

A Technique for Removal of the Visuoperceptual Component from Tracking Performance and Its Application to Parkinson's Disease

Richard D. Jones,* *Senior Member, IEEE*, Ivan M. Donaldson, and Neil B. Sharman

Abstract—Although it is well established that subjects with Parkinson's disease perform poorly on complex sensory-motor tasks, the extent to which this is due to visuoperceptual deficits is unclear. We measured the performance of 16 patients with Parkinson's disease, both on and off drugs, and 16 age and sex matched control subjects on preview and nonpreview tracking tasks and a nonmotor test of dynamic visuoperception. Order effects were controlled for by a randomized cross-over design. Performance on the perceptual task was measured in terms of *perceptual resolution* and was found impaired in the Parkinsonian group. The contribution of visuoperceptual function to tracking performance was removed using the concept of a visuoperceptual buffer-zone. The mean tracking error remained impaired on all tracking tasks and demonstrated that limitations in visuoperceptual function play only a minor role in the tracking errors in both Parkinsonian and control subjects. It is clear that the technique for determining the visuoperceptual component of performance on complex sensory-motor tasks has considerable scope for application in studies of a variety of brain disorders.

I. INTRODUCTION

IT is well established that subjects with Parkinson's disease (PD) perform poorly on complex sensory-motor tasks such as tracking [1]–[11]. This is generally considered to be due to a combination of slowness in initiating (i.e., prolonged reaction times) [3], [7], [9], [12]–[18] and executing movements (i.e., reduced speed of ballistic movements) [3], [5], [6], [12], [13], [18], and impaired motor planning [3]–[5], [11], [19]–[23]. Lack of dopamine in the basal ganglia of the brain underlies the characteristic Parkinsonian features of rigidity, bradykinesia (i.e., slowed initiation and execution of movement), and rest tremor.

In addition, there is evidence that visuoperceptual function is also impaired in PD [24]–[34] although this has not been confirmed by some studies [15], [35]–[41]. Visual sensation has also been demonstrated impaired in PD in terms of contrast sensitivity [42]–[46], low-contrast visual acuity [46], [47],

Manuscript received August 1, 1994; revised June 13, 1996. This work was supported by the Accident Compensation Corporation, the New Zealand Lotteries Board, and the Canterbury Area Health Board. *Asterisk indicates corresponding author.*

*R. D. Jones is with the Department of Medical Physics & Bioengineering, Christchurch Hospital, Christchurch, New Zealand (e-mail: r.jones@chmeds.ac.nz).

I. M. Donaldson is with the Department of Neurology, Christchurch Hospital, and the Department of Medicine, Christchurch School of Medicine, Christchurch, New Zealand.

N. B. Sharman is with the Department of Medical Physics & Bioengineering, Christchurch Hospital, Christchurch, New Zealand.

Publisher Item Identifier S 0018-9294(96)07257-6.

and high-contrast visual acuity [48]. Furthermore, we have demonstrated that visuoperception function is impaired in PD after taking the impaired visual acuity into account [34].

This raises the likelihood that poor performance on complex sensory-motor tasks in PD is partially due to impaired visual function. Before addressing this directly, it is worth reviewing techniques and approaches used elsewhere to separate and quantify various components of complex sensory-motor performance, such as visual perception, cognition, motor planning, and motor execution. Specifically, which techniques were applied in previous studies to measure and separate nonmotor functions from tasks requiring a motor response.

Reaction time is probably the most common parameter studied in sensory-motor dysfunction. The usual approach is to measure a series of reaction times using varying stimulus and/or response complexity. Thus, by changing only perceptual or cognitive elements of a task while maintaining the same motor response (such as depression of a key), the perceptual or cognitive component of total reaction time can be isolated. This approach has been applied successfully to visual perception, motor planning [7], [14], [15], [49], [50], and cognition [14] in Parkinson's disease. Analysis of covariance is an alternative approach, whereby it is possible to remove the reaction and movement times from visuoperceptual and perceptual-motor tasks dependent on a motor response [16]. Whereas the above studies compare reaction times between tasks, others have studied cognitive and motor functions by fractionating single reaction times into premotor (central) and motor (peripheral) components [9], [51].

Pursuit tracking is the major category of continuous sensory-motor task used to fractionate performance into multiple components (i.e., sensory, perceptual, cognitive, motor planning, and motor execution) [52]. Two main approaches have been previously used to isolate and quantify causes of abnormal tracking performance. The first involves breaking the ballistic response in step tracking into reaction times, movement times, overshoot, and settling time [3], [13], [53]–[55]. This allows indirect deductions about cognitive, motor planning, and motor execution functions, although the distinction between cognitive and motor elements often remains imprecise. The second approach, analogous to the primary methodology mentioned above for reaction time analysis, involves calculation of differentials in tracking performance from inter-trial alterations in target and/or controlled system dynamics. This has been successfully used to study predictive motor planning [3], [5],

[7], [11], [20], acquisition/modification of motor sets [8], and reliance on visual feedback [3], [4], [8] in Parkinson's disease. Conversely, Kondraske *et al.* have developed techniques based upon their hierarchical elemental resource model [56] which allow prediction of performance on high level tasks from performance on a number of lower level tasks [56]–[58].

No one, however, has estimated the extent to which sensory, perceptual, cognitive, planning, and execution elements are responsible for errors in a single tracking task. More specifically, in the context of this paper, no one has identified the extent to which visuoperceptual deficits can alter tracking performance.

This paper proposes an analytical technique whereby the contribution of visuoperceptual function to tracking performance can be isolated and quantified. It then describes the application of this technique to the performance of a group of Parkinsonian subjects on visuoperceptual (nonmotor) and tracking tasks.

II. APPARATUS

The system hardware comprised a PDP-11/34 computer¹ with a VT11 dynamic vector graphics system (1024 × 1024 resolution, 279 mm × 228-mm screen) for displaying test stimuli (eye-screen distance 132 cm). A steering wheel [395-mm diameter, minimal 1.0-N friction at perimeter, angular position sampled at 28.6 Hz (1/35 ms)] was used to measure subject's motor output. The dynamic perception and tracking tasks were generated and analyzed by software written in FORTRAN IV,¹ except for display of moving stimuli for which the faster MACRO assembly language was necessary.

III. TESTS

A. Tracking Tasks

The three primary pursuit tracking tasks have been described elsewhere [53], so only a summary of the seven tracking tasks is provided here. Each task lasts 120 s and subjects are instructed to maintain an arrow point on the input target signal throughout the test. Rotation of the wheel moves the arrow horizontally. The sides of the head of the arrow are 5.6 mm long and are at 51° to the 14.3-mm shaft. Six of the tasks use preview and nonpreview versions of the three target signals. In the preview mode the target waveform descends from the top of the screen giving an 8.0-s preview before reaching the level of the subject's arrow and a 1.1-s postview (Fig. 1); the task commences after the descent of an initial vertical line on the screen which precedes and merges with the target waveform. In the nonpreview mode only the current position of the target, which moves horizontally, is shown (Fig. 2). Of a number of performance measures calculated from these tasks, only the mean absolute error and the average lag of subject's response relative to target are presented in this paper. The mean absolute error is

$$\text{Error} = \frac{1}{N} \sum_{i=1}^N (|x_{\text{arrow}} - x_{\text{target}}|). \quad (1)$$

¹Since the study, the sensory-motor test battery (including performance fractionation procedures) has been redeveloped (in Turbo Pascal) to run on a 386/486 PC-based system [59], [60].

The average lag corresponds to the peak of the cross-correlation between the target and the response signals over the full tracking run, with the peak being accurately determined by the fitting of a parabola to the five highest points of the cross-correlation function.

- *Random Tracking (Preview and Nonpreview)*: The input target signal is a random waveform of 0.21 Hz bandwidth (20 dB/dec rolloff). The task requires smooth movements over a 175° range of the steering wheel corresponding to 165 mm on the screen (Figs. 1 and 2).
- *Sinusoidal Tracking (Preview and Nonpreview)*: The input target signal is a sinusoidal waveform of 0.14 Hz (= 1.28 cycles on screen in preview mode). The task requires smooth movements over a 180° range of the steering wheel corresponding to 170 mm on the screen.
- *Step Tracking (Preview and Nonpreview)*: This task comprises 32 abrupt steps alternating between displacement from and return to center screen. In the nonpreview form (Fig. 2), spatial unpredictability is present in the outward steps through four randomly distributed amplitude/direction movements (large and small steps requiring 90° and 22.5° on wheel, respectively, and both to right and left of center) with temporal unpredictability achieved via four randomly distributed durations between steps (2.8, 3.4, 4.0, and 4.6 s).
- *Combination Tracking*: In combination tracking the stimulus alternately cycles between the preview random (Fig. 1) and nonpreview step (Fig. 2) tracking modes over 11-s cycles. Thus, while tracking the random target, the preview signal is abruptly and unpredictably replaced by a stationary vertical line at a distance horizontally displaced from the preview signal. The reverse applies at the end of the step tracking mode with the reappearance of the preview random target as if it had continued invisibly during the step mode. Combination tracking allows determination of the effect on performance of translation between two tracking modes at opposite ends of the sensory-motor spectrum.

B. Dynamic Perception Task

The dynamic perception task requires only a nonpaced verbal response, thus eliminating confounding effects due to motor deficits. It intentionally bears a close resemblance to the tracking tasks (in particular random preview) so that validity of comparisons between them is maximized. For this task the subject has to state whether a computer-controlled arrow point stays perfectly on a target signal identical to that of the preview random tracking task (Fig. 1). The duration of the 20 trials decreases from 10 s to 2 s and various error offsets are simulated (Table I). The test score is the number of incorrect on-off answers over the 20 trials.

To enable estimation, and subsequent removal, of errors in dynamic visual perception from performance on tracking tasks, it is necessary to translate the ordinal score of incorrect responses into a quantitative measure of, what we have termed, *dynamic perception resolution* [34]. At each screen update (35-ms interval) during a dynamic perception trial the closest

TABLE I
DESCRIPTION OF THE 20 TRIALS IN THE DYNAMIC PERCEPTION TEST

Trial	Duration (s)	Auto-tracking error			
		Type	Offset ¹ (mm)	Disjointed sections ²	Max spacing ³ (mm)
1	10	Zero	-	None	-
2	10	Left-shift	2.18	None	2.18
3	10	right-shift	2.18	Disjoint#1	2.18
4	10	Zero	-	None	-
5	10	Lag	1.78 (70 ms)	None	1.68
6	5	Lead	1.78 (70 ms)	None	1.71
7	5	Zero	-	None	-
8	5	Sine ⁴	2.18	None	1.91
9	5	Left-shift	1.09	None	1.09
10	5	Right-shift	1.64	Disjoint#2	1.57
11	5	Zero	-	None	-
12	5	Lag	1.78 (70 ms)	None	1.62
13	2	Right-shift	2.18	None	2.09
14	2	Zero	-	None	-
15	2	Lag	1.78 (70 ms)	None	1.71
16	2	Left-shift	1.64	Disjoint#3	1.64
17	2	Sine ³	2.18	None	1.91
18	2	Zero	-	None	-
19	2	Lead	1.78 (70 ms)	Disjoint#4	1.70
20	2	Sine ³	1.64	None	1.30

¹All offsets were in integer values of pixels: One horizontal pixel = 0.273 mm, One vertical pixel = 0.223 mm.
²Disjointed errors [i.e., auto-tracking errors present for only part(s) of a trial] were as follows: Disjoint#1 = On-40%, Off-20%, On-40%; Disjoint#2 = On-20%, Off-40%, On-40%; Disjoint#3 = On-50%, Off-50%; Disjoint#4 = Off-50%, On-50%. Sharp transitions between on- and off-line sections (which would otherwise produce an undesired tell-tale *jump*) were eliminated through the use of smooth transitions.
³Maximum spacing is defined as the largest "spacing" (see text and Fig. 4) occurring during a trial.
⁴All sinusoidal auto-tracking errors had a frequency of 0.45 Hz.

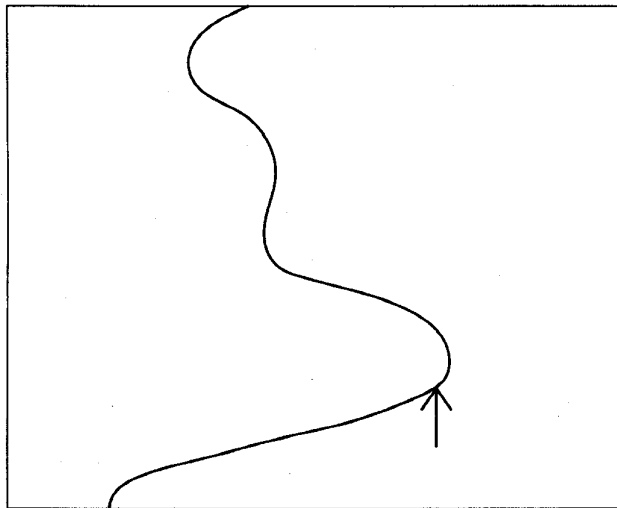


Fig. 1. Visual display for the preview tracking task (random target) and for the dynamic perception test. The arrow moves horizontally in response to movement of steering wheel by subject. The target scrolls down the screen, taking 9.1 s from top to bottom.

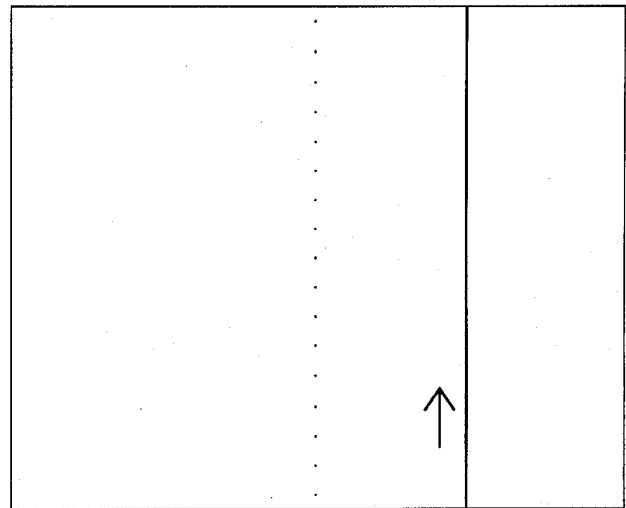


Fig. 2. Visual display for all nonpreview tracking tasks (i.e., random, sine, and step). The stationary line of dots was present only during the step tracking task and introduced spatial predictability into the *return* steps to center screen (cf. spatial unpredictability of the *outward* steps).

distance between the point of the arrow and the straight line segments making up the target waveform on the screen—that is, the *spacing*—is calculated (Fig. 3). The *maximum spacing* occurring during each trial is then determined (Fig. 4). The

dynamic perception resolution (DPR) is defined as the smallest of maximum spacings over the 20 trials (Table I) and beyond which a subject is always able to perceive the arrow as being off the target at some stage during the target's

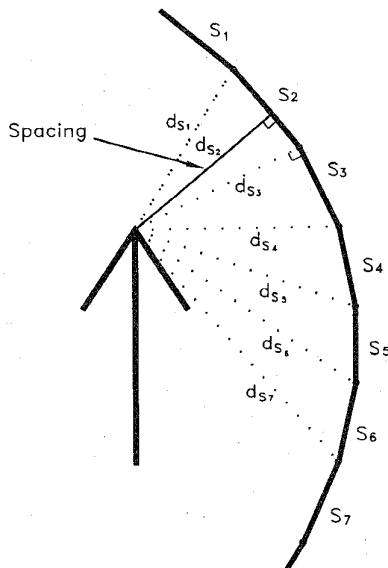


Fig. 3. Close-up of the dynamic perception test showing the shortest distances ($d_{s1} \dots d_{s_m}$) between the point of the arrow and the straight-line segments ($S_1 \dots S_m$) making up the target waveform. A shortest distance can be either perpendicular to its line segment (d_{s2}) or be to its nearest endpoint ($d_{s1}, d_{s4}, \dots, d_{s7}$) or both (d_{s3}). The *spacing* is the minimum of these distances.

descent. It should be noted that, except for inaccuracies due to quantization of the maximum spacings, a subject's DPR is independent of the length (in particular the fixed vertical component) of target segments. DPR has a test-retest reliability of 0.84 for normal subjects.

IV. REMOVAL OF VISUOPERCEPTUAL COMPONENT FROM TRACKING PERFORMANCE

Visual perception can be considered as one of several lower-level performance resources utilized during high-level sensory-motor tasks [52], [56]. Thus, on the reasonable assumption that dynamic visual perception is utilized maximally and to the same extent during tracking as it is in the dynamic perception task, it should be possible to define, and subsequently remove, the visuoperceptual contribution to overall performance on any of the tracking tasks. This can be achieved by considering the DPR to be constant during a particular session and by introducing the concept of a *visuoperceptual buffer-zone* extending either side of the tracking target. If subjects can hypothetically track the target perfectly except for visuoperceptual limitations, they will be within or, at worst, on the boundary of the visuoperceptual zone at all times. Consequently, to remove the contribution of visuoperceptual limitations from a real tracking response trajectory, each sample of the trajectory is moved toward the target by the width of the visuoperceptual zone at the level of the arrow.

In the nonpreview case, determination of the position of the visuoperceptual zone is straightforward in that it simply extends either side of target by a distance equal to the subject's DPR (Fig. 5). Each sample of the subject's arrow position is then looked at in turn. If the arrow is anywhere inside the zone (i.e., $E < \text{DPR}$, where E is the raw tracking error

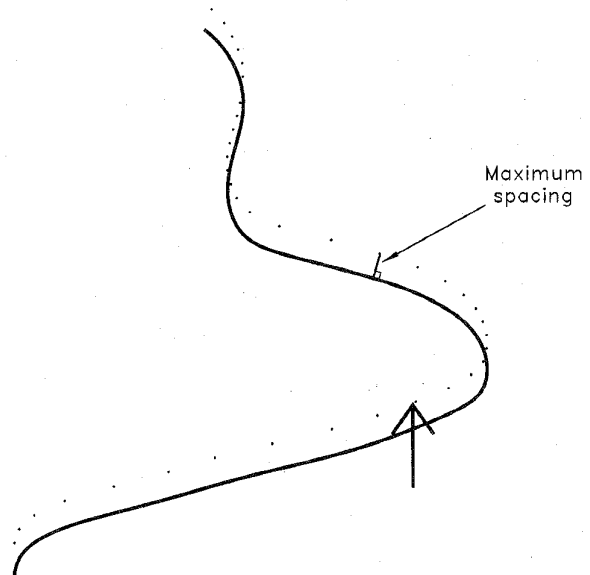


Fig. 4. A snap-shot of the dynamic perception test showing the maximum spacing for this trial. In this example, the arrow automatically followed the descending target with a constant lag (the dots indicating the arrow's trajectory were not displayed during the actual task).

$|x_{arrow} - x_{target}|$), the subject would have been unable to perceive that it was not correctly placed and so the subject's arrow is regarded as being exactly on target, resulting in a zero error. Alternatively, if the arrow is outside of the visuoperceptual zone, it is regarded as being moved toward the target by an amount equal to DPR. That is, the position of the arrow becomes

$$x_{new} = \begin{cases} x_{target} & E \leq \text{DPR} \\ x_{arrow} + \text{DPR} & E > \text{DPR} \text{ and } x_{arrow} < x_{target} \\ x_{arrow} - \text{DPR} & E > \text{DPR} \text{ and } x_{arrow} > x_{target} \end{cases} \quad (2)$$

The modified tracking output data can then be reanalyzed to give a reasonable estimate of performance equivalent to what the subject would have achieved with perfect visual acuity and perception.

A similar approach can be applied to removal of the visuoperception component from the tracking response to preview targets. However, in this case, the width of the visuoperceptual zone on the horizontal, E_{VP} , varies as one moves along the target but can be no less than DPR (Fig. 6). For example, our standard 0.14-Hz sinusoidal target has a maximum gradient of 73.5° from the vertical and, hence, a maximum E_{VP} of $3.52 \times \text{DPR}$ (i.e., $1/\cos 73.5^\circ$); likewise, our standard random target has a maximum gradient of 75.9° and hence a maximum E_{VP} of $4.10 \times \text{DPR}$. Therefore, to remove the visuoperceptual component from the response to preview targets, it is first necessary to calculate and store the visuoperceptual zone along the full length of the target. This process is equivalent to running a circle of radius DPR along the full length of the target with the area swept out being

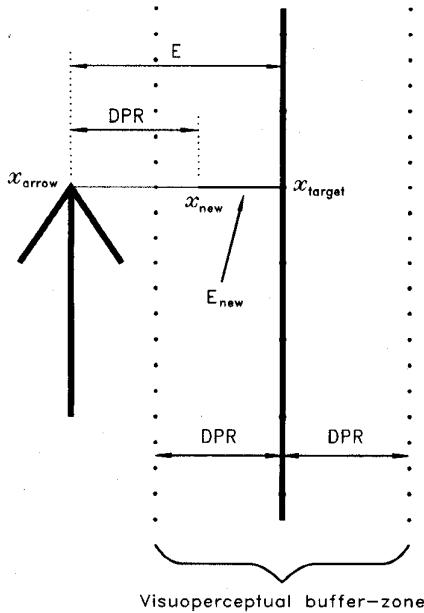


Fig. 5. Schematic of the nonpreview tracking task showing the invisible visuoperceptual zone and the DPR-compensated position, x_{new} , of the subject's response.

the visuoperceptual zone. Its implementation involves several steps.

- A) The creation of two real arrays, specifying boundaries of the visuoperceptual zone, and initialization to the target values.
- B) The generation of a circle of radius DPR but with storage only being necessary of those points corresponding to integer y -values (in vertical pixels).
- C) Stepping the circle along each point of the target and pushing visuoperceptual zone boundaries out to perimeter of circle (Fig. 8). This results in most visuoperceptual zone boundary points being in their correct final position (i.e., filled dots *A* in Fig. 7). However, because the circle is moved up in discrete steps, rather than continuously, some boundary points remain too close to the target (e.g., open dots *B* and *D* in Fig. 7); this effect is particularly noticeable for long target vectors.
- D) Pushing the boundary points out further, where necessary, to lie on tangents to DPR-circles centered at ends of consecutive target vectors (e.g., filled dots *C* and *F* in Fig. 7).

Having established the boundaries of the visuoperceptual zone, the visuoperceptual component can be removed from the tracking response as is done for the simpler nonpreview case. That is, each sample of subject's arrow position x_{arrow} is moved horizontally toward the target by a distance of up to E_{VP} at that level (Fig. 8) (cf. DPR in nonpreview case) and becomes

$$x_{new} = \begin{cases} x_{target} & E \leq E_{VP} \\ x_{arrow} + E_{VP} & E > E_{VP} \text{ and } x_{arrow} < x_{target} \\ x_{arrow} - E_{VP} & E > E_{VP} \text{ and } x_{arrow} > x_{target} \end{cases} \quad (3)$$

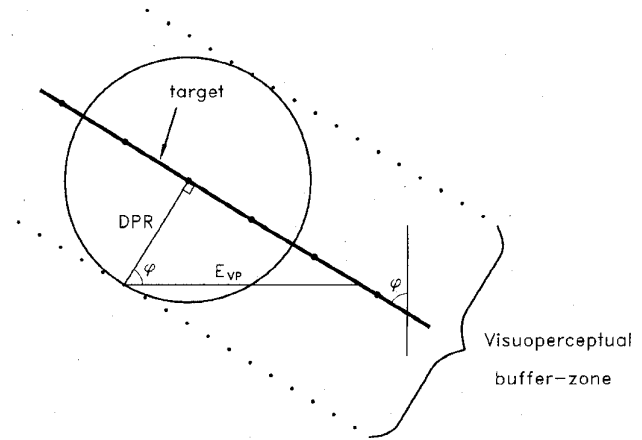


Fig. 6. Idealized case of a preview target with a constant gradient φ from the vertical on screen, in which the width of the visuoperceptual zone on the horizontal, E_{VP} , is simply $DPR/\cos(\varphi)$. This illustrates that, irrespective of the target signal, E_{VP} can be no less than DPR (e.g., when target stationary) and will often be considerably greater.

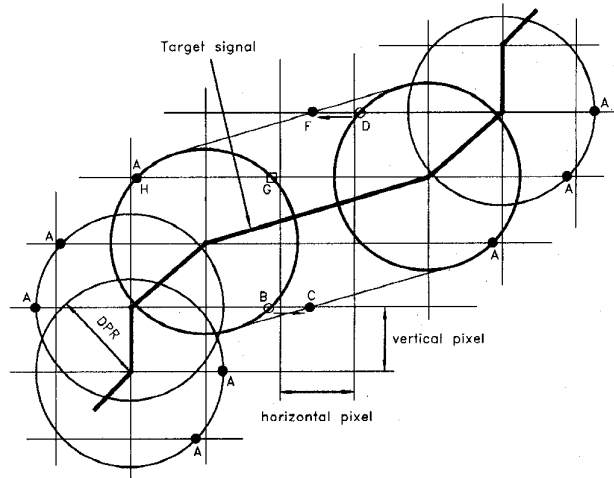


Fig. 7. Calculation of points defining boundary of visuoperceptual zone: Filled dots = correct final positions; Open dots = temporarily incorrect positions following initial "pushing" out of boundary points by circles (radius DPR) at end of target vectors but *prior* to pushing the boundary points out further, where necessary, to lie on tangents to DPR-circles centered at ends of consecutive target vectors (e.g., $B \rightarrow C$, $D \rightarrow F$). Note that the two sides of a DPR circle do not necessarily intercept the horizontal line through a target end-point on opposite sides of that end-point (e.g., *G* and *H* are both to the left of their target end-point); in this case, the boundary point closest to the target end-point is ignored (e.g., *G*).

The data can then be re-run through the standard error analysis to get "DPR-removed" values of desired performance measures.

V. EXPERIMENTAL STUDY

A. Subjects

The experimental group comprised 16 patients with PD made up of nine males and seven females. Ages ranged from 38–72 years (mean 57.2 years). All were within grades I–III on the Hoehn–Yahr scale [61] (two on I, five on II and nine on III), were not suffering from "on-off," and had no dyskinesia.

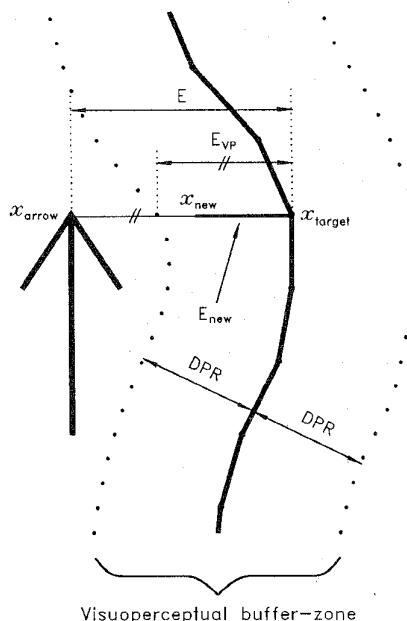


Fig. 8. Schematic of the preview tracking task showing the visuo-perceptual zone and the DPR-compensated position, x_{new} , of the subject's response calculation. E_{VP} is the width of the visuo-perceptual zone on the horizontal at the level of the point of the arrow.

Duration of illness ranged from 0.4–12 years (mean 5.5 years). All patients were being treated with either L-dopa plus a decarboxylase inhibitor (6) or an anticholinergic (7) or both (3), and supplemented in some patients by either bromocriptine (1) or amantidine (4). Patients (and controls) were included only if they had a corrected visual acuity of 6/9 or better in one eye, and no visual field defect. All patients had normal ophthalmoscopic findings, appeared mentally normal and without evidence of depression on routine clinical assessment, were right-handed, and held current driving licenses (although were not necessarily driving).

The control group comprised 16 subjects who had no neurological symptoms or history. They were matched against the PD group (using a paired experimental design) for age (range 38–74 years, mean 57.7 years, NS), sex, handedness, and driving status.

B. Procedures

Subjects were assessed clinically and quantitatively on two sessions, one week apart. The patients were on their normal drug regime for one session and off their anti-Parkinsonian medication for 24 h on the other session. Function was quantified in the right arm only. The 16 patients and their matched controls were evenly allocated to two subgroups in a randomized cross-over design to eliminate any between-session order effects (primarily learning) in determination of the effect of medication (on versus off drugs) on performance.

The nonparametric Wilcoxon matched-pairs statistic [62] was used for both between-group and within-subject comparisons due to its greater robustness over its parametric paired t-test equivalent, with only minimal loss of power. This is important due to many sensory-motor measures having very

skewed distributions as well as different variances between normal and patient groups.

VI. RESULTS

A. On Versus Off Drugs

No significant difference in performance was found between on-drugs and off-drugs on the dynamic perception test ($p = 0.72$) or any of the tracking measures ($p > 0.05$). Consequently, the following results represent averaged data from the on-drug and off-drug sessions for all subjects.

B. Dynamic Visual Perception

In terms of incorrect responses, the Parkinsonian subjects were considerably impaired on the dynamic perception test relative to the normal control group (4.87 versus 2.19, 122%, $p < 0.01$). When converted to perceptual resolutions, the scores again indicate impaired dynamic perception in the Parkinsonian group (1.84 versus 1.45 mm, 27%, $p < 0.001$).

C. Tracking Performance

In terms of overall performance (measured by mean absolute error), the PD group were worse than the control subjects on all seven tracking tasks, ranging from 24% on the nonpreview step task to as high as 118% on the preview sine task (Table II). General slowness is clearly a major contributor to their poor performance as indicated by much longer lags (Table II).

To estimate the influence of visuo-perceptual deficits to poor tracking performance in the PD group, the raw tracking data for each subject in the PD and control groups was reanalyzed following removal of the visuo-perceptual resolution (i.e., DPR). The subsequent DPR-corrected tracking error scores were, of course, smaller but remained impaired in the PD group on all seven tracking tasks (Table II). Furthermore, the differences between the two groups remained reasonably similar. This indicates that visuo-perceptual deficits play only a minor part in the poor performances of PD subjects on tracking tasks.

As with the raw data, lags were longer in the PD group than the control subjects on the DPR-corrected data on all tracking tasks. Differences between the two groups remained similar (mean differences of 88 and 102% for raw and DPR-corrected error scores, respectively) indicating that visuo-perceptual deficits are not a primary (and possibly not even a minor) cause of the slowness displayed by PD subjects on tracking tasks.

D. Functional Decomposition of Tracking Performance

Finally, it is possible to take performance on any one of the tracking tasks and break the mean error score up in to its components. This is shown, for example, for the nonpreview random task in Table III. The difference between the raw and DPR-corrected error scores of 1.69 and 1.28 mm for PD and control groups, respectively, do not, however, equal their respective DPR scores of 1.84 and 1.45 mm. This discrepancy

TABLE II
PERFORMANCE ON TRACKING TASKS IN TERMS OF MEAN ABSOLUTE ERRORS AND AVERAGE LAGS

Tracking task	Raw scores				DPR-corrected scores			
	PD	Normal	Diff (%)	p	PD	Normal	Diff (%)	p
Random (pv) - Error (mm)	7.62	3.69	107	***	5.35	2.05	161	***
- Lag (ms)	234	81.4	187	***	163	51	220	***
Random (npv) - Error (mm)	8.54	5.02	70	***	6.85	3.74	83	***
- Lag (ms)	301	169	78	***	253	142	78	***
Sine (pv) - Error (mm)	14.50	6.63	118	***	11.03	4.09	169	***
- Lag (ms)	213	94.3	126	**	161	57	182	**
Sine (npv) - Error (mm)	10.78	6.12	76	***	9.09	4.78	87	***
- Lag (ms)	165	107	54	**	137	83	65	**
Step (pv) - Error (mm)	6.50	4.20	54	**	5.24	3.41	54	*
- Lag (ms)	544	257	112	**	516	243	112	**
Step (npv) - Error (mm)	12.53	10.13	24	***	11.36	9.36	21	***
- Lag (ms)	1563	1269	23	***	1541	1262	22	***
Combination - Error (mm)	16.35	11.71	40	***	14.31	10.29	39	***
- Lag (ms)	1534	1155	33	***	1503	1120	34	***

pv = preview, npv = non-preview, DPR = dynamic perception resolution, * p<0.05, ** p<0.01, *** p<0.001.

TABLE III
FUNCTIONAL DECOMPOSITION OF RANDOM (NONPREVIEW) MEAN ABSOLUTE ERROR SCORES

Function	Parkinsonian (mm)	Normal (mm)
Visuoperceptual		
Dynamic Perception	1.84	1.45
'Inside perceptual zone'	-0.15	-0.17
	1.69 (19.8%)	1.28 (25.5%)
Lack of preview	1.50 (17.6%)	1.69 (33.7%)
Remainder (motor, etc)	5.35 (62.6%)	2.05 (40.8%)
Tracking error score	8.54	5.02

is a consequence of subjects being occasionally and unintentionally inside their perceptual buffer zone and, hence, requires the addition of "inside perceptual zone" adjustment factors of -0.15 and -0.17 mm.

Both the PD and control groups improved their error scores by 1.50 (p < 0.01) and 1.69 mm (p < 0.001), respectively, when given a preview of the target. The remaining component of the overall error scores (5.35 and 2.05 mm) must therefore

be due to limitations in one or more areas of nonvisuoperceptual cognition, motor planning (other than preview-based predictive planning), and motor execution.

VII. DISCUSSION

Sensory-motor tests of dynamic visuoperception and tracking performance have been undertaken by a group of PD subjects leading to confirmation of previous studies that visuoperceptual performance [24]–[33] and tracking performance [1]–[11] are considerably worse than that of matched controls.

The primary purpose of this paper has, however, been to propose and validate a technique for quantification and removal of the visuoperceptual component in tracking performance, specifically one-dimensional (1-D) pursuit tracking with and without preview. This procedure is particularly pertinent to the study of brain disorders in which both visuoperceptual and motor functions can be impaired at the same time, such as in PD, Huntington's disease [63], Alzheimer's disease [26], stroke [64], [65], and head injury [66].

This is the first study to demonstrate that impaired visuoperceptual function in Parkinsonian subjects is responsible for only a small part of their poor performance on tracking tasks and, by extension, complex sensory-motor tasks in general. This finding is not unexpected, considering the relative subtlety of visuoperceptual dysfunction compared with the severity of motor deficits in PD. Nevertheless, it has not been possible to objectively confirm this hypothesis previously. The much greater deficits on preview over nonpreview tracking (e.g., 166% versus 83% on Random), following removal of the visuoperceptual component, supports our earlier finding that Parkinsonian subjects are less able to make use of explicit advance information to improve performance [11]. It also demonstrates that it is unlikely this deficiency can be attributed solely to poorer visuoperception of the more complex target-response in preview tracking.

The ability to fractionate a single tracking performance into its visuoperceptual, motor planning, and motor execution components is clearly a powerful tool but its results need to be interpreted with caution. For example, the breakdown of the nonpreview random tracking errors in Table III by way of *percentages* could be misleading. Difficulties with motor execution are clearly the greatest source of the overall difference in tracking errors between the PD and control groups, as indicated by the absolute Remainder terms (5.35 and 2.05 mm, respectively) (note, however, that this study has not explicitly demonstrated that motor execution makes up the major proportion of the Remainders). As a percentage, the PD group's Remainder (i.e., 62.6% of total error score) distorts the apparent contribution to errors from other functions. Hence, although the visuoperceptual contribution to tracking errors is smaller in the control than the PD group in absolute terms (1.28 and 1.69 mm, respectively), the reverse is true of the visuoperceptual contribution as a proportion of the overall tracking error (25.5 and 19.8%).

Although there are clearly a number of areas of study of brain function/dysfunction which could substantially benefit from application of the fractionation technique described, it is important that its limitations be kept in perspective. First,

the accuracy of the perceptual resolution for a particular subject will be limited by the quantization of the maximum spacings in the dynamic perception test (see Table I). Second, it would be a gross over-simplification to suggest that visuoperception can be fully quantified by measures such as perceptual resolution [34]; other factors are clearly involved in the dynamic perception test such as arrow shape, whether arrow overlaps/crosses the target signal, and duration for which the maximum spacing is presented. Third, as described, the technique can only be applied to 1-D tracking (i.e., response marker can only move on a single axis). Nevertheless, despite these limitations, the utility of the present approach has been well demonstrated in this paper. Furthermore, with minor modification, the fractionation technique could be applied to other 1-D tracking tasks (e.g., having a response marker other than an arrow) and indeed to two-dimensional tracking tasks, with the most critical requirement being for an associated dynamic perception task which closely parallels the visual characteristics of the tracking task.

It is somewhat surprising that withdrawal of medication did not affect performance on either the visuoperceptual or tracking tasks, especially as there is incontrovertible evidence for the role of dopamine in the visual and motor pathways and for the benefits of L-dopa administration on motor function [67]. As has been discussed elsewhere for visuoperceptual function [34], [48], this may indicate that reserves of dopamine were not depleted at relevant brain sites 24 h after L-dopa withdrawal or that any drop was insufficient to affect performance.

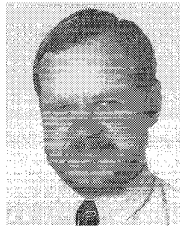
In summary, the visuoperception removal technique has considerable scope and potential for application in further studies of PD as well as a variety of other studies of brain disorders, such as stroke, head injury, Huntington's disease, and dementias, in which there are complex sensory-motor deficits. It could also be applied to study of the normal aging process in which interpretation of reduced performance on sensory-motor tasks [68]–[70] is complicated by diminished visual acuity [71], [72] and visual perception [68], [71], [73], [74]. The utility of the technique could be further enhanced by applying it with other techniques for fractionation of sensory-motor performance, such as for visuoperceptual function [34] and procedures aimed at breaking down the "Remainder" term (see Table III) into its motor execution, motor planning (other than preview), and other components. Such techniques also have considerable application in the validation and refinement of models of sensory-motor performance, such as Kondraske's elemental resource model [56] and Neilson *et al.*'s control-systems-based adaptive model theory [75].

REFERENCES

- [1] R. W. Angel, W. Alston, and J. R. Higgins, "Control of movement in Parkinson's disease," *Brain*, vol. 93, pp. 1–14, 1970.
- [2] K. Cassell, K. Shaw, and G. Stern, "A computerised tracking technique for the assessment of Parkinsonian motor disabilities," *Brain*, vol. 96, pp. 815–826, 1973.
- [3] K. A. Flowers, "Visual "closed-loop" and "open-loop" characteristics of voluntary movement in patients with Parkinsonism and intention tremor," *Brain*, vol. 99, pp. 269–310, 1976.
- [4] J. D. Cooke, J. D. Brown, and V. B. Brooks, "Increased dependence on visual information for movement control in patients with Parkinson's disease," *Canada J. Neurol. Sci.*, vol. 5, pp. 413–415, 1978.

- [5] B. L. Day, J. P. R. Dick, and C. D. Marsden, "Patients with Parkinson's disease can employ a predictive motor strategy," *J. Neurol. Neurosurg. Psychiatry*, vol. 47, pp. 1299-1306, 1984.
- [6] A. Baroni, F. Benvenuti, L. Fantini, T. Pantaleo, and F. Urbani, "Human ballistic arm abduction movements: Effects of L-dopa treatment in Parkinson's disease," *Neurol.*, vol. 34, pp. 868-876, 1984.
- [7] C. A. Bloxham, T. A. Mindel, and C. D. Frith, "Initiation and execution of predictable and unpredictable movements in Parkinson's disease," *Brain*, vol. 107, pp. 371-384, 1984.
- [8] C. D. Frith, C. A. Bloxham, and K. N. Carpenter, "Impairments in the learning and performance of a new manual skill in patients with Parkinson's disease," *J. Neurol. Neurosurg. Psychiatry*, vol. 49, pp. 661-668, 1986.
- [9] M. R. Sheridan, K. A. Flowers, and J. Hurrell, "Programming and execution of movement in Parkinson's disease," *Brain*, vol. 110, pp. 1247-1271, 1987.
- [10] T. Warabi, H. Noda, N. Yanagisawa, K. Tashiro, and R. Shindo, "Changes in sensorimotor function associated with the degree of bradykinesia of Parkinson's disease," *Brain*, vol. 109, pp. 1209-1224, 1986.
- [11] R. D. Jones and I. M. Donaldson, "Tracking tasks and the study of predictive motor planning in Parkinson's disease," in *Proc. Annu. Int. Conf. IEEE Eng. Med. Biol. Soc.*, 1989, vol. 11, pp. 1055-1056.
- [12] S. A. K. Wilson, "Some disorders of motility and of muscle tone, with special reference to the corpus striatum," *Lancet*, vol. ii, pp. 1-10, 53-62, 169-178, and 215-219, 1925.
- [13] E. V. Evarts, H. Teräväinen, and D. B. Calne, "Reaction time in Parkinson's disease," *Brain*, vol. 104, pp. 167-186, 1981.
- [14] R. D. Rafal, M. I. Posner, J. A. Walker, and F. J. Friedrich, "Cognition and the basal ganglia: Separating mental and motor components of performance in Parkinson's disease," *Brain*, vol. 107, pp. 1083-094, 1984.
- [15] G. E. Stelmach, J. G. Phillips, and A. W. Chau, "Visuo-spatial processing in Parkinsonians," *Neuropsychologica*, vol. 27, pp. 485-493, 1989.
- [16] F. Girotti, P. Soliveri, F. Carella, G. Geminiani, G. Aiello, and T. Caraceni, "Role of motor performance in cognitive processes of Parkinsonian patients," *Neurology*, vol. 38, pp. 537-540, 1988.
- [17] S. L. Pullman, R. L. Watts, J. L. Juncos, T. N. Chase, and J. N. Sanes, "Dopaminergic effects on simple and choice reaction time performance in Parkinson's disease," *Neurology*, vol. 38, pp. 249-254, 1988.
- [18] S. L. Pullman, R. L. Watts, J. L. Juncos, and J. N. Sanes, "Movement amplitude choice reaction time performance in Parkinson's disease may be independent of dopaminergic status," *J. Neurol. Neurosurg. Psychiatry*, vol. 53, pp. 279-283, 1990.
- [19] K. A. Flowers, "Some frequency response characteristics of Parkinsonism on pursuit tracking," *Brain*, vol. 101, pp. 19-34, 1978.
- [20] ———, "Lack of prediction in the motor behavior of Parkinsonism," *Brain*, vol. 101, pp. 35-52, 1978.
- [21] Y. Stern, R. Mayeux, J. Rosen, and J. Ilson, "Perceptual motor dysfunction in Parkinson's disease: A deficit in sequential and predictive voluntary movement," *J. Neurol. Neurosurg. Psychiatry*, vol. 46, pp. 145-151, 1983.
- [22] Y. Stern, R. Mayeux, and J. Rosen, "Contribution of perceptual motor dysfunction to construction and tracing disturbances in Parkinson's disease," *J. Neurol. Neurosurg. Psychiatry*, vol. 47, pp. 983-989, 1984.
- [23] C. Robertson and K. A. Flowers, "Motor set in Parkinson's disease," *J. Neurol. Neurosurg. Psychiatry*, vol. 53, pp. 583-592, 1990.
- [24] F. Boller, D. Passafiume, N. C. Keefe, K. Rogers, L. Morrow, and Y. Kim, "Visuospatial impairment in Parkinson's disease: Role of perceptual and motor factors," *Arch. Neurol.*, vol. 41, pp. 485-490, 1984.
- [25] X. L. Blonder, R. E. Gur, R. C. Gur, A. J. Saykin, and H. I. Hurtig, "Neuropsychological functioning in hemiparkinsonism," *Brain. Cogn.*, vol. 9, pp. 244-257, 1989.
- [26] E. Mohr, I. Litvan, J. Williams, P. Fedio, and T. N. Chase, "Selective deficits in Alzheimer and Parkinsonian dementia: Visuospatial function," *Canada J. Neurol. Sci.*, vol. 17, pp. 292-297, 1990.
- [27] E. Mohr, J. Juncos, C. Cox, I. Litvan, P. Fedio, and T. N. Chase, "Selective deficits in cognition and memory in high-functioning Parkinsonian patients," *J. Neurol. Neurosurg. Psychiatry*, vol. 53, pp. 603-606, 1990.
- [28] B. E. Levin, M. M. Llabre, W. J. Weiner, J. Sanchez-Ramos, C. Singer, and M. C. Brown, "Visuospatial impairment in Parkinson's disease," *Neurol.*, vol. 41, pp. 365-369, 1991.
- [29] G. Ransmayr, B. Schmidhuber-Eiler, E. Karamat, S. Engler-Ploer, W. Poewe, and K. Leidlmaier, "Visuoperception and visuospatial and visuorotational performance in Parkinson's disease," *J. Neurol.*, vol. 235, pp. 99-101, 1987.
- [30] V. A. Bradley, J. L. Welsh, and D. J. Dick, "Visuospatial working memory in Parkinson's disease," *J. Neurol. Neurosurg. Psychiatry*, vol. 52, pp. 1228-1235, 1989.
- [31] S. A. Raskin, J. C. Borod, J. Wasserstein, I. Bodis-Wollner, L. Coscia, and M. D. Yahr, "Visuospatial orientation in Parkinson's disease," *Int. J. Neurosci.*, vol. 51, pp. 9-18, 1990.
- [32] S. D. Netherton, J. W. Elias, N. N. Albrecht, C. Acosta, J. T. Hutton, and J. W. Albrecht, "Changes in the performance of parkinsonian patients and normal aged on the Benton Visual Retention Test," *Exp. Aging Res.*, vol. 15, pp. 13-18, 1989.
- [33] D. Testa, V. Fetoni, P. Soliveri, M. Musicco, E. Palazzini, and F. Girotti, "Cognitive and motor performance in multiple system atrophy and Parkinson's disease compared," *Neuropsychologica*, vol. 31, pp. 207-210, 1993.
- [34] R. D. Jones and I. M. Donaldson, "Fractionation of visuoperceptual dysfunction in Parkinson's disease," *J. Neurolog. Sci.*, vol. 131, pp. 43-50, 1995.
- [35] R. G. Brown and C. D. Marsden, "Visuospatial function in Parkinson's disease," *Brain*, vol. 109, pp. 987-1002, 1986.
- [36] S. Sala, G. di Lorenzo, A. Giordano, and H. Spinnler, "Is there a specific visuo-spatial impairment in Parkinsonians?," *Neurol.*, vol. 49, pp. 1258-1265, 1986.
- [37] B. E. Levin, M. M. Llabre, and W. J. Weiner, "Cognitive impairments associated with early Parkinson's disease," *Neurol.*, vol. 39, pp. 557-561, 1989.
- [38] J. A. Cooper, H. J. Sagar, N. Jordan, N. S. Harvey, and E. V. Sullivan, "Cognitive impairment in early, untreated Parkinson's disease and its relationship to motor disability," *Brain*, vol. 114, pp. 2095-2122, 1991.
- [39] I. Daum and N. Quinn, "Reaction times and visuospatial processing in Parkinson's disease," *J. Clin. Exp. Neuropsychol.*, vol. 13, pp. 972-982, 1991.
- [40] R. S. Wilson, D. W. Gilley, C. M. Tanner, and C. G. Goetz, "Ideational fluency in Parkinson's disease," *Brain Cognition*, vol. 20, pp. 236-244, 1992.
- [41] M. Richards, L. J. Cote, and Y. Stern, "The relationship between visuospatial ability and perceptual motor function in Parkinson's disease," *J. Neurol. Neurosurg. Psychiatry*, vol. 56, pp. 400-406, 1993.
- [42] W. Skrandies and I. Gottlob, "Alterations of visual contrast sensitivity in Parkinson's disease," *Hum. Neurobiol.*, vol. 5, pp. 255-259, 1986.
- [43] C. Bulens, J. D. Meerwaldt, G. J. van der Wildt, and C. J. Keemink, "Contrast sensitivity in Parkinson's disease," *Neurol.*, vol. 36, pp. 1121-1125, 1986.
- [44] C. Bulens, J. D. Meerwaldt, and G. J. van der Wildt, "Effect of stimulus orientation on contrast sensitivity in Parkinson's disease," *Neurology*, vol. 38, pp. 76-81, 1988.
- [45] I. Bodis-Wollner, M. S. Marx, S. Mitra, P. Bobak, L. Mylin, and M. Yahr, "Visual dysfunction in Parkinson's disease. Loss in spatiotemporal contrast sensitivity," *Brain*, vol. 110, pp. 1675-1698, 1987.
- [46] D. Regan and C. Maxner, "Orientation-selective visual loss in patients with Parkinson's disease," *Brain*, vol. 110, pp. 415-432, 1987.
- [47] D. Regan and D. Neima, "Low-contrast letter charts in early diabetic retinopathy, ocular hypertension, glaucoma, and Parkinson's disease," *Brit. J. Ophthalmol.*, vol. 68, pp. 885-889, 1984.
- [48] R. D. Jones, I. M. Donaldson, and P. L. Timmings, "Impairment of high-contrast visual acuity in Parkinson's disease," *Mov. Dis.*, vol. 7, pp. 232-238, 1992.
- [49] C. A. Bloxham, D. J. Dick, and M. Moore, "Reaction times and attention in Parkinson's disease," *J. Neurol. Neurosurg. Psychiatry*, vol. 50, pp. 1178-1183, 1987.
- [50] G. E. Stelmach, C. J. Worrington, and E. A. Strand, "Movement preparation in Parkinson's disease: The use of advance information," *Brain*, vol. 109, pp. 1179-1194, 1986.
- [51] J. C. Anson, "Fractionated simple reaction time as a function of movement direction and level of pre-stimulus muscle tension," *Int. J. Neurosci.*, vol. 35, p. 140, 1987.
- [52] R. D. Jones, "Measurement of sensory-motor control performance capacities," in *The Biomedical Engineering Handbook*, J. D. Bronzino, Ed. Boca Raton, FL: CRC, 1995, ch. 145, pp. 2187-2208.
- [53] R. D. Jones and I. M. Donaldson, "Measurement of sensory-motor integrated function in neurological disorders: Three computerized tracking tasks," *Med. Biol. Eng. Comput.*, vol. 24, pp. 536-540, 1986.
- [54] G. V. Kondraske, K. Behbehani, M. Chwialkowski, R. Richmond, W. Von Maltzahn, S. S. Smith, and V. Mooney, "A system for human performance measurement," *IEEE Eng. Med. Biol. Mag.*, vol. 7, pp. 23-28, 1988.
- [55] K. Behbehani, G. V. Kondraske, and J. R. Richmond, "Investigation of upper extremity visuomotor control performance measures," *IEEE Trans. Biomed. Eng.*, vol. 35, pp. 518-525, 1988.

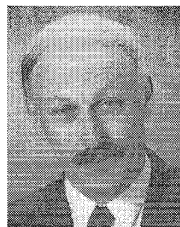
- [56] G. V. Kondraske, "A working model for human system-task interfaces," in *The Biomedical Engineering Handbook*, J. D. Bronzino, Ed. Boca Raton, FL: CRC, 1995, ch. 143, pp. 2157-2174.
- [57] ———, "Computation of functional capacity: Strategy and example for shoulder," in *Proc. Annu. Int. Conf. IEEE Eng. Med. Biol. Soc.*, 1987, vol. 9, pp. 477-478.
- [58] P. J. Vasta and G. V. Kondraske, "Performance prediction of an upper extremity reciprocal task using nonlinear causal resource analysis," in *Proc. Annu. Int. Conf. IEEE Eng. Med. Biol. Soc.*, 1994, vol. 16.
- [59] R. D. Jones, N. B. Sharman, R. W. Watson, and S. R. Muir, "A PC-based battery of tests for quantitative assessment of upper-limb sensory-motor function in brain disorders," in *Proc. Annu. Int. Conf. IEEE Eng. Med. Biol. Soc.*, 1993, vol. 15, pp. 1414-1415.
- [60] ———, "A PC-based battery of tests for measurement of sensory-motor function in brain disorders," in *Proc. 1st Med. Eng. Week World Conf.*, Taipei, Taiwan, 1994, vol. 1, pp. 223-226.
- [61] M. M. Hoehn and M. D. Yahr, "Parkinsonism: Onset, progression, and mortality," *Neurol.*, vol. 17, pp. 427-442, 1967.
- [62] S. Siegel and N. J. Caspellan, *Nonparametric Statistics for the Behavioral Sciences*, 2nd ed. New York: McGraw-Hill, 1988.
- [63] E. Mohr, P. Brouwers, J. J. Claus, U. M. Mann, P. Fedio, and T. N. Chase, "Visuospatial cognition in Huntington's disease," *Mov. Dis.*, vol. 6, pp. 127-132, 1991.
- [64] R. D. Jones, I. M. Donaldson, and P. J. Parkin, "Impairment and recovery of ipsilateral sensory-motor function following unilateral cerebral infarction," *Brain*, vol. 112, pp. 113-132, 1989.
- [65] K. J. Ottenbacher and S. Jannell, "The results of clinical trials in stroke rehabilitation research," *Arch. Neurol.*, vol. 50, pp. 37-44, 1993.
- [66] M. Rosenbaum, N. Lipsitz, J. Abraham, and T. Najenson, "A description of an intensive treatment project for the rehabilitation of severely brain-injured soldiers," *Scand. J. Rehab. Med.*, vol. 10, pp. 1-6, 1978.
- [67] "New strategies in the treatment of Parkinson's disease," in *Acta Neurol. Scand.*, U. K. Rinne, H. Pakkenberg, and N. O. Jensen, Eds. 1989, supp. no. 126.
- [68] R. D. Jones, L. R. T. Williams, and J. E. Wells, "Effects of laterality, sex, and age on computerized sensory-motor tests," *J. Hum. Mot. Stud.*, vol. 12, pp. 163-182, 1986.
- [69] A. R. Potvin, K. Syndulko, W. W. Tourtellotte, J. A. Lemmon, and J. H. Potvin, "Human neurologic function and the aging process," *J. Amer. Geriatr. Soc.*, vol. 28, pp. 1-9, 1980.
- [70] R. A. Schmidt, *Motor Control and Learning: A Behavioral Emphasis*. Champaign, IL: Human Kinetics, 1982.
- [71] M. C. Cristarella, "Visual functions of the elderly," *Amer. J. Occupat. Therapy.*, vol. 31, pp. 432-440, 1977.
- [72] P. W. Weymouth, "The effect of age on visual acuity," in *Vision of the Aging Patient*, M. T. Hirsch and R. E. Wick, Eds. Chilton: Radnor, 1960.
- [73] P. F. Farver and T. Farver, "Performance of normal older adults on tests designed to measure parietal lobe functions," *Amer. J. Occupational Therapy*, vol. 36, pp. 444-449, 1982.
- [74] J. Botwinick, "Neuropsychology of aging," in *Handbook of Clinical Neuropsychology*, S. B. Filskov and T. Boll, Eds. New York: Wiley, 1981.
- [75] P. D. Neilson, M. D. Neilson, and N. J. O'Dwyer, "Adaptive model theory: Application to disorders of motor control," in *Approaches to the Study of Motor Control and Learning*, Advances in Psychology Series, J. J. Summers, Ed. Amsterdam, The Netherlands: Elsevier, 1992, ch. 17, pp. 495-548.



Richard D. Jones (M'87-SM'90) received the B.E. (Hons) and M.E. degrees in electrical and electronic engineering from the University of Canterbury, Christchurch, New Zealand, in 1974 and 1975, respectively, and the Ph.D. in medicine from the Christchurch School of Medicine, University of Otago, Christchurch, in 1987.

He is a Biomedical Engineer and Head of the Diagnostic Physics and Bioengineering Section in the Department of Medical Physics and Bioengineering at Christchurch Hospital, a member of the Faculty of Engineering at the University of Canterbury, and a member of the Faculty of Medicine at the Christchurch School of Medicine. His research interests include measurement and modeling of sensory motor function, particularly in certain brain disorders, and signal processing in clinical neurophysiology.

Dr. Jones is a Registered Engineer, a Member of the Institution of Professional Engineers New Zealand, and a Fellow and currently President of the Australasian College of Physical Scientists and Engineers in Medicine. He was Representative for the Asia/Pacific Region on the Administrative Committee of the IEEE Engineering in Medicine and Biology Society in 1993/1994 and has been a member of the EMBS's International Program Committee since 1988. He is currently an Associate Editor of IEEE TRANSACTIONS ON BIOMEDICAL ENGINEERING.



Ivan M. Donaldson received the M.B Ch.B. degree in 1966 and the MD degree in 1976, both from the University of Otago, New Zealand. He passed examinations to become a Member of the Royal Australasian College of Physicians in 1970 and a Member of the Royal College of Physicians (United Kingdom) in 1972. In 1975, he was made a Fellow of the Royal Australasian College of Physicians and in 1986 a Fellow of the Royal College of Physicians (London).

He is a Neurologist at Christchurch Hospital and Associate Professor at the Christchurch School of Medicine. His research interests include movement disorders and motor disabilities.



Neil B. Sharman received the Dip.Sc degree and M.Sc. (Hons) degree in computer science from the University of Canterbury, Christchurch, New Zealand, in 1990 and 1992, respectively.

He has been writing software for the medical area since 1987 and is currently working in the Computer Science department at the University of Melbourne in Australia. His research interests include text and image compression, and the management of very large collections of documents.