that a common internal timing system is invoked across a variety of tasks if those tasks have similar computational requirements.

We have replicated the perception-of-duration results with both auditory and visual stimuli (Ivry & Gopal, in press). We have also found that patients with cerebellar lesions are impaired on a perceptual task in which judgments are made about the velocity of a moving stimulus (Ivry & Diener, 1991). This latter task was selected for two reasons. First, velocity, by definition, involves a computation that occurs over time. Second, lesions of the cerebellum have been associated with eye movement disorders (Aschoff & Cohen, 1971; Ritchie, 1976), and we were interested in whether these disorders might, in part, reflect impaired perception of a to-be-tracked stimulus.

Moreover, we have argued that the cerebellar timing hypothesis can account for a number of disparate functions associated with the cerebellum (Keele & Ivry, in press). Some of these are summarized in Table 15.2. Together with our empirical results, we believe that a compelling argument can be made that timing control can be viewed as a component operation of coordination.

Slope Analysis of Timing Variability

The Wing-Kristofferson model provides one way to partition variance on the repetitive tapping task. Wing and Kristofferson have typically referred to the two subcomponents as clock and implementation (or motor delay). However, as discussed earlier, only the implementation component is theoretically derived; once this is

Table 15.2 Generalization of the Timing Hypothesis to Other Functions Associated With the Cerebellum

Deficit	Timing interpretation and selected reference
Hypermetria in rapid movements	Loss of ability to temporally coordinate agonist/antagonist activity, especially antagonist onset (Hallett, Shahani, & Young, 1985)
Locomotion ataxia	Loss of ability to coordinate phase-phase relations between different limbs (Arshavsky, Gelfand, & Orlovsky, 1983)
Abolition of conditioned learning	Loss of ability to represent temporal relationship of conditioned stimulus to unconditioned stimulus necessary for making conditioned response adaptive (Thompson, 1986)
Efference copy	Loss of ability to anticipate afferent information (Gellman, Gibson, & Houk, 1985)
Cerebellar dysarthria	Deficit in temporally coordinating interarticulatory actions (Ivry & Gopal, in press)

estimated, the remaining variance is, by default, attributed to
turn to an alternative approach to partitioning variance on timi

Logic of Approach and Background/Previous Work

We call this approach a slope analysis. The basic idea is quite
employed by others (e.g., Getty, 1975; Killeen & Weiss, 1987
variability of a timing system is assumed to increase with the du
being timed. Thus, if timing variability is measured as a function
the slope of this function provides an estimate of clock variabil
rests on one critical assumption, namely, that the only duratio
involved in a timing task is the clock. As in the Wing-Krist
observed variability is assumed to be the sum of the contribut
independent processes. One of these processes is a timing syst
may be involved in implementing responses, and there may
processes. However, for the slope analysis to be valid, we mu
of the processes other than the timing system are independent
If the subject is tapping repetitive intervals of 350 ms or 55
due to the implementation component is invariant.

This assumption is also an essential part of the Wing-Krist
model assumes independence of clock and implementation
estimate the implementation variability. Wing (1980) reports o
in which clock and motor delay estimates were derived for tap
target intervals over the range 220 to 490 ms. As predicted, the
was essentially constant over the different intervals. In contr
clock variance was highly dependent on the produced interval,
of approximately 95 ms^2, when the intertap interval was 22
approximately 465 ms^2, when the intertap interval was 490 ms
of the strongest sources of support for the Wing-Kristofferson

While the basic assumptions of the Wing-Kristofferson mo
the clock estimate will vary with interval duration, the exact for
is dependent on additional assumptions about the mechani
Killeen & Weiss, 1987). Two-process clock-counter models
linear relationship between variance and interval duration (Ab
1962). Alternative models in which the time-dependent variab
a single process such as variable activation times (e.g., Gros
1989; also Rosenbaum, 1990) predict that the linear relations
the standard deviation and interval duration (general form of We
1975). Wing (1980) plotted the estimate of clock variance as a
duration and observed a significant linear fit. Over 96% of the va
for by the linear component. However, a linear relationship a
strong when the data are replotted with standard deviation dep
(Figure 15.6, a and b). Indeed, the proportion of clock varian
a linear component actually increases slightly.

Obviously, from a regression analysis it is unclear whether th
of timing variability as a function of interval duration is in t
standard deviation. However, the second parameter of the reg

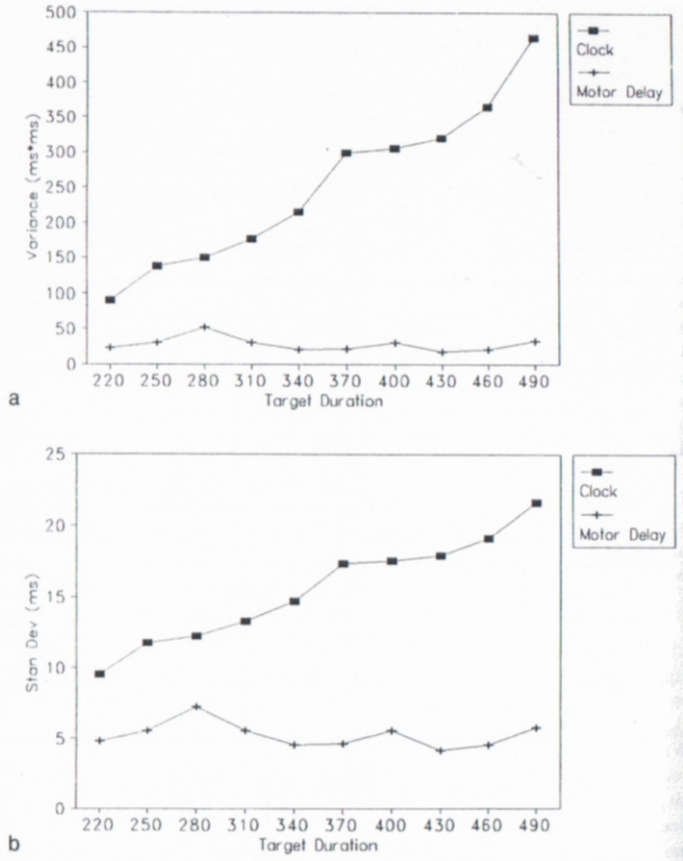

Figure 15.6 Clock and motor delay estimates as a function of target interval. Data are estimated from Wing (1980). Variance is plotted on the ordinate in (a), and standard deviation is plotted on the ordinate in (b).
Note. From "The Long and Short of Timing in Response Sequences" by A.M. Wing. In *Tutorials in Motor Behavior* by G. Stelmach and J. Requin (Eds.), 1980, New York: Elsevier Science Publishers. Adapted by permission.

intercept, may be informative. In the Wing (1980) data, the intercept for the clock variance function is -155 ms^2. In contrast, the intercept for the clock standard-deviation function is essentially 0 ms. On intuitive grounds, we would expect the intercept to be zero: as the interval to be timed approaches zero, the variance should also become negligible. The large negative value yielded by the regression equation

for the variance function is problematic, especially considerin
definition must be positive. Thus, our working hypothesis i
deviation function is more accurate.

The variance–standard deviation debate is secondary to our curr
less of the outcome, the basic point is that, assuming linearity
provides an alternative way to estimate clock variability. This ap
of strengths in comparison to the Wing-Kristofferson model. F
(1980) has plotted the clock estimate, there is no need to perfor
into clock and motor delay. The slope analysis can be perfor
observed data. This bypasses error that will be introduced by the e
This is especially important given that the clock and motor delay
estimated independently; in the Wing-Kristofferson model, the cl
by subtraction. Any error in estimating the motor delay componer
error in the clock estimate. The slope analysis eliminates the de

Second, the slope analysis provides an alternative method fo
timing with perceptual timing. The strength of the Wing-Kris
motor timing was that it provided an analytic tool for separating c
sources of variability. The perception task used in our patient stu
for a similar decomposition: All of the variability is treated as a lu
it is reasonable to assume that there is also peripheral, or n
contributing to performance on this task. For example, there m
the perceived onset of the tones. The slope analysis can provide
for decomposing performance on either motor or perceptual tim
provides an estimate of the clock component. If a common interv
in both tasks, then the slope values should be comparable. The
contain a preliminary report of two experiments testing this pre

Slope Experiment 1

In the first experiment, subjects performed the repetitive tapping
tion task at four different target durations, 325 ms, 400 ms, 47
To improve the stability of the data, each subject completed four
of eight blocks each. Four blocks involved tapping, one bloc
Each block consisted of a practice trial and six test trials. Th
tested time perception in which the four durations served as the s
blocks. The order of blocks was counterbalanced with the con
and perception for a given duration were paired, with the tap
preceding the perception block. To date, five subjects have cor

Figure 15.7, a and b, presents the variability data plotted as a f
Variance is plotted on the ordinate in Figure 15.7a, and standard
on the ordinate in Figure 15.7b. Separate functions are shown for
ity on the tapping task as well as estimated clock and motor
Note that the current data replicate Wing (1980) and provide str
basic slope prediction derived from the Wing-Kristofferson mod
estimate varies minimally with duration, whereas the clock estim
tonically.

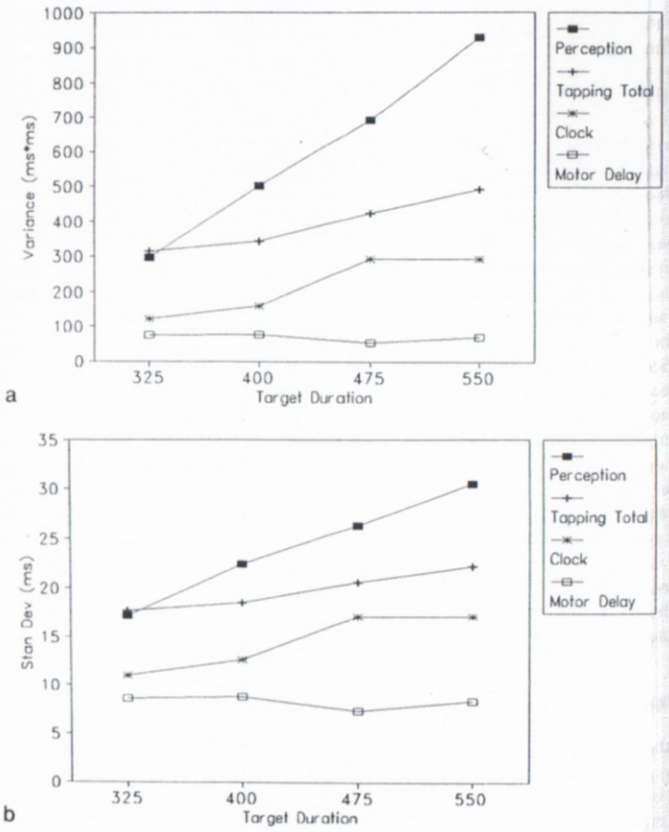

Figure 15.7 Results of Slope Experiment 1. Data from the repetitive tapping task are plotted in terms of overall variability (a), and standard deviation (b), with estimates of clock and motor delay components from the Wing-Kristofferson model.

Table 15.3 summarizes the regression analyses. A linear component accounts for over 85% of the variance for all of the functions except the motor delay estimates. As in our reanalysis of the Wing (1980) data, the intercepts indicate that the standard deviation functions are more meaningful. Large negative intercepts are obtained for both the clock and the perception variance functions. In contrast, the intercept values for the standard deviation functions are in agreement with a couple of different predictions. First, the clock intercept is close to zero. Second, the intercept for the

Table 15.3 Regression Analysis for Slope Experiment 1

Measure	Slope (ms)	Interc (ms
Standard deviation		
Tapping total	0.0213	10
Clock	0.0311	1
Motor delay	−0.0032	9
Perception	0.0584	−1
Variance		
Tapping total	0.8483	28
Clock	0.8824	−163
Motor delay	−0.0527	90
Perception	2.7893	−615

overall variability data on the tapping task is slightly larger, 1 motor delay estimate of 8.2 ms. This is predicted because include not only the motor delay component, but also oth variability that are not duration dependent. One troubling a scores, however, is that the perception intercept is negative, − was predicted to estimate nonclock variability on the percep that the negative intercept reflects measurement error. It is a sources of variability on the perception task are negligible.

Given the intercept results, we focus on the slopes for t functions.[1] Contrary to our prediction, the mean slope estin the tapping and the perception functions. Even though on completed this task, the difference is marginally significant, two-tailed test. The perception slope is almost three times a slope. The difference is reduced if the clock slope for the tapp for the overall scores. However, the perception slope is still 8 data, the slope analysis does not provide converging evidence system is invoked in the repetitive-tapping and the perceptio

Slope Experiment 2

There are a number of possible explanations for the differ although each subject completed four sessions, the data m enough for this type of analysis; the slope values are heavily v

[1]The intercept of the function for overall variance on the tapping is positiv functions may be viable. However, not only is the variance function for th but, by our approximations, the variance function in Wing (1980) yields a ms². The intercept for the standard deviation function from Wing's data is

and slowest target durations, and error in estimating these data points would distort the functions. Second, the central assumption underlying the slope analysis may not be correct. Sources of variability other than the clock may contribute to the slope values. Third, the timing demands in the tapping and perception tasks may not be comparable. In particular, each trial in the tapping task requires the production of a series of 42 consecutive intervals, 12 with a pacing signal and 30 unpaced. In contrast, each trial on the perception task requires a comparison between 2 isolated intervals, the standard interval and the comparison interval. It is possible that the repetitive aspect of the tapping task serves to stabilize the operation of an internal timing system. This would produce a decrease in variability on the tapping task.

We thus modified the procedures in a second experiment to make the two tasks more comparable. The modified tapping task began with a paced phase in which the computer generated a single interval marked by two 50-ms tones. The word *tap* then appeared on the screen, and the subject made two key presses, attempting to reproduce the target interval. This procedure was repeated until the subject had produced 12 isolated intervals following the presentation of the target interval. Following this, the tones were eliminated, and the subject produced 30 more intervals, each individually initiated after the word *tap* was displayed on the computer. The response-stimulus interval was randomly varied to prevent subjects from adopting a rhythmic mode of responding. After producing 30 unpaced intervals, the subject was provided with feedback. A block consisted of one practice and six test trials. Each subject completed four blocks, one at each of the four target durations.

After completing a block of tapping, the subjects were tested on a modified version of the perception task. On each trial, only a single test interval was presented. The subject judged whether the interval was shorter or longer than an implicit standard. To help the subjects establish an implicit standard, the first 10 trials of a block involved relatively easy comparisons. For example, if the target interval was 400 ms, the durations used in the first 10 trials were either less than 325 ms or greater than 475 ms. Subjects rarely made errors with these values when performing the perception task with a standard interval. In addition, the preceding tapping trials were expected to establish an appropriate standard interval, because the subjects had just completed a set of tapping trials at that duration.

To summarize, in the second slope experiment, the intervals were generated individually rather than repetitively. Correspondingly, the perceptual judgments were made on isolated intervals. The same five subjects were tested. One subject completed the 2nd slope experiment prior to the 1st slope experiment.

The variability functions and regression analyses are summarized in Figure 15.8, a and b, and Table 15.4, respectively. Over 88% of the variance is accounted for by a linear function for both functions when plotted by variance (Figure 15.8a) and when plotted by standard deviation (Figure 15.8b). Of primary interest, the slope values are essentially identical for the tapping and perception functions, $t(4) = 0.18$. These results support the hypothesis that a common clock is used for both tasks. This finding is in accord with our correlational studies with normal subjects (Keele et al., 1985, 1987) and patient research (Ivry & Keele, 1989). The earlier studies, however, had assessed performance only at a single interval. In the current approach, the properties of the clock lead to the expectation that the variability of the timing process should increase in a systematic manner as the target interval is lengthened.

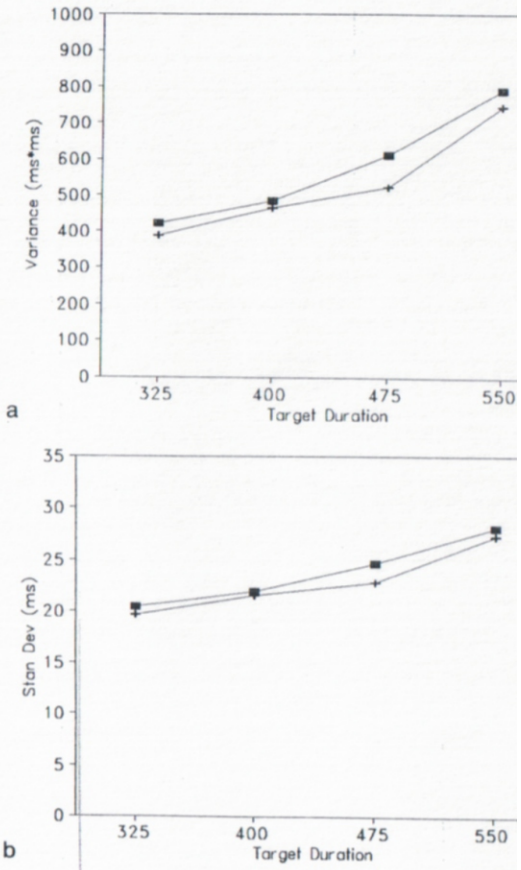

Figure 15.8 Results of Slope Experiment 2 in terms of variance (tion (b) functions. Only overall tapping variability is depicted beca production of isolated intervals.

Moreover, we expected the increase to be the same for both mo if a common timing system was involved. These predictions

Clock and motor delay functions are not shown in Figu be estimated, because the Wing-Kristofferson model requi consecutive intervals. However, as stated previously, an e sources that are not duration dependent (i.e., nonclock variabili from the intercept values. Once again, the data favor models

Table 15.4 Regression Analysis for Slope Experiment 2

Measure	Slope (ms)	Intercept (ms)	r^2
Standard deviation			
Tapping	0.0387	5.7	0.91
Perception	0.0340	9.0	0.97
Variance			
Tapping	1.8088	−271.9	0.89
Perception	1.6480	−145.0	0.96

when standard deviation is plotted on the ordinate. Both the tapping and the perception values are positive and of seemingly reasonable magnitude. In contrast, the variance functions yield large negative intercepts.

Summary of the Different Slicings of the Timing Pie

Figure 15.9, a and b, summarizes two ways of slicing up the variability pie on timing tasks. The Wing-Kristofferson model (Figure 15.9a) slices off the implementation component and attributes the remaining variability to central processes, most notably a clock. The slope analysis (Figure 15.9b) slices off the clock component and attributes the remaining variability to all other processes involved in the tasks. Taken together, the two methods provide converging operations to analyze timing variability.

Moreover, the methods may facilitate a more finely grained analysis of the different components involved in timing tasks. For example, on the repetitive tapping task, an estimate of central processes that are not part of the clock component can be obtained by examining performance on both tasks. This component can be inferred from the disjunctive set (see Figure 15.9c), the region of the pie that is not directly estimated by either procedure. If timing is attributed to the clock component, and the implementation of the response is attributed to the motor delay component, what computational process might generate this remaining source of variability? One possibility is that a timing process can be conceived of as having more than one component, only one of which is concerned with the actual timing. For example, clock-counter models postulate a second component that keeps track of the number of outputs of the clock. An alternative two-component model assumes variability in a process that accesses the timer. Consider an analogy in which a foot race is to be timed with a stopwatch. An erroneous time could be attributed to either a problem with the stopwatch or a problem with starting or stopping the stopwatch. Indeed, in track and field, the latter type of error is sufficiently great that hand-held timings are generally not accepted as official.

The preceding statements begin a natural extension of our component analysis of coordination. The 1st step led to a decomposition of variability into components

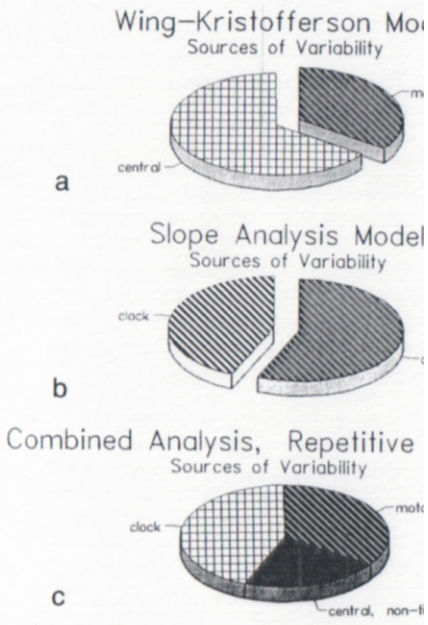

Figure 15.9 Three different slicings of the variability pie on tim

such as force and timing. A 2nd step, based on the Win decomposes timing variability into central and peripheral analysis provides a 3rd step for further decomposing the ce example, manipulations that only affect the intercept can timing components that are duration independent. Accessin to be such a process. On the other hand, manipulations that ably reflect properties of the duration-dependent componen To date, we have tested patients only at a single duration. If used, we predict that steeper slopes would be obtained for the lateral cerebellum than for control subjects or other pat deficits. In this manner, we anticipate that the slope analys for identifying the components of an internal timing system the different sources of variability manifest in the operation

STUDIES OF FORCE VARIAB

As reviewed earlier, a strong case can be made that an i common to both perception and production, involves the cereb

a brief summary of preliminary research exploring the neural systems involved in force control. The evidence suggests that the basal ganglia play a crucial role in this component operation. The nature of this computation, however, is unclear. We do not postulate that the basal ganglia control the recruitment of motor units. Rather, we expect that the contribution of the basal ganglia in force regulation is less direct. One possibility is that the basal ganglia computation is more related to shifts in motor set that may precede or trigger motor unit recruitment (Mink & Thach, 1991; Wing, 1988).

Force Control in Parkinson's Disease

A large body of research with both animals and humans has investigated the effects of basal ganglia dysfunction on movement kinematics and kinetics. The most consistent finding is that lesions of the substantia nigra or globus pallidus, two of the basal ganglia nuclei, reduce the speed at which movements occur. For example, Horak and Anderson (1984) found that kainic acid injections into the globus pallidus in monkeys led to a slowing of movement time and that this deficit became more pronounced as the pathological consequences of the injections advanced. This finding matches the clinical observations of Parkinson's disease in humans. This disease, in which extensive cell death in the substantia nigra is observed, is characterized by a slowness in movement and rigidity. This slowness, or bradykinesia, has been documented in numerous studies (e.g., Benecke, Rothwell, Dick, Day, & Marsden, 1987; Hallett & Khoshbin, 1980; Stelmach, Teasdale, Phillips, & Worringham, 1989).

Superficially, bradykinesia might be interpreted as a timing deficit, because the movements are abnormally slow. Such an interpretation would appear to be at odds with our findings on the tapping and time perception tasks in which Parkinson patients performed as well as age-matched healthy subjects (Ivry & Keele, 1989). It may be necessary to make a distinction between tasks that require explicit timing control and those in which temporal properties arise as the result of an interaction of many processes, only one of which may be the operation of an internal clock. However, examining the variability of the movements produced by Parkinson patients is informative. Teasdale, Phillips, and Stelmach (1990) measured movement time and movement time variability in a group of Parkinson patients. Although the patients moved more slowly than the control subjects, the patients' movement times were not more variable once the differences in absolute movement time were taken into account. This result meshes with our null findings on the tapping task. Indeed, we chose a relatively slow tapping rate (ITI = 550 ms) to ensure that the Parkinson patients were able to keep up with the pace. Given this allowance, the temporal characteristics of the patients' movements were as consistent as for healthy subjects. Teasdale et al. (1990) obtain the same result in a unidirectional movement task.

Force variability in Parkinson patients has been examined in two recent studies. Stelmach and Worringham (1988) first measured the maximum isometric force capability for Parkinson and control subjects. Then each subject was asked to produce force pulses to match targets that were either 25%, 50%, or 75% of that person's

maximum capability. Three different accuracy conditions w
jects' responses were scored as to whether they fell withi
region. Although not significant, the maximum force prod
patients was about 25% lower than the maximum force pr
Most interesting, the Parkinson patients were as accurate a

Rather than simply determine if the produced force fel
target area, Stelmach et al. (1989) used a quantitative meas
in which they recorded the actual forces produced for each
as a coefficient of variation (standard deviation of peak force
ences were observed between the Parkinson patients and th
coefficient of variation for both groups averaged about 10
forces tested.

We have conducted a similar experiment using our for
Parkinson patients and 11 age-matched control subjects
tapping study, the Parkinson patients were tested under
conditions, the latter being when the patients skipped the
cycles. Thus, this design provided a between-subject comparis
sons) and a within-subject comparison for the patients (*on*

As described in the section on the task-specific approach
were produced by isometric contractions of the index finger.
the same for all subjects, corresponding to 1, 3, 5, and 7 N. T
well below maximum force capability for all of the subject
of six force pulses with feedback and six force pulses with
completed six trials at each of the four targets.

The mean forces produced by the control subjects and the
the *on* and *off* medication conditions approximated the target
patients under both conditions tended to produce slightly less
subjects, and this effect was more pronounced when the patien

The variability data are presented in Figure 15.10, a and b.
for the three groups at each target level is plotted in Figu
are replotted in Figure 15.10b as coefficient-of-variation me
variability as a percentage of the target. In accord with previo
Newell, this volume, chap. 2), the coefficient-of-variation fu
sloping and concave. Most important for our present concern
coefficient-of-variation functions are essentially identical fo
in the Stelmach studies, these results would suggest that the
no more variable than controls at regulating force.

However, Figure 15.10 does not reflect one important asp
Parkinson patients were much slower in generating the force
control subjects. Moreover, this effect was greatly magnifi
manipulation. The mean contraction time for the control subj
mean contraction times for the Parkinson subjects were 483
on and *off* conditions, respectively. A number of researche
theoretically derive the relationship of movement variability
and temporal properties of the movement (e.g., Carlton &
chap. 2; Meyer, Smith, & Wright, 1982; Newell & Carlton, I
nik, & Frank, 1978). In each of these models, variability is

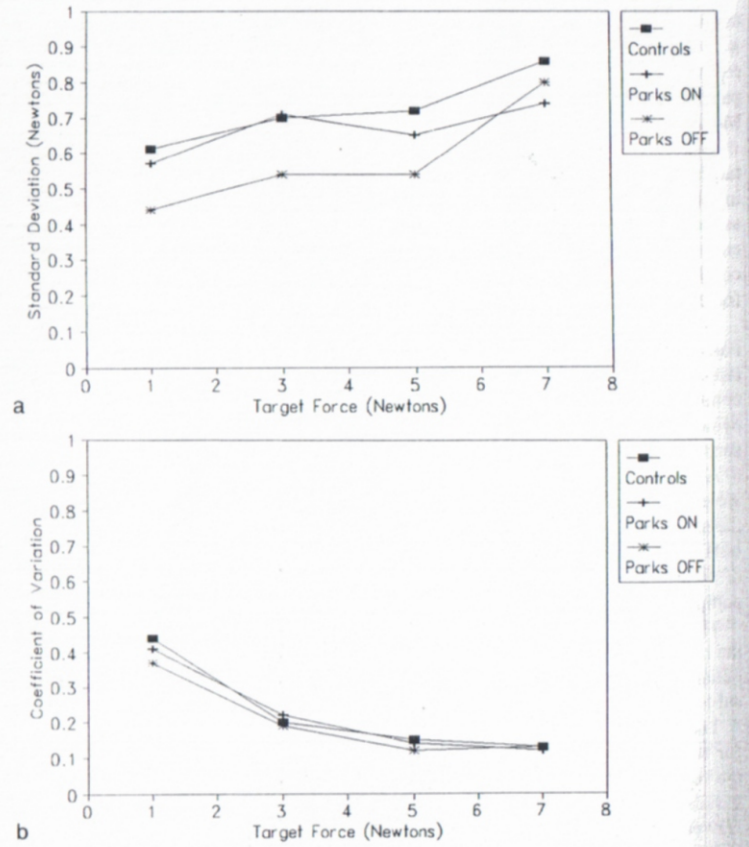

Figure 15.10 Results of Force Control Experiment 1. Mean standard deviation of peak force is plotted as a function of target force (a). The data are transformed into coefficient of variation measures (SD/Force) in (b).

with force (or distance), as was found in the current experiment. In addition, all of these models predict that variability should be inversely related to impulse duration (or movement time). In other words, there should be a speed-accuracy trade-off.

Assuming that this trade-off was operative, the data in Figure 15.10 can be reinterpreted as demonstrating a force control deficit in the Parkinson patients. In the *off* condition, the Parkinson patients took over 80% longer to complete their force pulses in comparison to the control subjects. Despite this increase in impulse duration, the variability of the two groups was essentially identical. It is possible

that increased force variability in the patients is offset b variability resulting from the slower generation of the force powerful demonstration of this finding is seen in the within- 50% increase in impulse duration when the task was perform led to no observed decrease in variability. Again, we assu variability caused by the drug manipulation is obscured by the resulting from the longer impulse durations.

The preceding argument is admittedly post hoc: We had o a force variability deficit could be identified by examining th However, when the temporal differences were considered, it analysis was too simplistic. The argument of a force deficit in P emerges when the data are analyzed in light of current models is possible, however, that the Parkinson patients do not ha rather that, for some unknown reason, their movements do no speed-accuracy trade-off.

This possibility was tested in a second experiment. Four of whom had been in the preceding experiment, were teste task under two different instructions. In both conditions, the p to make single, smooth force pulses, trying to match the ta condition, the patients were instructed to generate rapid for condition, the patients were instructed to move more slowly single pulse. Subjects completing the *slow* condition first we about twice as fast in the *fast* condition; subjects completing were instructed to move about twice as slow in the *slow* con

The subjects were able to follow the instructions. The m for the *slow* and *fast* conditions were 588 ms and 252 ms, respe data are shown in Figure 15.11, a and b. The Parkinson p strong dependency of variability on impulse duration. Both of variability (Figure 15.11a) and the relative measure of varia reveal greater variability for the faster impulses. These res performance of Parkinson patients does follow a general spe Thus, the results of this experiment strengthen the interpr ganglia lesions of Parkinson's disease impair force control.

In summary, the evidence reviewed in this section sugg coordination in Parkinson's disease may reflect an increas different component operation than that observed in the studi patients. It should be reemphasized that to call the deficit a may be misleading. A more accurate description of the Park in terms of regulating the force-time profile of an isometri not think the Parkinson patients adopt a strategy of moving compensating for increased variability in force control. Rath disease impairs their ability to produce normal force-time p viously, the computation performed by the basal ganglia indirectly to force control. The computational process may be transitions between different states of muscular activity. N model the proposed deficit is not attributed to a process i

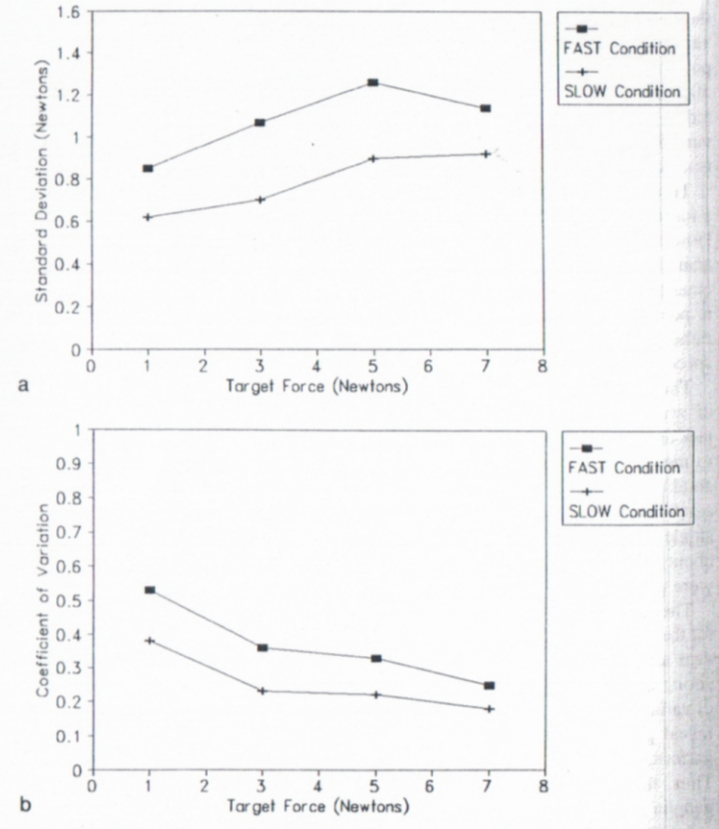

Figure 15.11 Results of Force Control Experiment 2 in terms of mean standard deviation of peak force plotted as a function of target force in (a), and coefficient of variance measures (SD/Force) in (b).

regulating temporal aspects of the contraction, a process we would expect to involve the cerebellum.

Force and Timing Deficits in Clumsy Children

We have recently applied a component analysis to a developmental issue (Lundy-Ekman, Ivry, Keele, & Woollacott, 1991); namely, Why are some children clumsy,

and can the motor variability in clumsy children be attribute processes? Different subtypes of clumsiness have been note 1970), although an etiological framework for these differen lated. One hypothesis concerning developmental motor probl may reflect mild forms of brain dysfunction (Tupper, 198 abnormalities are not found (Henderson, 1987), the child symptoms that are similar to the problems seen in patients w the term *soft neurological signs* has been employed to desc contrast to the "hard signs" that can be ascribed to specifi

Lundy-Ekman et al. (1991) identified a group of clumsy them for the presence of soft neurological signs. From a g eight-year-old children who showed some element of clums for further study. Fourteen of these children demonstrated so cerebellar dysfunction, such as dysmetria and intentional demonstrated soft signs consistent with basal ganglia dysfu basal ganglia signs included choreiform, athetoid, and synk signs are not associated with Parkinson's disease, but rath ganglia disorder, Huntington's disease. The signs observed cluded from the study were either mixed or inconsistent.

The 25 clumsy children and 10 normal children were teste tapping, perception-of-duration, and perception-of-loudness and b). The results provided a striking double dissociation. C children with soft cerebellar signs were more variable than e or the children with basal ganglia signs (Figure 15.12a). This r on the perception-of-duration task, but not on the control involving loudness discrimination. In contrast, the children signs were more variable on the force control task than were (Figure 15.12b). The force control deficit for the children w signs can be assessed more directly in this experiment than in because both groups of clumsy children produced force pu the same duration.

SUMMARY OF COMPONENT AN OF VARIABILITY

The research reviewed in this chapter demonstrates the stre analysis. We began with a set of mental operations that wer involved in the performance of coordinated action. Correlation subjects were conducted to assess the validity of these putat studies were then followed by neuropsychological investigati ducting these studies was twofold: First, we sought converg existence of the operations identified in the correlational worl to explore the neural basis of the operations. The patient r strong evidence for the role of the cerebellum in timing fu work has implicated the basal ganglia in the regulation of force correlated with force such as shifts in motor set (e.g., equilib

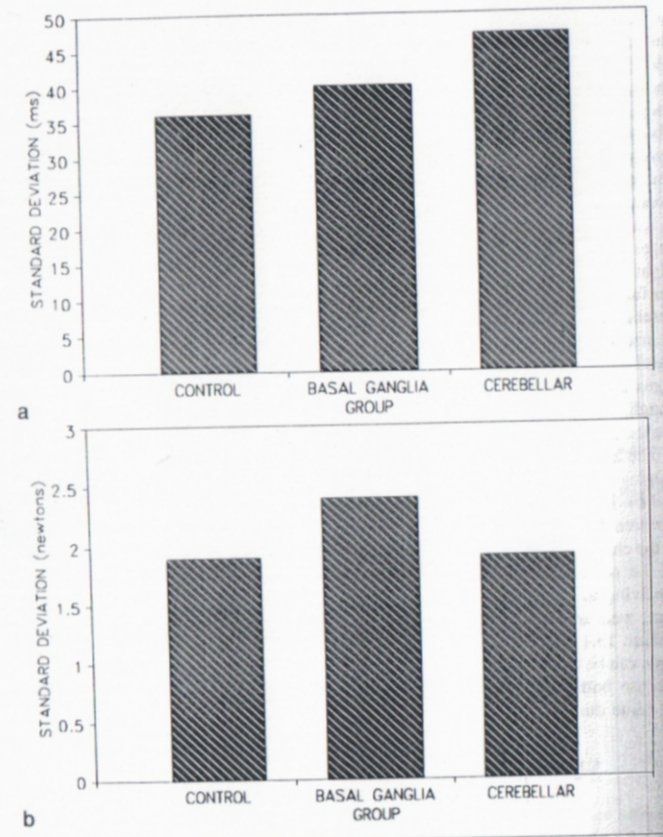

Figure 15.12 Mean standard deviation on the (a) tapping and (b) force control tasks for clumsy children.

Note. From "Timing and Force Control Deficits in Clumsy Children" by L. Lundy-Ekman, R. Ivry, S. Keele, and M. Woollacott, 1991, *Journal of Cognitive Neuroscience*, **3**, pp. 368-377. Adapted by permission.

The dissociation between the contributions of the cerebellum and the basal ganglia in timing and force aspects of movement emphasizes the usefulness of a component analysis for understanding variability. As shown in neuroscience textbooks (e.g., Ghez & Fahne, 1985), these structures are part of two primary subcortical pathways of the motor system. It is noteworthy that there is little interaction between these

two pathways, at least prior to motor and premotor cortex Assuming the computations performed within these pathwa would be expected that variability that arises within each path dent. However, if the observable behavior requires the succe of these pathways (in addition to other pathways), then the va cannot be attributed to a single source. A component analysis for identifying the appropriate pieces of the variability pie.

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