Effects of Alzheimer’s Disease on Visual Enumeration

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Speeded enumeration of visual objects typically produces fast and accurate performance for up to 3 to 4 items (subitization) but slower and less accurate performance thereafter (counting). We investigated enumeration ability in patients with Alzheimer’s disease (AD) and in age-matched controls. AD patients were slower overall than controls. The subitizing span was significantly reduced in AD patients compared with controls (2.3 vs 3.5 items) and counting rate was significantly slower (451 vs 349 ms/item). Error rates were similar in the two groups except at numerosity 3, when AD patients made errors but controls did not (consistent with their subitizing spans).

Within the AD patient group, several aspects of performance correlated significantly with Mini-Mental State Examination scores. Together, the results provide a striking contrast with studies showing preservation of enumeration ability in normal aging.

Enumeration tasks typically require participants to determine as quickly as possible how many items are present in a visual display. When the resulting response times (RTs) are plotted as a function of numerosity, a bilinear function generally emerges, with a discontinuity or flex point at around three or four items. Thus, RTs increase relatively little (usually < 100 ms/item) up to about three or four items, and performance is also accurate over this range. For larger numerosities, however, RTs increase considerably more with each additional item (~350 ms/item), and errors are increasingly likely to occur (e.g., Trick & Pylyshyn, 1993, 1994). Subitization refers to the fast and accurate enumeration of small numbers of items, whereas counting refers to the slower and less accurate enumeration of larger numbers of items (e.g., Kaufman, Lord, Reese, & Volkmann, 1949; Mandler & Shebo, 1982; Sagi & Julesz, 1984; Trick & Pylyshyn).

Various explanations have been proposed to account for subitization. These include the involvement of a preverbal counting system that is accurate and reliable only for small numbers of items (Gallistel & Gelman, 1992), the use of pattern information as a cue to numerosity (Mandler & Shebo, 1982), and the ability of the visual system to selectively tag up to about four items that are individuated at a preattentive level of processing (Trick & Pylyshyn, 1993, 1994). Beyond the subitizable range, items appear to be counted in a spatially serial manner, leading to linear increases in RT as a function of numerosity (e.g., Trick & Pylyshyn, 1994).

Enumeration in Normal Aging

Several studies have investigated the effects of old age on enumeration (e.g., Basak & Verhaeghen, 2003; Geary & Lin, 1998; Kotary & Hoyer, 1995; Nebes, Brady, & Reynolds, 1992; Sliwinski, 1997; Trick, Enns, & Brodeur, 1996; Watson, Maylor, & Bruce, in press; Watson, Maylor, & Manson, 2002). However, methodologies have differed somewhat across studies, with some using limited ranges of numerosities, some including distractors in the displays, and some adopting a two-alternative forced-choice procedure. Nevertheless, the general picture emerging from these studies is that enumeration efficiency is surprisingly unaffected by old age. Thus, in the absence of distractors, although older adults may be slightly slower overall than young adults, they usually show similar subitizing spans, subitizing rates, and counting rates (see Watson et al., 2002, in press, for summaries of the evidence and also a discussion of exceptions).

These findings for enumeration are particularly striking in view of marked age-related deficits in other visual tasks. For example, when older adults search for a single target among varying numbers of distractors, their search rate can be much slower than that of young adults (see Madden & Whiting, 2004, for a summary). Thus, visual search data are largely consistent with a generalized slowing account of aging (e.g., Birren, 1965; Cerella, 1985, 1990; Cerella, Poon, & Williams, 1980; Salthouse, 1985) such that age differences increase with task difficulty (in this case, with increasing display size). In contrast, enumeration rates are unaffected by normal aging as evidenced by parallel RT–numerosity functions for young and older adults (e.g., Watson et al., 2002, in press).

Enumeration in Dementia

The unusual sparing of enumeration in normal aging raises the interesting possibility that if enumeration were impaired by dementia, this would be a rare example of a qualitative difference between the effects of normal and abnormal aging (see Nebes, 1992; Nebes & Madden, 1988, on the importance of such differences); it would, for example, contrast with largely quantitative differences between the effects of normal and abnormal aging on visual search (e.g., Foster, Behrmann, & Stuss, 1999; Nebes & Brady, 1989). Existing evidence on enumeration in dementia is, however, sparse and/or equivocal. With untimed tasks, some researchers have found no difference in accuracy for counting up to 9 dots between patients with Alzheimer’s disease (AD) and age-matched controls (Fujimori, Imamura, Yamashita, Hirono, & Mori, 1997; Halpern, McMillan, Moore, Dennis, & Grossman, 2003), whereas others...
have found deficits with dementia for counting up to 30 dots (Seron et al., 1991).

As far as we are aware, only three studies have examined speeded enumeration in patients with AD. Boone and colleagues (2002) presented displays containing between 7 and 28 dots. They did not examine counting rates (milliseconds per item), but they did find that AD patients were slower overall than controls, with minimal difference in accuracy. Nebes and associates (1992) compared healthy young and older adults with depressed geriatric and AD patients on speeded enumeration (using vocal RTs) of between 1 and 4 randomly arranged dots. Their interest was in enumeration rate as an index of cognitive slowing. Slopes calculated over numerosities 1 through 4 were similar for both groups of healthy adults and depressed patients, but the slope for the AD patients was significantly greater, which the authors interpreted as evidence of “the presence of a cognitive slowing in Alzheimer’s disease, but not in depression” (p. 331). Subitization is not mentioned in the study by Nebes and associates, but a visual inspection of their data in Figure 1 and Table 2 suggests another possibility, namely, that AD patients differ from the other groups not in subitization rate but rather in subitization span. Thus, whereas the healthy adults and depressed patients were relatively fast and accurate for numerosities 1 through 3, both RTs and errors increased markedly after numerosity 2 for AD patients.

Finally, Kaufmann and colleagues (2002) compared AD patients and controls in enumerating between one and nine dots, and they found significant group differences in overall RT for both small numbers (up to four) and larger numbers (five to nine). Moreover, it appears from their Figure 1 that the group difference was approximately twice as great for larger numbers as for small numbers, which is consistent with generalized slowing (e.g., Nebes & Madden, 1988) but contrary to the result for normal aging (e.g., Watson et al., 2002). Thus the results of the studies of both Nebes and associates (1992) and Kaufmann and associates indicate that there may indeed be qualitative differences between enumeration performance in normal and abnormal aging.

The Present Study

In view of the paucity of enumeration data in dementia, and the intriguing hints from the aforementioned studies that enumeration is preserved in normal aging but not in abnormal aging, we investigated visual enumeration in AD patients and age-matched controls. In order to examine performance over both the subitizing and counting ranges, we assessed the speeded enumeration of between one and nine randomly arranged circles. In particular, our aim was to formally test the hypotheses that subitization span (i.e., the flex point of the bilinear function dividing fast accurate subitizing and slow serial counting) and counting rate are reduced by AD.

Methods

Participants

We recruited patients from a residential home specializing in the care and treatment of people with dementia. Residents of this home had all received medical diagnoses of dementia prior to admission, mostly from psychiatrists at the District Hospital’s Elderly and Mentally Infirm Unit. (Care home staff members were therefore not involved in diagnosis.) From this home, we specifically selected 18 residents as having been formally diagnosed as suffering from progressive cognitive deterioration that was due to probable AD. Of these 18 AD patients, 6 failed to complete the experiment (see the Results section), and we therefore did not include their data in the analyses; the remaining 12 AD patients (4 men and 8 women) had a mean age of 80.8 years ($SD = 4.8$; range $= 73–88$). We recruited 8 control participants living independently in the community from a panel of older volunteers taking part in studies of cognitive aging at the University of Warwick, England. There were 3 men and 5 women, with a mean age of 79.1 years ($SD = 4.1$; range $= 75–87$). The groups did not differ significantly in terms of age, $t(18) < 1$, but the AD patients scored significantly lower on the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) than did the controls ($M = 17.3$, $SD = 4.9$, range $= 9–24$, for the AD patients; $M = 29.4$, $SD = 0.7$, range $= 28–30$, for the controls; $t(18) = 6.88$, $p < .0001$). As we expected, AD patients tended to be less well educated than controls (see, e.g., Fratiglioni & Rocca, 2001), leaving school on average at the age of 15.3 years ($SD = 1.65$) compared with an average of 16.6 years ($SD = 1.62$) for controls. (Education data were unavailable for 2 AD patients and 1 control.) However, school-leaving age did not differ significantly between groups, $t(15) = 1.64, p = .12$. All participants were recruited on a voluntary basis and received no payment for taking part in the study.

Apparatus and Stimuli

We controlled the experiment by using a 1-GHz Pentium III based laptop computer with a 14-in. (35.56-cm) screen at a resolution of $1,024 \times 768$ pixels. The screen was placed directly in front of the participant at eye level, with a viewing distance of approximately 60 cm. The computer measured RTs to the nearest millisecond and synchronized all display changes to the screen retrace. The participants’ response key was the spacebar of the laptop keyboard. The experimenter keyed in numerical responses by using an externally connected numeric keypad.

The stimuli were light red open circles (8-bit RGB values = 250, 164, and 176, respectively) displayed against the black screen. Each circle measured 8.5 mm $\times$ 8.5 mm ($0.81^2 \times 0.81^2$) with a continuous line width of 2 mm. We generated displays by placing the stimuli randomly into the cells of an invisible $6 \times 6$ matrix (120 mm $\times$ 120 mm, 11.42$^2 \times 11.42^2$). The interelement spacing was 75 pixels (21 mm, 2$^\circ$) and, to avoid any perception of a regular matrix structure, we displaced each circle by up to 10 pixels (2.5 mm, 0.24$^\circ$) in the vertical and horizontal directions.

Design and Procedure

The experimenter tested all participants individually in a quiet illuminated room free from disturbances or distractions, either in their residential care home (AD patients) or in their own homes (controls). The testing session began with the administration of the MMSE. This was followed by the instructions for the enumeration task, a demonstration block of trials, three blocks of practice trials, and finally three blocks of experimental trials. Each trial of the enumeration task began with a blank screen for 1,000 ms followed by a white (RGB values = 200, 200, and 200, respectively) central fixation cross for 750 ms. The
.enumeration display of between one and nine light red circles then appeared and remained until a response was made. Participants were required to determine the number of circles present as quickly as possible and respond by pressing the spacebar of the computer keyboard at the same time as saying aloud their numerical response. The experimenter, who was seated in a position so that she could not see the computer screen, then entered the participant’s response by means of the remote numeric keypad. We measured RT from the onset of the enumeration display to the participant’s press of the spacebar. Note that a similar technique has been used successfully in the past with both patients (Watson & Humphreys, 1999) and older adults (Watson et al., 2002). It has a number of advantages over other methodologies used in studies of enumeration. For example, some require participants to press a number key themselves corresponding to numerosity (e.g., Basak & Verhaeghen, 2003; Sliwinski, 1997), which contaminates enumeration times with times to locate and select the appropriate key, and increases task demands. Others use voice keys (e.g., Nebes et al., 1992), but these do not reliably detect acoustic onsets (see Rastle & Davis, 2002) and also occasionally trigger in response to other noises or fail to trigger in response to the participant’s voice onset.

The experimenter used the demonstration block, consisting of 9 trials (1 for each numerosity), to explain the task. When the experimenter was satisfied that the participant understood the procedure, she administered the practice blocks. Each practice block contained 18 trials (2 for each numerosity). Finally, this was followed by the three experimental blocks, each of 45 trials (five replications of the nine numerosities). The trials within a block were presented in a random order. There were opportunities for rest breaks between blocks of trials; the next block of the experiment proceeded only if the participant expressed a willingness to continue.

RESULTS

There were 4 AD patients who dropped out of the experiment after completing one block of experimental trials, and a further 2 AD patients who dropped out after two blocks (3 failed to understand or follow the task instructions, and 3 did not wish to continue). Of these 6 excluded AD patients, 5 had MMSE scores between 9 and 11. For the remaining participants, we pooled the data across the three experimental blocks and calculated mean correct RTs and error rates for each numerosity for each participant. We adopted the usual procedure in enumeration studies of excluding the largest numerosity (nine) from the statistical analyses to prevent contamination by end effects (see Mandler & Shebo, 1982; Trick & Pylyshyn, 1994). In all analyses of variance (ANOVAs) with a repeated measure, where there was evidence of departure from the sphericity assumption, we report Greenhouse–Geisser corrections.

Response Times

Figure 1 shows the overall means for correct RTs as a function of numerosity for AD patients and controls. We can see that the controls displayed the usual bilinear pattern of relatively little increase in RT as a function of numerosity up to about three or four items, with a much steeper function beyond this range. The AD patients were clearly slower overall than the controls, with mean RTs for numerosities 1–8 of 2,947 and 1,536 ms, respectively. In addition, the AD patients’ RT–numerosity function was (a) less flat in the range up to three to four items and (b) steeper than that of the controls beyond that range. An ANOVA with group as the between-subjects factor and numerosity (1–8) as the within-subjects factor confirmed a significant effect of numerosity (1–8) as the within-subjects factor confirmed a significant effect of group, $F(1, 18) = 16.87, MSE = 4,529,094.75, p < .001$, a significant effect of numerosity, $F(1, 51, 27, 23) = 105.96, MSE = 584,342.91, p < .001$, and a significant interaction, $F(1, 51, 27, 23) = 4.78, MSE = 584,342.91, p < .05$.

An additional feature of Figure 1 is the decrease in RT (though not significant) from numerosity 1 to 2 for AD patients. This was largely attributable to just 2 patients with RT(1) – RT(2) differences of 1,224 and 1,327 ms, both exceeding the overall mean difference by more than 2.5 SD. The reason for this unusual end effect is unclear. One possibility is that these patients rechecked the displays when they could find only one item, in a similar way that some observers recheck target-absent displays in visual search tasks (e.g., Plude & Doussard-Roosevelt, 1989). Without these 2 participants, there was a mean increase from numerosity 1 to 2 for AD patients of 68 ms (i.e., within the traditional subitizing rate), which was significantly less than the increase of 461 ms from numerosity 2 to 3, $t(9) = 2.64, p < .03$. We repeated all the analyses reported here without these 2 patients, with qualitatively similar results in every case.

Subitizing Span

We determined the span of subitization for each participant by fitting a bilinear function to the mean RTs of the enumeration function for numerosities 1 to 8. We achieved the best fit by using an optimization procedure (see Watson et al., in press, for details) that minimized the mean square error between the model prediction and observed data by modifying the model’s four free parameters (two slopes and two intercepts). We calculated the flex point of the resulting bilinear function and took it to represent the point at which the subitization process was replaced by the slower serial counting process. This produced one obviously anomalous flex point of 4.34 for an AD patient who showed no evidence of subitization (RT slopes of 448 and

![Figure 1. Mean correct response times (RTs) for the patients with Alzheimer’s disease (AD) and controls as a function of numerosity. Error bars indicate ± 1 standard error of the mean.](http://psychsocgerontology.oxfordjournals.org/)

436 ms/item for numerosities 1–3 and 5–8, respectively). Her data were therefore not included in our analyses of the subitizing span.

We found the bilinear model to be a good fit to the data, with mean $R^2$ values of 0.988 for AD patients and 0.987 for controls. The mean subitization span was 2.32 (SD = 0.63) for the AD patients and 3.45 (SD = 0.30) for the controls; an independent samples $t$ test showed that these differed significantly, $t(17) = 4.66$, $p < .0005$.

**Counting Rate**

For each participant, we determined the slope of the RT–numerosity function by linear regression of the mean RTs for numerosities 5 to 8 to provide a measure of the counting rate. This was significantly slower for AD patients ($M = 451.4$ ms/item; $SD = 98.6$) than for controls ($M = 348.5$ ms/item; $SD = 49.7$), $t(18) = 2.71$, $p < .02$.

**Errors**

Percentage error rates as a function of numerosity for AD patients and controls are displayed in Figure 2. Note that there was no evidence of any trade-offs between speed and accuracy. Thus, participants were both faster and more accurate at smaller numerosities than at larger numerosities. In addition, AD patients were both significantly slower and at least numerically less accurate than controls, with overall mean error rates (excluding numerosity 9) of 3.40% ($SD = 0.30$) for the AD patients and 2.08% ($SD = 1.89$) for controls.

Errors were rare for numerosities 1 to 2 for AD patients and nonexistent for numerosities 1 to 3 for controls, but they increased steadily thereafter. A 2 (group) × 8 (numerosity) mixed ANOVA confirmed a significant effect of numerosity, $F(3.45, 62.09) = 5.82, MSE = 33.44, p < .002$, but there was no overall effect of group, $F(1, 18) = 1.58, MSE = 42.26, p > .2$, and no interaction, $F < 1$. However, on the basis of a significant difference in subitizing span between groups, we conducted a post hoc comparison of error rates at numerosity 3 only, which was within the subitizing span for controls but not for AD patients. This revealed a significantly greater error rate for AD patients than for controls, $t(18) = 2.45$, $p < .03$.

**Correlations With MMSE**

To examine whether performance on the enumeration task in AD patients was related to the severity of their dementia, we performed correlations with MMSE scores. Higher MMSE scores were significantly associated with both faster overall RTs, $r(11) = -.871$, $p < .0001$, and lower overall error rates, $r(11) = -.693$, $p < .02$. In addition, MMSE scores were significantly correlated with subitizing span, $r(10) = .608$, $p < .05$, and there was a marginally significant correlation between MMSE and counting rate, $r(11) = -.516$, $p < .09$, such that as dementia increased in severity, subitizing span decreased, and counting rate tended to be slower.

**DISCUSSION**

To summarize our main findings, AD patients with a mean age of 81 years were approximately 1.4 s slower overall than age-matched controls, but no less accurate overall, in determining how many items were present in a visual display. More importantly, there were significant group differences in the pattern of performance for both small and large numerosities. First, the range over which the RT–numerosity function was relatively flat and performance was accurate (i.e., subitization) was significantly reduced in AD patients in comparison with controls. Second, for larger numerosities, counting was significantly slower in AD patients than in controls. In addition, within the AD patient group, these deficits were associated with severity of dementia as indicated by scores on the MMSE.

Before discussing the effects of AD on enumeration, it is worth noting that the data from the control group are highly similar to our earlier findings from both young and older adults (Watson et al., 2002, in press), despite some methodological differences such as the use in the present study of a laptop computer to collect data in participants’ own homes and the need to test somewhat older adults (by approximately 10 years). Thus, the control group showed the usual bilinear RT–numerosity function with a mean subitization span of 3.5 items, a mean subitization rate (although not reported herein) for numerosities 1 to 3 of 21 ms/item, and a counting rate of 349 ms/item. These findings therefore lend further support to the conclusion that enumeration is remarkably intact in normal aging.

Regarding the effects of AD, the first point to note is that, as in the study by Boone and colleagues (2002) of dot counting, patients were slower overall than controls, as expected from previous RT studies of AD (e.g., Nestor, Parasuraman, & Haxby, 1991; see Amieva, Phillips, Della Sala, & Henry, 2004 and Parasuraman & Haxby, 1993 for summaries). In addition, the present interaction of patient group with numerosity is consistent with dementia-related generalized slowing such that the absolute difference in RT between patients and controls increases as task complexity increases, regardless of the particular cognitive processes required by the task (e.g., Nebes & Brady, 1992).

As far as we are aware, the present study is the first systematic investigation of AD and speeded enumeration incorporating both the subitizing and counting ranges of numerosities. However, the results confirm our informal observations from...
AD patients had both a smaller subitizing span and a slower counting rate in AD. It is worth noting that there was minimal overlap between the distributions of scores from the present two groups. Only 2 AD patients had subitizing spans within the controls’ range (2.94–4.00), and only 2 AD patients had counting rates within the controls’ range (259–387 ms/item). Moreover, none of the 12 AD patients had subitizing spans within the controls’ range (2.94–4.00), and only 2 AD patients had counting rates within the controls’ range (259–387 ms/item). Moreover, none of the 12 AD patients had both a subitizing span and a counting rate within the controls’ range. In summary, the present study was successful in demonstrating striking deficits in speeded enumeration caused by abnormal aging, which contrasts sharply with spared enumeration performance in normal aging.

How can we explain AD patients’ smaller subitizing spans and slower counting rates? First, the currently most comprehensive account of subitization is the FINST (for fingers of instantiation) theory of Trick and Pylyshyn (1993, 1994). This proposes that the visual system is able to selectively tag or assign pointers to items in a display in a spatially parallel fashion (see Pylyshyn, 1989). It is further assumed that the number of such visual indexes (called FINSTs) is limited to around four. Small numbers of items can be enumerated by directly determining the total number of FINSTs currently bound to items in the visual field—hence subitization. However, when the number of items exceeds the number of FINSTs, enumeration proceeds by means of a serial process of disengaging and reassigning FINSTs (or a single focus of attention) to the remaining items, maintaining a running total, recording which items have been counted, and so on. These additional relatively complex operations result in a substantial and linear increase in RT as numerosity increases.

FINST theory successfully accounts for subitization (Trick & Pylyshyn, 1993, 1994), in addition to other findings (e.g., Pylyshyn & Storm, 1988). Moreover, there are several reasons why an efficient visual system requires the ability to tag multiple items simultaneously. Some examples include the efficient relocation of attention around multiple relevant stimuli, the computation of spatial relationships and the integration of information across saccades (e.g., see Pylyshyn, 1989, 1998, 2001). Thus, any reduction in the number of FINSTs would be expected to have an impact on the efficiency of visual functioning in everyday tasks beyond those related to enumeration. In terms of FINST theory, the observed decrease in subitizing span with AD is tentatively interpreted as a reduction in the number of available FINSTs. It is therefore predicted that AD patients should be similarly impaired in other tasks where visual indexing is crucial, such as multiple object tracking and subset-selected visual searches (see Pylyshyn, 2000, for examples). Indeed, fewer FINSTs could be the source of several attentional or cognitive deficits seen in AD (e.g., see Amieva et al., 2004; Perry & Hodges, 1999; Rizzo, Anderson, Dawson, & Nawrot, 2000).

Interestingly, similar reductions in subitizing span were observed by Dehaene and Cohen (1994) in a study of 5 simultanagnosic patients with predominantly parietal lesions. These authors ruled out low-level visual deficits as an explanation on the grounds that these could not account for the preservation of subitization (albeit with a smaller span). Instead, they suggested that their results were at least consistent with “a postlesional reduction in the number of FINSTs” but also noted that “variables affecting the number of available FINSTs remain largely unspecified” (p. 973). The present study suggests that AD can be added to the list of such variables, which already includes development (e.g., Starkey & Cooper, 1995) and individual differences (e.g., Trick & Pylyshyn, 1994), but excludes normal aging (e.g., Watson et al., in press) and working memory span (Tuholski, Engle, & Baylis, 2001).

Before leaving the discussion of subitization, we should mention another possibility, namely, that the reduced subitizing span in AD patients is a consequence of failing to switch between modes of enumeration from one trial to the next (see Starkey & Cooper, 1995, for evidence of this in young children) and thereby occasionally counting subitizable numerosities. This seems a plausible hypothesis in view of set-shifting deficits in AD (see reviews by Amieva et al., 2004; Parasuraman & Haxby, 1993; Perry & Hodges, 1999). However, testing this possibility would involve a detailed analysis of RT distributions at each numerosity for evidence of bimodality in relation to sequential effects, which would require many more trials from each participant than we were able to obtain.

Turning next to counting, we suggest that there are several possible interpretations of the slower (but no less accurate) rate of enumerating larger numerosities seen in AD patients relative to controls. As Dehaene and Cohen (1994) commented, “counting is a complex process, and there are many ways in which it might be impaired . . . patients might have difficulty isolating objects from the background, reciting the verbal labels, or memorizing the items already counted” (p. 960). One interpretation of the present data was considered earlier, namely, generalized slowing such that all processes involved are slowed by AD to the same proportional degree. These processes are likely to include the disengagement or shifting of visuospatial attention away from the currently attended location to another, which is known to be impaired by AD (e.g., Parasuraman, Greenwood, Haxby, & Grady, 1992), and subvocalization, which is also slowed by AD (e.g., Hulme, Lee, & Brown, 1993; Nebes, Halligan, Rosen, & Reynolds, 1998).

The present findings from AD patients with respect to counting contrast sharply with those of Dehaene and Cohen (1994) from their simultanagnosic patients. Not only were the patients extremely slow in counting, they were also highly error prone. Their poor performance was attributed to a difficulty in keeping track of previously explored spatial locations, with some patients failing to count some of the items and others.
counting the same items over and over again. This was apparently not a particular problem for the present AD patients in view of similar error rates for patients and controls over the counting range (though error rate was significantly associated with dementia severity in AD patients).

Instead, the present findings for counting resemble more those of Tuholski and associates (2001; Experiment 1), who compared young adults with high and low working memory spans on speeded enumeration of 1 to 12 randomly arranged yellow vertical bars. Whereas the two groups did not differ in terms of subitizing span (see the earlier discussion), counting rate (calculated over 5–12 bars) was significantly faster for participants with high working memory span (295 ms/item) than for those with low working memory span (373 ms/item), with no difference in accuracy. Tuholski and associates argued that counting is an attention-demanding process that may be more efficient in those with greater working memory capacity because of their superior ability to inhibit previously viewed locations. Note that AD is also associated with a decline in working memory capacity (e.g., Baddeley, Bressi, Della Sala, Logie, & Spinnler, 1991; Baddeley, Logie, Bressi, Della Sala, & Spinnler, 1986; Belleville, Perez, & Malenfant, 1996; Collette, Van der Linden, Bechet, Belleville, & Salmon, 1998), and so for the same reason we would expect AD to lead to a reduced counting ability, as we found. Our data further suggest that the inhibition applied during counting might involve, at least in part, controlled (active) inhibition given previous research showing the relative preservation of automatic inhibitory mechanisms in AD, including inhibition of return (e.g., Amieva et al., 2004; Danckert, Maruff, Crowe, & Currie, 1998; Faust & Balota, 1997; but see also Castel, Pratt, & Craik, 2003).

In conclusion, the present study provides a rare example of a qualitative difference between the effects of normal and abnormal aging. Thus, at least for this particular enumeration task, whereas performance remains relatively intact in old age, both subitizing span and counting rate are impaired by AD (or, at least, probable AD: a definite diagnosis is not possible prior to autopsy), with the degree of impairment related to dementia severity. Further studies will be required to explore possible explanations, boundary conditions, behavioral consequences, and the diagnostic potential of these deficits. In particular, longitudinal studies are necessary to chart the course of decline in subitizing span and counting rate, and their relation to other cognitive deficits as the disease progresses.

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