Does Old Age or Parkinson’s Disease Cause Bradyphrenia?

James G. Phillips, Tanya Schiffler, Michael E. R. Nicholls, John L. Bradshaw, Robert Iansek, and Lauren L. Saling

1Department of Psychology, Monash University, Clayton, Australia
2Department of Psychology, University of Melbourne, Parkville, Australia.
3Geriatric Research Unit, Kingston Centre, Cheltenham, Australia.

Background. Age-related declines in intellectual functioning have been linked to slower processing of information. However, any slowness with advancing age could simply reflect slower movement rather than impaired cognition. To assess any age-related decline in cognitive speed, we used an accuracy-based task that does not require a speeded motor response and that measures the time required to acquire information (inspection time). To identify possible biological mechanisms of cognitive slowing, this task was also applied to patients with Parkinson’s disease, a basal ganglia disorder that reportedly causes bradyphrenia (slower thought processes).

Methods. In one experiment, 16 young (mean age 22.4 years) and 16 older adults (mean age 71.6 years) matched for intelligence and education completed an inspection time task. The task required judgments as to order of onset of two lights, where the interval between onsets ranged from 20–250 msec. A second experiment compared 16 patients diagnosed with idiopathic Parkinson’s disease and 16 age-matched controls upon the same task.

Results. Older adults demonstrated significant cognitive slowing compared to younger adults. Medicated nondemented Parkinsonian patients were not impaired on this task compared to age-matched controls.

Conclusions. Clinical and empirical impressions of bradyphrenia in Parkinson’s disease may instead reflect advancing age or slower movement, because the effects of age may be greater in some cases than the effects of basal ganglia disease once motor dysfunction has been allowed for.

There are problems in addressing any age-related decline in intellectual functioning. The concept of intelligence is somewhat descriptive (1), and conventional intelligence tests may be inappropriate for older age groups (2). Nevertheless, any age-related deterioration of intellectual functioning occurs upon performance subscales of intelligence tests, rather than vocabulary subscales (3). Because age-related decline in intellectual functioning is associated with a slowing of the processing of information (3), this study addressed mechanisms determining information processing rate. To this end, parallels have been drawn between the slowing of performance in old age and that of Parkinson’s disease (PD; (4)), as such subcortical syndromes exhibit bradyphrenia, a slowing of information processing rates (5). This study, therefore, considered whether speed of information processing declines in older adults and in patients with PD.

Experiment One

Age-related cognitive slowing is frequently reported; however, most tests of cognitive function (such as Digit-Symbol Substitution Test [DSST] and reaction time [RT]) require a speeded motor response. Because aging often involves motor slowing (6), the use of such tests could lead to spurious impressions of poor cognitive function. This investigation employed an index (inspection time) that is independent of motor involvement (7) because cognitive speed is inferred from accuracy rather than speed of response.

Inspection time is defined as the time required to make an accurate and reliable judgment in a readily discernible task. It is the minimum stimulus exposure duration required to attain a near perfect accuracy (8). Inspection time (9) is correlated with psychometric intelligence (10). However, there are problems with the few studies that have employed the inspection time paradigm to investigate cognitive slowing in aged populations. For instance, Kirby and Nettelbeck (11) found that older adults had longer inspection times and lower intelligence scores; however, this may have reflected their cohort’s educational opportunities.

To control for some of the factors affecting such cross-sectional comparisons (3), the present study matched young and older adults for estimated verbal intelligence and education. In addition, there have been some previous concerns of participants using post-stimulus apparent motion cues during tachistoscopic stimulus presentation (12), which reduce relationships between inspection time and intelligence. To preclude the use of strategies that use apparent motion, we used a novel version of the inspection time paradigm, where inspection time is operationalized as a temporal order judgment in which two lights flash on in rapid succession and participants decide which illuminated first.

The inspection time task employed the method of constant stimuli where fixed numbers of trials are conducted at each of several specific interstimulus intervals (ISI). Response accuracy for each ISI was measured, with the knowledge that participants with faster mental speeds would require shorter ISI to make accurate judgments of order of onset. The ISI at which criterion performance is reached (95% accuracy) was used to derive an index of mental speed (inspection time). Using a task that is not susceptible to motor slowing enabled us to consider...
whether age-related differences in performance are largely due to changes in cognitive speed with age or to peripheral factors such as motor slowing.

METHODS

Participants.—Sixteen young adults (mean age 22.4 years) and 16 older adults (mean age 71.6 years), participated (see Table 1). There were equal numbers of males and females within each group. Younger and older adults were matched for years of education and intelligence using the National Adult Reading Test (NART), which is highly correlated with general intelligence (13) and provides a measure of general intellectual ability uncontaminated by cohort differences in age. Participants had normal or corrected-to-normal vision.

Apparatus and task.—The stimulus display consisted of a central red light-emitting diode (LED) 7mm in diameter and two lateral green LEDs (10 mm in diameter) mounted on a matt-black display panel measuring 110 × 195 mm. The two lateral LEDs were situated 80 mm to the left and right of the midline of the central LED. The stimulus display was located at eye level 600 mm away from the seated participant. A Toshiba 486 laptop computer controlled stimulus presentation and data collection.

Participants recorded their responses by pressing one of two buttons mounted on a panel (measuring 110 by 55 mm) situated 400 mm from the display. Buttons were 15 mm in diameter and raised 10 mm from the panel, allowing responses to be made using the forefingers of each hand.

The task consisted of judging the order of onset of two LEDs situated on either side of a central fixation point. Participants responded to the LED first illuminated by pressing a button on the corresponding side. If participants could not discern which LED had been activated first, they were required to guess. Each trial began with the presentation of a cue (1 msec before stimulus presentation) that was the activation of the central red LED. The stimulus consisted of consecutive activation of the two lateral green LEDs. These remained illuminated until a response was made, thus masking any apparent motion. The interval between the onset of the two lights (ISI) was: 20, 40, 60, 80, 100, 125, 150, 175, 200, or 250 msec.

Procedure.—The experiment was conducted in a well-lit room. A black cloth backdrop was used to minimize visual distraction. Participants completed two blocks of eight practice trials as a familiarization with the required response and exposure duration. Each testing session consisted of 20 blocks of 10 trials (i.e., 200 trials). Pencil-and-paper tests were administered midway through the session.

RESULTS

Mean accuracy for each condition was analyzed using a mixed model analysis of variance (ANOVA) with a between-subjects factor of Group (young vs older adults) and a within-subjects factor of Interstimulus Interval (20, 40, 60, 80, 100, 125, 150, 175, 200, 250 msec).

Accuracy.—Accuracy scores (out of 20) for the different ISI are shown in Figure 1. As ISI increased in duration, significantly fewer errors were made \([F(9,270) = 43.65, p < .01]\). Trend analysis revealed a significant linear component to the effect of ISI \([F(1,270) = 263.01, p < .01]\) upon accuracy, indicating a steady improvement in accuracy as the ISI increased.

Younger adults performed at significantly higher levels of accuracy (mean = 19.0) than older adults (mean = 17.6); \([F(1,30) = 15.60, p < .01]\). At the smallest ISI, both younger and older adults performed at chance levels of accuracy. A significant Group by ISI interaction \([F(9,270) = 3.50, p < .001]\) indicated that the two groups differed in the rate of improvement in accuracy with greater intervals between the two stimuli.

Inspection time.—Accuracy data were used to determine inspection time (IT). IT is calculated by fitting a cumulative normal ogive to each participant’s data (8). Because a cumulative normal ogive is an exponential function, a log transform was applied to accuracy scores (to 1 minus the probability correct), and then a linear regression was performed upon accuracy and ISI. The time required for a criterion level (95%) of accuracy was then determined from the fitted function. Significant fits were obtained for 87.5% of functions overall, with no significant difference in the proportion of variance accounted for by the fitted curves for each age group.

Inspection time is the stimulus exposure for accurate performance and is an index of speed of information processing. Inspection times were significantly shorter for young (82.9 msec) than older (158.6 msec) adults, \([t(30) = -4.07, p < .01]\). Young adults required stimulation of briefer duration for accurate judgments.

The usual significant correlations were found between age and DSST \((r(30) = -.66, p < .01)\) such that older adults required more time on this transcription task. As the DSST may be affected by any age-related motor slowing, it is interesting to note a significant correlation between age and IT \((r(30) = .62, p < .01)\), with young adults taking less time to acquire information. This suggests that poorer performance on such tests is not simply due to slower movement. While IT correlated with Age and tests of performance, it did not correlate with estimates of crystallized intelligence [i.e., NART, education; (3)].

DISCUSSION

Although age-related slowing of performance has been reported, it has often reflected differences in factors such as education, motivation, and intelligence. This study investigated speed of elementary processing operations as indexed by inspection time, unconfounded by factors such as differences in...
movement speed. Older adults were significantly slower than younger adults at processing simple temporal order information, and required longer interstimulus intervals to accumulate sufficient evidence to make an accurate judgment about the order in which two LEDs were activated.

Older adults were less able to judge order of stimulus onset, implying that they had slower "mental clocks." However, these age differences in mental speed were not due to cohort differences in educational level or intelligence, as participants were matched on these variables. Cognitive slowing in older adults was also not attributable to impaired cognitive status, as participants were screened for dementia. Participants were also screened for epilepsy, stroke, head injuries, and psychiatric disorders; thus, these factors did not influence age-related differences in mental speed. Because the estimate of mental speed (IT) was derived from accuracy, our data indicate that a sizable cause of slowing is of central rather than a peripheral (motor) origin.

**Experiment Two**

Experiment One supported clinical impressions of a slowing of information processing with age. Indeed, parallels have been drawn between the slowness of age and Parkinson's disease (4). Although primarily a movement disorder affecting the basal ganglia (BG), PD is also characterized by bradyphrenia (5), and up to 40% of patients (14) are reported to be demented. An investigation of Parkinsonian bradyphrenia may contribute to an understanding of information processing rates and potentially identify biological mechanisms underlying cognitive slowing.

Experiment Two considered whether the disturbances of BG function caused by PD affect the speed of information processing, over and above that of normal aging. Because inspection time measures cognitive speed independently of motor involvement, this task is useful in studying PD, which reportedly causes bradyphrenia. As we were specifically interested in the contribution of the BG to cognitive speed, we excluded PD pa-

---

Table 2. Clinical Data for Patients with Parkinson's Disease

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Disease Duration (years)</th>
<th>Disease Severity*</th>
<th>Medication</th>
<th>Dose (mg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>62</td>
<td>F</td>
<td>12</td>
<td>14</td>
<td>Sinemet</td>
<td>1500/150</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>200/50</td>
</tr>
<tr>
<td>2</td>
<td>61</td>
<td>M</td>
<td>17</td>
<td>14</td>
<td>Madopar</td>
<td>100/25</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pernox</td>
<td>1.875</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>400/100</td>
</tr>
<tr>
<td>3</td>
<td>78</td>
<td>M</td>
<td>5</td>
<td>12</td>
<td>Madopar</td>
<td>800/200</td>
</tr>
<tr>
<td>4</td>
<td>74</td>
<td>F</td>
<td>10</td>
<td>8</td>
<td>Sinemet</td>
<td>725/100</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>300/75</td>
</tr>
<tr>
<td>5</td>
<td>69</td>
<td>F</td>
<td>6</td>
<td>9</td>
<td>Sinemet</td>
<td>50/12.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>800/200</td>
</tr>
<tr>
<td>6</td>
<td>73</td>
<td>F</td>
<td>20</td>
<td>13</td>
<td>Madopar</td>
<td>300/75</td>
</tr>
<tr>
<td>7</td>
<td>71</td>
<td>F</td>
<td>5</td>
<td>7</td>
<td>Eldepryl</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Madopar</td>
<td>600/150</td>
</tr>
<tr>
<td>8</td>
<td>74</td>
<td>F</td>
<td>5</td>
<td>7</td>
<td>Eldepryl</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Madopar</td>
<td>300/75</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>500/125</td>
</tr>
<tr>
<td>9</td>
<td>52</td>
<td>F</td>
<td>6</td>
<td>5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>67</td>
<td>M</td>
<td>13</td>
<td>11</td>
<td>Madopar Q</td>
<td>250/62.5</td>
</tr>
<tr>
<td>11</td>
<td>66</td>
<td>M</td>
<td>12</td>
<td>11</td>
<td>Sinemet</td>
<td>1000</td>
</tr>
<tr>
<td>12</td>
<td>67</td>
<td>M</td>
<td>5</td>
<td>7</td>
<td>Madopar</td>
<td>600/150</td>
</tr>
<tr>
<td>13</td>
<td>48</td>
<td>M</td>
<td>8</td>
<td>9</td>
<td>Sinemet</td>
<td>1000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>100/25</td>
</tr>
<tr>
<td>14</td>
<td>55</td>
<td>M</td>
<td>6</td>
<td>7</td>
<td>Artane</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Deprenyl</td>
<td>10</td>
</tr>
<tr>
<td>15</td>
<td>49</td>
<td>F</td>
<td>10</td>
<td>10</td>
<td>Madopar</td>
<td>1000/250</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinemet CR</td>
<td>150/37.5</td>
</tr>
<tr>
<td>16</td>
<td>71</td>
<td>F</td>
<td>15</td>
<td>6</td>
<td>Cogentin</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Madopar</td>
<td>400/100</td>
</tr>
</tbody>
</table>

*Rating of Parkinsonian symptoms using Webster scale (16).
†Predominantly tremorous patient not medicated.
patients exhibiting dementia, as other structures are implicated in such patients (15).

Hitherto, speed of information processing in PD has typically been assessed with tasks involving a motor component; as PD is primarily a motor disorder, such procedures may overestimate cognitive impairments in this population. If PD causes bradyphrenia, PD patients will require longer ITs even when controlling for motor impairments. On the other hand, if bradyphrenia in PD is merely an artifact of aging and slower movements of these patients, then PD patients will not require longer ITs.

METHOD

Participants.—Participants were screened for prior head injury, stroke, and other neurological or psychiatric conditions in addition to Parkinson’s disease. Sixteen patients (7 male, 9 female) diagnosed with idiopathic Parkinson’s disease participated. Mean duration of disease was 9.7 years (SD = 4.8); symptom severity was measured using the Webster Scale (16); clinical characteristics may be seen in Table 2. As outlined in Table 3, 16 control participants were matched to PD patients for age, sex, years of education, and premorbid intelligence [estimated using NART (13)]. Participants had normal or corrected-to-normal vision.

All participants were screened for dementia using the Mini-Mental State Examination [MMSE; (17)]. While no participants were excluded on the grounds of dementia, a significant independent sample t test indicated the PD group (mean = 29.6; t(30) = −2.18, p < .05) had slightly poorer mental status than the controls (mean = 28.6; t(30) = −2.18, p < .05).

Apparatus, task, and procedure.—The apparatus and task were the same as in Experiment One. The procedure was also the same except that Webster scales were administered at the beginning of each testing session, and the DSST was not administered because its large motor component potentially invalidates its use as a test of cognitive function in the PD group.

RESULTS

Accuracy.—Mean accuracy scores for the different ISI are shown in Figure 2. A two-way ANOVA (Group × ISI) revealed a significant effect of ISI [F(9, 270) = 35.41, p < .01], with both groups responding more accurately as the ISI increased. Although controls (17.4) tended to be more accurate for each ISI than PD patients (16.9), there was no significant group effect [F(1, 30) = 0.81, p < .05]. There was no interaction between group and ISI [F(9, 270) = 1.22, p > .05], indicating that the rate of improvement in accuracy with greater intervals between the two stimuli was similar for both groups.

Inspection time.—Measures of IT were derived from the accuracy data. Significant fits were obtained for 90% of functions in this sample, with no significant difference in the degree of fit obtained for PD and control groups. There were no appreciable differences in mean IT between the PD group (mean = 206.1 msec) and controls (mean = 204.1 msec; t(30) = .03, p > .05).

A significant correlation between IT and MMSE [r(30) = −.42, p < .05] indicated that poorer mental status was associated with slower cognitive speed. The relationships between clinical measures of disease severity and IT were also examined. There were, however, no significant correlations between IT and symptom severity [r(14) = −.25, p > .05] or disease duration [r(14) = −.29, p > .05], indicating that changes in cognitive speed when uncontaminated by motor slowing were unrelated to severity of PD symptoms.

Table 3. Characteristics of Patients With Parkinson's Disease (PD) and the Controls

<table>
<thead>
<tr>
<th></th>
<th>PD Patients</th>
<th>Controls</th>
<th>t tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>t(30)</td>
</tr>
<tr>
<td></td>
<td>66.1 (11.0)</td>
<td>66.4 (11.4)</td>
<td>−0.08, p &gt; .05</td>
</tr>
<tr>
<td>Education (yrs)</td>
<td>10.9 (2.7)</td>
<td>11.3 (2.7)</td>
<td>−0.33, p &gt; .05</td>
</tr>
<tr>
<td>Verbal Intelligence (National Adult Reading Test)</td>
<td>110.7 (7.7)</td>
<td>112.4 (5.2)</td>
<td>−0.81, p &gt; .05</td>
</tr>
</tbody>
</table>

Figure 1. Accuracy as a function of interstimulus intervals for both younger (closed circles) and older adults (open circles).

Figure 2. Accuracy as a function of interstimulus intervals for both Parkinson's disease (PD, open circles; and control (closed circles) groups).
DISCUSSION

Although Parkinson’s disease is clinically reported to cause bradyphrenia (5), the assessment of cognitive slowing may reflect the behavioral measures employed, as cognitive speed is typically inferred from movements when performing cognitive tasks. Given that PD is a motor disorder, clinical impressions of bradyphrenia in PD may to some extent reflect the diminished capacity of these patients to express themselves.

This study investigated information processing speed in a group of nondemented PD patients using a task that measured cognitive speed independently of movement. Results indicated that nondemented Parkinsonian patients did not exhibit a cognitive slowing beyond that found in normal aging when controlling for motor impairment. In other words, PD patients did not exhibit bradyphrenia when controlling for akinesia (difficultly initiating movement), bradykinesia (difficulty maintaining movement), or dementia.

Disorders of the basal ganglia are thought to cause a subcortical dementia (5). Parkinson’s disease affects dopaminergic cells in the Substantia Nigra Pars Compacta (SNc) regulating the basal ganglia circuits (18). While the central and caudal end of the SNc, which projects to the putamen, is predominantly affected, the disorder is not so localized that other structures are uninvolved. Indeed, the juxtaoperation of motor and cognitive circuits and the involvement of other structures suggest that cognitive dysfunction is likely.

Nevertheless, reports of cognitive impairment in Parkinson’s disease remain controversial. Some studies report a slowing of thought processes (19,20), while others do not (21,22). Given this controversy, a generalized slowing of cognition is perhaps unlikely. Since cognition is complex, it is feasible that only specific processing operations are affected (14). For example, Duncombe and associates (22) found no slowing of the mental rotation of 2D symbols, but others have found a slowness mentally rotating 3D shapes (19). Moreover, the assessment of cognitive functioning is further complicated by medication status (23). Recent work suggests that dopamine may improve tests of frontal lobe function (24) and assist in the reproduction of temporal intervals (25).

Although it might be desirable to assess cognitive function in patients off medications, this can be difficult for ethical and practical reasons.

To address cognitive functions of the basal ganglia, our previous studies have assessed cognitive function in nondemented patients with Parkinson’s disease and have not found cognitive deficits. Although selecting nondemented patients has practical and theoretical appeal, it may be restrictive and unrealistic, because cognitive status reflects a continuum rather than a dichotomy. We found a significant correlation between inspection time and cognitive status (measured using MMSE), indicating that better intellectual functioning can be associated with faster mental speed. Although this aspect of our data confirms previous reports of a relationship between cognitive slowing and intellectual status (26,27), it indicates that the strength of such a relationship is reduced when controlling for motor slowing and educational levels. Although about 27% of patients with Parkinson’s disease are demented (28), this dementia is associated with cortical dysfunction (15) and in 90% of cases with cortical pathology (28). Although the concept of subcortical dementia has clinical merit, there is a need to clarify this concept and consider factors contributing to levels of cognitive function.

CONCLUSION

The speed of information processing may underlie age-related differences in measured intelligence. Information processing rate is typically inferred from behavioral measures of performance. Most tasks purporting to measure information processing rate involve a motor component reducing their construct validity as measures of cognitive speed. A substantial slowing in information processing rate was found in older adults compared to younger adults matched for education and intelligence. Age-related differences in cognitive speed were also not influenced by requirements for a speeded motor response, as our index of cognitive speed was inferred from accuracy rather than reaction time.

Cognitive slowing exists in older adults independent of differences in educational or motor capacity. As cognitive slowing was not salient in PD, the mechanisms of age-related cognitive slowing are unlikely to originate from basal ganglia dysfunction. A generalized slowing of information processing was not apparent in these patients with Parkinson’s disease. There is a need to consider age and motor instability when addressing clinical manifestations of bradyphrenia.

ACKNOWLEDGMENTS

This study was supported by grants from the Australian Research Council and the Alzheimer’s Association of Australia.

We are grateful for the assistance of the people with Parkinson’s disease who gave so freely of their time and the Kingston Centre for the use of their facilities.

Address correspondence to Dr. James G. Phillips, Department of Psychology, Monash University, Clayton VIC 3168, Australia. E-mail: psy193g@alpaha1.cc.monash.edu.au

REFERENCES


Received December 31, 1997
Accepted October 15, 1998