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by

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Abstract

A MODEL FOR SPATIAL DISORDERS IN PARKINSON'S DISEASE

by

Yaakov Stern

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Parkinson's disease (PD) is a progressive disorder of movement associated with degeneration in the basal ganglia and substantia nigra. In addition to motor deficits such as tremor, rigidity, bradykinesia, and postural instability, parkinsonian patients also have deficits of sequential and predictive movement that appear to represent a disorder in the higher-order control of movement. Perceptual motor deficits on cognitive tasks such as construction are commonly observed. In this study, patients with PD and controls completed a tracing task in which they traced patterns of increasing complexity presented on a vertical screen. Some patterns were presented with segments deleted and subjects were required to fill in missing segments. Tracing movements were quantified using digitizing equipment. Subjects also completed a brief test of general intellectual function, a construction test and an assessment for depression, and severity of patients' signs and symptoms of PD was rated. Patients performed more poorly than controls on complete patterns, and their errors increased more sharply than controls on patterns with missing segments. Patients' but not controls' performance on patterns with missing segments was related to performance on the construction tasks. These findings suggest that there is a

perceptual motor deficit in PD that affects performance in both the tracing and construction tasks. This deficit may represent the defective participation at the basal ganglia in an efference copy system which correlates motor and sensory information in order to generate an accurate spatial representation of the environment.

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Cognitive deficits in Parkinson's disease (PD) are most often encountered on perceptual motor or visuospatial tasks (e.g. Botez & Barbeau, 1975). Since many of these tasks require discrete voluntary motor responses, it might be argued that observed deficits reflect nothing more than the movement disorders endemic to PD. If so, these tasks provide no additional information concerning the existence of an independent cognitive dysfunction. This argument could be countered by delineating specific learned voluntary movement disturbances in PD that are not related to its motor symptoms. There is wide ranging evidence from studies in animals (e.g. Divac, 1972) and man (e.g. Stern, Mayeux, Rosen & Ilson, 1983) that suggests that the basal ganglia, the area most likely to be affected in PD, may be involved in cognitive processes that impact upon the processing of spatial information. The present study examined deficits in construction and another voluntary movement task, their interrelationship, and their relation to the clinical motor signs of PD. It attempted to delineate a specific perceptual motor deficit in PD that is separate from the movement disorder of PD.

After a brief review of PD, constructional apraxia, and the functional neuroanatomical connections of the basal ganglia, evidence suggesting that the basal ganglia are involved in cognitive processes affecting perceptual motor function will be discussed. The pilot study which was carried out to develop the techniques and analyses used in this study will then be described.

Parkinson's Disease

PD is a neurological disorder that was first described by James Parkinson in 1817. The primary motor symptoms of PD consist of tremor,

rigidity, bradykinesia, and postural instability. Tremor occurs at rest, at a rate of 4-8 cycles-per-second (Fahn, 1977). Rigidity, or resistance to passive movement, in the limbs or axial muscles of the neck and trunk often has a cogwheeling, or ratchet-like, quality. Bradykinesia is more difficult to define; it consists of difficulty in initiation and paucity of movement -- or akinesia, and slowness of movement. Postural instability results in problems of balance and gait.

PD can occur in the aftermath of encephalitis or as a consequence of the administration of certain drugs but, more often, it has no known cause (Fahn, 1977). The neuropathology of idiopathic PD consists primarily of neuronal loss in the substantia nigra and locus coeruleus, with changes in the basal ganglia due to loss of nigral neurons (Fahn, 1977). The biochemical basis for PD appears to involve cell loss in the substantia nigra and the concomitant depletion of dopamine generally supplied to the basal ganglia via nigro-striatal projections. The depletion of dopamine may serve to release the basal ganglia from inhibitory modulation (Fahn, 1977). Consequently, partly cholinergic neural systems become more dominant, leading to abnormal striatal activity. Drug treatment for PD therefore combines replacement of dopamine via levodopa with administration of anticholinergic medication (Fahn, 1977).

Depression often occurs in PD, and estimates of its incidence range from 40 to 90% (Mjones, 1949; Mindham, 1970), with 50% being the most frequently cited figure (Brown & Wilson, 1972; Mayeux, Stern, Rosen & Leventhal, 1981a). Whether this depression is situational or endogenous, or is characteristically different than depression as

defined in the Diagnostic and Statistical Manual (Third Edition) (DSM-III, American Psychiatric Association, 1980) is not clear, but in one study those patients that met DSM-III criteria for major depression had neurochemical changes similar to those seen in depressed psychiatric patients (Mayeux, Cote, Stern & Williams, 1983). Since the severity of depression correlates with intellectual function in patients with PD (Mayeux et al., 1981a); it is important to assess depression in any study of intellectual function in PD.

Investigators have typically described the cognitive dysfunction associated with PD as a form of dementia, with incidence rates ranging from 20-80% (Lieberman, Dzialotowski, Kupersmith, Serby, Goodgold, Kerein & Goldstein, 1979). Some attribute the dementia of PD to concomitant Alzheimer's disease occurring independent of and superimposed upon the PD in older patients (Hakim & Mathieson, 1979), while others see it as a dementia that, while similar to Alzheimer's disease, does not share its neuropathological changes (Lieberman et al., 1979). Alzheimer's disease is characterized by a gradual progression of amnesia, aphasia and apraxia, with cognitive deterioration. An alternate view is that PD represents a subcortical dementia similar to that described in progressive supra-nuclear palsy and Huntington's disease (Albert, 1978). Subcortical dementia is poorly defined. Its features have been characterized as slowed but not defective memory and cognition often accompanied by prominent affective symptoms, gait and postural changes, involuntary movements, and dysarthria but not aphasia.

While the general characterization of intellectual changes as dementia is useful, it does not establish the specific characteristics

of the intellectual changes associated with a specific disease. This is especially important in PD since deficits in tasks such as construction or tracking are often seen in patients that would not meet DSM-III criteria for dementia (Mayeux, Stern, Rosen & Benson, 1981). Lowered performance on the Wechsler Adult Intelligence Scale (WAIS) is mainly found on performance subtests (Loranger, Goodel, McDowell, Lee & Sweet, 1972). Deficits have been noted on (1) construction tasks such as drawing, block design, and puzzle assembly (Mayeux & Stern, in press; Botez & Barbeau, 1975; Joubert & Barbeau, 1969); (2) spatial tasks such as the vertical orientation and trail-following in which the subject must use a map to guide his movement along an array of markers on the floor (Teuber & Proctor, 1964; Bowen, Hoen & Yahr, 1972); and (3) tracking tasks in which the subject must match his movement to that of a display (Flowers, 1978a, 1978b).

A central hypothesis in this study is that patients' defective performance on tasks only occurs under specific conditions. The evidence to be presented throughout this introduction will suggest that if there is sufficient external information (or exteroceptive stimuli) to guide performance, then patients can perform these tasks successfully. When this external information is not present, successful performance depends on internal coordination of spatial information and patients' performance will be defective. Several types of evidence to support this hypothesis will be reviewed after two additional issues are briefly discussed. First, since perceptual motor deficits in PD are commonly found on construction tasks, the nature of constructional apraxia will be described. Second, since many of the studies to be discussed deal with manipulations of the basal ganglia and areas it is

interconnected with, the functional neuroanatomy of the basal ganglia will be briefly described. This is also important since, while the major locus at neuropathology in PD is in the substantia nigra, evidence suggests that a system of interrelated structures including the basal ganglia and portions of the frontal lobes is disrupted.

Constructional Apraxia

Benton (1969) defined constructional apraxia as a disturbance in activities such as assembling, building, drawing, in which the spatial form of the final product is inaccurate even though the individual movements made during construction were not defective. While it might be expected that any test that requires discrete motor responses would be affected by the motor signs of PD, it would be necessary to demonstrate construction deficits unrelated to those motor symptoms in order to posit a higher-order disturbance of the nature that Benton defines.

Many types of tests have been used to elicit constructional apraxia, including drawing from a model and spatial tasks. While construction deficits are most commonly associated with damage to the parietal lobe, specific types of construction difficulties have been described in patients with frontal lobe lesions. Luria and Tsvetkova (1964) suggested that, in parieto-occipital lesions, the general factor underlying constructive disturbances is a loss of the spatial organization of the elements, while in lesions of the frontal lobe the general factor underlying constructive disturbances is a loss of programming and regulating sequential behavior, instability of the primary intention or program, and the inability to compare results with the preliminary intention. Therefore, differences exist between

construction deficits in patients with frontal and parietal lobe lesions. Since the following discussion of the neuroanatomic connections of the basal ganglia will describe intimate connections between them and the frontal association cortex, it might be expected that construction deficits associated with basal ganglia dysfunction would be similar to those seen in patients with frontal lesions. This similarity will be discussed further below.

Functional Neuroanatomy of the Basal Ganglia

This review of the neuroanatomy of the basal ganglia and connecting structures, and the depiction in Figure 1, are based primarily on Webster (1975). The largest components of the basal ganglia are the striatum (caudate and putamen), and the globus pallidus which is divided into an internal and external segment. Generally the striatum is afferent and projects to both parts of the globus pallidus which is efferent to other structures. The other nuclei included in the basal ganglia are the substantia nigra which consist of the pars compacta and pars reticulata, and the subthalamic nucleus. The external globus pallidus projects to the subthalamic nucleus, which in turn projects to the internal globus pallidus. The projection of the pars compacta of the substantia nigra to the striatum is the dopaminergic nigro-striatal pathway that appears to be the key locus of degeneration in PD. The globus pallidus in turn has projections to the pars compacta of the substantia nigra. In addition, the striatum projects to the pars reticulata of the substantia nigra, which in turn projects to dorsal thalamic nuclei.

Most pallidal efferents end in nuclei of the dorsal thalamus, which in turn project to the motor and granular frontal cortex. Other fibers from the dorsal thalamus end in the nucleus centrum medianum which projects back to the striatum. Some pallidal efferents project directly to the centrum medianum which in turn projects to the striatum. All the projections discussed maintain topical ordering throughout all of the interconnections. In addition, it should be apparent by now that there is a large amount of "feedback" between the structures involved. Thus, the striatum both receives projections from and projects to the substantia nigra, and pallidal input to the thalamus is paired with thalamic input to the striatum. To complete this picture, all areas of the association cortex project to the caudate of the striatum, again in a topically ordered manner. The basal ganglia's main sensory input is indirect information via the cerebral cortex, and some indirect input from the reticular formation. Its main output is via the thalamus to the motor and frontal granular cortex. (The presence of output via the thalamus to the rest of the association cortex is still debated.) Thus, there is another "loop" consisting of the association cortex projecting to the striatum, striatum to the globus pallidus, globus pallidus to dorsal thalamus, and dorsal thalamus to at least the motor and granular frontal cortex. The functional nature of the cortico-striatal projections will be further discussed below. In addition, it will be suggested that the organization of the system into "feedback loops" in some way alows the coordination and monitoring of perceptual motor activity.

The major impact of PD on the basal ganglia and its interconnections is neurochemical. With the degeneration of the substantia nigra, there

is a reduction of dopamine in the striatum, which generally is the area of highest concentration of this biogenic monoamine in the brain. The dopaminergic input from the nigro-striatal pathway probably generally serves to inhibit striatal activity, while cholinergic input from other sources has an excitatory capacity. Other neurochemical changes in the brains of patients with PD exist but are not as well understood.

A review of studies of the behavioral consequences of basal ganglia manipulation will support the contention that the basal ganglia play a role in the internal coordination of perceptual motor activity.

Basic Behavioral Studies in Animals

Gross ablation or stimulation of the basal ganglia disrupts ongoing behavior and impairs simple task performance, suggesting that this area is not only important in control of movements, but may affect goal directed behaviors as well. For example, Hassler (1978) found that unilateral stimulation of the putamen in freely moving cats produced arrest of all activities, opening of the eyes, turning to the stimulated side, and suppression of spontaneous turning to the non-stimulated side. In contrast, pallidal stimulation elicited the excitatory effects of turning to the contralateral side, and dilation of the pupils. He suggested that the putamen might function to focus attention and excitability to the stimulated side by suppressing all other events, including those coming from the opposite side.

The motor inhibitory or attentional modulating effects of stimulation to the striatum are complicated. Mild electrical or chemical stimulation to the rat caudate during bar-pressing for liquid reinforcement inhibited this response, although the animal was still capable of

other behaviors such as grooming or exploring (Buchwald, Wyers, Okuma & Heuser, 1961). Introduction of a novel stimulus during stimulation disinhibited bar-pressing. Unilateral stimulation often led to replacement of contralateral with ipsilateral bar-pressing (Buchwald, Hull & Trachtenberg, 1964). Stimulation thus appears to affect behavior in a fashion that is dependent on the external environment or situation.

Gross bilateral ablation of the caudate in cats produced no gross neurological symptoms, but marked compulsory approach to prominent stimuli and peculiar postural adjustment were noted (Villablanca, Marcus & Olmstead, 1976). On bar-pressing alternation and T-maze alternation tasks, where animals had to alternate between two responses for reinforcement, responses were slowed, and there was a marked inability to alternate responses; animals persisted in the response that produced the last reward (Olmstead, Villablanca, Marcus & Avery, 1976). The animal became stimulus bound to the most intensive stimulus and was unable to make fluent alternating responses between behaviors that were present in his repertoire. Thus, the animal was only influenced by external cues and could not coordinate task performance internally.

Since these studies used rather gross ablation and stimulation techniques, the specific behavioral roles of areas of the striatum and pallidum could not be distinguished. Still, both concepts suggest that the basal ganglia are important for the sequencing of responses when they are not cued by external stimuli.

Basal Ganglia - Cortical Systems

Investigations of specific behaviors reveal a unique pattern of

deficit associated with lesions of different areas of the basal ganglia. Observed deficits are similar to those found following ablation of cortical areas projecting to each particular site in the basal ganglia.

As mentioned above, anatomic evidence indicates that there are topically ordered projections from the entire association cortex to specific areas of the striatum (Divac, 1972; Webster, 1975). Table 1 summarizes three cortico-striatal projection systems that have been investigated: orbitofrontal cortex to ventrolateral head of caudate, dorsolateral frontal cortex to anterodorsal head of caudate, and inferotemporal cortex to the tail of caudate. As indicated, topical relations are also maintained in projections from caudate to other basal ganglia and lower structures (Teuber, 1976).

Performance on specific behavioral tasks is affected by experimental manipulation of one cortico-striatal system while remaining unaffected by manipulation of the other systems. This dissociation is found at the level of cortex, caudate, and lower structures as well (see Table 1). Representative tasks affected by manipulation of each system are also listed in Table 1. These findings have been best documented in monkey but have also been observed in dogs, cats and rats (Divac, 1972).

Destruction or stimulation of the anterodorsal head of caudate or of dorsolateral frontal cortex yield deficits in delayed response and delayed spatial alternation (Divac, 1968). In the delayed response paradigm, the animal watches food being placed in one of two identical food wells. A delay period follows, in which an opaque screen blocks the animal's view of the well. The screen is then raised and the animal

chooses one of the wells. In the delayed spatial alternation paradigm, a similar procedure is used, but the correct food well is always the incorrect well of the previous trial.

Lesions or stimulation to the orbito-frontal cortex or to the ventrolateral head of caudate produce deficits in tasks requiring response inhibition or impulse control such as object reversal and go-no go tasks (Battig, Rosvold & Mishkin, 1962). On the first trials of an object reversal task, the animal receives a reward for choosing either of two objects. Then the rewarded object is switched and the animal must learn to choose the previously incorrect stimulus. In go - no go tasks, a response must be initiated to one stimulus and inhibited to another.

Experimental manipulation of the tail of caudate or inferotemporal cortex yields deficits in visual discrimination tasks (Divac, Rosvold & Szwarcbart, 1967). In these tasks the animal is presented with two visual stimuli, such as a triangle and a circle, and is rewarded for choosing one of the stimuli regardless of its position.

Two features are notable in these studies: the degree of specificity of behavioral function of various parts of the caudate, and the similarity of effects yield from manipulation basal ganglia area and projecting cortical areas. One study that demonstrated both of these features combined aspects of visual discrimination and delayed response paradigms (Cohen, 1972). Two different visual stimuli were briefly displayed via back projection on two opaque displays, which then became opaque again. After a delay, the animal had to choose the display on which the correct visual stimulus had originally been projected. Monkeys were implanted bilaterally on in two of these four areas: dorsolateral frontal cortex, anterodorsal head of the caudate,

inferotemporal cortex, and tail of caudate. Stimulation to one of these regions was administered during task performance. Stimulation to both dorsolateral frontal cortex or anterodorsal head of caudate affected performance only if administered during the delay period. In contrast, stimulation to tail of caudate or inferotemporal cortex only affected performance if administered during the presentation of the visual stimuli. Thus, the anatomical systems were functionally dissociated while the anatomically related structures within the systems showed functional equivalence.

The specific deficit seen following destruction of the anterodorsal head of caudate or the dorsolateral frontal cortex for delayed response tasks and delayed spatial alternation again stresses the participation of these areas in tasks in which there is a paucity of external cues. In these tasks the animal must make a spatial judgment between two food wells without external guidance. If this guidance is even indirectly provided, for example by making the food wells slightly different in shape, the lesioned animal can perform successfully. A similar pattern of performance in humans with diseases that affect the basal ganglia would lend further support to this interpretation of delayed response and spatial alternation performance. While it is possible that deficits could be found in humans that parallel those seen with lesions to other basal ganglia-cortical systems, attention here is limited to the basal ganglia-frontal system responsible for delayed response and delayed spatial alternation deficits.

Specific lesions to the basal ganglia occur infrequently in man. PD and Huntington disease (HD), among others, are used as human models of basal ganglia dysfunction. Although the basal ganglia are not the only

structures affected in these diseases (Boller, Mizutani, Roessmann & Gambetti, 1980; Hakim & Mathieson, 1979; Ruberg, Ploska, Javoy-Agid & Agid, 1982; Victor, 1978), similarities in behavioral abnormalities, as well as parallel results from animal studies, suggest a primary involvement for the basal ganglia in some of the observed changes. In addition, there is evidence for depletion of dopamine in the frontal cortex in PD (Javoy-Agid & Agid, 1980) and when dopamine is depleted in the frontal lobes of animals, performance on delayed spatial alternation tasks is impaired (Brozoski, Brown Rosvold & Goldman, 1979). This suggests that the fronto-basal ganglia system may be the one affected in PD.

Behavioral tasks used in human studies may be analogous to those used with animals, but must be redesigned for human study. For example, the delayed response task used for animals is inappropriate because the position of the reward can be verbally encoded and retained not only over the delay period, but for a long period after testing (Teuber, 1972). Typically, tasks devised for humans are designed to examine intellectual processes that may be similar to those tapped in animal tasks. Development of appropriate tasks demands analysis of the salient features of the animals' tasks. This analysis has been most fully explored for delayed response tasks.

Delayed response task performance requires a choice based on spatially encoded information with no external cues to guide it. If the baited food well had some identifying characteristic besides its location in space performance is not affected (Pohl, 1969). The spatial deficit was examined using maze tasks in which the correct path had a specific relationship to the animal's location. While lesioned animals

performed poorly on this task, they learned an equally difficult maze in which the correct path did not depend on the animal's position in space (Potegal, 1969). This suggests that a salient feature of delayed response tasks is the maintenance of spatial orientation to the baited food well relative to the position of the subject's body (Potegal, 1972). It is possible that as the animal moves during the delay period, it must take these movements into account in order to maintain its orientation to the baited food well. Potegal (1971) demonstrated a similar deficit in HD patients on a task in which they stood in front of a target position, were blindfolded, shifted position, and then attempted to touch the target position from their new location. Patients were unable to compensate for the change in body position in this simple spatial localization task.

The Aubert task (1961) is another spatial task which also demands compensation for body movement in a situation where there are no external cues present to guide performance. In a darkened room, subjects sit in a chair tilted to the left or right of the vertical, upright position and attempts to adjust a luminous rod to vertical. Both PD and frontal lesioned patients make larger than average errors on this task (Teuber & Proctor, 1964). The dependence of perceptual-motor performance on exteroceptive stimuli has also been demonstrated using a block design task. While Luria and Tsvetkova (1964) found that both patients with frontal and parietal lobe lesions performed poorly on a block design task, frontal but not parietal patient performance could be improved by supplying the patient with external guidance in performing the task. First, the separate blocks in the pattern to be copied were clearly delineated. Second, the patients were supplied with instructions that

stressed a logical sequencing of constructive activity and constant checking of the accuracy of blocks put into place. In a pilot study (Stern, unpublished), 10 PD patients who had difficulties with WAIS block design were given a version of the test in which the individual blocks were outlined. With this aid, four of the subjects could complete all of the designs. The remaining subjects were supplied with a strategy for completing the task which consisted of constructing one row of blocks at a time and then comparing that row with the sample and correcting it if necessary before continuing. With this aid all subjects could complete every WAIS block design pattern. This work suggests a similarity between the construction deficits in patients with PD and frontal lobe damage. Further, it demonstrates that both groups benefit from external guidance.

These studies demonstrate that analogous deficits can be isolated in humans with PD and frontal lesions and in animals with lesions to the dorsolateral frontal cortex or anterodorsal head of caudate. The similar effects of cortical and basal ganglia lesions in both animal and human studies raises the question of their functional relationship. Divac (1972) has suggested two alternatives: (1) the cortex and basal ganglia may be working in parallel, with each individual structure capable of handling the behavioral demands of the tasks, or (2) associated areas may work in series, with each contributing a component of the necessary processing. While the behavioral consequences of damage to associated areas are similar, they are not identical. For example in adult monkeys electrical stimulation to dorsolateral frontal cortex disrupted delayed response task performance only when applied in the first few seconds of the delay period. In

contrast, stimulation to anterodorsal head of caudate reduced performance to chance level when administered at any point in the delay period (Stam, 1969). In humans, although both PD and frontal lobe lesioned patients were impaired on the Aubert task, frontal patients were defective only when sitting on a tilted chair. Parkinsonians made errors even while sitting on a chair in its upright position and, further, parkinsonians had more difficulty in adjusting their own bodies to an upright position (Teuber, 1976).

These findings suggest that Divac's second alternative is more likely; areas of the basal ganglia and their corresponding cortical areas work in series, such that each structure contributes a component of the processing necessary for a particular behavior. Lesions to each area result in similar, but not identical behavioral deficits.

Higher-order Deficits in the Control of Voluntary Movement

To this point, the involvement of the basal ganglia in carrying out behavioral tasks that have a cognitive component has been discussed. Various studies have described deficits in the control of voluntary motor activity that relate to changes in the basal ganglia and that also appear to have an intellectual component. These can best be described as deficits in the generation of sequential and predictive voluntary movement.

Although parkinsonian patients were capable of performing voluntary movements individually, they were impaired when required to perform two movements simultaneously, such as squeezing a bulb with one hand while drawing designs with the other (Schwab, Chafetz & Walker, 1954; Perret, 1968). Movements became increasingly deficient when the complexity of

one of the tasks was increased (De L. Horne, 1973). Clinically, a deficit in diadokinetic or double simultaneous movement is observed in PD. A similar deficit was found in cats with lesions in the caudate; animals were unable to bar-press for fluid and drink the fluid simultaneously (Olmstead et al., 1976). Both human and animal difficulties may be related to deficits in the monitoring and coordinating of motor information with sensory information needed to sequence task performance.

Not only sequences of movement, but single movements are affected by changes in the basal ganglia. Although Kornhuber (1974) suggested that slow movements are controlled by the basal ganglia while fast, ballistic movements are programmed by the cerebellum, evidence suggests that ballistic movements are also impaired by diseases of the basal ganglia. Healthy subjects adjust the velocity of their movement to make ballistic movements of varying distances in the same amount of time. In contrast, parkinsonians maintain a constant velocity and, consequently, movement time increases (Flowers, 1975). EMG studies of the normal ballistic movement found that there was a single triphasic burst of agonist, antagonist, and then agonist muscles during the movement. Parkinsonians generated a variable number of triphasic burst groupings (Hallet, Shahani & Young, 1977); this may represent a deficit in the planning or monitoring of movements. Analysis of changes in velocity of ballistic movement of patients on a step tracking task revealed that each movement actually consisted of groups of separate movements (Draper & Johns, 1964). If the position of the target was shifted as the patients moved toward it, inability to correct movements toward the target's new position occurred before they had reached its original position (Draper

& Johns, 1964). When patients tracked a target moving in a sine wave pattern across an oscilloscope screen, tracking accuracy decreased as speed of the target's movement increased (Flowers, 1978a). Flowers (1978a) suggested that patients can only make "closed loop" movements, which were guided by exteroceptive stimuli, in this case constant visual feedback. This is what occurred when the target moved slowly. Tracing the sine wave pattern at a higher speed demanded "open loop" movement, where the subject had to predict the movement of the target and generate that movement on his own. That is, the subject had to generate his own sine wave movement, based on a prediction of where the target would travel in the future. Patients could not generate these predictive movements, since by their nature the movements could not be guided by external stimuli. These deficits in predictive movement were not a function of slowed movement in general, because patients could track "noise" patterns, which allowed only closed loop movements, as well as controls did (Flowers, 1978a).

Similar tracking deficits have also been demonstrated in non-human primates with lesions to various areas of the basal ganglia. Bilateral cyrogenic lesions of the caudate impaired monkey's ability to track a moving light with their paw; the animals lagged behind the target, and erred when reaching for the target even when it was stationary (Bowen, 1969). In another study, unilateral cooling of the globus pallidus in rhesus monkeys impaired rapid but not slow movements of the contralateral arm (Caan & Stein, 1979). Again, this study suggests that the basal ganglia are especially involved in open loop, or predictive movements, which must be planned before they are initiated, and must be carried out without external feedback.

A predictive tracking deficit can also be demonstrated in humans and animals with basal ganglia lesions by removing visual feedback. For example, unilateral cooling of the globus pallidus in monkeys produced a breakdown in flexion-extension elbow movements of the contralateral arm only when movements were made without visual guidance (Hore, Meyer-Lohmann & Brook, 1977). A follow-up study revealed deficits in these movements even with visual feedback. However, over time monkeys adapted a strategy of making slower, more accurate movements that were presumably under greater visual guidance (Hore & Villis, 1980). In parkinsonian patients, flexion and extension movements of the elbow also were inaccurate when visual feedback was removed (Cooke, Brown & Brooks, 1978). In a tracking task (Flowers, 1978b), subjects used a joystick to control a marker and track a target moving in a regular pattern across an oscilloscope screen. During testing, either the tracked target or the subject-controlled marker would briefly disappear from the screen. During this time the subject was required to continue tracking with the joystock as if the marker or target were still there. The poor performance shown by PD patients, relative to controls, again suggested a deficit in the generation of predictive movement.

Pilot Study

The deficits of sequential and predictive movement seen in humans and animals following damage to the basal ganglia suggest that the basal ganglia may be involved in aspects of planning and monitoring of movement. These deficits appear to have the same characteristics as the perceptual motor deficits which are seen in delayed response tasks in animals and in tasks such as construction in humans. If these

higher-order movement deficits have a similar underlying cause as cognitive deficits in PD, the two should occur together and relate in severity. We hypothesized that sequential and predictive movement disorders in PD would relate to construction performance (Stern et al., 1983). Flowers (1978b) had used a tracking task, described above, to study deficits in predictive movement deficits in PD. We developed a simplified task based on Flowers' to test our hypotheses. Patterns were mounted on a vertical, transparent screen (Figure 2) and subjects simply traced the pattern back and forth with their finger. Patterns 3, and 5-7 had portions missing. In pattern 3, the subject was instructed to move back and forth between the two endpoints in a straight line. Patterns 5-7 were similar to one another, but had progressively increasing deletions. Before tracing each of these patterns, pattern 4 was superimposed to demonstrate the completed pattern to the subject. The subject then traced each pattern, attempting to fill in the missing segments. Tracing performance was videotaped through the transparent screen for later analysis.

Eighteen PD patients and 14 healthy elderly controls were tested with this task. In addition, they were tested with a modified version of the Mini-mental Status Examination (Mayeux et al., 1981a) and with the Rosen Drawing Test (Rosen, 1981) in which the subject simply copied designs of varying complexity.

Videotaped tracing performance was rated by two neurologists. In rating tracing performance, we found that PD patients made two types of errors. In the first type of error, patients deviated from the displayed pattern and hesitated during tracing. These errors were not related to the complexity of the patterns; they occurred with similar frequency in

all patterns. Ratings of these errors did not correlate with performance on the mental status or construction tests and these errors were infrequent in the control group. We concluded that they were related to the non-cognitive motor deficits of PD.

Another type of error that was observed was called "loss-of-form" and consisted of distortions of the missing segments of patterns during attempts to trace their shape. PD patients made these errors on all patterns. Errors increased in severity as the deleted segments became more complex, and summed error ratings correlated with performance on both the mini-mental and construction tests. In addition, these errors were not related to severity of PD symptoms or to the first type of error we had found.

The parkinsonian patients' performance in this pilot study meets the criteria that have been described for a construction deficit; loss-of-form errors occurred that were not related to the motor deficits of PD. In addition, patients' performance in this study was similar to that described in other studies of perceptual motor tasks in patients with PD and animals with basal ganglia lesions; performance broke down when external guidance was removed. The relation of the loss-of-form errors to performance on the construction task suggests that performance on both tasks may be affected by a common cognitive deficit.

The major weakness in this pilot study is that subjective ratings were used to evaluate tracing movements. In the present study equipment was developed to quantify subjects' tracing movements. Subjects completed this modified task as well as a construction and mental status test. Parkinsonian symptoms were rated. As in the pilot study, the purpose of this study was to evaluate the relation between performance on

a sequential and predictive movement task and on a standard test of perceptual motor ability. In addition, the effect of parkinsonian symptoms on tracing performance was evaluated to differentiate between performance deficits that relate to these symptoms and those that may be representative of a true perceptual motor deficit.

Hypotheses

The pilot study (Stern et al., 1983) suggests that there is a relation between the higher-order movement deficits and perceptual motor deficits in PD such as construction. This may reflect a similarity in the underlying cause for the two types of performance deficits. This finding, and those in other studies of patients and animals, suggest the hypothesis that in PD there is an inability to monitor, evaluate, and generate accurate spatial performance in the absence of external guidance. This is because the basal ganglia can no longer effectively participate in the system that usually enables this performance. This deficit is hypothesized as the underlying cause of poor performance on on perceptual motor tasks in PD, and in animals with basal ganglia lesions.

Given the hypothesis in this rough form, several predictions were made about the results of this study:

(1) In the tracing task, as exteroceptive performance cues are removed by deleting segments of the pattern, patients' tracing accuracy should decrease. This decrease in accuracy, as more of the pattern is deleted, should be significantly greater in patients than in controls.

(2) Performance on tracing task designs with deleted segments should correlate with construction performance in parkinsonians but not

controls.

(3) Some aspects of tracing performance in patients should correlate with the severity of parkinsonian motor symptoms. However, symptom severity should not correlate with tracing performance on missing segments of patterns.

METHODS

Subjects

Sixteen patients with PD were chosen according to the following criteria:

(1) Appropriate clinical features of PD, including tremor, rigidity, bradykinesia, and postural instability, resulting in an established diagnosis of PD. For uniformity and ease of availability, only patients with idiopathic PD were tested.

(2) Stabilized medication for PD symptoms over the past two months. Medication was not altered for the purposes of this study.

(3) Ability to comprehend instructions and respond appropriately to tasks.

Four patients who were screened did not meet these criteria and were not included in the study.

Nine controls were selected according to the following criteria:

(1) No history or signs of neurologic or psychiatric disorder upon examination.

(2) No medications that could affect intellectual function.

(3) Approximate matching of age and education to those of PD patients.

All subjects were volunteers, and were provided with and signed a statement of informed consent. All subjects were English speaking and none had a history or signs of dementia according to DSM-III criteria upon neurologic evaluation or neuropsychological testing. These criteria include a loss of intellectual abilities of sufficient severity

to interfere with social or occupational functioning, memory impairment and some other disturbance of higher cortical function. A summary of demographic characteristics of patients is given in Table 2 and Appendix A.

Procedure

All tests and assessments were performed on a single day under appropriate testing conditions. Testing consisted of neurological, psychiatric, and neuropsychological testing, and the tracing task.

Neurologic Assessment. All subjects were examined by the same neurologist. The neurologist had no knowledge of subject performance or other aspects of the study. For the patients, the Columbia University Parkinson Disease Evaluation was used to rate signs and symptoms of PD. Each sign and symptom is rated according to defined criteria from 0 to 4 (0 = absence; 4 = greatest severity). For tremor and rigidity, each extremity is rated. The evaluation form and criteria are included in Appendix B. PD evaluation scores and duration of illness are included in Appendix C. Control subjects were evaluated and interviewed to ensure the absence of history, signs or symptoms of neurologic disorder.

Psychiatric Assessment. The Beck Depression Inventory (BDI) was used to rate depression (Beck, Ward, Mendelson, Mock & Erbaugh, 1961). This is a self report instrument in which the subject, in the presence of the examiner, denies or affirms to various degrees 21 statements associated with symptoms of depression. BDI scores are included in Appendix A.

Neuropsychological Testing. The following tests were administered:

(1) Modified Mini-Mental Status Examination (MMS). This is a version of the Mini-mental Status Examination (Folstein, Folstein & McHugh, 1975), as modified by Mayeux et al. (1981a). Modifications include the following items: digit span (forward and backward); recall of present and four previous presidents of the U.S.; naming to confrontation of ten pictured objects from the Boston Naming Test (Kaplan, Goodglass & Weintraub, 1976); a second sentence for verbal repetition; copying two picture designs. These modifications were included to broaden the depth of the assessment and evaluate language more adequately. The modified MMS can be divided into subtests representing different aspects of intellectual function. We have found the modified MMS to be a useful measure of intellectual function in PD (Mayeux et al., 1981a; Mayeux, Stern, Rosen & Benson, 1981b). Maximum score is 57 and a score of 25 or more was required to continue in this study; preliminary investigation suggests that a score below 25 suggests a global dementing process. Modified MMS scores are included in Appendix A. A copy of the modified MMS is included as Appendix D.

(2) The Rosen Drawing Test (Rosen, 1981). In this test the subject copies 15 designs of increasing complexity. Designs were selected to test different levels of spatial representation ranging from topographic to Euclidean to perspective. A copy of the Rosen Drawing Test is included as Appendix E.

Tracing Task. The following tracing task, based on that of Flowers (1978b) and similar to that of Stern et al. (in press), was administered.

(1) Apparatus. The tracing apparatus consisted of a clear screen (4 feet x 4 feet) mounted perpendicular to a testing table. Paths drawn on clear plastic were affixed to the screen (Figure 2). The lines of the paths were .79 mm wide. As before, Path 3 consisted of 2 endpoints which subjects connected in a straight line. Paths 4-7 were the same sawtooth patterns, and Paths 5-7 had progressively increasing deletions. The subject traced the paths by moving a stylus along one side of the screen.

Mounted on the tracing screen was an "L" shaped microphone array of a sonic digitizer (Scientific Accessories Corporation GP6-25H). This array was connected to the digitizing unit. The stylus that is moved along the patterns during tracing was also connected to the digitizing unit. This stylus emitted a noise that was picked up by the microphone array and transformed to the digitizing unit. The digitizing unit converted this sonic information into a Cartesian, X-Y coordinate that describes the position of the stylus at that point in time accurate to within .01 cm. These coordinates were generated approximately 100 times per second. The information was transmitted for storage to a digital disc recorder (Sykes ComStor 1) and later transferred to a computer

(Digital MINC-11) for analysis. Digitizing equipment is further described in Appendix F.

(2) Procedure. The subject was presented with patterns in the same sequence shown in Figure 1, except that pattern 2 was not included in order to create a more straightforward relation between complete patterns and those with missing segments. The subject was instructed to trace the pattern with the stylus starting from the right side, and at the end of the pattern, return to the starting point. Left hand performance followed right. On patterns with deleted segments, the subject was instructed to attempt to trace the pattern filling in the missing segments.

(3) Analysis of tracing performance. A series of computer programs were written in BASIC to analyze tracing performance. Printouts and documentation for these programs can be found in Appendix F. Two aspects of tracing performance, tracing accuracy and velocity, were quantified.

Tracing accuracy was measured in two ways: (1) Area Error. Computer paradigms contrasted the stylus position at each point with the position of the closest point on the pattern. The area between the subject's tracing movement and the pattern being traced was calculated. Tracing accuracy for a missing segment of the pattern could be separately calculated as loss-of-form error. (2) Path Crossings. Another measure of tracing accuracy was the number of times the subject crossed the path

while tracing. With greater accuracy this measure might be expected to increase, since the subject is closer to the path and crosses it more often.

Since points are digitized at a regular rate, the velocity of tracing movement could be calculated. Velocity was measured in two ways: (1) Digitized Points. One velocity measure was simply the number of points digitized during the tracing of a specific extent of a path. Number of digitized points increased with tracing time. (2) Velocity. Another velocity measure used a paradigm which calculated the distance traversed from each digitized point to the next. Those point-to-point velocity values were converted to values representing the velocity at each digitized point by taking the mean of the point-to-point velocities toward and away from the point in question.

Data Analysis

All tracing measures were calculated separately for both sweeps of each path and for the right and left hand. A sweep consisted of the tracing of a path in one direction. Path 3 was analyzed in the same way as Path 1. In the sawtooth paths with deleted segments, (Paths 5-7), performance was quantified in two different ways. One approach was to calculate the tracing measures only for the missing segments, in order to generate a pure "loss-of-form" measure. In this case, the corresponding portions of Path 4, the complete sawtooth path, were used as a baseline for comparison. A second approach was to quantify performance for entire sweeps of Paths 5-7, under the assumption that the presence of the missing segment affected performance along the whole sweep. In this case, the entire Path 4 could be used as a baseline for comparison.

The data analysis itself addressed several questions. Preliminary questions included evaluating possible differences between left and right hand performance and performance on sweep 1 and sweep 2 of each pattern. Thee issues were addressed using t-tests for repeated measures. Another preliminary analysis investigated the relationship between tracing velocity and tracing accuracy. This relationship was tested using correlations.

A major thrust of the data analysis was to compare performance of PD and control groups on the tracing task. This was done separately for each tracing measure in each sweep in the straight line paths (Paths 1 and 3), and the sawtooth paths (Paths 4-7). In both cases, repeated measures ANOVA's were used to evaluate changes in each tracing measure as segments of the paths were deleted. In the straight line analyses, effects in the ANOVA included Path (Path 1 vs. 3), Group (PD vs. control), Hand (Right vs. Left), and the apropriate interactions of these effects.

Two similar analyses were carried out for measures of tracing performance in the sawtooth paths. In the first, separate ANOVA's compared measure for missing segments of each of Paths 5-7 to the corresponding segment of Path 4. This comparison is a logical way to examine the effects of deleting segments, but observations in pilot work suggested that it may obscure important aspects of the effects of deleting segments on tracing. It was noted that not only was tracing of the deleted segment affected but that of the remainder of the path. This occurred in different subjects for various reasons: (1) subjects adjusted or slowed their tracing movements before entering the missing segments, (2) subjects paused prior to beginning to trace the missing

segments, (3) while subjects concentrated on filling in missing segments, performance on remaining portions was not as carefully executed, and (4) when filling in missing segments incorrectly, subjects often could not rejoin the path in a way that would allow them continue tracing it accurately. While none of these events occurred consistently, they did suggest that when evaluating performance only in the deleted segment itself, information about the effects of deleting segments on tracing performance could be missed. Therefore, a second analysis compared the tracing performance measures of entire sweeps of Paths 4-7. This analysis also allowed all four paths to be compared at once, which was not possible in the first type of analysis since different segments were deleted in each path. In this way, trends in performance could be examined as progressively increasing deletions in the sawtooth path were made. In both straight line and sawtooth path analyses, the experimental hypothesis predicted Group X Path interactions, reflecting increasing error by the patients as more of the path was deleted.

Analysis then focused on the relation between tracing performance and performance on cognitive tests. This is a central issue in this study, since it is hypothesized that aspects of tracing performance and performance on tests such as construction should both be affected by a perceptual motor deficit in patients with PD. This relationship was investigated with correlations.

Finally, the relation between tracing performance and measures of PD severity was explored using correlations.

RESULTS

Parkinsonians were significantly younger and better educated than controls, with the average age and years of education 60.5 and 13.2, respectivley, in the PD group, and 67 and 11.2 in the control group. Since age and education affect performance on cognitive tests, it would be expected that MMS and Rosen Drawing Test scores would be higher in the PD group. However, scores on these tests were comparable in the two groups, with PD patients averaging 50.7 and 11.1 on the MMS and Rosen, respectively, and the control group averaging 53.2 and 12.5, respectively (see Table 2). Since age and level of education have been found in the past to be related to performance on these tests (Mayeux et al., 1981), it is possible that if controls and parkinsonians were more closely matched for age and education, controls would have performed significantly better on the MMS and Rosen. Alternatively, it is useful to have the groups matched for performance on these tasks in order to demonstrate relative deficits in the tracing task in the face of comparable performance on these paper and pencil tasks.

Since depression is common in PD, it was important to compare the severity of depression in the two groups. While the average Beck Depression Inventory score was higher in the PD group (PD = 7.9; control = 3.8), the scores did not differ significantly (see Table 2). Thus, it can be assumed that depression did not play a major role in intergroup differences in performance.

Preliminary Analyses

Right vs. left hand and first vs. second sweep performance

Since all paths were traced for two sweeps and with both left and right hands, error and velocity measures were compared for differences between the first and second sweep, and between left and right hand performance. These comparisons were done with t-tests for repeated measures and calculated separately for each path. In comparing right and left hand performance, there were no significant differences between any performance measure on any path. However, there were significant differences between performance on the first and second sweeps in almost every case. Therefore, in correlation analyses investigating the interrelationship of variables in a single path, left and right performance were combined while first and second sweep performance was analyzed separately. Left- and right-hand performance was collapsed for correlational analyses despite occasional significant Hand effects in the repeated-measures ANOVA's. The justification for this is that right- and left-hand performance was combined on the basis of lack of significant differences between left- and right-hand performance within performance on a particular path and correlational analyses were also only calculated for one path at a time. The Hand effect in the ANOVA's assesses combined right- and left-hand performance difference across two or more paths, a different type of comparison.

Relation between tracing error and velocity

Studies of ballistic movement have shown relationships between velocity and accuracy of movement. While movements in the tracing task were slower and would not be considered ballistic, it was expected that a similar relationship would exist. In both PD and control groups, there

were some significant correlations between velocity measures (velocity and digitized points) and tracing error measures (area and path crossings). Significant correlations are presented in Tables 3 and 4 for controls and patients respectively. These correlations indicate that the aspects of tracing movement measured in this study can yield interrelationships that are similar to those seen in other studies of movement.

Since velocity and tracing error did intercorrelate, increased tracing error might in some cases be attributable to an increase in the speed of tracing. Because the experimental hypothesis predicts greater tracing error in the PD group, it is important to evaluate the possibility that this difference is due only to increased tracing speed in that group. This might in turn suggest tht the patients make greater tracing error only because they have difficulty modulating the speed of their tracing movement. This possibility can be excluded, however, since a group difference in velocity existed in only one case, the ANOVA comparing Path 1 to Path 3 performance in the first sweep (See Table 5 and Figure 4). Further, in that case the group difference was seen only in the digitzed points measure of velocity, which did not correlate with tracing accuracy in the PD group.

Comparison of Tracing Performance in PD and Control Groups

Various aspects of tracing performance in parkinsonians and controls were evaluated using repeated measure analysis of variance. Three main effects and their interactions were included: Group, Hand, and Path. Straight line and sawtooth paths were analyzed separately, and separate analyses were performed for sweep 1 and sweep 2.

Straight line analyses, Path 1 vs. Path 3

Significant factors for each performance variable are summarized in Table 5 and illustrated in Figures 3 and 4.

For the area measurement of tracing error in sweep 1, the patients' mean error was 16.7 and 35.13 in Paths 1 and 3 respectively, while controls' mean error was 5.79 and 17.13 respectively. Group, Path and Group X Path interaction effects were significant. Post hoc analysis showed a significant decline in performance (i.e., increase in area measure) from Path 1 to 3 only in the PD group ($p < .05$). This accounts for the significant interaction effect. The group effect indicates poorer performance in the PD group in general (see Figure 2).

For the area error measurement of tracing error in sweep 2, patients' mean error was 19.1 and 36.9 while controls' was 6.3 and 17.9 in Paths 1 and 3 respectively. Only Group and Path effects were significant, but not the interaction effect. The Group effect indicates poorer performance by the PD group (see Figure 3). The Path effect indicates that combined patient and control performance was worse in Path 3.

Patients crossed the Path a mean of 8.13 and 2.7 times, and controls 39.69 and 16.0 times in sweep 1 at Paths 1 and 3 respectively. Four effects were significant: Group, Path, Group X Path, and Group X Hand. The Group X Hand interaction effect was due to the significant change in path crossings in the control group from Path 1 to 3 ($p < .05$) as compared to the nonsignificant change in the PD group. The Group X Hand effect was due to a significant difference in left- and right-hand performance in the control but not in the PD group ($p < .05$). Both of these interaction effects are probably due to the nature of the path crossings

measure. Figure 5 demonstrates that tracing performance must be relatively accurate for path crossings to occur. A slight increase in area error results in a minimum number of path crossings. In the patient group there was a floor effect, with performance poor enough in Path 1 that there were few path crossings. In Path 3, patients' tracing performance was poorer than in Path 1 as measured by area error, but this decline could not be reflected in the path crossing measure.

Similarly, in sweep 2 of Paths 1 and 3, mean path crossings in the PD group was 6.04 and 3.22 and in the control group 35.81 and 16.1 respectively. In the ANOVA, Group, Path, and Group X Path effects were significant and followed the same pattern as in sweep 1. Since it appeared that path crossings were not a sensitive measure of tracing error when error increased, this variable was not analyzed in the sawtooth path ANOVA's.

Turning to velocity, the mean velocity in the patient group was 0.104 and 0.123, and in the controls, 0.065 and 0.106 in sweep 1 of Paths 1 and 3 respectively (Figure 4). Only the Path effect was significant, indicating a higher velocity in Path 3 for the combined PD and control groups. Only the Path effect was significant in sweep 2 as well. Mean velocity in sweep 2 for the PD group was 0.11 and 0.13, and for the control group was 0.072 and 0.112 in Paths 1 and 3 respectively.

Mean digitized points in sweep 1 for the PD group was 676.2 and 521.1, and for the control group was 980.2 and 629.6 for Paths 1 and 3 respectively. There were significant Group, Path and Hand effects, representing more rapid tracing in the PD group, in Path 3 and with the right hand respectively. In sweep 2, mean digitized points in the PD

group were 743.6 and 521.9, and in the control group, 823.0 and 571.9 in Paths 1 and 3 respectively. Only the Path effect was significant, indicating faster tracing overall in Path 3.

Thus, for the two velocity measures, velocity and digitized points, the Path effect was significant in all four cases, indicating slower tracing in Path 3. In one case, digitized points in sweep 1, there were also significant Group and Hand effects. Since the two velocity measures paralleled each other, only the digitized points measure was used in the sawtooth analyses.

Sawtooth path analyses

Analysis of performance sawtooth paths was carried out in two ways. First, tracing performance measures for the missing segment of Paths 5-7 were individually contrasted with the measures for the comparable area of the Path 4, the complete sawtooth path. Then tracing performance measures for entire sweeps of sawtooth Paths 4-7 were then compared in a single repeated measures analysis of variance. For the reasons explained above, only the area measure of tracing error and the number of digitized points measure of velocity were used in these analyses.

Tables 6-8 and Figures 6-13 summarize the significant effects found in the first type of sawtooth analysis. In comparing the area error measurement of missing segments in Path 5 and the corresponding section of Path 4 (Table 6, Figures 6 and 7), mean area error in sweep 1 for the PD group was 6.26 and 10.6 and for the control group was 1.87 and 4.67 for Paths 4 and 5 respectively (Figure 6). Group and Path effects were significant, indicating greater overall error in the PD group and greater error by both groups in Path 5. In sweep 2, mean area error for the PD group was 7.62 and 14.0, and for the controls 2.62 and 6.8 for

Paths 4 and 5 respectively. Group, Path and Hand effects were significant, indicating greater overall error in the PD group, and greater error by both groups in Path 5 and when using the left hand.

No effects were significant in the ANOVA's investigating digitized points in sweep 1 of Paths 5 and 6. In sweep 2 there was a significant Hand effect, indicating more rapid tracing with the right hand.

In comparing area error measurements in the missing segment of Path 6 and the corresponding segment of Path 4 (Table 7, Figures 8 and 9), mean error error in sweep 1 for the PD group was 4.94 and 10.6, and for the control group, 1.8 and 4.67 for Paths 4 and 6 respectively. There was a significant Path effect, indicating poor performance by both groups in Path 6; no other effects were significant. In sweep 2, mean area error for the PD group was 7.2 and 13.15, and for the control group 2.19 and 6.1 for Paths 4 and 6 respectively. Group, Path, and Hand effects were significant, indicating poorer overall performance by the PD group and poorer performance by both groups on Path 6 and with the left hand.

No significant effects were found for digitized points in sweep 1 of Paths 4 and 6 but in sweep 2 there was a significant Hand effect, indicating faster tracing with the right hand.

Table 8 and Figures 10 and 11 present the significant effects in the comparison of tracing performance measures on the missing segments of Path 7 and the corresponding sections of Path 4. In the first sweep, mean area error in the PD group was 7.16 and 32.96, and in the control group 3.32 and 14.27 in Paths 4 and 7 respectively. Three effects were significant, Group, Path, and Group X Path. The Group X Path interaction is due to a significant increase in error in the PD group

from Path 4 to Path 7 (P .05) while this comparison is not significant in the control group. In addition, overall performance is worse in the PD group and on Path 7. In the second sweep, mean area error for the PD group was 9.5 and 31.49, and for the control group, 4.15 and 19.3 respectively. There are significant Path, Hand, Group X Hand, and Group X Path X Hand interactions. Path and Hand effects indicate poorer overall performance in Path 7 and with the left hand. The Group X Hand interaction indicates relatively poorer performance by the PD group with the left hand ($p < .05$), and the Group X Path X Hand interaction indicates a larger decrement in performance by the PD group from Path 4 to Path 7, particularly for left-hand performance. The later interactions were found in the place of the Group and Group X Path interaction effect seen in sweep 1.

In the velocity analyses, no effects were significant except for a hand effect in sweep 2, indicating slower tracing with the left hand.

In summary, the first type of sawtooth analysis found that PD patients had greater tracing error, and that performance in both groups decreased from Path 4 to any of the paths with deleted segments. The decrease in performance from Path 4 to Path 7 was greater in the PD group. The only factor affecting velocity in some cases was the hand used for tracing. The pattern of tracing error performance in the sawtooth patterns is similar to that seen in the straight line: all subjects perform more poorly on the path with deleted segments, PD patients perform more poorly than controls, and the decrease in performance from the complete path to the path with deleted segments is more severe in the PD group. However, velocity decreased from Path 1 to Path 3 but did not change significantly from Path 4 to any sawtooth path

with deleted segments.

A second analysis of tracing performance in sawtooth paths compared performance measures of the entire sweep, as opposed to measures only of the missing segment performance. This approach allowed all four sawtooth paths to be included in a single analysis. Results are presented in Table 9 and Figures 12 and 13. In sweep 1, area error in the PD group was 23.9, 28.5, 28.9, and 49.7, and in the controls, 10.7, 12.8, 15.9, and 20.54 for Paths 4-7 respectively. Group, Path, Hand, and Group X Path effects were significant. The Group X Path interaction was due to a significant increase in area error in the PD group from Paths 6-7 ($p < .05$) that was not present in the control group. In addition, patients' performance was generally poorer than controls', and overall performance was better in the earlier paths and with the right hand. In sweep 2, mean area error in the PD group was 26.3, 33.7, 30.2, and 47.5, and in the controls, 12.5, 16.19, 16.36, and 29.5 in Paths 4-7 respectively. Group, Path, Hand, and Group X Path X Hand effects were significant. Area error increased significantly in the PD group from Path 6 to 7 in both hands ($p < .05$), but in the control group a significant increase was seen only in the right hand. Overall, patients performed more poorly than controls, performance was better on the early than the later forms and with the right vs. the left hand. As with the comparison of Paths 1 and 3 and Paths 4 and 7, a significant Group X Path interaction was seen again in this case. The emergence of this consistent pattern of results argues for a specific deficit in the PD patients for performance on paths with deleted segments.

The analysis of number of digitized points reveals no significant effects in sweep 1 but significant Path and Hand effects in sweep 2.

These indicate slower tracing with left than the right hand, which was also seen in the first type of analysis, and a tendency for subjects to trace faster in the later forms, which was not significant in the first type of analysis, but barely reaches significance here.

Relation of Tracing Performance to Cognitive Tests and Demographic Variables

Correlations were calculated to assess the relationship between tracing performance measures and the age and education of subjects and their performance on the MMS and Rosen Drawing Test. These calculations were performed separately for each form, group and sweep, but collapsed over left and right hand (Tables 10 and 11). In the control group, tracing performance was correlated with many variables, and no clear pattern of relationship between tracing performance and particular cognitive variables could be discerned. In the parkinsonian group, a similar pattern prevails until performance in Paths 6 and 7, the more difficult paths, was analyzed. In these paths, measures of tracing performance correlate almost exclusively and in every instance with the construction segment of the MMS, and with performance on the Rosen Drawing Test. In contrast, controls' performance on these paths correlate with other subtests of the MMS that measure general intelligence, and sometimes with the age, level of education, and depression ratings of the patients, but never with the construction segment of the MMS or the Rosen Drawing Test.

Relation of Tracing Performance in PD Patients to Measures of PD Severity

Correlations were calculated between measures of PD severity and measures of tracing performance and are presented in Table 12. In the straight line paths (Paths 1 and 3), parkinsonian symptoms correlate primarily with velocity measures, while, in the sawtooth paths, correlations are also seen with some of the error measures. It was not possible to isolate a clear pattern of interrelationships between specific tracing measures and specific measures of PD symptom severity.

DISCUSSION

Tracing Task Performance

The data in this study present a pattern which confirms and expands upon findings in previous studies. The major findings in this study which will be discussed more fully in the following sections are: (1) The increasing errors of the parkinsonians on paths with deleted segments. Although patients performed more poorly than controls even on complete paths, their decline in performance was greater than controls' on paths with missing segments. (2) The close relation of loss-of-form errors (errors on paths with missing segments) in the PD group to performance on the Rosen Drawing Test and the construction items of the MMS. This relation did not exist in the control group. (3) The additional effect of parkinsonian symptoms on performance of the PD patients, especially on velocity of tracing.

Parkinsonian patient performance on paths with deleted forms. Repeated measure ANOVA's revealed a consistent pattern of parkinsonian patient performance as compared to controls. First, as would be expected, patient performance was generally worse than control group performance. Also, both patients and controls performed more poorly on paths with deleted segments than on complete paths. Significant Group X Path interaction effects are key evidence in demonstrating that despite general lowered performance by PD patients, their performance declined even more than the controls when they traced patterns with missing segments. This interaction effect was present in three important contrasts: comparison of straight line, Paths 1 and 3, comparisons of Path 4 vs. Path 7 for the performance measures of the

deleted segment only, and the comparison of performance measures over the complete path on Paths 4 through 7.

Relation of tracing performance to other variables. The consistent correlation of parkinsonians' tracing performance measures, particularly those in Paths 6 and 7, with performance on construction tasks, suggests that the tracing task taps similar cognitive processes as construction tasks. In contrast, tracing performance in controls correlated with indices of general intelligence and almost never with the construction tasks. This suggests that while poorer performance in controls was related to intelligence factors, in patients it was related to a general perceptual motor deficit that affects both tracing and construction performance. Another factor affecting performance exclusively in PD patients is the severity of the PD symptoms. In the pilot study (Stern et al., in press), the effect of PD motor symptoms was implied, but no direct correlations were noted. In this study, it is clearly seen through significant correlations that PD symptom severity affects performance. However, it was not possible to isolate particular symptoms that affect specific aspects of performance; correlations were wide-ranging. The most clearly seen relation is between tracing time or velocity and PD severity symptoms.

The most stringent general interpretation of findings in this study is that the motor symptoms of PD accounted for the general lower performance of the patients while the perceptual motor deficit of PD accounted for the additional decline in performance on paths with deleted segments. However, the generally lower performance of the PD patients could, in part, also reflect the patients' perceptual motor deficits.

Perceptual Motor Coordination

The relation between patients' errors on paths with deleted segments and performance on the construction tasks suggests that those tasks are mediated by a similar process that might be best described as perceptual motor deficit. This deficit could represent an inability to coordinate perception with certain motor functions in order to generate movements based on an internal concept of space (or a motor plan). Perception refers to the ability to discriminate and integrate external sensory information, in this case visual. The motor functions consist of a series of movements which are sequentially performed. The coordination of these two activities in order to fulfill a motor plan may represent a higher order of motor control.

The tracing task in this study required both sequential and predictive movements. Specific movements had to be accomplished in the absence of visual feedback, forcing the subject to generate predictive movements using only internal guidance. Flowers (1978b) and others have also demonstrated this deficit in the generation of movement in the absence of visual guidance in both PD and non-human primates with lesions in the basal ganglia (Bowen, 1969; Cooke et al., 1978; Hore et al., 1977; Caan & Stein, 1979). The tracing task also demands that the movements be generated in their proper sequence, beginning and terminating at their proper time. As noted above, deficits in the sequencing of movement have often been demonstrated in PD.

In the construction task, the subjects again were required to generate sequences of movements that satisfied the spatial demands set by the design to be copied. In this case some visual guidance was present in the form of the designs. However, the designs were often

sufficiently complex as to challenge the subjects' ability to organize the movements necessary to reproduce them. As noted earlier, when some external organization for construction is supplied, patients can successfully perform construction tasks. Other investigtors have demonstrated construction deficits in PD which may also reflect this inability to organize or sequence the movements necessary for successful performance (Joubert & Barbeau, 1969; Botez & Barbeau, 1975). Other tracking studies demonstrate that even when patients are informed that a target is moving in a specific sequence they cannot use this information to improve their tracking accuracy (Flowers, 1978b; Marsden, 1982).

Efference Copy, Corollary Discharge, and Perceptual Motor Coordination

According to Marsden (1982), the basal ganglia may be responsible for the automatic execution of learned motor plans; that is, they take part in sequencing the motor programs needed to accomplish a motor plan. The mechanism through which the basal ganglia may aid in this activity is unknown, but hypothetical systems that could serve this purpose have been described.

One hypothetical process that may underlie perceptual motor coordination, and, additionally, represent the functional role of the basal ganglia-cortical systems in behavior, is corollary discharge or efference copy. This concept has been best described in terms of its contribution to movement, and to the type of spatial localization required for delayed response and similar tasks.

Teuber (1966, 1972, 1976) hypothesized that, in voluntary movement, efferent extrapyramidal motor commands flow to both motor effectors and to central receptors. This centrally monitored information helps the

system prepare for the consequences of concurrent or impending actions. For example, sensory systems could be prepared for input that would be produced as a consequence of a motor movement. Corollary discharge refers to the concurrent discharge of efferent signals centrally. Another term that has been used to describe this type of system is "efference copy," which refers to a record of efferent motor commands which is retained centrally (Angel, 1976). Similar systems have been proposed by many investigators (Sperry, 1950; von Holst, 1954).

The existence of a corollary discharge system has been studied in many ways. When the eye makes a saccadic movement, the visual world is not perceived as jumping. However, when the occular muscles are paralyzed and the eye is moved manually, the visual field does move (von Helmholtz, 1925). This suggests that the sensory system is normally prepared to compensate for the consequences of motor movement.

Another study made use of step-tracking task, in which subjects moved a marker controlled by a joystick to follow a target line which moved rapidly from the center to the right or left of an oscilloscope screen. When subjects were induced into making errors on this task, they corrected them without any external feedback about their occurrence. Thus, the motor commands for the movement must have been monitored centrally and corrected (Angel, 1976). This error correction, which could be based on efference copy, was slowed in PD (Angel, Alston & Higgins, 1970; Angel, Alston & Garland, 1971). Single unit recordings from cells in the basal ganglia of monkeys during tracking studies also revealed that the majority of cells were not involved in the initiation of movement, but were responsive during corrective movement (Aldridge,

Anderson & Murphy, 1980; Anderson, Aldridge & Murphy, 1979; Dolbakayan, Hernandez-Mesa & Bures, 1977). Both of these studies implicate the basal ganglia in the process of corollary discharge. As noted earlier, the basal ganglia are involved in neuroanatomical systems that are functionally arranged as feedback systems (Webster, 1975). Possibly, they could mediate a system of this nature.

Prism-adaptation provides conditions for the operation of corollary discharge, and demonstrates its role in spatial perception and perceptual motor coordination. In prism-adaptation studies, the subject wears prisms over his eyes which displace the visual field in some regular fashion. Initially the subject errs in his directed movements by reaching toward his displaced view of an object. Over time, the subject compensates for the displacement and makes correct reaching movements. This adaptation occurs even if no direct feedback about the accuracy of his movements is received. For example, monkeys with dorsal rhizotomies, which eliminated kinesthetic feedback from the arms, showed prism adaptation in a task where they reached for an object even though they were not allowed to see their arms or check the accuracy of their movement (Bossom & Ommaya, 1968). This suggests that a central mechanism such as corollary discharge is monitoring movements and their consequences, yielding information for prism adaptation. In this case, eye movements and their visual consequences are probably being monitored. The role of corollary discharge in prism adaptation is elucidated by two other findings: first, subjects whose limbs are passively moved as opposed to initiating their own limb movements cannot prism-adapt, and second, subjects must be allowed to make movements that correspond to their intended movements in order to prism-adapt (Held &

Freedman, 1963). These findings demonstrate the importance of the subject's generating his own efferent motor commands that can be monitored via corollary discharge, and of those commands actually representing the movement that is carried out. The efferent information is monitored centrally, and is correlated with an accompanying expectation of the sensory consequence of the movements. When a subject is wearing prisms, this expectancy is not met. The lack of correlation between the expected and actual consequences of the movement is sufficient information to allow the system to adapt. In effect, the sensory feedback leads to modification of later motor movement. Successful prism-adaptation requires intact basal ganglia. Adaptation failed to occur in monkeys with basal ganglia lesions (Bossom, 1965; Bossom & Ommaya, 1968), and in the one reported case of a person with a unilateral caudate lesion (Potegal, 1972).

Prism-adaptation studies demonstrate the role of the basal ganglia not only in corollary discharge, but in the related process of perceptual motor coordination. The adaptation process involves coordinating and integrating motor and sensory information to form a new perceptual representation of the spatial environment. This spatial representation is therefore not just sensory but involves a motor component as well. Therefore, basal ganglia dysfunction may affect not only movement, but internal spatial representation as well. The basal ganglia could contribute to perceptual motor coordination as part of a corollary discharge system, or actually be the locus for the correlation of sensory and motor information. Alternatively, since damage to the basal ganglia can affect movement, the process of perceptual motor coordination could be disrupted because of the difficulty of correlating the irregular

motor information with sensory information. In either case, the intact basal ganglia would be needed as part of the perceptual motor system to guide behavior when no external guidance is available.

Teuber suggested that many of the deficits noted after lesions to the basal ganglia-frontal lobe system may also be viewed as a failure of perceptual motor coordination (Teuber & Proctor, 1964; Teuber, 1966, 1972, 1976). For example, as noted earlier, delayed response tasks demand accurate spatial representation of the environment relative to the animal's own position in the absence of exteroceptive stimuli. Potegal (1972) similarly suggested that the caudate serves as part of an "egocentric orientation" system in which movements of the head and eyes are encoded for spatial localization. Internal representation of any point in space would consist of the motor programs which would turn the head and eyes to that point, given the position of the torso.

The important contribution of the basal ganglia for perceptual motor coordination may be seen in cognitive tasks as well. Even non-motor tasks requiring the evaluation and subsequent modificiation of behavior based on expected outcome may be affected (Teuber, 1972, 1976). For example, in human neuropsychological tasks such as the Wisconsin Card Sort, subject's behavior must be guided by an analysis of past performance that must be performed without any external guidance. When sorting behavior is not rewarded, performance must be evaluated and then changed in order to be correct. Patients with diseases of the basal ganglia such as PD, as well as those with frontal lobe lesions, perform poorly on this task (Bowen, 1976).

Another important role of perceptual motor coordination is its contribution to the intellectual processes involved in spatial

representation of the environment. Defective spatial representation can affect performance on tasks such as construction of geometric forms. It appears, then, that defective participation of the basal ganglia in the process of perceptual motor coordination could account for both the predictive and sequential movement deficits seen in the tracing task and the deficits noted on the construction tasks.

While the construct of a deficit in perceptual motor coordination in PD is hypothetical, it does have predictive utility. In one study, it was hypothesized that this deficit, if present, should affect not only performance on visuomotor tasks, but also on tasks in other sensory modalities. PD patients and controls were given a test of tactile perception that had no visual component (Mayeux, Tomaino, Rosen, Stern & Gerstman, in press). Subjects traced the rough, raised outline of various geometric shapes with their finger and then were asked to select each shape from a visual array of assorted shapes. In some cases subjects actively generated their own tracing movements, while in others their finger was passively guided along the outline of the shape. In the latter situation, parkinsonians and controls performed comparably. In the former situation patients' performance was the same as in the passive guidance condition while the controls' performance was better. Thus, the PD patients did not benefit from generating their own exploratory movements while the controls did. In a perceptual motor coordination model, the motor commands which are required for self generated motor movements, would be correlated with the resulting tactile sensation in order to obtain a more accurate perception of the explored shape. In this study, the identical performance of the PD patients under self-guided or passive conditions suggests a deficit in the perceptual

motor system which consequently gives rise to an inaccurate perception. This model, therefore, seems to be a useful one for explaining intellectual deficits in patients with PD.

Conclusion

While the basal ganglia have long been associated with movement, investigations of the consequences of manipulations of the basal ganglia suggest that they have a role in cognitive processes. The cognitive tasks used in investigations often have a motor component, but they also demand that behavior be internally monitored and guided in the absence of external cues. This is necessary for successful performance on delayed response tasks in the rat, horizontal orientation task in humans, as well as on tracing and construction tasks used in this study. The present study suggests that the basal ganglia are important for perceptual motor coordination, a process that is crucial for perception and representation of the spatial environment. Thus, deficits in perceptual motor coordination can affect not only tasks with a distinct motor component, but other intellectual tasks as well.

In this study, performance of patients with PD on a tracing task related to their performance on construction tasks. The patients performed more poorly than controls on the tracing task when external guidance for tracing was removed, and patients' but not controls' performance on this task was related to their construction ability. These findings in PD may be representative of the cognitive processes which the basal ganglia play a role in mediating.

Anatomically, perceptual motor coordination would depend on a feedback system which correlates and integrates motor and sensory

information. Available evidence suggests that the basal ganglia could play an important role in this system.

Plans for Future Studies

The model of a deficit in perceptual motor coordination being a consequence of basal ganglia dysfunction is important and useful in guiding the investigation of intellectual deficits in patients with diseases of the basal ganglia.

Based on the findings in the present study, several investigations are being planned. One study directly addresses the issue of basal ganglia involvement in the perceptual motor deficits that have been described. While it is logical to assume that basal ganglia dysfunction underlies these deficits in PD, this has not been directly tested. In the planned study various C-T measures of basal ganglia size will be obtained in PD and Alzheimer's disease patients. It is anticipated that some of these measures will correlate with perceptual motor performance in the PD patients.

In a related study, the theme of the similarity of frontal lobe lesioned and parkinsonian patients' perceptual motor deficits will be investigated by comparing these two groups on a battery of perceptual motor tasks. In addition, patients with parietal lobe lesions and Alzheimer's disease will be included to investigate the differences in the patterns of perceptual motor performance in these groups.

Finally, a prism-adaptation study comparing parkinsonians and normal controls is being carried out to test the hypothesis that the patients' prism-adaptation will be defective in comparison to controls.

These anticipated studies emphasize the heuristic value of the model generated by the present study for creating testable hypotheses and predictions.

APPENDIX A

Individual Test Scores and Handedness Information

Subject	Handedness	Beck Depression Inventory	Modified Mini-Mental Status	Rosen Drawing Test
Parkinson's Disease				
1	R	.	55	14
3	R	3	51	9
4	R	6	42	9
6	L	10	49	8
7	R	5	52	9
8	R	10	47	13
11	R	10	41	13
13	R	0	46	10
14	R	10	53	13
28	R	1	54	13
29	R	11	53	8
30	ambidex	4	56	11
31	R	12	54	9
32	R	27	43	9
33	R	2	54	15
34	R	16	57	14
Control				
15	R	1	56	12
16	R	5	53	14
17	R	0	54	15
18	R	6	55	12
20	R	3	49	9
21	R	.	54	12
22	R	1	55	14
23	R	6	52	13
24	R	8	51	12

APPENDIX B

Columbia University Parkinson Rating Scale

DEFINITION OF 0-4 SCALE

1. Mentation:
   0 = normal
   1 = impaired cognition, e.g. difficulty with arithmetic or abstraction. Normal memory.
   2 = mild to moderate impairment of cognition and memory but preserved orientation
   3 = severe impairment of cognition, memory and orientation
   4 = global dementia (impairment in all intellectual functions)

2. Motivation/Initiative
   0 = normal
   1 = less assertive than usual; more passive
   2 = loss of initiative or disinterest in elective (non-routine) activities
   3 = loss of initiative or disinterest in day to day (routine) activities
   4 = withdrawn, complete loss of motivation

3. Depression: (sad, blue, down-in-dumps, exclude reactive)
   0 = not present
   1 = periods of sadness or guilt, never sustained for days or weeks
   2 = sustained depression (1 week or more)
   3 = sustained depression with vegetative symptoms (insomnia, anorexia, weight loss, loss of interest)
   4 = sustained depression with vegetative symptoms and suicidal thoughts or intent

4. Swallowing:
   0 = normal
   1 = rare choking
   2 = occasional choking
   3 = requires soft food
   4 = requires NG tube

5. Cutting food:
   0 = normal
   1 = somewhat slow and clumsy, but no help needed
   2 = can cut most foods, although clumsy and slow
   3 = food must be cut by someone, but can still feed slowly
   4 = needs to be fed

6. Hygiene:
   0 = normal
   1 = somewhat slow, but can manage alone
   2 = unable to shower alone; very slow in hygienic care
   3 = requires assistance for washing, brushing teeth, combing hair
   4 = foley catheter

7. Dressing:
   0 = normal
   1 = slow, but no help needed
   2 = occasional assistance with buttoning, gettings arms in sleeves
   3 = considerable help required, but can do some things alone
   4 = helpless

8. Turning in Bed:
   0 = normal
   1 = slow, but not help needed
   2 = can turn alone, but with great difficulty
   3 = can initiate, but not turn by himself
   4 = helpless

9. Sialorrhea:
   0 = none
   1 = slight but definite excess of saliva in pharynx; patient may be unaware thereof, no drooling
   2 = moderately excessive saliva with minimal drooling, if any
   3 = marked excess of saliva with some drooling
   4 = marked drooling, requires special measures

10. Tremor:
   0 = absent
   1 = slight and infrequently present
   2 = moderate in amplitude but only intermittently present
   3 = moderate and present most of the time
   4 = marked in amplitude and present most of the time

11. Rigidity: (Judged on passive movement of major joints with patient relaxed in sitting position. Cogwheeling to be ignored.)
   0 = absent
   1 = slight or detectable only when activated by mirror or other movements
   2 = mild to moderate
   3 = marked, but full range of motion easily achieved
   4 = severe, range of motion achieved with difficulty

12. Speech Disorder:
   0 = none
   1 = slight loss of expression, diction and/or volume
   2 = monotone, slurred but understandable
   3 = marked impairment, difficult to understand
   4 = unintelligible

13. Facial Expression:
   0 = normal
   1 = minimal hypomimia, could be normal "Poker Face"
   2 = slight but definitely abnormal diminution of facial expression
   3 = moderate hypomimia
   4 = masked or fixed facies with severe or complete loss of facial expression

14. Foot Agility: (Patient taps heel on ground in rapid succession.)
   0 = normal
   1 = somewhat slow rapid succession movements
   2 = definitely slow rapid succession movements
   3 = very slow rapid succession movements
   4 = can barely move feet

15. Finger Dexterity: (Patient taps thumb with forefinger in rapid succession. Then he taps thumb with each finger in rapid succession.)
   0 = normal
   1 = slightly slow
   2 = slow
   3 = markedly slow
   4 = unable

16. Diadokokinesis: (Patient opens and closes hands, pats thigh and pronates and supinates hands in rapid succession.)
   0 = normal
   1 = slightly slow
   2 = slow
   3 = markedly slow
   4 = unable

17. Bradykinesia: (Combining both slowness and poverty of movement in general.)
0 = none
1 = minimal slowness giving movement a deliberate character; could be normal for some persons
2 = mild degree of slowness and poverty of movement which is definately abnormal
3 = moderate slowness with occasional hesitation on initiating movement and arrests of on-going movement
4 = marked slowness and poverty of movement, with frequent freezing and long delays in initiating movement

18. Arising from Chair: (Straight-back wood or metal chair)
0 = normal
1 = slow
2 = pushes self up from arms or seat
3 = tends to fall back and may have to try several times but can get up without he
4 = unable to arise without help

19. Posture:
0 = normal erect
1 = not quite erect, slightly stooped posture; could be normal for older person
2 = moderate simian posture, definitely abnormal
3 = marked simian posture with kyphosis
4 = severe flexion with extreme abnormality of posture

20. Postural Stability: (Judged by response to sudden posterior displacement produced by push on sternum while patient standing as erect as possib with eyes closed.)
0 = normal
1 = retropulsion but recovers unaided
2 = absence of postural response; would fall if not caught by examiner
3 = very unstable, tends to fall spontaneously on the Romberg test
4 = unable to stand without assistance

21. Gait Disturbance:
0 = freely ambulatory, good stepping, turns readily
1 = walks slowly, may shuffle with short steps but no festination or propulsion
2 = walks with great difficulty with festination, short steps, freezing and propulsion but requires little or no assistance
3 = severe disturbance of gait requiring assistance
4 = cannot walk at all, even with help

22. Dyskinesia:
0 = none
1 = infrequent, mild, low amplitude dyskinesia
2 = occasional (daily) low amplitude dyskinesia
3 = frequent sustained, medium amplitude dyskinesia
4 = severe, persistant, high amplitude dyskinesia

23. Psychosis:
0 = not present
1 = rare illusionary phenomena, insight retained
2 = frequent illusions, rare hallucinations that impair daily activities
3 = frequent hallucinations/delusion without insight impairing daily activities
4 = persistant hallucinations/delusion unable to care for self

APPENDIX C

Parkinson's Disease Evaluation Scores and Duration of Illness

Subject	Total PDE*	RUE+ Tremor	LUE+ Tremor	Total Tremor	RUE Rigidity	LUE Rigidity	Total Rigidity	Brady-Kinesia	Postural Stability	Duration of Illness
1	12.5	.5	0	.5	1	.5	4	1	.5	3
3	19	.5	.5	1	1	1	4	0	1	9
4	62	1	0	1.5	2	2	11.5	6.5	6.5	7
6	49	2	2	7	1	1	6.5	2	2	10
7	28	.5	.5	2	1	2	6.5	1.5	1	2
11	32	.5	.5	2	0	0	1	2	2	9
13	49.5	0	0	0	1	.5	2.5	2	3	16
14	23	0	1	1	1	1	3	1	1	4
28	3.5	0	0	0	.5	0	0	0	0	18
29	38	0	0	0	1	.5	3.5	2	2	6
30	30	0	0	0	1.5	1	6	1	0	5
31	29	0	0	0	0	0	0	2	2	5
32	46	2	0	4	1	1	7	3	2	16
33	14	.5	0	.5	1.5	0	2.5	.5	0	2.5
34	30	1	1	4	1.5	.5	5	3	1.5	3.5

*Total does not include first 3 items of the PDE.
+RUE = right upper extremity; LUE = left upper extremity.

APPENDIX F

Technical Discussion of Digitizing Equipment Including Programs and Documentation

The Scientific Accessories Corporation Graph Pen GPG-25H outputs a continuous stream of data in ASCII at 9600 Baud. Data was collected through the modem port of the ComStor I recorder with switches set on 9600 Baud for both terminal and modern ports. Recording in the ComStor is initiated and terminated by a write switch and files saved in this matter are automatically catalogued and sequentially labeled. Each two sweep trial of each path for each hand was saved as an individual file. Data for one subject (12 files) typically filled a disk.

Data analysis began by interfacing the recorder to a DecWriter IV Printer terminal and cataloguing each disk to determine the number of characters in each file, an important piece of information for the data analysis programs. The recorder was then interfaced through its modem port at 1200 Baud to the following computer setup: a MINC-11 computer with 2 RX-02 disk drives, a VT 125 terminal, the DecWriter IV for printout, and and a DecWriter Graphics Printer for graphic printout. Transfer of data and its analysis was accomplished via programs written in MINC BASIC.

The 10 programs presented here are chained together to sequentially perform the transfer and analysis functions for a maximum of 10 files at a time. This configuration was typically run overnight since data transfer from ComStor to MINC took several hours. Other versions of these programs were also implemented in a menu driven format to enable a

particular analysis, or an analysis of a particular segment of tracing performance. The MINC Command EXTRA-SPACE must be in effect before most of these programs are used. The printouts here are of working copies that may contain slight modifications made for different purposes during their use. These programs should be carefully checked before they are used.

TRANS 1 initiates the transfer of data from the ComStor to the MINC and stores the data. The program stores the data in two virtual array files, one for X coordinates and one for Y, appending "X" and "Y" to the input file name. After inputting information about the files to be transferred (lines 80-95), the program initiates a loop to receive the data from the ComStor. Since the ComStor sends data too quickly for this loop to handle, a switch on the recorder that causes it to pause momentarily after each coordinate is sent must be enabled. After up to 10 files are transferred and stored, the program chains to TRANS 2.

TRANS 2 calculates velocity at each digitized point. First, the velocity to and from a particular point is calculated, then the values are averaged to calculate the velocity at the point itself. Velocity values for each point are saved in a file with the same name as the X and Y coordinate files, but with a V appended.

TRANS 3 simply finds and stores the point at which the second sweep begins in the tracing of a path.

TRANS 4 accesses the velocity files created in TRANS 2 and calculates summary statistics for the tracing velocity and digitized points in both sweeps of a traced path.

TRANS 5 calculates the area error measures for the straight line paths. In this case these calculations are performed for three segments

in each sweep. The basic paradigm will be explained in the discussion of TRANS A.

TRANS 6 calculates area error for entire sweeps of a sawtooth path. The error calculation paradigm follows the tracing movement until it touches or crosses the path. It then calculates the area between the tracing movement and the path. This is done by treating the tracing movement coordinates and the intersected coordinates of the path as if they outline an irregular polygon and calculating the area of this polygon. After this area is calculated, tracing movement is once again followed until the path is crossed again or until the end of the sweep. Thus, both an area measure and the number of times the path is crossed is calculated.

TRANS 7 is similar to TRANS 6 except that it yields three area error measures for each sweep, the second being the area error for the missing segment of the path, and the first and third the area error for surrounding portions. Thus, Paths 5-7 are each treated differently in this program. Path 4 was run through a similar program three times, to analyze it as if it were Paths 5, 6 and 7 to derive area error measures for portions of Path 4 similar to those missing in later paths.

TRANS 8 graphs and prints the tracing movement and velocity of straight line paths, and TRANS 9 does the same for sawtooth paths.

TRANS A is similar to TRANS 5. It calculates area error for straight line paths (Paths 1-3). This program also uses the strategy of defining and finding the area of irregular polygons consisting of tracing movement coordinates and the path itself, but this strategy is implemented in a simpler manner.

```
TRANS1    01-DEC-82  03:05:55

10 REM TRANS1
20 PRINT 'This program transfers a series of files from comstor to disk.'
30 PRINT 'Make sure the proper current comstor file is set.' \ PRINT
40 PRINT 'How many files will you transfer' \ INPUT F
50 DIM P(10)
60 COMMON F$(10),F,Z(10)
70 FOR I=1 TO F
80 PRINT 'Input name of file ';I;' as in redbook'; \ INPUT F$(I)
90 PRINT 'Input number of characters in file ';I; \ INPUT P
95 PRINT 'Input form number (1,3-7)'; \ INPUT Z(I)
100 P(I)=INT((P-1)/9)
110 IF P(I)>4000 THEN PRINT 'MODIFY DIMENSION STATEMENTS BEFORE CONTINUING'
120 NEXT I
130 G$=CHR$(17)
140 FOR A=3 TO F
150 OPEN 'SY1:'+F$(A)+'X' AS FILE #1 \ DIM #1,X(4000)=5
160 OPEN 'SY1:'+F$(A)+'Y' AS FILE #2 \ DIM #2,Y(4000)=5
170 C=0
180 B=1
190 REM INITIATING RETRIEVAL PROCESS
200 CIN(,S$,1,,,1)
210 COUT(,G$)
220 FOR I=1 TO P(A)
230 CIN('RETRIEVE',S$)
240 X(I)=VAL(SEG$(S$,1,4))/100
250 Y(I)=ABS((VAL(SEG$(S$,5,8))/100)-90)
260 C=C+1
270 PRINT F$(A);C,X(I);Y(I)
280 NEXT I
290 X(0)=P(A)
300 CLOSE
310 NEXT A
320 CHAIN 'TRANS2'
330 END
```

```
TRANS2    01-DEC-82  03:06:49

10 REM TRANS2
20 COMMON F$(10),F,Z(10)
47 FOR B=1 TO F
50 OPEN 'SY1:'+F$(B)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
60 OPEN 'SY1:'+F$(B)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
70 OPEN 'SY0:WORK' AS FILE #5 \ DIM #5,D(4000)=5
80 OPEN 'SY1:'+F$(B)+'V' FOR OUTPUT AS FILE #4 \ DIM #4,V(4000)=5
90 OPEN 'LP:' FOR OUTPUT AS FILE 1
95 PRINT 'VELOCITY CALCULATION: ';F$(B)
100 PRINT #1,""
110 PRINT #1,"-------------------------------------------------------------"
120 PRINT #1,"SUMMARY: VELOCITY CALCULATONS FOR FILE ";F$(B)
130 PRINT #1,""
140 T=0 \ REM TOTAL OF POINT VELOCITIES
150 M=0 \ REM TOTAL OF SQUARED VELOCITIES
160 DEF FNV(X,A,Y,B)=SQR((X-A)^2+(Y-B)^2)
170 N=1
180 N1=X(0)
260 V(0)=N
270 V(1)=N1
280 N4=N+1
290 N5=N1
300 GOSUB 370
310 CLOSE
320 KILL 'WORK'
330 PRINT  \ PRINT
340 NEXT B
360 GO TO 560
370 REM ************************************************
380 REM         VELOCITY CALCULATION ROUTINE
390 REM ************************************************
400 FOR I=N4 TO N5
410 D(I)=FNV(X(I-1),X(I),Y(I-1),Y(I))
420 NEXT I
```

```
430 FOR I=N4 TO N5-1
440 V(I)=INT(((D(I)+D(I+1))/2)*1000+.5)/1000
445 IF V(I)>5 THEN PRINT #1,'ERROR ';I
450 T=T+V(I)
460 M=M+V(I)^2
470 NEXT I
480 PRINT #1,'A TOTAL OF';(N5-N4+2);'POINTS WERE STUDIED'
490 PRINT #1,'RANGING FROM POINT';N;'TO POINT';N1
500 PRINT #1,'YEILDING';(N5-N4);'POINT VELOCITIES'
510 PRINT #1,'MEAN VELOCITY= ';(T/(N5-N4))
520 A=(M-(N5-N4)*(T/(N5-N4))^2)/(N5-N4)
530 PRINT #1,'VARIANCE= ';A
540 PRINT #1,'STANDARD DEVIATION= ';SQR(A)
550 RETURN
560 CHAIN 'TRANS3'
570 END
```

```
TRANS3    01-DEC-82  03:07:56

10 REM TRANS3
40 COMMON F$(10),F,Z(10)
90 FOR T=1 TO F
95 PRINT 'TURNING POINT CALCULATION: ';F$(T)
100 OPEN 'SY1:'+F$(T)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
110 OPEN 'SY1:'+F$(T)+'Y' AS FILE #1 \ DIM #1,Y(4000)=5
120 I=X(0)
130 FOR M=1 TO I
140 IF X(M)>70 THEN  GO TO 160
150 NEXT M
160 FOR L=M TO I
170 IF X(L)<X(L-1) THEN IF X(L)>X(L+1) THEN IF X(L)>X(L+2) THEN  GO TO 200
180 NEXT L
190 IF L<>I THEN  GO TO 200
192 CLOSE #2
194 OPEN 'LP:' FOR OUTPUT AS FILE 1
196 PRINT #1,'NO TURNING POINT, FILE ';F$(T)
198 Y(0)=I/2
199 GO TO 220
200 Y(0)=(L-1)
220 CLOSE
230 NEXT T
270 CHAIN 'TRANS4'
280 END
```

```
TRANS4    01-DEC-82  03:08:34

10 REM TRANS4
50 COMMON F$(10),F,Z(10)
90 FOR L=1 TO F
95 PRINT 'SWEEP VELOCITY: ';F$(L)
100 OPEN 'SY1:'+F$(L)+'V' FOR INPUT AS FILE #4 \ DIM #4,V(4000)
110 OPEN 'SY1:'+F$(L)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)
120 OPEN 'SY1:'+F$(L)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)
130 OPEN 'LP:' FOR OUTPUT AS FILE 1
140 PRINT #1,""
150 PRINT #1,"-------------------------------------------------------------"
160 PRINT #1,"SWEEP VELOCITY STATISTICS FOR FILE ";F$(L)
170 PRINT #1,""
180 N=Z
190 N1=Y(0)
200 N2=Y(0)
210 N3=X(0)-1
220 PRINT #1,'SWEEP 1:'
230 N4=N \ N5=N1 \ GOSUB 320
240 PRINT #1,'SWEEP 2:'
250 N4=N2 \ N5=N3 \ GOSUB 320
260 CLOSE
270 NEXT L
280 GO TO 470
290 REM ************************************************
300 REM         VELOCITY CALCULATION ROUTINE
310 REM ************************************************
320 T=0 \ REM TOTAL OF POINT VELOCITIES
330 M=0 \ REM TOTAL OF SQUARED VELOCITIES
340 FOR I=N4 TO N5
350 T=T+V(I)
360 M=M+V(I)^2
370 NEXT I
380 R=N5-N4+2
```

```
390 PRINT #1,'A TOTAL OF';(R);'POINT VELOCITIES WERE STUDIED'
400 PRINT #1,'RANGING FROM POINT';N4;'TO POINT';N5
410 PRINT #1,'MEAN VELOCITY= ';(T/R)
420 A=(M-(R)*(T/R)^2)/R
430 PRINT #1,'VARIANCE= ';A
440 PRINT #1,'STANDARD DEVIATION= ';SQR(A)
450 PRINT #1,''
460 RETURN
470 CHAIN 'TRANS5'
480 END
```

```
TRANS5    01-DEC-82  03:09:41

10 REM TRANS5
20 COMMON F$(10),F,Z(10)
30 F1=F
80 FOR Z=1 TO F1
85 IF Z(Z)>3 THEN  GO TO 1240
87 PRINT 'SEGMENTAL AREA: ';F$(Z)
90 OPEN 'SY1:'+F$(Z)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
100 OPEN 'SY1:'+F$(Z)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
110 OPEN 'SY1:'+F$(Z)+'V' FOR INPUT AS FILE #4 \ DIM #4,V(4000)=5
120 OPEN 'LP:' FOR OUTPUT AS FILE 1
130 DIM D(500)
140 REM ****SET ENDPOINTS
150 PRINT #1,""
160 PRINT #1,"------------------------------------------------------------"
170 PRINT #1,"ERROR STATISTICS FOR FILE ";F$(Z)
180 PRINT #1,""
190 N=1
200 N1=Y(0)
210 N2=Y(0)
220 N3=X(0)
230 FOR I=N TO N1
240 IF X(I)>40.59 THEN N7=(I-1) \ GO TO 260
250 NEXT I
260 FOR I=N7 TO N1
270 IF X(I)>55.59 THEN N8=(I-1) \ GO TO 290
280 NEXT I
290 FOR I=N2 TO N3
300 IF X(I)<55.59 THEN N9=(I-1) \ GO TO 320
310 NEXT I
320 FOR I=N9 TO N3
330 IF X(I)<40.59 THEN 01=(I-1) \ GO TO 350
340 NEXT I
350 N5=N \ N6=N7 \ GOSUB 420
```

```
360 N5=N7 \ N6=N8 \ GOSUB 420
370 N5=N8 \ N6=N1 \ GOSUB 420
380 N5=N2 \ N6=N9 \ GOSUB 420
390 N5=N9 \ N6=01 \ GOSUB 420
400 N5=01 \ N6=N3 \ GOSUB 420
410 GO TO 1220
420 E=0 \ REM DIFFERENCE SCORES
430 G=0 \ REM AREA TOTAL
440 H=0 \ REM POLYGON COUNTER
450 F=0 \ REM SET FLAG
460 C=1 \ REM COUNTER FOR DIFFERENCE ARRAY
470 READ X1,Y1,X2,Y2
480 DATA 23.57,21.42,72.61,21.25
490 DEF FNV(X)=Y1+(((Y2-Y1)*(X-X1))/(X2-X1))
500 Z1=X(N5)
510 Z2=FNV(X(N5))
520 B=N5 \ REM POLYGON ENDPOINT
530 REM **********************************
540 REM       START MAIN LOOP
550 REM **********************************
560 FOR I=N5 TO N6
570 D(C)=Y(I)-FNV(X(I))
580 A=SGN(D(C))
590 IF F=0 THEN  GO TO 660
600 IF F=1 THEN  GO TO 620
610 IF F=-1 THEN  GO TO 640
620 IF A<>1 THEN  GOSUB 1260
630 GO TO 670
640 IF A<>-1 THEN  GOSUB 1260
650 GO TO 670
660 IF A=0 THEN B=I \ Z1=X(I) \ Z2=FNV(X(I))
670 F=A
680 C=C+1
690 IF C=500 THEN  GOSUB 1470
700 NEXT I
```

```
710 REM ************************************
720 REM        FINISH LOOP AND MOP UP
730 REM ************************************
740 GOSUB 1470
750 IF F=0 THEN  GO TO 980 \ REM NO NEED FOR END ROUTINE
760 IF B<>I THEN  GO TO 850
770 X3=X(I) \ REM **** START END ROUTINE NUMBER 1
780 Y3=FNV(X3)
790 M=(Z1+X(I))*(Z2-Y(I))
800 P=(X(I)+X3)*(Y(I)-Y3)
810 Q=(X3+Z1)*(Y3-Z2)
820 G=G+ABS((M+P+Q)/2)
830 H=H+1
840 GO TO 970
850 L=0 \ REM **** START END ROUTINE NUMBER 2
860 FOR R=B+1 TO I
870 L=L+(X(R-1)+X(R))*(Y(R-1)-Y(R))
880 NEXT R
890 X3=X(I)
900 Y3=FNV(X(I))
910 M=(X(I)+X3)*(Y(I)-Y3)
920 P=(X3+Z1)*(Y3-Z2)
930 Q=(Z1+X(B))*(Z2-Y(B))
940 G=G+ABS((L+M+P+Q)/2)
950 H=H+1
970 REM ******************
980 REM SUMMARY STATISTICS
990 REM ******************
1000 PRINT #1,"**********"
1010 IF N5=N THEN PRINT #1,"IN SWEEP 1"
1020 IF N5=N2 THEN PRINT #1,"IN SWEEP 2"
1030 PRINT #1,"A TOTAL OF ";(N6-N5+1);" POINTS WERE STUDIED"
1040 PRINT #1,"FROM POINT";N5;"TO POINT";N6
1050 PRINT #1,"AREA CALCULATION ROUTINE WAS RUN ";H;" TIMES"
1060 PRINT #1,"TOTAL AREA MEASUREMENT = ";G
1070 PRINT #1,"MEAN SUM OF SQUARED DEVIATIONS= ";(E/(N6-N5+1))
```

```
1080 PRINT #1,""
1090 RESTORE
1100 REM ***VELOCITY****
1110 T=0 \ T1=0
1120 IF N5=N THEN N5=N5+1
1130 IF N6=N3 THEN N6=N6-1
1140 FOR I=N5 TO N6
1150 T=T+V(I)
1160 T1=T1+V(I)^2
1170 NEXT I
1180 T3=N6-N5+2
1185 PRINT #1,'MEAN VELOCITY= ';(T/T3)
1190 PRINT #1,'STANDARD DEVIATION= ';SQR((T1-T3*(T/T3)^2)/T3)
1200 PRINT #1,''
1210 RETURN
1220 CLOSE
1230 RESTORE
1240 NEXT Z
1245 F=F1
1250 GO TO 1560
1260 REM ****************************
1270 REM AREA CALCULATION SUBROUTINE
1280 REM ****************************
1290 L=0 \ M=0 \ P=0 \ Q=0
1300 FOR R=(B+1) TO I-1
1310 L=L+(X(R-1)+X(R))*(Y(R-1)-Y(R))
1320 NEXT R
1330 IF X(I-1)-X(I)=0 THEN X3=X(I) \ GO TO 1370
1340 S1=(Y1-Y2)/(X1-X2)
1350 S2=(Y(I-1)-Y(I))/(X(I-1)-X(I))
1360 X3=((Y1-S1*X1)-(Y(I-1)-S2*X(I-1)))/(S2-S1)
1370 Y3=FNV(X3)
1380 M=(X(I-1)+X3)*(Y(I-1)-Y3)
1390 P=(X3+Z1)*(Y3-Z2)
1400 Q=(Z1+X(B))*(Z2-Y(B))
1410 G=G+ABS((L+M+P+Q)/2)
```

```
1420 B=I
1430 Z1=X3
1440 Z2=Y3
1450 H=H+1
1460 RETURN
1470 REM ***************************
1480 REM DIFFERENCE ARRAY SUMMATION
1490 REM ***************************
1500 FOR R=1 TO C
1510 E=E+D(R)^2
1520 D(R)=0
1530 NEXT R
1540 C=1
1550 RETURN
1560 CHAIN 'TRANS6'
1570 END
```

```
TRANS6    01-DEC-82  03:11:51

10 REM TRANS6
12 COMMON F$(10),F,Z(10)
15 FOR Z=1 TO F
20 IF Z(Z)<>4 THEN  GO TO 1280
30 PRINT 'AREA: ';F$(Z)
40 OPEN 'SY1:'+F$(Z)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
50 OPEN 'SY1:'+F$(Z)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
60 OPEN 'LP:' FOR OUTPUT AS FILE 1
70 DIM E(5,1)
80 PRINT #1,""
90 PRINT #1,"-------------------------------------------------------------"
100 PRINT #1,"ERROR STATISTICS FOR FILE ";F$(Z)
110 PRINT #1,""
120 REM *******SET ENDPOINTS********
130 N=1
140 N1=Y(0)
150 N2=Y(0)
160 N3=X(0)
280 REM ******CYCLE CONTROL******
290 N5=N
300 N6=N1
310 GO TO 350
320 N5=N2
330 N6=N3
340 REM ***** SET UP VARIABLES *****
350 B=N5 \ REM POLYGON ENDPOINT
360 D=0 \ REM DIFFERENCE VALUE
370 G=0 \ REM AREA TOTAL
380 H=0 \ REM POLYGON COUNTER
390 L=1 \ REM LAST SAWTOOTH SEGMENT
400 V=1 \ REM FIRST SAWTOOTH SEGMENT
410 I=N5 \ REM CURRENT POINT
420 READ E(0,0),E(0,1),E(1,0),E(1,1),E(2,0),E(2,1),E(3,0),E(3,1)
430 READ E(4,0),E(4,1),E(5,0),E(5,1)
```

```
440 DATA 22,16.32,38.95,26.28,38.89,16.23,55.83,26.4,55.95,16.32,72.81,26.44
450 DATA 72.81,26.44,55.95,16.32,55.83,26.4,38.89,16.23,38.95,26.28,22,16.32
460 DEF FNA(X)=E(0,1)+(((E(1,1)-E(0,1))*(X-E(0,0)))/(E(1,0)-E(0,0)))
470 DEF FNB(Y)=E(1,0)+(((E(2,0)-E(1,0))*(Y-E(1,1)))/(E(2,1)-E(1,1)))
480 DEF FNC(X)=E(2,1)+(((E(3,1)-E(2,1))*(X-E(2,0)))/(E(3,0)-E(2,0)))
490 DEF FND(Y)=E(3,0)+(((E(4,0)-E(3,0))*(Y-E(3,1)))/(E(4,1)-E(3,1)))
500 DEF FNE(X)=E(4,1)+(((E(5,1)-E(4,1))*(X-E(4,0)))/(E(5,0)-E(4,0)))
510 GOSUB 1540
520 Z1=Z5 \ Z2=Z6
530 REM ***********************************
540 REM            MAIN LOOP
550 REM ***********************************
560 FOR I=(N5+1) TO N6
570 FOR A=L TO 5
580 IF X(I-1)-X(I)=0 THEN Z3=X(I) \ GO TO 630
590 S1=(E(A-1,1)-E(A,1))/(E(A-1,0)-E(A,0))
600 S2=(Y(I-1)-Y(I))/(X(I-1)-X(I))
610 IF S1=S2 THEN  GO TO 770
620 Z3=INT((((E(A-1,1)-S1*E(A-1,0))-(Y(I-1)-S2*X(I-1)))/(S2-S1))*100)/100
630 IF Z3<=E(A,0) THEN IF Z3>=E(A-1,0) THEN  GO TO 660
640 IF Z3<=E(A-1,0) THEN IF Z3>=E(A,0) THEN  GO TO 660
650 GO TO 780
660 IF Z3>=X(I-1) THEN IF Z3<=X(I) THEN  GO TO 690
670 IF Z3>=X(I) THEN IF Z3<=X(I-1) THEN  GO TO 690
680 GO TO 780
690 Z4=E(A-1,1)+(((E(A,1)-E(A-1,1))*(Z3-E(A-1,0)))/(E(A,0)-E(A-1,0)))
700 Z4=INT(Z4*100)/100
710 IF Z4<=E(A,1) THEN IF Z4>=E(A-1,1) THEN  GO TO 740
720 IF Z4<=E(A-1,1) THEN IF Z4>=E(A,1) THEN  GO TO 740
730 GO TO 780
740 IF Z4>=Y(I-1) THEN IF Z4<=Y(I) THEN  GO TO 800
750 IF Z4>=Y(I) THEN IF Z4<=Y(I-1) THEN  GO TO 800
760 GO TO 780
770 IF X(I-1)=Z1 THEN IF Y(I-1)=Z2 THEN Z3=X(I) \ Z4=Y(I) \ GO TO 840
780 NEXT A
790 GO TO 840
```

```
800 IF X(I)=X(I-1) THEN IF Y(I)=Y(I-1) THEN  GO TO 840
810 IF Z1=Z3 THEN IF Z2=Z4 THEN  GO TO 840
820 L=A
830 GOSUB 1320
840 NEXT I
850 REM *************************
860 REM        END ROUTINES
870 REM *************************
880 L=5
890 IF B<>I THEN  GO TO 980
900 GOSUB 1540 \ REM **** START END ROUTINE NUMBER 1
910 Z3=Z5 \ Z4=Z6
920 M=(Z1+X(I))*(Z2-Y(I))
930 P=(X(I)+Z3)*(Y(I)-Z4)
940 Q=(Z3+Z1)*(Z4-Z2)
950 G=G+ABS((M+P+Q)/2)
960 H=H+1
970 GO TO 1140
980 S=0 \ P=0 \ P1=0 \ P2=0 \ REM **** START END ROUTINE NUMBER 2
990 FOR R=B+1 TO I
1000 S=S+(X(R-1)+X(R))*(Y(R-1)-Y(R))
1010 NEXT R
1020 GOSUB 1540
1030 Z3=Z5 \ Z4=Z6
1040 M=(X(I)+Z3)*(Y(I)-Z4)
1050 IF L=V THEN P=(Z3+Z1)*(Z4-Z2) \ GO TO 1110
1060 P=(Z3+E(L-1,0))*(Z4-E(L-1,1))
1070 FOR R=(V+1) TO (L-1)
1080 P2=P2+(E(R,0)+E(R-1,0))*(E(R,1)-E(R-1,1))
1090 NEXT R
1100 P1=(E(V,0)+Z1)*(E(V,1)-Z2)
1110 Q=(Z1+X(B))*(Z2-Y(B))
1120 G=G+ABS((S+M+P+P1+P2+Q)/2)
1130 H=H+1
```

```
1140 REM ******************
1150 REM SUMMARY STATISTICS
1160 REM ******************
1170 PRINT #1,"**********"
1180 IF N6=N1 THEN PRINT #1,"IN SWEEP 1"
1190 IF N6=N3 THEN PRINT #1,"IN SWEEP 2"
1200 PRINT #1,"**********"
1210 PRINT #1,"A TOTAL OF ";(N6-N5+1);" POINTS WERE STUDIED"
1220 PRINT #1,"FROM POINT";N5;"TO POINT";N6
1230 PRINT #1,"AREA CALCULATION ROUTINE WAS RUN ";H;" TIMES"
1240 PRINT #1,"TOTAL AREA MEASUREMENT = ";G
1250 PRINT #1,""
1260 IF N6=N1 THEN  GO TO 320
1270 CLOSE
1275 RESTORE
1280 NEXT Z
1310 GO TO 1630
1320 REM ****************************
1330 REM      AREA CALCULATION
1340 REM ****************************
1350 S=0 \ M=0 \ P=0 \ P1=0 \ P2=0 \ Q=0
1360 FOR R=(B+1) TO (I-1)
1370 S=S+(X(R-1)+X(R))*(Y(R-1)-Y(R))
1380 NEXT R
1390 M=(X(I-1)+Z3)*(Y(I-1)-Z4)
1400 IF L=V THEN P=(Z3+Z1)*(Z4-Z2) \ GO TO 1460
1410 P=(Z3+E(L-1,0))*(Z4-E(L-1,1))
1420 FOR R=(V+1) TO (L-1)
1430 P1=P1+(E(R,0)+E(R-1,0))*(E(R,1)-E(R-1,1))
1440 NEXT R
1450 P2=(E(V,0)+Z1)*(E(V,1)-Z2)
1460 Q=(Z1+X(B))*(Z2-Y(B))
1470 G=G+ABS((S+M+P+P1+P2+Q)/2)
1480 B=I
1490 Z1=Z3
1500 Z2=Z4
```

```
1510 V=L
1520 H=H+1
1530 RETURN
1540 REM *********************************
1550 REM      ENDPOINT CALCULATION
1560 REM *********************************
1570 IF L=1 THEN Z5=X(I) \ Z6=FNA(X(I))
1580 IF L=2 THEN Z5=FNB(Y(I)) \ Z6=Y(I)
1590 IF L=3 THEN Z5=X(I) \ Z6=FNC(X(I))
1600 IF L=4 THEN Z5=FND(Y(I)) \ Z6=Y(I)
1610 IF L=5 THEN Z5=X(I) \ Z6=FNE(X(I))
1620 RETURN
1630 CHAIN 'TRANS7'
1640 END
```

```
TRANS7    01-DEC-82  03:14:28

10 REM TRANS7
20 COMMON F$(10),F,Z(10)
30 FOR Z=1 TO F
40 IF Z(Z)<5 THEN  GO TO 1550
50 PRINT 'SEGMENTAL AREA: ';F$(Z)
60 OPEN 'SY1:'+F$(Z)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
70 OPEN 'SY1:'+F$(Z)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
80 OPEN 'SY1:'+F$(Z)+'V' FOR INPUT AS FILE #4 \ DIM #4,V(4000)=5
90 OPEN 'LP:' FOR OUTPUT AS FILE 1
100 DIM E(5,1)
110 DATA 22,16.32,38.95,26.28,38.89,16.23,55.83,26.4,55.95,16.32,72.81,26.44
120 DATA 72.81,26.44,55.95,16.32,55.83,26.4,38.89,16.23,38.95,26.28,22,16.32
130 PRINT #1,""
140 PRINT #1,"-------------------------------------------------------------"
150 PRINT #1,"ERROR STATISTICS FOR FILE ";F$(Z);" ANALYZED AS PATTERN ";Z(Z)
160 PRINT #1,""
170 N=1
180 N1=Y(0)
190 N2=Y(0)
200 N3=X(0)
210 IF Z(Z)=5 THEN X1=40.51 \ X2=54.51
220 IF Z(Z)=6 THEN X1=55.91 \ Y1=21.52 \ X2=65.85
230 IF Z(Z)=7 THEN X1=47.58 \ X2=65.85
240 FOR I=N TO N1
250 IF Z(Z)=6 THEN  GO TO 280
260 IF X(I)>=X1 THEN N7=(I-1) \ GO TO 300
270 GO TO 290
280 IF Y(I)<=Y1 THEN IF X(I)>53 THEN N7=(I-1) \ GO TO 300
290 NEXT I
300 FOR I=N7 TO N1
310 IF X(I)>=X2 THEN N8=(I-1) \ GO TO 330 .
320 NEXT I
330 FOR I=N2 TO N3
340 IF X(I)<=X2 THEN N9=(I-1) \ GO TO 360
```

```
350 NEXT I
360 FOR I=N9 TO N3
370 IF Z(Z)=6 THEN  GO TO 400
380 IF X(I)<=X1 THEN O1=I \ GO TO 420
390 GO TO 410
400 IF Y(I)>=Y1 THEN IF X(I)<=59 THEN O1=(I-1) \ GO TO 420
410 NEXT I
420 READ E(0,0),E(0,1),E(1,0),E(1,1),E(2,0),E(2,1),E(3,0),E(3,1)
430 READ E(4,0),E(4,1),E(5,0),E(5,1)
440 N5=N \ N6=N7
450 IF Z(Z)=6 THEN L=1 \ L1=4 \ GO TO 470
460 L=1 \ L1=3
470 GOSUB 770
480 N5=(N7+1) \ N6=N8
490 IF Z(Z)=5 THEN L=3 \ L1=3
500 IF Z(Z)=6 THEN L=4 \ L1=5
510 IF Z(Z)=7 THEN L=3 \ L1=5
520 GOSUB 770
530 N5=(N8+1) \ N6=N1
540 L1=5
550 IF Z(Z)=5 THEN L=3
560 IF Z(Z)=6 THEN L=5
570 IF Z(Z)=7 THEN L=5
580 GOSUB 770
590 READ E(0,0),E(0,1),E(1,0),E(1,1),E(2,0),E(2,1),E(3,0),E(3,1),E(4,0)
600 READ E(4,1),E(5,0),E(5,1)
610 N5=N2 \ N6=N9-1
620 L=1
630 IF Z(Z)=5 THEN L1=3
640 IF Z(Z)=6 THEN L1=1
650 IF Z(Z)=7 THEN L1=1
660 GOSUB 770
670 N5=N9 \ N6=(O1-1)
680 IF Z(Z)=5 THEN L=3 \ L1=3
690 IF Z(Z)=6 THEN L=1 \ L1=2
700 IF Z(Z)=7 THEN L=1 \ L1=3
```

```
710 GOSUB 770
720 N5=01 \ N6=N3
730 IF Z(2)=6 THEN L=2 \ L1=5 \ GO TO 750
740 L=3 \ L1=5
750 GOSUB 770
760 GO TO 1530
770 B=N5 \ D=0 \ G=0 \ H=0 \ V=L \ I=N5
780 DEF FNA(X)=E(0,1)+(((E(1,1)-E(0,1))*(X-E(0,0)))/(E(1,0)-E(0,0)))
790 DEF FNB(Y)=E(1,0)+(((E(2,0)-E(1,0))*(Y-E(1,1)))/(E(2,1)-E(1,1)))
800 DEF FNC(X)=E(2,1)+(((E(3,1)-E(2,1))*(X-E(2,0)))/(E(3,0)-E(2,0)))
810 DEF FND(Y)=E(3,0)+(((E(4,0)-E(3,0))*(Y-E(3,1)))/(E(4,1)-E(3,1)))
820 DEF FNE(X)=E(4,1)+(((E(5,1)-E(4,1))*(X-E(4,0)))/(E(5,0)-E(4,0)))
830 GOSUB 1780
840 Z1=Z5 \ Z2=Z6
850 REM  ************ MAIN LOOP **************
860 FOR I=(N5+1) TO N6
870 FOR A=L TO L1
880 IF X(I-1)-X(I)=0 THEN Z3=X(I) \ GO TO 930
890 S1=(E(A-1,1)-E(A,1))/(E(A-1,0)-E(A,0))
900 S2=(Y(I-1)-Y(I))/(X(I-1)-X(I))
910 IF S1=S2 THEN  GO TO 1070
920 Z3=INT((((E(A-1,1)-S1*E(A-1,0))-(Y(I-1)-S2*X(I-1)))/(S2-S1))*100)/100
930 IF Z3<=E(A,0) THEN IF Z3>=E(A-1,0) THEN  GO TO 960
940 IF Z3<=E(A-1,0) THEN IF Z3>=E(A,0) THEN  GO TO 960
950 GO TO 1080
960 IF Z3>=X(I-1) THEN IF Z3<=X(I) THEN  GO TO 990
970 IF Z3>=X(I) THEN IF Z3<=X(I-1) THEN  GO TO 990
980 GO TO 1080
990 Z4=E(A-1,1)+(((E(A,1)-E(A-1,1))*(Z3-E(A-1,0)))/(E(A,0)-E(A-1,0)))
1000 Z4=INT(Z4*100)/100
1010 IF Z4<=E(A,1) THEN IF Z4>=E(A-1,1) THEN  GO TO 1040
1020 IF Z4<=E(A-1,1) THEN IF Z4>=E(A,1) THEN  GO TO 1040
1030 GO TO 1080
1040 IF Z4>=Y(I-1) THEN IF Z4<=Y(I) THEN  GO TO 1100
1050 IF Z4>=Y(I) THEN IF Z4<=Y(I-1) THEN  GO TO 1100
1060 GO TO 1080
```

```
1070 IF X(I-1)=Z1 THEN IF Y(I-1)=Z2 THEN Z3=X(I) \ Z4=Y(I) \ GO TO 1140
1080 NEXT A
1090 GO TO 1140
1100 IF X(I)=X(I-1) THEN IF Y(I)=Y(I-1) THEN  GO TO 1140
1110 IF Z1=Z3 THEN IF Z2=Z4 THEN  GO TO 1140
1120 L=A
1130 GOSUB 1580
1140 NEXT I
1150 REM        END ROUTINES
1160 L=L1
1170 IF B<>I THEN  GO TO 1260
1180 GOSUB 1780 \ REM **** START END ROUTINE NUMBER 1
1190 Z3=Z5 \ Z4=Z6
1200 M=(Z1+X(I))*(Z2-Y(I))
1210 P=(X(I)+Z3)*(Y(I)-Z4)
1220 Q=(Z3+Z1)*(Z4-Z2)
1230 G=G+ABS((M+P+Q)/2)
1240 H=H+1
1250 GO TO 1420
1260 S=0 \ P=0 \ P1=0 \ P2=0 \ REM **** START END ROUTINE NUMBER 2
1270 FOR R=B+1 TO I
1280 S=S+(X(R-1)+X(R))*(Y(R-1)-Y(R))
1290 NEXT R
1300 GOSUB 1780
1310 Z3=Z5 \ Z4=Z6
1320 M=(X(I)+Z3)*(Y(I)-Z4)
1330 IF L=V THEN P=(Z3+Z1)*(Z4-Z2) \ GO TO 1390
1340 P=(Z3+E(L-1,0))*(Z4-E(L-1,1))
1350 FOR R=(V+1) TO (L-1)
1360 P2=P2+(E(R,0)+E(R-1,0))*(E(R,1)-E(R-1,1))
1370 NEXT R
1380 P1=(E(V,0)+Z1)*(E(V,1)-Z2)
1390 Q=(Z1+X(B))*(Z2-Y(B))
1400 G=G+ABS((S+M+P+P1+P2+Q)/2)
1410 H=H+1
1420 REM SUMMARY STATISTICS
```

```
1430 PRINT #1,"************************"
1440 IF N5=N THEN PRINT #1,"IN SWEEP 1" \ PRINT #1,''
1450 IF N5=N2 THEN PRINT #1,"IN SWEEP 2" \ PRINT #1,''
1460 PRINT #1,"A TOTAL OF ";(N6-N5+1);" POINTS WERE STUDIED"
1470 PRINT #1,"FROM POINT";N5;"TO POINT";N6
1480 PRINT #1,"AREA CALCULATION ROUTINE WAS RUN ";H;" TIMES"
1490 PRINT #1,"TOTAL AREA MEASUREMENT = ";G
1500 PRINT #1,""
1510 GOSUB 1840
1520 RETURN
1530 CLOSE
1540 RESTORE
1550 NEXT Z
1560 GO TO 1930
1570 REM       AREA CALCULATION
1580 S=0 \ M=0 \ P=0 \ P1=0 \ P2=0 \ Q=0
1590 FOR R=(B+1) TO (I-1)
1600 S=S+(X(R-1)+X(R))*(Y(R-1)-Y(R))
1610 NEXT R
1620 M=(X(I-1)+Z3)*(Y(I-1)-Z4)
1630 IF L=V THEN P=(Z3+Z1)*(Z4-Z2) \ GO TO 1690
1640 P=(Z3+E(L-1,0))*(Z4-E(L-1,1))
1650 FOR R=(V+1) TO (L-1)
1660 P1=P1+(E(R,0)+E(R-1,0))*(E(R,1)-E(R-1,1))
1670 NEXT R
1680 P2=(E(V,0)+Z1)*(E(V,1)-Z2)
1690 Q=(Z1+X(B))*(Z2-Y(B))
1700 G=G+ABS((S+M+P+P1+P2+Q)/2)
1710 B=I
1720 Z1=Z3
1730 Z2=Z4
1740 V=L
1750 H=H+1
1760 RETURN
1770 REM     ****** ENDPOINT CALCULATION
1780 IF L=1 THEN Z5=X(I) \ Z6=FNA(X(I))
```

```
1790 IF L=2 THEN Z5=FNB(Y(I)) \ Z6=Y(I)
1800 IF L=3 THEN Z5=X(I) \ Z6=FNC(X(I))
1810 IF L=4 THEN Z5=FND(Y(I)) \ Z6=Y(I)
1820 IF L=5 THEN Z5=X(I) \ Z6=FNE(X(I))
1830 RETURN
1840 REM    ****** VELOCITY SUBROUTINE
1850 T=0 \ M=0
1860 FOR I=(N5+1) TO (N6-1)
1870 T=T+V(I) \ M=M+V(I)^2
1880 NEXT I
1890 R=N6-N5-1
1900 PRINT #1,'' \ PRINT #1,'MEAN VELOCITY= ';(T/R)
1910 PRINT #1,'STANDARD DEVIATION= ';SQR(((M-(R)*(T/R)^2)/R))
1920 RETURN
1930 CHAIN 'TRANS8'
1940 END
```

```
TRANS8    01-DEC-82  03:17:19

10 REM TRANS8
50 COMMON F$(10),F,Z(10)
90 DIM X1(500),Y1(500),V1(500)
100 FOR R=1 TO F
105 IF Z(R)>3 THEN  GO TO 510
110 OPEN 'SY1:'+F$(R)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
120 OPEN 'SY1:'+F$(R)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
130 OPEN 'SY1:'+F$(R)+'V' FOR INPUT AS FILE #4 \ DIM #4,V(4000)=5
140 N=Y(0)
150 N1=X(0)
160 G=INT(N/500)+1
170 H=INT((N1-N)/500)+1
180 PRINT 'GRAPHING FILE ';F$(R)
190 PRINT CHR$(27)+'[5i'
195 PRINT CHR$(12)
200 PRINT '                          FILE ';F$(R)
210 PRINT ''
220 PRINT CHR$(27)+'[4i'
230 FOR I=1 TO 500
240 K=I*G
250 IF K>N THEN  GO TO 280
260 X1(I)=X(K) \ Y1(I)=Y(K) \ V1(I)=V(K)
270 NEXT I
280 REGION("UPPER",1)
290 REGION("LOWER",2)
300 WINDOW("EXACT",20,20,80,23,1)
310 WINDOW("EXACT",20,0,80,.5,2)
320 GRAPH(,I,X1(1),Y1(1),,,1)
330 GRAPH(,I,X1(1),V1(1),,,2)
340 DISPLAY_COPY
350 DISPLAY_CLEAR
360 PRINT 'GRAPHING FILE ';F$(R)
370 FOR I=1 TO 500
```

```
380 K=N+(I*H)
390 IF K>N1 THEN  GO TO 420
400 X1(I)=X(K) \ Y1(I)=Y(K) \ V1(I)=V(K)
410 NEXT I
420 REGION("UPPER",1)
430 REGION("LOWER",2)
440 WINDOW("EXACT",20,20,80,23,1)
450 WINDOW("EXACT",20,0,80,.5,2)
460 GRAPH(,I,X1(1),Y1(1),,,1)
470 GRAPH(,I,X1(1),V1(1),,,2)
480 DISPLAY_COPY
490 DISPLAY_CLEAR
500 CLOSE
510 NEXT R
580 END
```

```
TRANS9    01-DEC-82  03:18:57

10 REM TRANS9
50 COMMON F$(10),F,Z(10)
90 DIM X1(500),Y1(500),V1(500)
100 FOR R=1 TO F
105 IF Z(R)<4 THEN  GO TO 580
110 OPEN 'SY1:'+F$(R)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
120 OPEN 'SY1:'+F$(R)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
130 OPEN 'SY1:'+F$(R)+'V' FOR INPUT AS FILE #4 \ DIM #4,V(4000)=5
140 N=Y(0)
150 N1=X(0)
160 G=INT(N/500)+1
170 H=INT((N1-N)/500)+1
180 PRINT CHR$(27)+'[5i'
185 PRINT CHR$(12)
190 PRINT '                                FILE ';F$(R)
200 PRINT '                                SWEEP 1'
210 PRINT ''
220 PRINT CHR$(27)+'[4i'
230 FOR I=1 TO 500
240 K=I*G
250 IF K>N THEN  GO TO 280
260 X1(I)=X(K) \ Y1(I)=Y(K) \ V1(I)=V(K)
270 NEXT I
280 WINDOW("EXACT",20,10,80,30)
290 GRAPH(,I,X1(1),Y1(1))
300 DISPLAY_COPY
310 REGION("UPPER",1)
320 REGION("LOWER",2)
330 WINDOW("EXACT",0,0,500,.5,1)
335 WINDOW("EXACT",0,20,500,80,2)
340 GRAPH(,I,,V1(1),,,1)
350 GRAPH(,I,,X1(1),,,2)
360 DISPLAY_COPY
370 DISPLAY_CLEAR
```

```
380 PRINT CHR$(27)+'[5i'
390 PRINT '                                SWEEP 2'
400 PRINT ''
410 PRINT CHR$(27)+'[4i'
420 FOR I=1 TO 500
430 K=N+(I*H)
440 IF K>N1 THEN  GO TO 470
450 X1(I)=X(K) \ Y1(I)=Y(K) \ V1(I)=V(K)
460 NEXT I
470 WINDOW("EXACT",20,10,80,30)
480 GRAPH(,I,X1(1),Y1(1))
490 DISPLAY_COPY
500 REGION("UPPER",1)
510 REGION("LOWER",2)
520 WINDOW("EXACT",0,0,500,.5,1)
525 WINDOW("EXACT",0,20,500,80,2)
530 GRAPH(,I,,V1(1),,,1)
540 GRAPH(,I,,X1(1),,,2)
550 DISPLAY_COPY
560 DISPLAY_CLEAR
570 CLOSE
580 NEXT R
590 CHAIN 'TRANSA'
650 END
```

```
TRANSA    01-DEC-82  03:23:55

10 REM TRANSA
20 COMMON F$(10),F,Z(10)
30 F1=F
80 FOR Z=1 TO F1
85 IF Z(Z)>3 THEN  GO TO 980
87 PRINT 'AREA CALCULATION: ';F$(Z)
90 OPEN 'SY1:'+F$(Z)+'X' FOR INPUT AS FILE #2 \ DIM #2,X(4000)=5
100 OPEN 'SY1:'+F$(Z)+'Y' FOR INPUT AS FILE #3 \ DIM #3,Y(4000)=5
110 OPEN 'LP:' FOR OUTPUT AS FILE 1
120 DIM D(500)
130 REM ****SET ENDPOINTS
140 PRINT #1,""
150 PRINT #1,"------------------------------------------------------------"
160 PRINT #1,"ERROR STATISTICS FOR FILE ";F$(Z)
170 PRINT #1,""
180 N=1
190 N1=Y(0)
200 N2=Y(0)
210 N3=X(0)
220 N5=N
230 N6=N1
240 GO TO 280
250 N5=N2
260 N6=N3
270 REM ***** SETUP VARIABLES
280 E=0 \ REM DIFFERENCE SCORES
290 G=0 \ REM AREA TOTAL
300 H=0 \ REM POLYGON COUNTER
310 F=0 \ REM SET FLAG
320 C=1 \ REM COUNTER FOR DIFFERENCE ARRAY
330 READ X1,Y1,X2,Y2
340 DATA 23.57,21.42,72.61,21.25
350 DEF FNV(X)=Y1+(((Y2-Y1)*(X-X1))/(X2-X1))
360 Z1=X(N5)
```

```
370 Z2=FNV(X(N5))
380 B=N5 \ REM POLYGON ENDPOINT
390 REM ***********************************
400 REM       START MAIN LOOP
410 REM ***********************************
420 FOR I=N5 TO N6
430 D(C)=Y(I)-FNV(X(I))
440 A=SGN(D(C))
450 IF F=0 THEN  GO TO 520
460 IF F=1 THEN  GO TO 480
470 IF F=-1 THEN  GO TO 500
480 IF A<>1 THEN  GOSUB 1000
490 GO TO 530
500 IF A<>-1 THEN  GOSUB 1000
510 GO TO 530
520 IF A=0 THEN B=I \ Z1=X(I) \ Z2=FNV(X(I))
530 F=A
540 C=C+1
550 IF C=500 THEN  GOSUB 1210
560 NEXT I
570 REM ************************************
580 REM        FINISH LOOP AND MOP UP
590 REM ************************************
600 GOSUB 1210
610 IF F=0 THEN  GO TO 820 \ REM NO NEED FOR END ROUTINE
620 IF B<>I THEN  GO TO 710
630 X3=X(I) \ REM **** START END ROUTINE NUMBER 1
640 Y3=FNV(X3)
650 M=(Z1+X(I))*(Z2-Y(I))
660 P=(X(I)+X3)*(Y(I)-Y3)
670 Q=(X3+Z1)*(Y3-Z2)
680 G=G+ABS((M+P+Q)/2)
690 H=H+1
700 GO TO 830
710 L=0 \ REM **** START END ROUTINE NUMBER 2
720 FOR R=B+1 TO I
```

```
730 L=L+(X(R-1)+X(R))*(Y(R-1)-Y(R))
740 NEXT R
750 X3=X(I)
760 Y3=FNV(X(I))
770 M=(X(I)+X3)*(Y(I)-Y3)
780 P=(X3+Z1)*(Y3-Z2)
790 Q=(Z1+X(B))*(Z2-Y(B))
800 G=G+ABS((L+M+P+Q)/2)
810 H=H+1
830 REM ******************
840 REM SUMMARY STATISTICS
850 REM ******************
860 PRINT #1,"**********"
870 IF N6=N1 THEN PRINT #1,"IN SWEEP 1"
880 IF N6=N3 THEN PRINT #1,"IN SWEEP 2"
890 PRINT #1,"A TOTAL OF ";(N6-N5+1);" POINTS WERE STUDIED"
900 PRINT #1,"FROM POINT";N5;"TO POINT";N6
910 PRINT #1,"AREA CALCULATION ROUTINE WAS RUN ";H;" TIMES"
920 PRINT #1,"TOTAL AREA MEASUREMENT = ";G
930 PRINT #1,"MEAN SUM OF SQUARED DEVIATIONS= ";(E/(N6-N5+1))
940 PRINT #1,""
950 IF N6=N1 THEN RESTORE  \ GO TO 250
960 CLOSE
970 RESTORE
980 NEXT Z
985 F=F1
990 GO TO 1300
1000 REM ****************************
1010 REM AREA CALCULATION SUBROUTINE
1020 REM ****************************
1030 L=0 \ M=0 \ P=0 \ Q=0
1040 FOR R=(B+1) TO I-1
1050 L=L+(X(R-1)+X(R))*(Y(R-1)-Y(R))
1060 NEXT R
1070 IF X(I-1)-X(I)=0 THEN X3=X(I) \ GO TO 1110
1080 S1=(Y1-Y2)/(X1-X2)
```

```
1090 S2=(Y(I-1)-Y(I))/(X(I-1)-X(I))
1100 X3=((Y1-S1*X1)-(Y(I-1)-S2*X(I-1)))/(S2-S1)
1110 Y3=FNV(X3)
1120 M=(X(I-1)+X3)*(Y(I-1)-Y3)
1130 P=(X3+Z1)*(Y3-Z2)
1140 Q=(Z1+X(B))*(Z2-Y(B))
1150 G=G+ABS((L+M+P+Q)/2)
1160 B=I
1170 Z1=X3
1180 Z2=Y3
1190 H=H+1
1200 RETURN
1210 REM ***************************
1220 REM DIFFERENCE ARRAY SUMMATION
1230 REM ***************************
1240 FOR R=1 TO C
1250 E=E+D(R)^2
1260 D(R)=0
1270 NEXT R
1280 C=1
1290 RETURN
1300 PRINT 'This job is completed. If you turn off the recorder, please remove'
1305 PRINT 'the disk first.  I will be here soon to straighten things out.'
1307 PRINT '                                                    Yaakov'
1310 END
```

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Table 1

Basal Ganglia-Cortical Systems:

Behavioral Tasks Affected Following Lesions

Affected Tasks	object reversal go-no go	delayed response delayed spatial alternation prism adaptation	visual differentiation
Lesioned Areas	orbitofrontal cortex	dorsolateral frontal cortex	infertemporal cortex
	ventrolateral of caudate	anterodorsal head of caudate	tail of caudate
		lateral globus pallidus	medial globus pallidus
		subthalamic nucleus	thalamic nuclei

Table 2

Subject Summary:

Demographic Characteristics, Neuropsychological

Test Scores and Parkinson's Disease Symptom Severity

(Values in parentheses are standard deviations,

p values are for t-test comparisons)

	PD	(N=16)	Controls	(N=9)	p
Age	60.58	(10.4)	67	(3.16)	.02
Education	13.2	(2.5)	11.2	(1.1)	.01
Mini Mental State	50.7	(5.1)	53.2	(2.0)	n.s.
Rosen Drawing	11.05	(2.35)	12.5	(1.7)	n.s.
Beck Depression Inventory	7.9	(6.9)	3.75	(2.9)	n.s.
PD Evaluation	30.9	(4.8)			
Tremor	1.79	(2.1)			
Rigidity	4.4	(2.8)			
Bradykinesia	1.44	(1.0)			
Duration of Illness	7.9	(5.0)			

Table 3

Relation Between Tracing Error and Velocity:

Controls

Path	Sweep	Error Measure	Velocity Measure	r	p
1	1	Path Crossings	Digitized Points	.635	.008
	2	Path Crossings	Digitized Points	.587	.016
3	2	Path Crossings	Digitized Points	.522	.037
	2	Path Crossings	Velocity	-.59	.01
4	1	Path Crossings	Digitized Points	.583	.01
	1	Path Crossings	Velocity	-.609	.007
5	2	Area	Digitized Points	.568	.032
6	2	Area	Digitized Points	-.52	.03

Table 4

Relation Between Tracing Error and Velocity:

Parkinson's Disease Patients

Path	Sweep	Error Measure	Velocity Measure	r	p
1	1	Area	Velocity	.42	.04
	1	Path Crossings	Velocity	.49	.015
5	2	Area	Digitized Points	-.4	.049
6	1	Area	Digitized Points	-.431	.05
7	1	Area	Digitized Points	-.58	.004

Table 5

ANOVA Comparing Tracing Measures in Parkinsonians and Controls: Paths 1 and 3

(Values are F, p <)

Effect	Area	Path Crossings	Velocity	Digitized Points
		Sweep 1		
Group	12.57, .002	26.57, .0001	n.s.	4.64, .05
Path	28.78, .0001	9.02, .01	18.59, .001	22.51, .001
Hand	n.s.	n.s.	n.s.	5.76, .03
Group X Path	4.42, .05	7.07, .02	n.s.	n.s.
Group X Hand	n.s.	5.8, .03	n.s.	n.s.
Path X Hand	n.s.	n.s.	n.s.	n.s.
Group X Path X Hand	n.s.	n.s.	n.s.	n.s.
		Sweep 2		
Group	16.42, .001	20.2, .0001	n.s.	n.s.
Path	23.22, .0001	6.79, .02	24.0, .0001	13.96, .002
Hand	n.s.	n.s.	n.s.	n.s.
Group X Path	n.s.	5.75, .03	n.s.	n.s.
Group X Hand	n.s.	n.s.	n.s.	n.s.
Path X Hand	n.s.	n.s.	n.s.	n.s.
Group X Path X Hand	n.s.	n.s.	n.s.	n.s.

Table 6

ANOVA Comparing Tracing Measures in Parkinsonians and Controls:

Paths 4 and 5, Equivalent Missing Segments

(Values are F, p <)

	Sweep 1		Sweep 2	
Effect	Area	Digitized Points	Area	Digitized Points
Group	10.69, .004	n.s.	6.16, .03	n.s.
Path	6.64, .02	n.s.	6.49, .02	n.s.
Hand	n.s.	n.s.	6.62, .02	8.48, .01
Group X Path	n.s.	n.s.	n.s.	n.s.
Group X Hand	n.s.	n.s.	n.s.	n.s.
Path X Hand	n.s.	n.s.	n.s.	n.s.
Group X Path X Hand	n.s.	n.s.	n.s.	n.s.

Table 7

ANOVA Comparing Tracing Measures in Parkinsonians and Controls:

Paths 4 and 6, Equivalent Missing Segments

(Values are F, p <)

	Sweep 1		Sweep 2	
Effect	Area	Digitized Points	Area	Digitized Points
Group	n.s.	n.s.	7.83, .02	n.s.
Path	15.93, .001	n.s.	13.56, .002	n.s.
Hand	n.s.	n.s.	9.82, .01	5.9, .03
Group X Path	n.s.	n.s.	n.s.	n.s.
Group X Hand	n.s.	n.s.	n.s.	n.s.
Path X Hand	n.s.	n.s.	n.s.	n.s.
Group X Path X Hand	n.s.	n.s.	n.s.	n.s.

Table 8

ANOVA Comparing Tracing Measures in Parkinsonians and Controls:

Paths 4 and 7, Equivalent Missing Segments

(Values are F, p <)

	Sweep 1		Sweep 2	
Effect	Area	Digitized Points	Area	Digitized Points
Group	10.47, .004	n.s.	n.s.	n.s.
Path	20.5, .0002	n.s.	16.68, .001	n.s.
Hand	n.s.	n.s.	4.88, .04	5.54, .05
Group X Path	4.37, .05	n.s.	n.s.	n.s.
Group X Hand	n.s.	n.s.	7.11, .02	n.s.
Path X Hand	n.s.	n.s.	n.s.	n.s.
Group X Path X Hand	n.s.	n.s.	7.09, .02	n.s.

Table 9

ANOVA Comparing Tracing Measures in Parkinsonians and Controls:

Paths 4-7, Entire Sweeps of Paths

(Values are F, p <)

	Sweep 1		Sweep 2	
Effects	Area	Digitized Points	Area	Digitized Points
Group	17.35, .001	n.s.	20.46, .0002	n.s.
Path	12.86, .0001	n.s.	7.04, .0005	4.86, .045
Hand	4.69, .05	n.s.	19.43, .0004	12.76, .0025
Group X Path	4.52, .01	n.s.	n.s.	n.s.
Group X Hand	n.s.	n.s.	n.s.	n.s.
Path X Hand	n.s.	n.s.	n.s.	n.s.
Group X Path X Hand	n.s.	n.s.	4.87, .006	n.s.

Table 10

Relation of Tracing Performance to Demographic Variables and Cognitive Tests: Parkinsonians

Path	Sweep	Tracing Variable	Related Variables	r	p
1	1	Path Crossings	Education	.546	.007
1	1	Digitized Points	MMS	-.462	.017
1	1		Digit Span Backward	-.459	.027
1	1		Calculations	-.572	.004
1	1		Naming Presidents	-.574	.004
1	2	Path Crossings	Orientation	-.42	.045
1	2		Recall	-.457	.028
1	2	Digitzed Points	MMS	-.492	.017
1	2		Digit Span Forward	-.502	.014
1	2		Calculations	-.421	.421
1	2		Language	-.434	.038
3	1	Area	Age		
3	1		Digit Span Backward	.487	.021
3	1		Recall	.513	.014
3	1		Construction	-.569	.0057
3	1		Rosen Drawing	-.552	.007
3	1	Path Crossings	Education	.431	.044
3	1		Recall	-.486	.021
3	2	Area	Orientation	-.480	.018
3	2		Recall	.492	.017
3	2		Language	-.564	.005

Table 10 (continued)

Path	Sweep	Tracing Variable	Related Variables	r	p
3	2		Construction	-.484	.019
3	2	Path Crossings	Education	.441	.034
3	2		Recall	-.645	.001
4	1	Area	Age	.598	.002
4	1		Digit Span Backward	.583	.003
4	1	Path Crossings	Digit Span Backward	-.599	.002
4	1		Calculation	-.511	.01
4	1		Recall	-.425	.038
4	2	Path Crossings	Recall	-.474	.022
5	2	Area (sweep)	Orientation	-.42	.04
6	1	Digitized Points (segment)	Construction	.488	.024
6	1		Rosen Drawing	.563	.007
6	1	Digitized Points (segment)	Construction	.559	.008
6	1		Rosen Drawing	.575	.006
6	2	Area (segment)	Construction	-.545	.012
6	2		Rosen Drawing	-.528	.016
6	2	Area (sweep)	Construction	-.46	.046
6	2		Rosen Drawing	-.501	.028
6	2		Digit Span Backward	.487	.034
6	2		Recall	.542	.01

Table 10 (continued)

Path	Sweep	Tracing Variable	Related Variables	r	p
6	2	Digitized Points (segment)	Construction	.66	.002
6	2		Rosen Drawing	.60	.005
6	2	Digitized Points (sweep)	Construction	.566	.012
6	2		Rosen Drawing	.549	.015
7	1	Area (segment)	Construction	-.431	.044
7	1		Rosen Drawing	-.444	.036
7	1	Area (sweep)	Construction	-.460	.031
7	1		Rosen Drawing	-.483	.023
7	1	Digitized Points (segment)	Construction	.635	.002
7	1		Rosen Drawing	.613	.002
7	1	Digitized Points (sweep)	Construction	.601	.003
7	1		Rosen Drawing	.541	.009
7	2	Area (segment)	Rosen Drawing	-.452	.045
7	2	Area (sweep)	Rosen Drawing	.44	.05
7	2	Digitized Points (segment)	Construction	.679	.001
7	2		Rosen Drawing	.664	.001
7	2	Digitized Points	Construction	.701	.001
			Rosen Drawing	.651	.002

Table 11

Relation of Tracing Performance to Demographic Variables and Cognitive Tests: Controls

Path	Sweep	Tracing Variable	Related Variables	r	p
1	1	Area	Naming Presidents	-.587	.016
1	2	Area	Education	-.499	.049
1	2		MMS	-.517	.039
1	2		Rosen Drawing	-.637	.008
1	2	Path Crossings	Rosen Drawing	.527	.035
3	1	Area	Naming Presidents	-.566	.022
3	1	Path Crossings	Naming Presidents	.553	.026
3	2	Area	Calculations	-.539	.031
3	2	Path Crossings	MMS	.56	.023
3	2		Education	.523	.037
5	2	Area (segment)	Naming Presidents	-.52	.024
6	2	Digitized Points (segment)	Age	.546	.023
7	1	Area (segment)	MMS	-.59	.014
7	1		Calculations	-.56	.023
7	1		Recall	-.622	.01
7	1	Area (sweep)	MMS	-.562	.023
7	1		Calculations	-.519	.039
7	1		Recall	-.641	.007
7	2	Area (segment)	Education	-.72	.002
7	2		Orientation	-.809	.001
7	2	Area (sweep)	Education	-.75	.01
7	2		Orientation	-.828	.001

Table 12

Relation of Tracing Performance in PD Patients to Measures of PD Severity

Path	Sweep	Tracing Variable	Related Variables	r	p
1	1	Digitized Points	Duration of illness	.44	.035
1	1		Bradykinesia	.416	.048
1	2	Digitized Points	PD severity	.528	.01
1	2		Rigidity	.608	.002
1	2		Bradykinesia	.607	.002
1	2		Postural stability	.433	.038
3	1	Digitized Points	Rigidity	.423	.043
3	2	Area	Bradykinesia	.479	.02
3	2	Digitized Points	Rigidity	.533	.009
5	2	Area (sweep)	Tremor	.425	.038
6	2	Digitized Points (segment)	PDE	-.467	.037

Figure 1. Neuroanatomical connections of the basal ganglia.

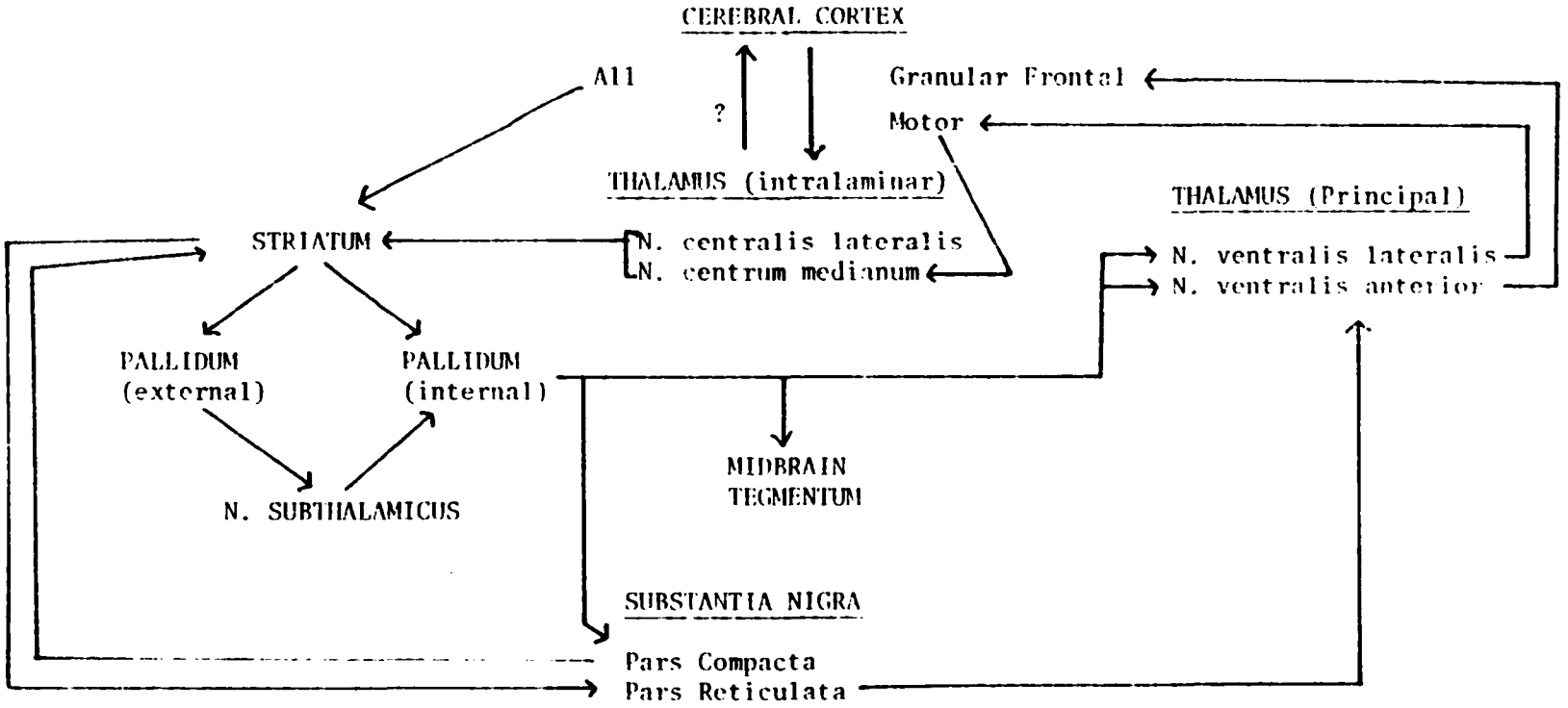

CEREBRAL CORTEX
All
?
Granular Frontal
Motor
THALAMUS (intralaminar)
THALAMUS (Principal)
STRIATUM
N. centralis lateralis
N. centrum medianum
N. ventralis lateralis
N. ventralis anterior
PALLIDUM (external)
PALLIDUM (internal)
MIDBRAIN TEGMENTUM
N. SUBTHALAMICUS
SUBSTANTIA NIGRA
Pars Compacta
Pars Reticulata

Figure 2. Paths used in tracing task in order of their administration.

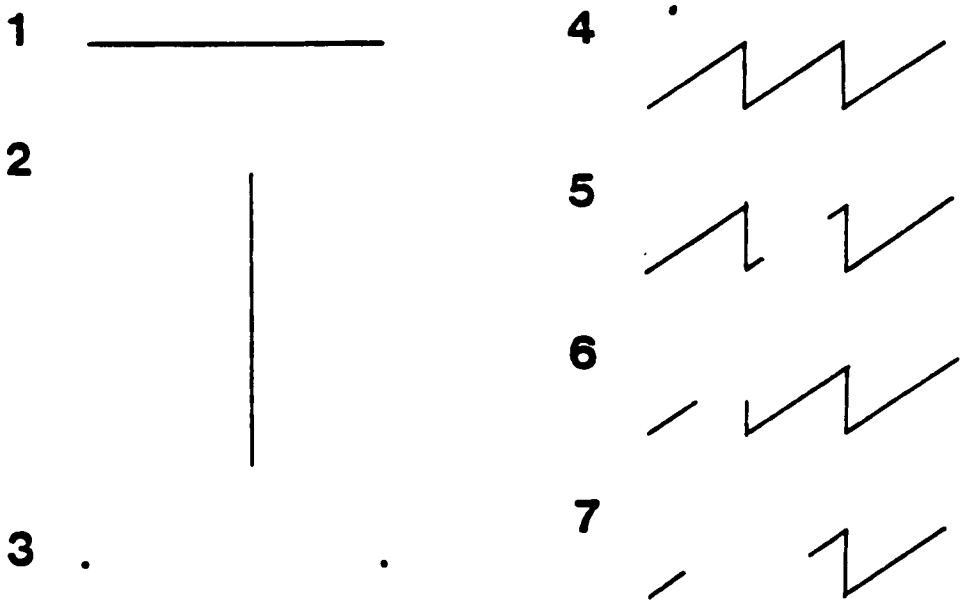

1
2
3
4
5
6
7

Figure 3. Path 1 and Path 3: Area error in Paths 1 and 3.

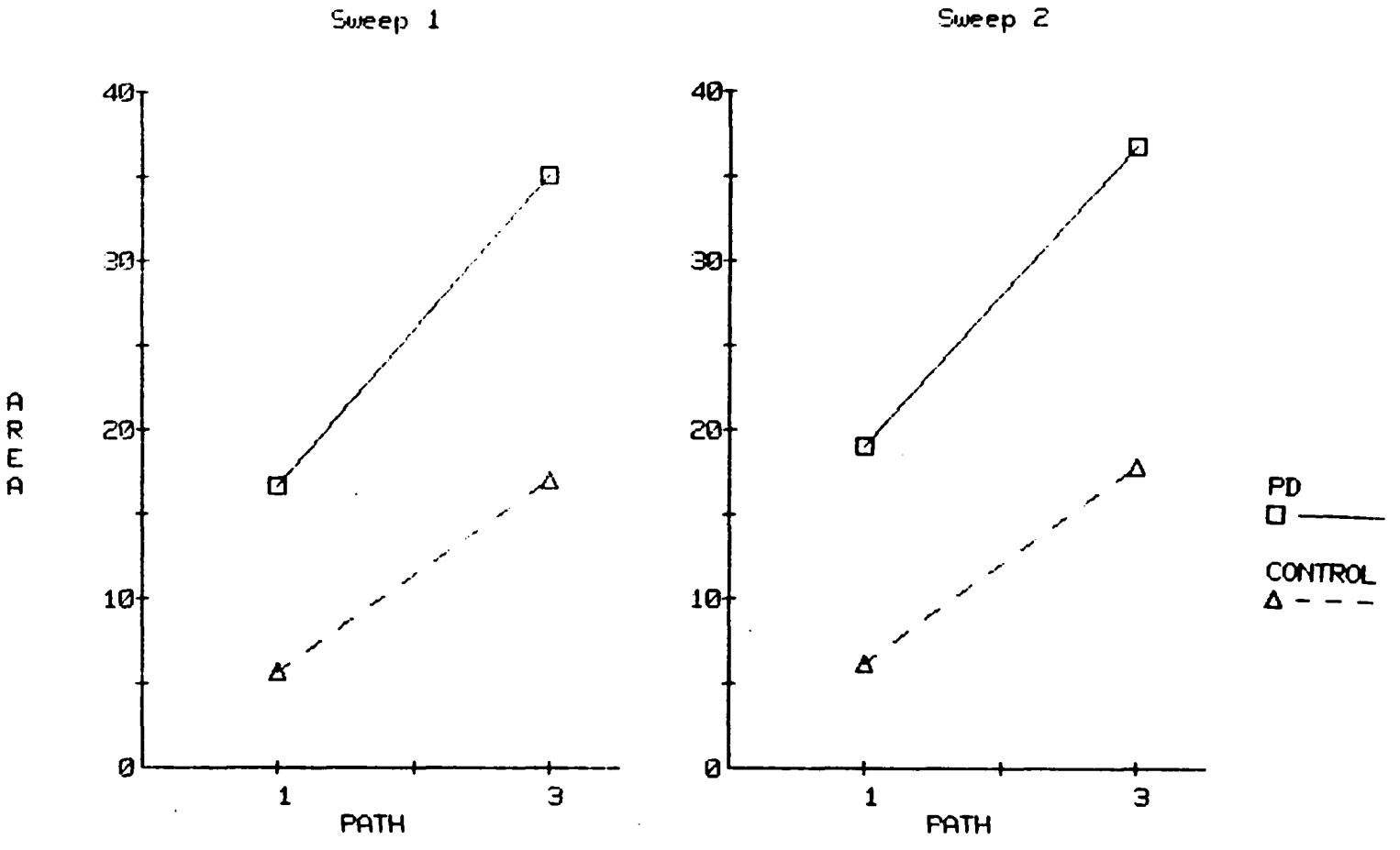

Sweep 1
Sweep 2
AREA
40
30
20
10
0
1
3
PATH
PD
CONTROL

Figure 4. Path 1 and Path 3: Total digitized points in Paths 1 and 3.

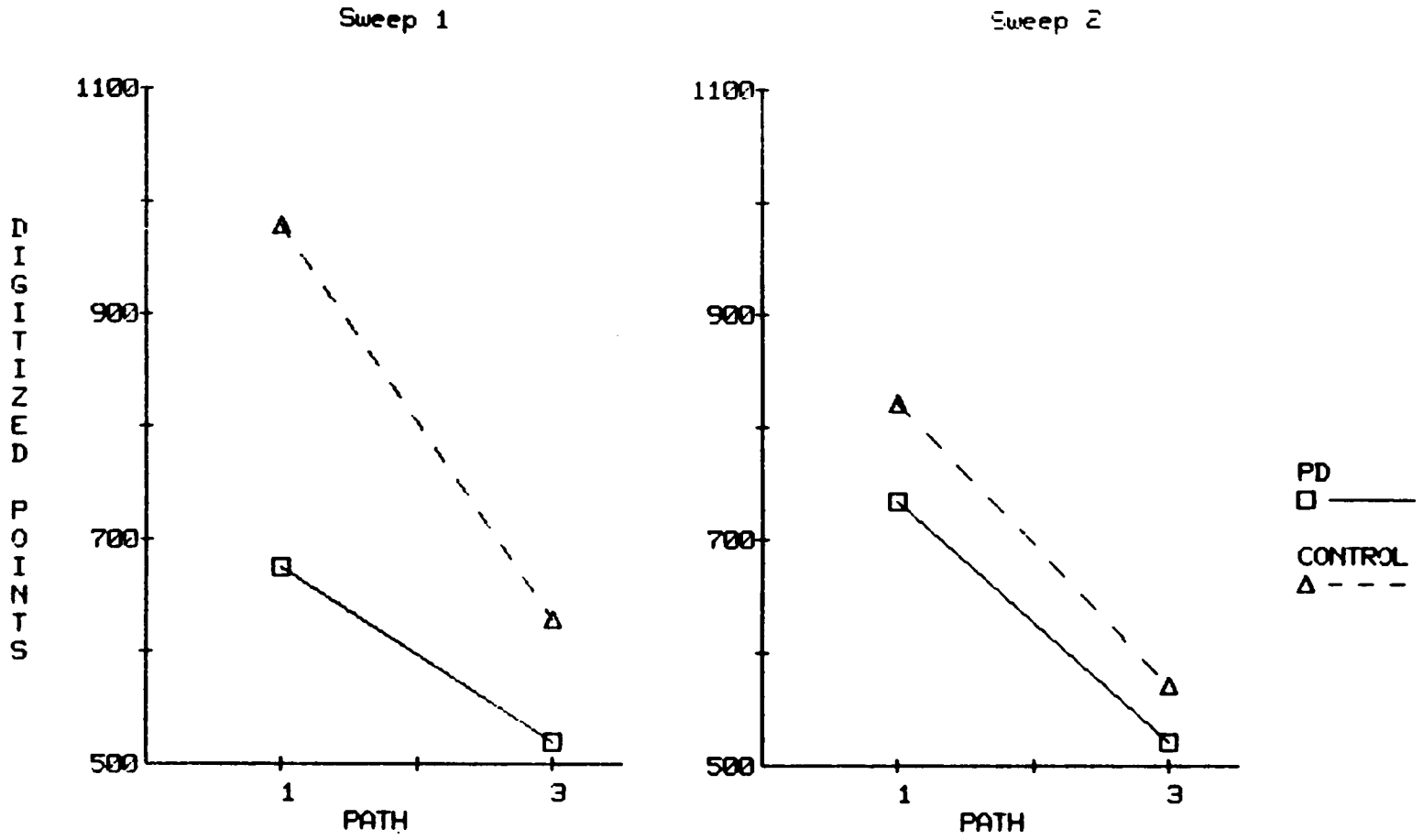

Sweep 1
Sweep 2
DIGITIZED POINTS
1100
900
700
500
1
3
PATH
PD
CONTROL

Figure 5. Sample scatter demonstrating the relationship between area error and path crossing measures of tracing error.

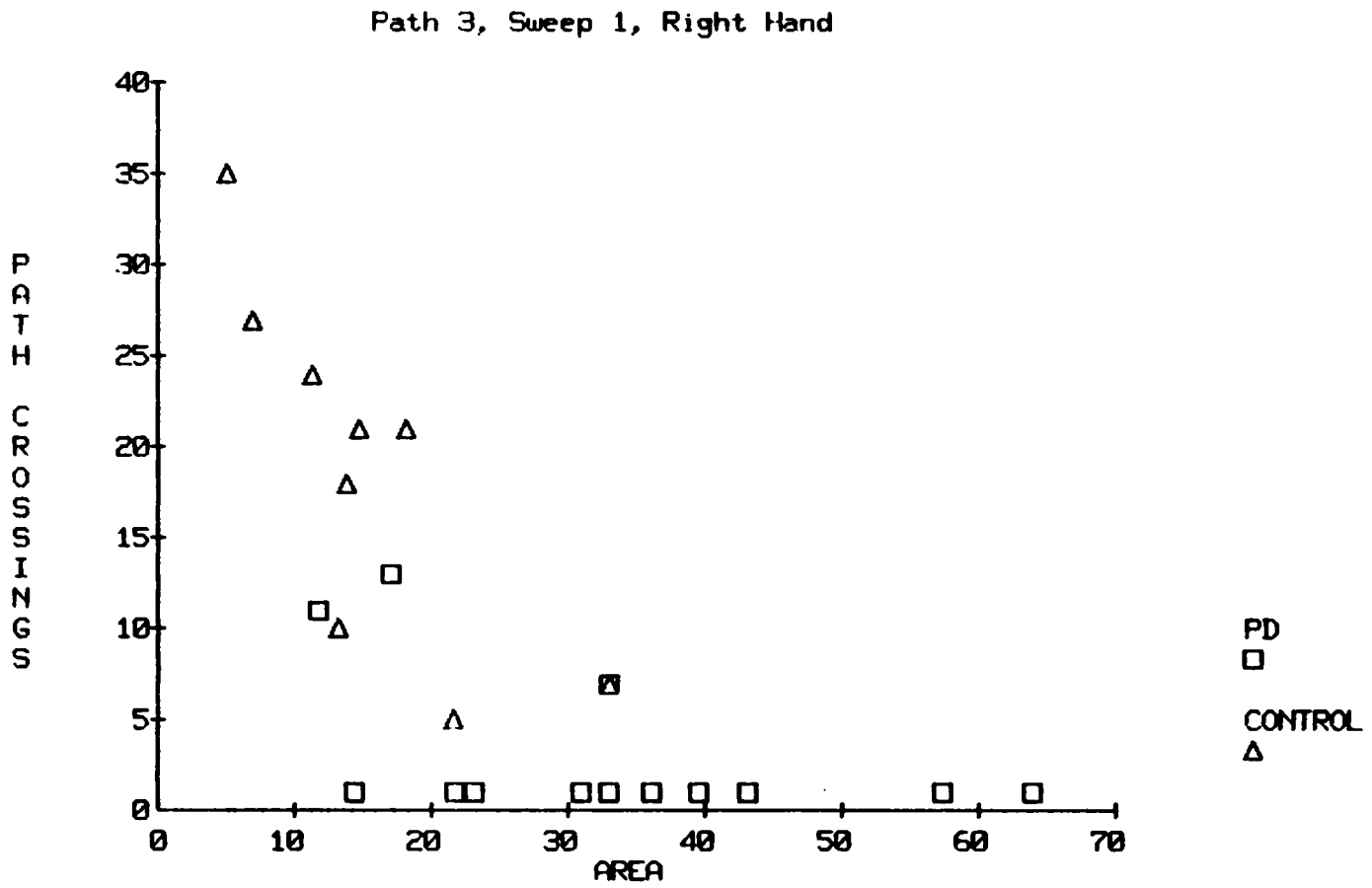

Path 3, Sweep 1, Right Hand
PATH CROSSINGS
40
35
30
25
20
15
10
5
0
0
10
20
30
40
50
60
70
AREA
PD
CONTROL

Figure 6. Path 4 and Path 5: Area error for missing segment in Path 5 and comparable portion of Path 4.

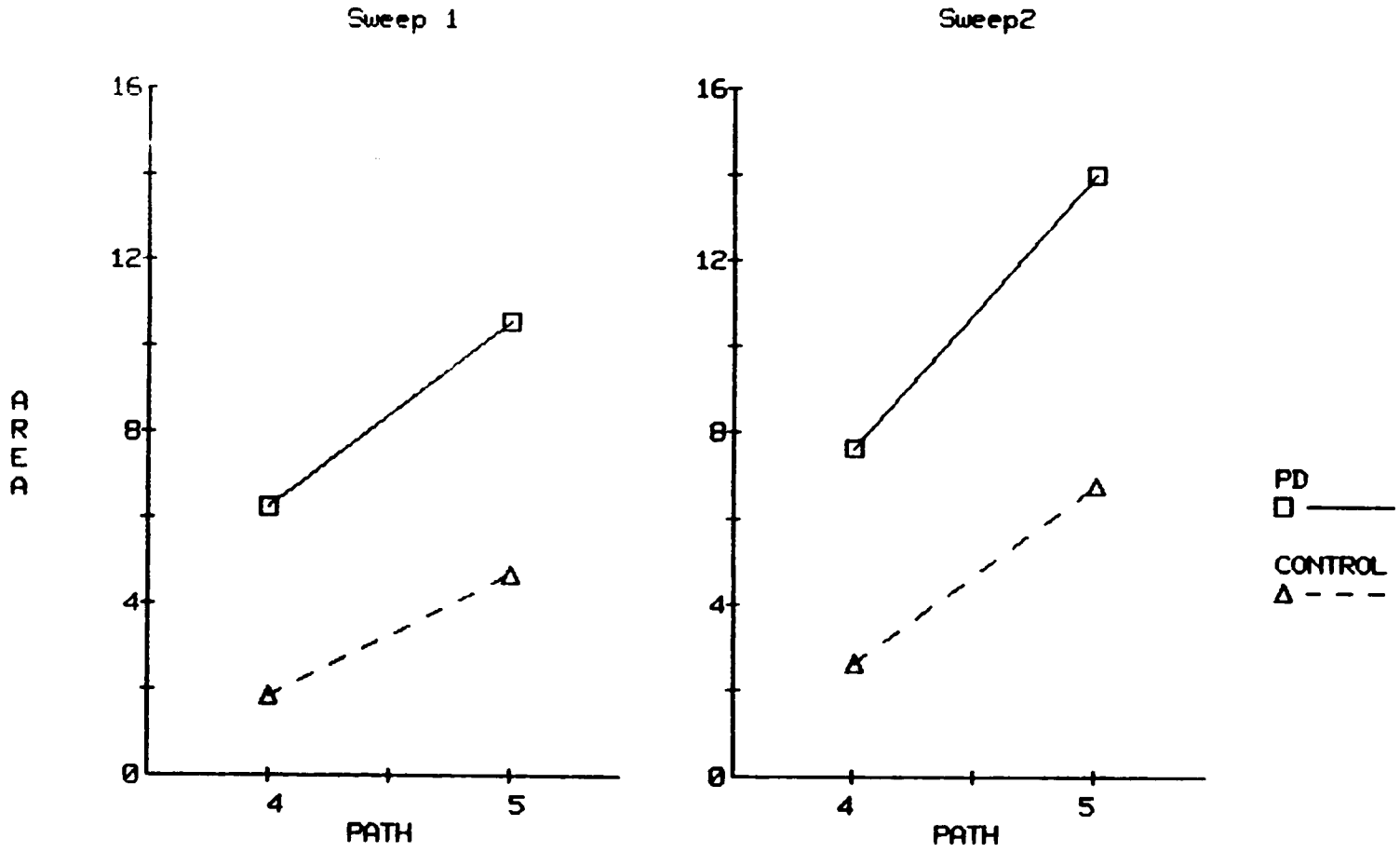

Sweep 1
Sweep2
AREA
16
12
8
4
0
4
5
PATH
PD
CONTROL

Figure 7. Path 4 and Path 5: Points digitized in missing segment of Path 5 and equivalent portion of Path 4.

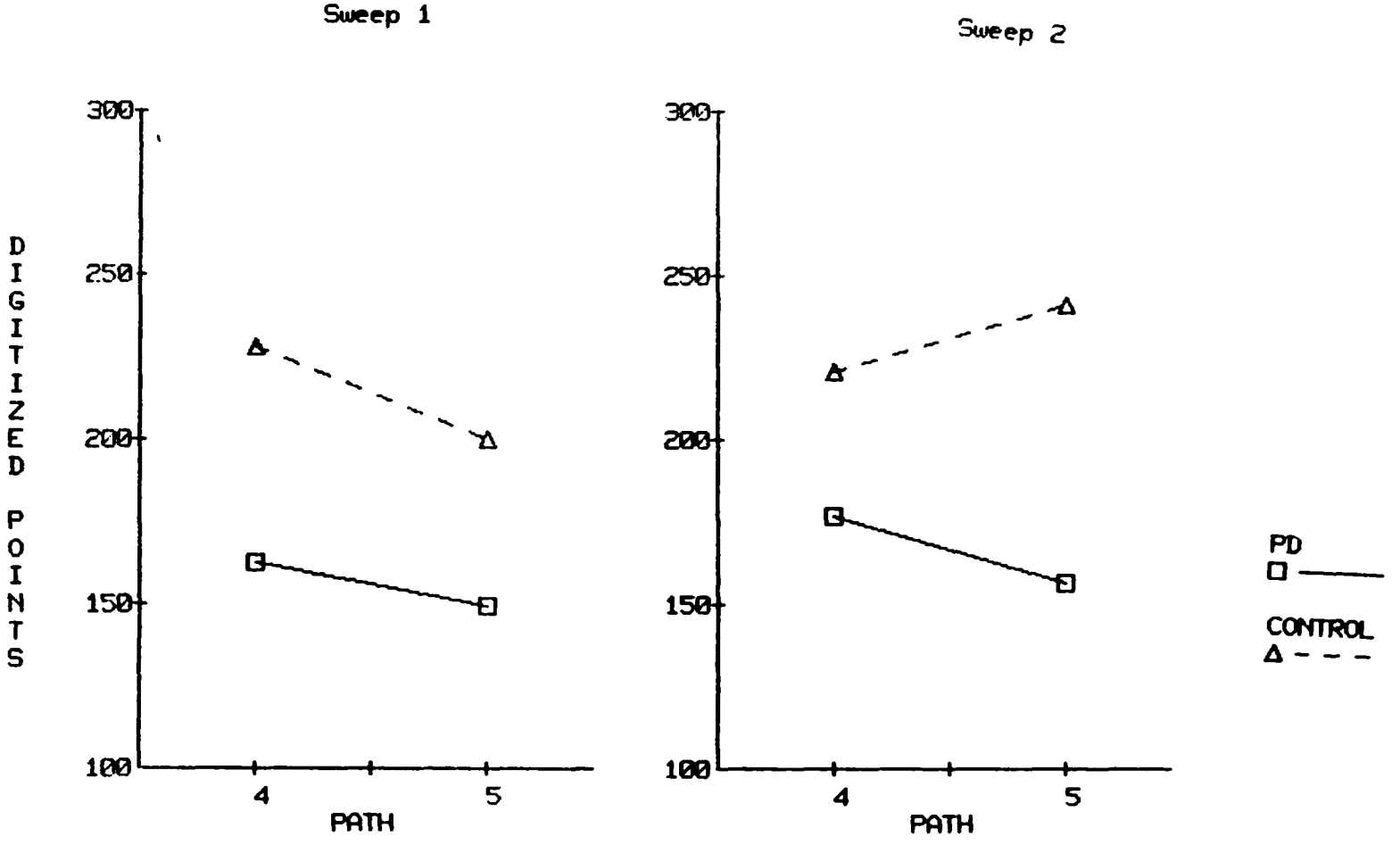

Sweep 1
Sweep 2
DIGITIZED POINTS
300
250
200
150
100
4
5
PATH
PD
CONTROL

Figure 8. Path 4 and Path 6: Area error for missing segment of Path 6 and equivalent portion of Path 4.

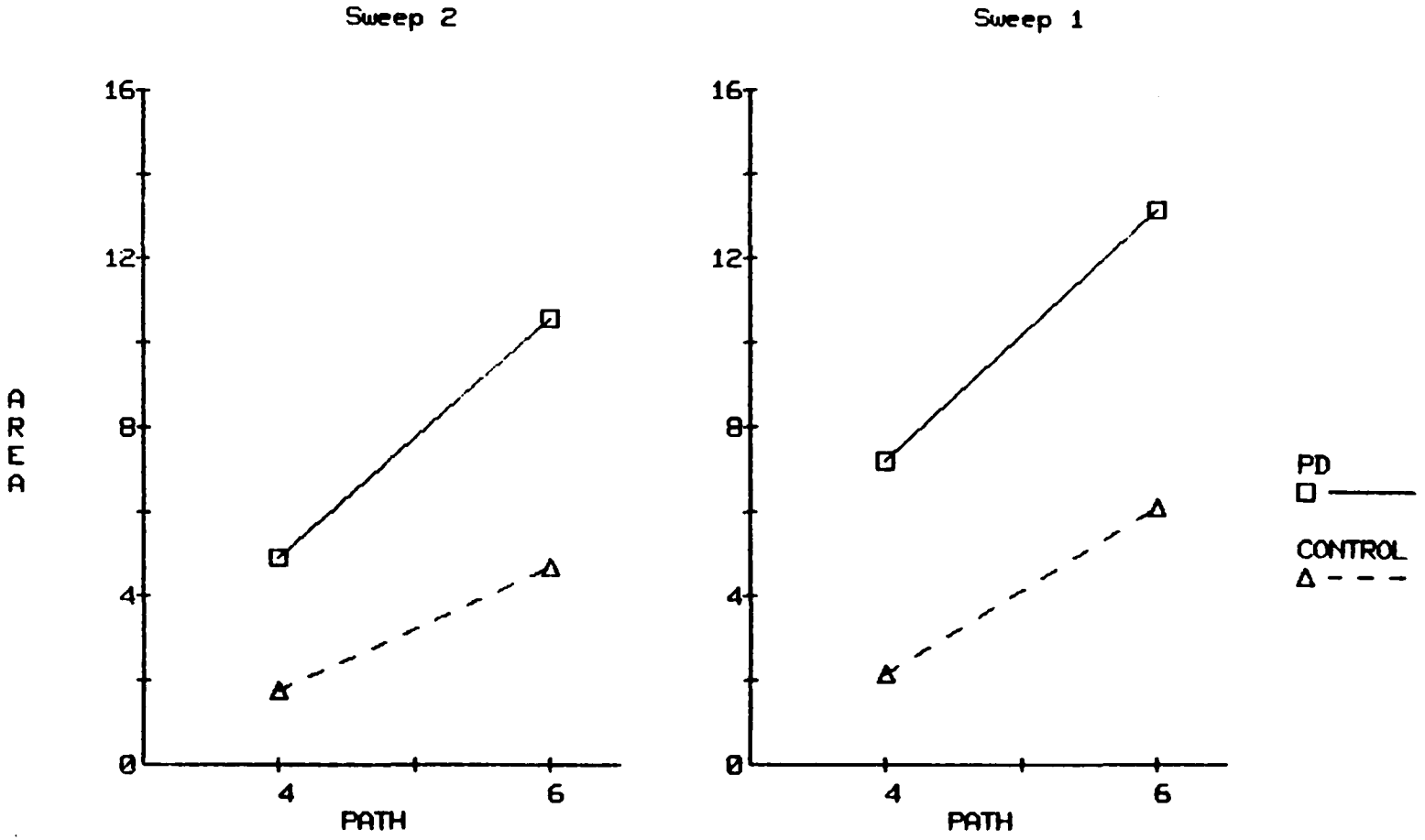

Sweep 2
Sweep 1
AREA
16
12
8
4
0
4
6
PATH
PD
CONTROL

Figure 9. Path 4 and Path 6: Points digitized in missing segment of Path 6 and equivalent portion of Path 4.

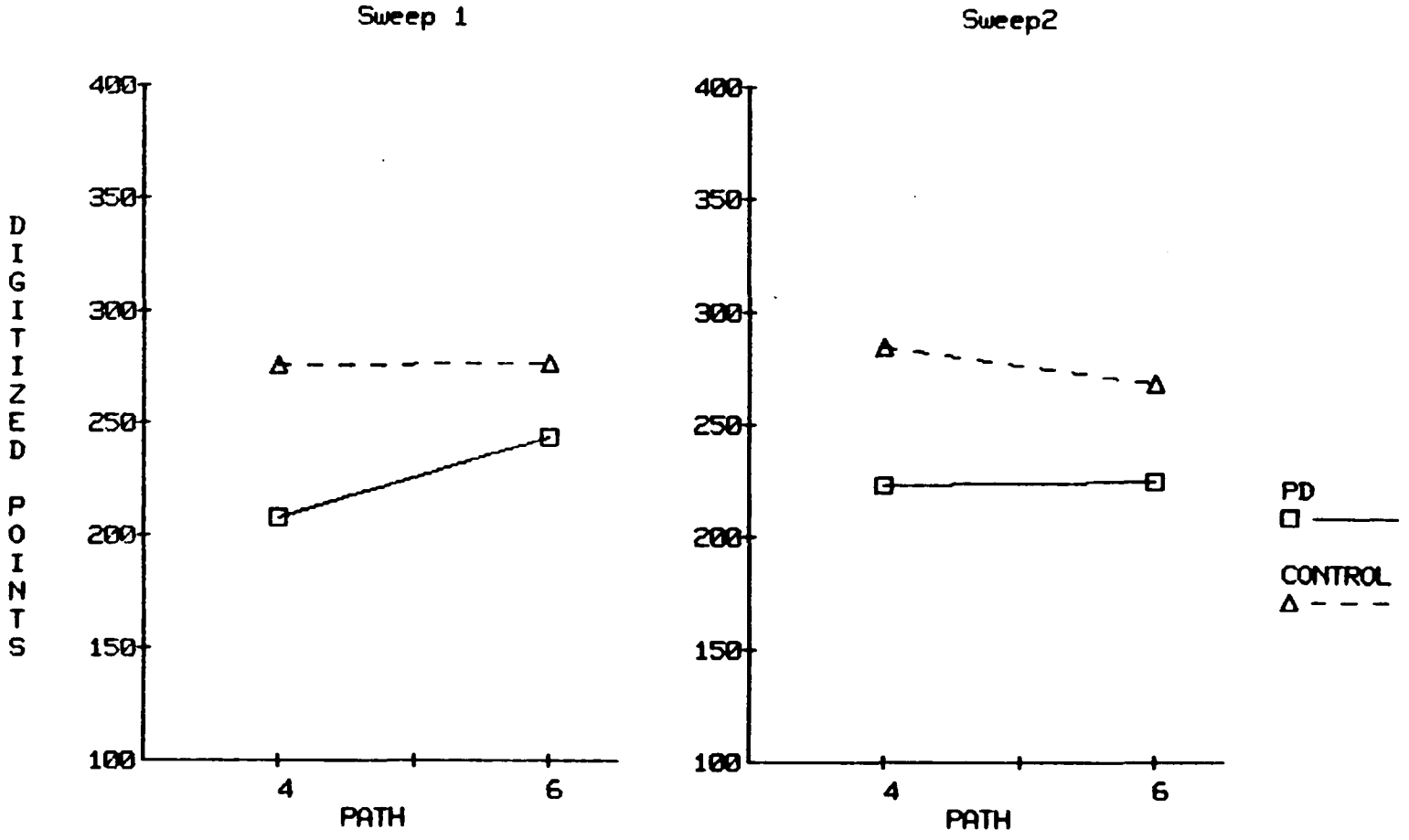

Sweep 1
Sweep2
DIGITIZED POINTS
400
350
300
250
200
150
100
4
6
PATH
PD
CONTROL

Figure 10. Path 4 and Path 7: Area error for missing segment of Path 7 and equivalent portion of Path 4.

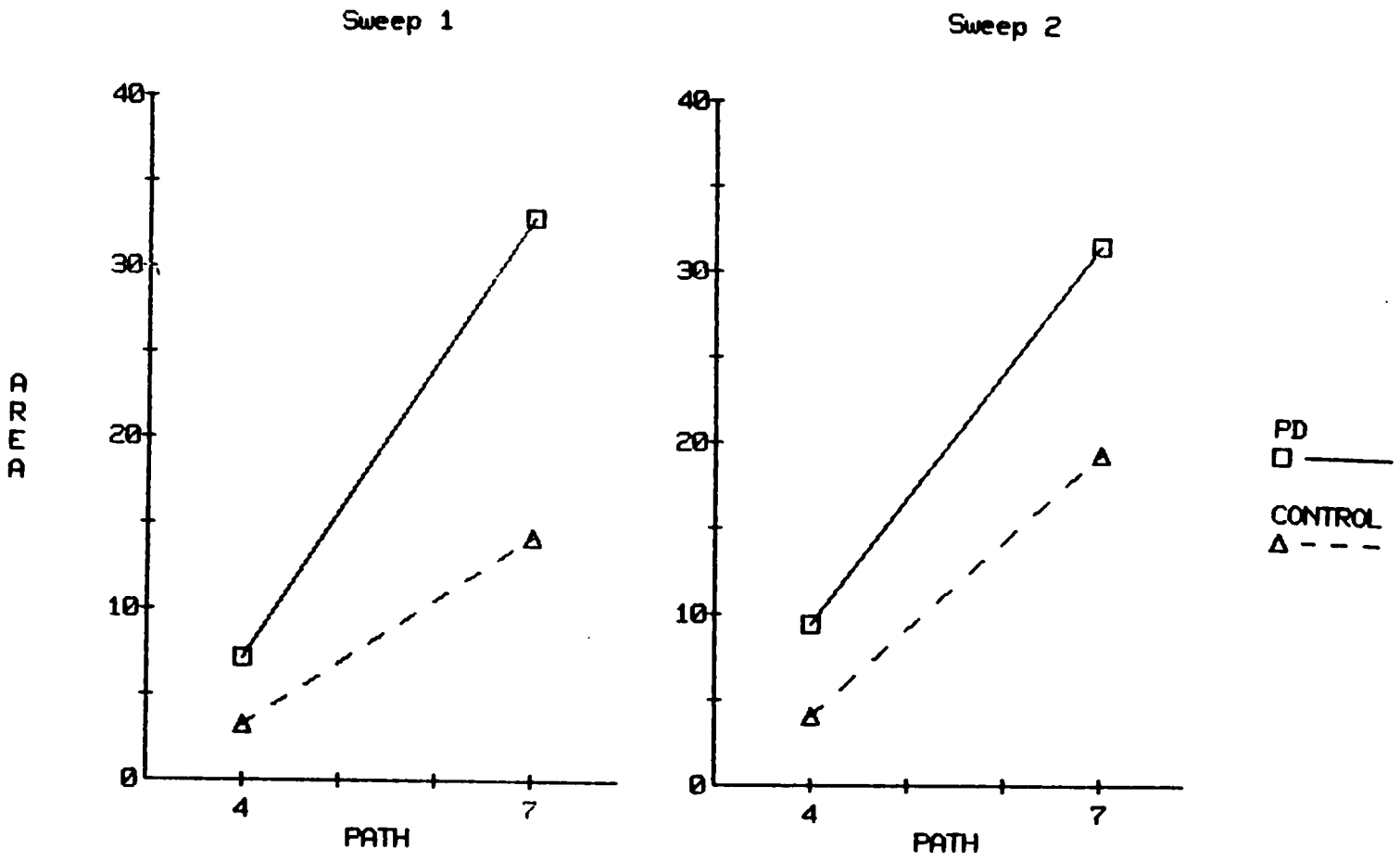

Sweep 1
Sweep 2
AREA
40
30
20
10
0
4
7
PATH
PD
CONTROL

Figure 11. Path 4 and Path 7: Points digitized in missing segment of Path 7 and equivalent portion of Path 4.

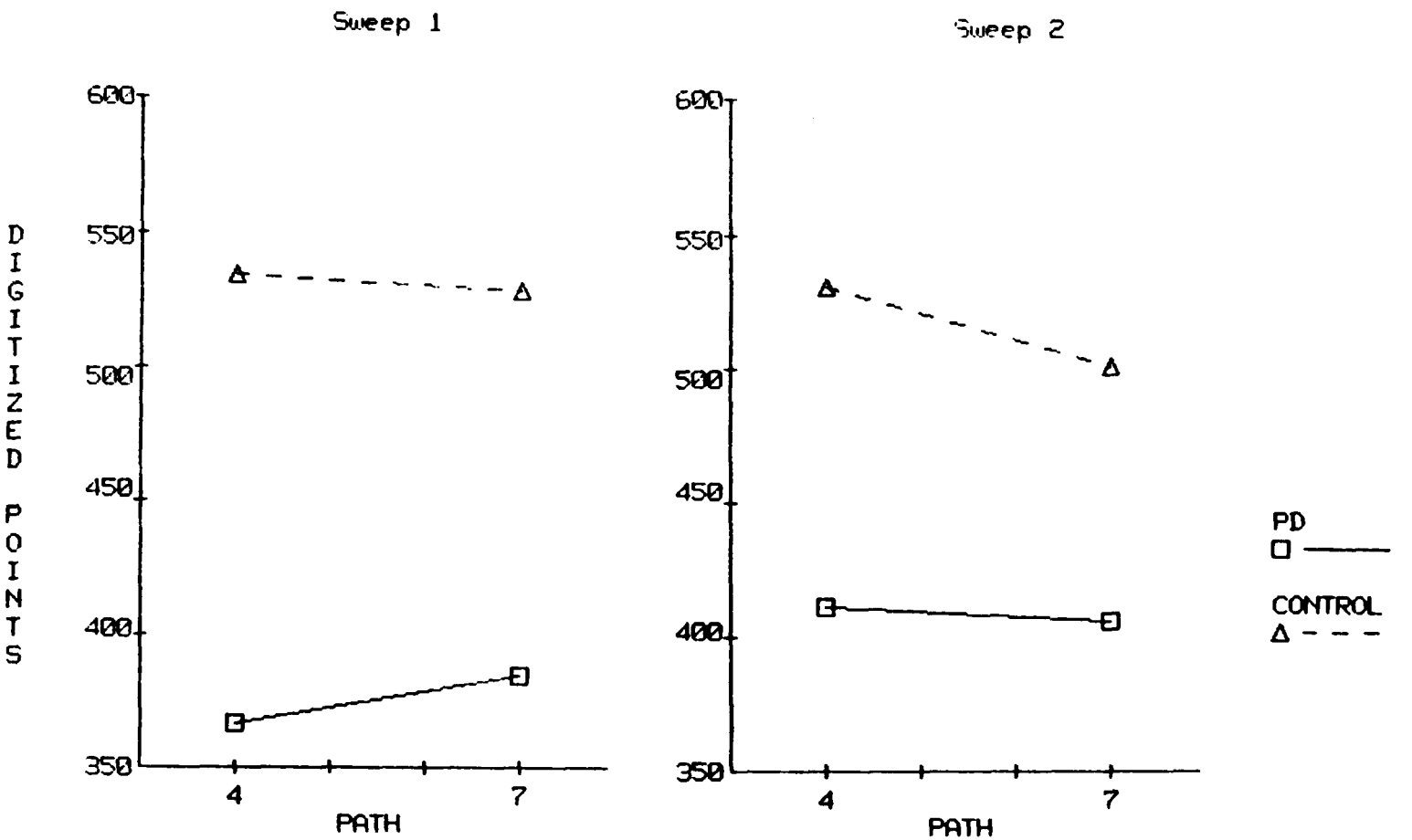

Sweep 1
Sweep 2
DIGITIZED POINTS
600
550
500
450
400
350
4
7
PATH
PD
CONTROL

Figure 12. Paths 4-7: Area error for entire sweeps of Paths 4 through 7. RH = Right Hand; LH = Left Hand; CON = Control.

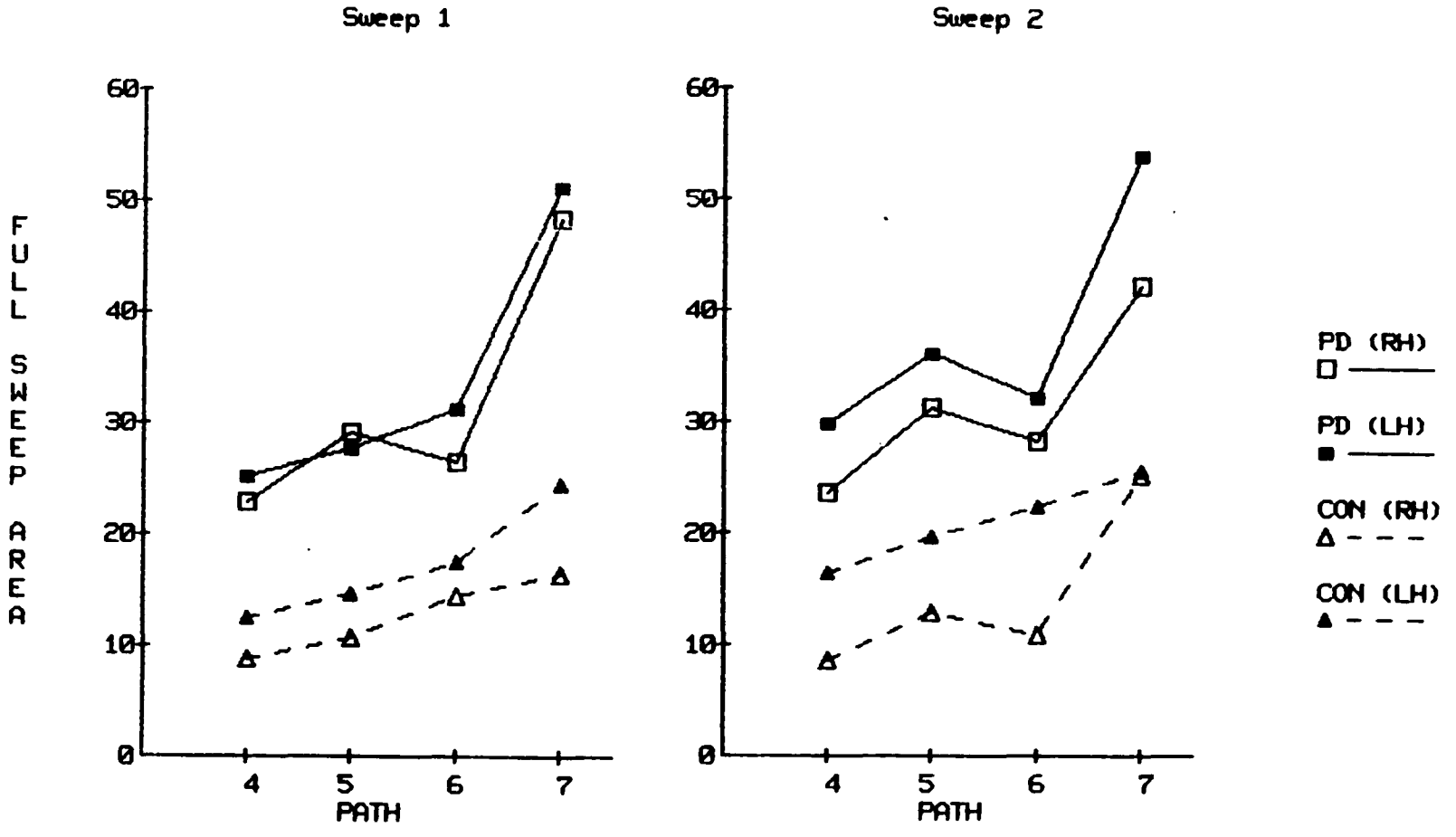
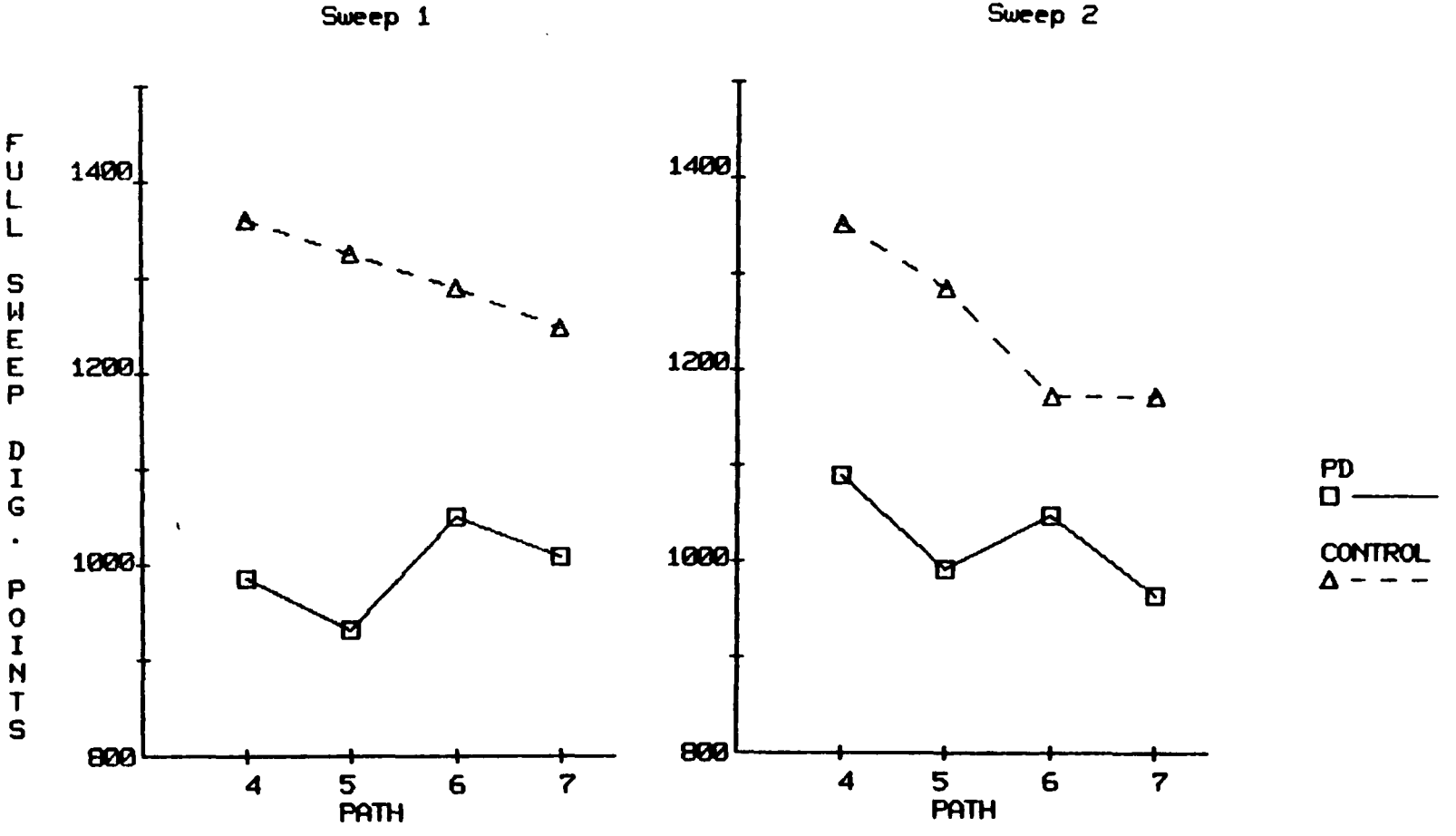

Sweep 1
Sweep 2
FULL SWEEP AREA
60
50
40
30
20
10
0
4
5
6
7
PATH
PD (RH)
PD (LH)
CON (RH)
CON (LH)

Figure 13. Paths 4-7: Points digitized for entire sweeps of Paths 4-7.


Sweep 1
Sweep 2
FULL SWEEP DIG. POINTS
1400
1200
1000
800
4
5
6
7
PATH
PD
CONTROL