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The Impact of Visual Acuity on Age-Related Differences in Neural Markers of Early Visual Processing

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Abstract

The extent to which age-related differences in neural markers of visual processing are influenced by changes in visual acuity has not been systematically investigated. Studies often indicate that their subjects had normal or corrected-to-normal vision, but the assessment of visual acuity seems to most frequently be based only on self-report. Consistent with prior research, to be included in the current study, subjects had to report normal or corrected-to-normal vision. Additionally, visual acuity was formally tested using a Snellen eye chart. Event-related potentials (ERPs) were studied in young adults (18–32 years old), young-old adults (65–79 years old), and old-old adults (80+ years old) while they performed a visual processing task involving selective attention to color. Age-related differences in the latency and amplitude of ERP markers of early visual processing, the posterior P1 and N1 components, were examined. All results were then re-analyzed after controlling for visual acuity. We found that visual acuity declined as a function of age. Accounting for visual acuity had an impact on whether older and younger adults differed significantly in the size and latency of the posterior P1 and N1 components. After controlling for visual acuity, age-related increases in P1 and N1 latency did not remain significant, and older adults were found to have a larger P1 amplitude than young adults. Our results suggest that until the relationship between age-associated differences in visual acuity and early ERPs is clearly established, investigators should be cautious when interpreting the meaning of their findings. Self-reports about visual acuity may be inaccurate, necessitating formal measures. Additional investigation is needed to help establish guidelines for future research, especially of very old adults.

Keywords

Aging; Visual Processing; Visual Acuity; ERPs

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1.0 Introduction

A large body of research suggests that there are age-related differences in visual processing (Ceponiene et al., 2008; Davis et al., 2008; De Sanctis et al., 2008; Dennis and Cabeza, 2008; Diaz and Amenedo, 1998; Dustman and Beck, 1966; Dustman and Snyder, 1981; Finnigan et al., 2011; Goh, 2011; Schmolesky et al., 2000; Spear, 1993; Yu et al., 2006; Zanto et al., 2010). There is also strong evidence of age-associated declines in visual acuity (Faubert, 2002; Kanthan et al., 2008; Klaver et al., 1998; Munoz et al., 2000; Rodriguez et al., 2002; Rubin et al., 1997; Spear, 1993). However, the extent to which age-related differences in measures of cortical visual processing are influenced by changes in visual acuity has not been systematically investigated. In this study, we focus on two early event-related potentials (ERPs), the posterior P1 and the posterior N1 components, and investigate whether the age-associated differences observed may be related to changes in visual acuity.

The posterior P1 and N1 components are mediated by extra-striate regions and index initial sensory-perceptual encoding (Hillyard et al., 1998b; Mangun et al., 1990; Natale et al., 2006; Schechter et al., 2005; Woldorff et al., 1997). The P1 component is hypothesized to reflect early cortical processing of stimuli sensitive to bottom-up influences such as stimulus salience and complexity (Hillyard et al., 1998a; Johannes et al., 1995), and the N1 component is theorized to reflect initial visual discrimination processing and early visual categorization (Hillyard et al., 1998a; Martinovic et al., 2011; Vogel and Luck, 2000). Both components are sensitive to spatial attention, but under most circumstances are not modulated by selective attention to non-spatial features such as color (Daffner et al., 2012a; Daffner et al., 2012b; Hillyard and Anllo-Vento, 1998; Hillyard and Munte, 1984).

Usually, age-related changes in P1 and N1 have been interpreted in terms of the impact that aging has on cortical visual processing (Ceponiene et al., 2008; De Sanctis et al., 2008; Diaz and Amenedo, 1998; Dustman and Beck, 1966; Dustman and Snyder, 1981; Finnigan et al., 2011; Zanto et al., 2010). Limited attention has been paid to the potential influence of age-associated differences in visual acuity on electrophysiological measures. Most investigations report that subjects had normal or corrected-to-normal vision (Ceponiene et al., 2008; Czigler and Balazs, 2005; De Sanctis et al., 2008; Falkenstein et al., 2006; Finnigan et al., 2011; Yordanova et al., 2004; Zanto et al., 2010). However, in most cases there is no indication that visual acuity was actually measured, and it is likely that the investigators largely relied on reports from subjects about their visual status. Only a few studies measured visual acuity (e.g., Celesia and Daly, 1977; Curran et al., 2001; De Sanctis et al., 2008; Diaz and Amenedo, 1998; Zanto et al., 2010). Of these studies, some set inclusion/exclusion cutoffs of 20/20 (Celesia and Daly, 1977), 20/30 (Diaz and Amenedo, 1998), or 20/40 (Zanto et al., 2010), whereas others did not establish clear cutoff scores (Curran et al., 2001; Werkle-Bergner et al., 2009). For example, Curran et al. (2001) reported that the mean visual acuity for their sample of older subjects (mean age 69.8) was 20/46, with a range from 20/20 to 20/100. We are not aware of any previous investigations that have accounted for differences in visual acuity when interpreting the significance of age-related differences in early visual ERPs.

To address this gap in the literature, we studied three age groups: young adults (18–32 years old), young-old adults (65–79 years old), and old-old adults (80+ years old) who performed a visual processing task involving selective attention to color. Consistent with many studies, all subjects were initially screened (by telephone interview) for normal or corrected-to-normal vision based on self-report. As part of the evaluation of participants, subjects underwent testing of visual acuity using a Snellen eye chart. Age-related differences in the latency and amplitude of the P1 and N1 components were examined. All results were then re-analyzed after controlling for visual acuity. The relative lack of attention to this factor in

the literature led to the expectation that it would have a limited impact on the findings. Nevertheless, we reasoned that if substantial changes in our results were found after controlling for visual acuity, it would suggest the need for investigators to be more cautious about interpreting age-associated differences in P1 and N1 components in terms of changes in cortical visual processing activity. Moreover, it would call upon researchers to include formal measures of visual acuity in future studies.

2.0 Methods

2.1 Participants

Subjects were recruited through community announcements in the Boston metropolitan area, including the Harvard Cooperative Study on Aging. All subjects underwent informed consent approved by the Partners Human Research committee. Participants were between 18 and 32 years or 65 and older. All subjects underwent an initial telephone screen in which they were asked about vision, hearing, and medical history. To be included in this study subjects had to report that they had normal vision or corrected-to-normal vision with glasses or contact lenses. In addition, inclusion criteria required that subjects be English-speaking and have 12 or more years of education, a Mini Mental State Exam (MMSE) score (Folstein et al., 1975) ≥ 26, and an estimated Intelligence Quotient (IQ) on the American National Adult Reading Test (AMNART) (Ryan and Paolo, 1992) ≥ 100. Subjects were excluded if they had a history of CNS diseases or major psychiatric disorders based on DSM-IV criteria (American Psychiatric Association, 1994), a history of clinically significant medical diseases, a history of clinically significant audiological disease, a Beck Depression Inventory (Beck and Steer, 1987) (for young subjects) or a Geriatric Depression Scale (Yesavage et al., 1982) (for old subjects) score of ≥ 10, were unable to distinguish between the color red and blue, or had focal abnormalities on neurological examination consistent with a CNS lesion. Subjects were paid for their time.

Binocular visual acuity was measured in all subjects with the Snellen 10 feet model wall chart, and recorded as a decimal representation of 20/x, such that 20/20 = 1.0 and represents “normal” visual acuity. Worse than normal vision was represented with a visual acuity value of less than 1.0 (e.g., 20/40 = 0.5). Better than normal vision was represented with a visual acuity value greater than 1.0. All subjects underwent a neuropsychological test battery that included the following: Digit Span Forward and Backward subtests of the Wechsler Adult Intelligence Scale-IV (WAIS-IV) (Wechsler, 2008), WAIS-IV Letter-Number Sequencing, WAIS-IV Digit-Symbol Coding, WAIS-IV Matrix Reasoning, Controlled Oral Word Association Test (COWAT) (Ivnik et al., 1996), Trail-Making Test Parts A and B (Reitan and Wolfson, 1985), Boston Naming test (Tombaugh and Hubley, 1997), Logical Memory II subtest of the Wechsler Memory Scale-III (Wechsler, 1997), and Visual Form Discrimination (Benton et al., 1983).

2.2 Experimental Procedures

A selective attention task was administered under low and high memory load. Under both loads, subjects were shown physically identical sets of stimuli, which consisted of individual letters presented in either the color red or the color blue. The low load task required subjects to respond by button press to one specific target letter. To help minimize group differences in performance on the high load task, demands were made easier for old subjects. For the high load task, the number of target letters chosen for each age group was based on pilot data: young subjects responded to 5 target letters and older subjects responded to 4 target letters. This was done to allow us to draw inferences about age-related differences in neural activity and not performance-related differences (Daffner et al., 2011; Daselaar and Cabeza, 2005; Riis et al., 2008). Subjects were instructed to pay attention to letters appearing in the

designated color while ignoring letters appearing in the other color, and respond to target letters appearing in the designated color only. Subjects were asked to respond as quickly and as accurately as possible to target letters. Practice trials preceded each set of experimental runs. All subjects participated in both tasks, whose order was counterbalanced. The hand used for the target response was counterbalanced across subjects.

Each task included 800 stimulus trials divided into 8 blocks. In both the high load and low load tasks, stimuli appeared one at a time within a fixation box that remained on the screen at all times and subtended a visual angle of ~3.5° × 3.5° at the center of a high-resolution computer monitor. Half of the stimuli appeared in the color red and half in the color blue, in randomized order. Target stimuli (7.5% in attend color; 7.5% in ignore color) were designated upper case letters and standard stimuli (70% overall; 35% in each color) were any non-target upper case letters. Fillers accounted for the remainder of the stimuli presented. Visual stimuli subtended an angle of ~2.5° along their longest dimension and were presented for 250 ms. The inter-stimulus interval (ISI) varied randomly between 815–1015 ms (mean ~ 915 ms) (see Fig. 1). For analytic purposes, trials were categorized in terms of whether the stimuli presented were in the attend or the ignore color. The Attend condition consisted of all stimuli in the designated color; the Ignore condition consisted of all stimuli in the non-designated color.

2.3 ERP Recordings

An ActiveTwo electrode cap (Behavioral Brain Sciences Center, Birmingham, UK) was used to hold to the scalp a full array of 128 Ag-AgCl BioSemi (Amsterdam, The Netherlands) “active” electrodes whose locations were based on a pre-configured montage. Electrodes were arranged in equidistant concentric circles from 10–20 position Cz. In addition to the 128 electrodes on the scalp, 6 mini bio-potential electrodes were placed over the left and right mastoid, beneath each eye, and next to the outer canthi of the eyes to check for eye blinks and vertical and horizontal eye movements. EEG activity was digitized at a sampling rate of 512 Hz.

2.4 Data Analysis

To create a composite score for neuropsychological tests, raw scores on each test were converted into z-scores based on the performance of all subjects. A composite z-score was computed for each subject by averaging his/her z-scores on all tests. Performance across groups was compared using ANOVA.

Median reaction time (RT), target hit rate, and false alarm rate were measured. Performance was compared using repeated measures ANOVA, with memory load as the within-subject factor and age group as the between-subject factor. A response was considered a hit if it occurred between 200–1000 ms after stimulus presentation. Behavioral and visual acuity data for the 3 groups were compared using ANOVA.

EEG data were analyzed using ERPLAB (www.erpinfo.org/erplab) and EEGLAB (Delorme and Makeig, 2004; http://sccn.ucsd.edu/eeglab) toolboxes that operate within the MATLAB framework. Raw EEG data were resampled to 256 Hz and referenced off-line to the algebraic average of the right and left mastoids. EEG signals were filtered using an IIR filter with a bandwidth of 0.03–40 Hz (12 dB/octave roll-off). Eye artifacts were removed through an independent component analysis. Individual bad channels were corrected with the EEGLAB interpolation function. EEG epochs for the two attention conditions (Attend and Ignore) were averaged separately. The sampling epoch for each trial lasted for 1200 ms, including a 200 ms pre-stimulus period that was used to baseline correct the ERP epochs. Trials were discarded from the analyses if they contained baseline drift or movement

artifacts greater than 90 μV. Only trials with correct responses were included in the analyses.

Regions of Interest (ROIs) across the scalp were designated, with each ROI reflecting a cluster of 7 electrode sites whose values were averaged (see Fig. 2). Consistent with prior research, measurement of the posterior P1 and N1 components focused on lateral posterior regions (Curran et al., 2001; De Sanctis et al., 2008; Nagamatsu et al., 2011; Natale et al., 2006; Vogel and Luck, 2000; Zanto et al., 2010), as represented by the left occipito-temporal (LOT) and right occipito-temporal (ROT) ROIs. ERPs to standard stimuli under the Attend condition are the focus of this report.[1] The latency of the P1 was measured as the local positive peak latency between 50–150 ms and latency of the N1 was measured as the local negative peak latency between 100–250 ms. Because of concerns about the potential impact of high frequency noise on peak latency values (Luck, 2005), time course analyses were performed on mean amplitude values recorded at 10 ms intervals between 70–130 ms for the P1 and 140–180 ms for the N1, collapsed across ROIs LOT and ROT. One-sample t-tests were carried out separately for the young, young-old, and old-old groups to determine the temporal intervals in which mean values differ significantly from 0 μV. The amplitude of the P1 and N1 was derived from the mean amplitude of the 30 ms interval centered at the component's mean peak latency for each age group. ERPs were analyzed using analysis of variance (ANOVA), with memory load, and ROI as within-subject variables, and age group as the between-subject variable. Given our interest in placing this data set within the context of the existing literature, much of which included only 2 age groups, we also report the results of the pairwise statistical analyses (young vs. young-old, young vs. old-old, young-old vs. old-old) regardless of the outcome of the overall effect of age. The Geisser-Greenhouse correction was applied for all repeated measures with greater than 1 degree of freedom. Statistical analyses of ERPs and behavioral variables were repeated controlling for visual acuity using analysis of covariance. Finally, regression analyses were used to explore relationships between age, visual acuity, ERP components, and behavioral performance.

3.0 Results

3.1 Participants

The young subject group included 25 subjects between the ages of 19 and 29 (mean age = 22.6 (2.3)). The young-old (y-old) subject group included 15 subjects between the ages of 67 and 79 (mean age = 73.9 (3.7)). The old-old (o-old) subject group included 22 subjects between the ages of 80 and 92 (mean age = 84.2 (3.4)). Table 1 provides a summary of information about subject characteristics. An additional 3 young, 3 young-old, and 3 old-old subjects participated in the experiment, but were excluded due to excessively noisy ERP data.

3.1.1 Neuropsychological Test Scores—The composite z-scores on neuropsychological tests differed across age groups ($F_{2,59} = 6.98$, $p < 0.005$), such that young adults had higher scores than young-old ($p < 0.05$) and old-old ($p < 0.05$) adults, with no difference between the latter two groups (Table 1). After controlling for visual acuity, the difference across groups became marginal ($F_{2,58} = 2.99$, $p < 0.06$). There were no differences across age groups in raw scores on Digit Span Backward, Controlled Oral Word Association Test, Logical Memory II, or the Boston Naming test. Age-related decline (all p's < 0.005) was observed in Matrix Reasoning, Visual Form Discrimination, Digit Symbol, Letter-Number Sequencing, Digit Span Forward, and Trail Making Parts A and B. After controlling for visual acuity, all of these results survived, except for the age-associated differences in Digit Span Forward and Trail Making Parts A and B.

3.1.2 Visual Acuity—Visual acuities ranged from 20/16 to 20/80, with an overall mean of 0.83 (0.26) ≈ 20/24 (Table 1). Fifty-eight out of 62 subjects had visual acuities of 20/40 or better. Table 2 presents a summary of the number of subjects in each age group who exhibited each of the different gradations of visual acuity from 20/16 to 20/80. An ANOVA for visual acuity revealed an effect of age ($F_{2,61} = 30.78$, $p < 0.001$), such that young (1.05 (0.20) ≈ 20/20) had better visual acuity than young-old (0.75 (0.17) ≈ 20/27) ($p < 0.001$) and old-old subjects (0.63 (.18) ≈ 20/32) ($p < 0.001$). Young-old subjects tended to have better visual acuity than old-old subjects ($p < 0.08$).

We were interested in determining whether the older individuals (young-old and old-old groups combined, n = 37) with worse visual acuity exhibited poorer performance on cognitive tests than their age-matched peers. There were no differences in the composite z-scores on neuropsychological tests between subjects with visual acuity better than 20/40 vs. those with 20/40 or worse, or between subjects with visual acuity better than 20/30 vs. those with 20/30 or worse. Raw scores on cognitive tests were also examined. Across the older subjects, the only test in which subjects with visual acuity better than 20/40 scored better than subjects with 20/40 or worse was Digit-Symbol Coding ($p < 0.05$). The only test in which older subjects with visual acuity better than 20/30 scored better than subjects with 20/30 or worse was Trail Making Test Part A ($p < 0.07$), and this was only a trend. In summary, among older subjects, impairment in visual acuity was not associated with worse cognitive performance, except perhaps on two timed tests highly dependent on visual processing.

3.2 Behavior

An ANOVA of target hit rate revealed no effect of age group (young: 93.0 (0.1) %, y-old: 96.0 (0.0) %, o-old: 93.2 (0.1) %), but an effect of memory load ($F_{1,59} = 34.13$, $p < 0.001$), and an age × memory load interaction ($F_{2,59} = 4.47$, $p < 0.05$). The effect of load was present because target hit rate was higher on the low load task (97.0 (5.2) %) than on the high load task (92.0 (6.7) %). The magnitude of this difference was larger for young adults than for the other two groups. Interestingly, after controlling for visual acuity, an effect of age emerged ($F_{2,58} = 3.72$, $p < 0.05$), which was present because older subjects had a higher target hit rate than young adult subjects (o-old > young ($p < 0.05$); y-old > young ($p < 0.01$); o-old = y-old ($p > 0.6$)). However, there was no longer an effect of memory load ($p > 0.3$), or an age × load interaction ($p > 0.1$).[2]

An ANOVA of false alarm rate revealed an effect of age ($F_{2,59} = 7.45$, $p < 0.005$) (young: 0.4% (0.5), y-old: 1.0% (0.9), o-old: 1.0% (1.0)), but no effect of memory load or interaction between age and memory load. Young subjects had a lower false alarm rate than old-old subjects ($p < 0.001$) and a marginally lower false alarm rate than young-old subjects ($p < 0.08$). Young-old and old-old did not differ in their false alarm rates. After controlling for visual acuity, there was no longer an effect of age on false alarms ($p > 0.1$).

An ANOVA of median RTs revealed a marginal effect of age ($F_{2,59} = 2.51$, $p < 0.1$), an effect of memory load ($F_{1,59} = 276.39$, $p < 0.001$), and no age × memory load interaction. Young subjects responded faster than old-old ($p < 0.05$) and marginally faster than young-old ($p < 0.1$) subjects. There was no difference between young-old and old-old in median reaction time ($p > 0.8$) (young: 536 (46) ms, y-old: 566 (52) ms, o-old: 568 (64) ms)). Subjects responded faster in the low load task (504 (7) ms) than the high load task (609 (8) ms). After controlling for visual acuity, there was no longer an effect of age on median reaction time ($p > 0.8$), but the effect of memory load survived.

3.3 ERPs

The grand average ERPs in response to standard stimuli under the Attend condition at the 8 lateral ROIs are presented for the low load task in Figure 3a and for the high load tasks in Figure 3b. Because there was no effect of load and no interaction between age group and load for the latency or amplitude of the P1 or N1 components, the results presented are collapsed across the low and high load tasks. Figure 4 presents the surface potential maps, illustrating the scalp distribution of the posterior P1 and posterior N1 for each group. (See Supplemental Figure 1 for an illustration of the scalp topographies of the difference waves between age groups.) Figure 5 illustrates the grand average ERPs for the 3 age groups collapsed across memory load and the lateral posterior ROIs, LOT and ROT. In presenting the results, effects of ROI or interactions between group and ROI are not provided unless the pattern changed after controlling for visual acuity.

3.3.1 Posterior P1 Component—An ANOVA for the P1 latency revealed an effect of age ($F_{2,59} = 3.42$, $p < 0.05$), which was present because P1 latency was earlier for young subjects (103.6 (2.6) ms) than for both young-old (113.0 (3.4) ms) and old-old subjects (112.1 (2.8) ms) (young < y-old ($p < 0.05$); young < o-old ($p < 0.05$); y-old = o-old ($p > 0.8$)). After controlling for visual acuity, the overall effect of age was no longer significant ($F_{2,58} = 2.27$, $p > 0.1$). Follow-up pairwise comparisons after controlling for visual acuity indicated that young subjects continued to exhibit an earlier P1 latency than young-old subjects ($p < 0.05$), but there were no longer reliable differences between young and old-old subjects ($p > 0.05$).

The amplitude of the P1 was measured as the mean value between 90–120 ms for young subjects and 100–130 ms for both groups of older subjects. An ANOVA revealed no effect of age ($p > 0.6$). However, controlling for visual acuity resulted in an effect of age ($F_{2,58} = 3.98$, $p < 0.05$), which was explained by both old-old and young-old subjects exhibiting a larger P1 amplitude than young subjects (y-old > young ($p < 0.05$); o-old > young ($p < 0.05$); o-old = y-old ($p > 0.9$)).

Time course analysis using one-sample t-tests, performed separately on the young, young-old, and old-old groups, revealed mean values significantly greater than 0 μV, consistent with a P1, between 70–80 ms for the young ($p < 0.001$), but not the old groups. Between 80–130 ms all groups generated a robust P1 component (all p's < 0.005). Results are presented in Supplemental Table 1. An ANOVA demonstrated that the size of the P1 was larger for young subjects than the other two groups from 70–80 ms ($p < 0.005$), with no group differences during any 10 ms interval from 80–130 ms. After controlling for visual acuity, the size of the P1of young subjects no longer differed from old subject from 70–80 ms. For each of the 10 ms intervals between 90–130 ms, both young-old and old-old subjects generated a larger P1 than young subjects (all p's < 0.05).

3.3.2 Posterior N1 Component—An ANOVA for N1 latency revealed no effect of age ($p > 0.1$), with the overall mean value being 161.7 (1.7) ms. Although the overall effect of age did not reach significance, pair-wise group analyses demonstrated that the latency of the N1 peaked earlier for young (163.5 (2.6) ms) than for old-old subjects (171.2 (2.7) ms), with no difference between young and young-old (168.2 (3.3) ms), or young-old and old-old (young < o-old ($p < 0.05$); young = y-old ($p > 0.2$); y-old = old-old ($p > 0.4$)). After controlling for visual acuity, an ANOVA revealed no effect of age ($p > 0.4$), and the difference in N1 latency between young and old-old subjects was no longer evident ($p > 0.1$).

The amplitude of the N1 was measured as the mean value between 150–180 ms for young, 155–185 ms for young-old, and 160–190 ms for old-old subjects. An ANOVA was

noteworthy for an effect of age ($F_{2,59} = 3.94$, $p < 0.05$) and an effect of ROI ($F_{1,59} = 11.27$, $p < 0.01$). The effect of age was explained by a smaller (muted) N1 response for old-old subjects as compared to young ($p < 0.05$) and young-old ($p < 0.05$) subjects, with no difference between young and young-old subjects ($p > 0.7$). The effect of ROI was present because the N1 amplitude was greater at ROI LOT. Controlling for visual acuity did not alter the effect of age ($F_{2,58} = 3.64$, $p < 0.05$). However, the effect of ROI was no longer significant ($p > 0.5$).[3,4,5]

Time course analysis using one-sample t-tests, performed separately on the young, young-old, and old-old groups, revealed mean values significantly greater than 0 μV, consistent with a N1, between 140–150 ms for the young ($p < 0.05$), but not the old groups. Between 150–180 ms young and young-old subjects generated a reliable N1 (all p's < 0.05). However, during no interval did the mean value for the old-old group differ from 0 μV. Results are presented in Supplemental Table 1. An ANOVA demonstrated that the size of the N1 was larger for young subjects than old-old subjects during each 10 ms interval from 140–170 ms (p's < 0.05). Young-old subjects generated a larger N1 than old-old subjects for each 10 ms interval from 160–180 ms. After controlling for visual acuity, the size of the N1 of young subjects no longer differed from old-old subject from 150–170 ms. Young-old subjects continued to exhibit a larger N1 than old-old between 160–180 ms (p's < 0.05).

3.4 Regression Analyses

Age strongly predicted visual acuity ($r^2 = 0.52$, $r = -0.72$, $p < 0.001$), such that the greater the age, the worse the visual acuity. Age also predicted performance on neuropsychological tests ($r^2 = .18$, $r = -0.43$, $p < 0.001$): the greater the age, the lower the composite z-score. This relationship survived controlling for visual acuity.

Collapsing across memory load, age predicted RT ($r^2 = 0.07$, $r = 0.27$, $p < 0.05$) and false alarm rate ($r^2 = 0.21$, $r = 0.46$, $p < 0.0005$), such that the greater the age, the worse the performance. After controlling for visual acuity, age no longer predicted median reaction time ($p > 0.7$), but continued to predict false alarm rate, although the relationship appeared less robust ($r^2 = 0.07$, $r = 0.26$, $p < 0.05$). There was no correlation between age and target hit rate ($p > 0.13$). However, after controlling for visual acuity, a correlation was observed ($r^2 = 0.09$, $r = 0.30$, $p < 0.05$): the greater the age, the higher the hit rate.

Age correlated with P1 peak latency ($r^2 = .10$, $r = .31$, $p < 0.05$) and N1 peak latency ($r^2 = .07$, $r = .26$, $p < 0.05$), such that the greater the age, the later the latencies. After controlling for visual acuity age continued to correlate with P1 latency ($r^2 = .07$, $r = .26$, $p < 0.05$), but no longer correlated with N1 latency ($r^2 = .03$, $r = .17$, $p > 0.17$). Age did not correlate with P1 amplitude ($r = .09$, $p > 0.49$) or N1 amplitude ($r = 0.20$, $p > 0.12$). Accounting for visual acuity resulted in age predicting P1 amplitude ($r^2 = .13$, $r = .37$, $p < 0.005$), but not N1 amplitude ($r = 0.16$, $p > 0.20$).

Visual acuity did not predict P1 latency, N1 latency, or N1 amplitude. Visual acuity marginally predicted P1 amplitude ($r^2 = .05$, $r = .23$, $p < 0.08$). After controlling for age, the relationship became robust ($r^2 = .17$, $r = .41$, $p < 0.005$); the better the visual acuity, the larger the P1 amplitude.

4.0 Discussion

The main purpose of this study was to investigate whether age-associated differences in ERP indices of early cortical visual processing were affected by differences in visual acuity. We found that controlling for visual acuity had an impact on whether older and younger adults differed significantly in the size and latency of the posterior P1 and N1 components. After

accounting for visual acuity, age-related increases in P1 and N1 latency did not remain significant, and older adults were found to have a larger P1 amplitude than young adults. Controlling for differences in vision also had an effect on behavioral variables. Despite intentionally making task demands easier for older subjects, there were age-associated increases in RT and false alarm rate. However, after accounting for visual acuity, these differences did not remain significant, and older subjects were found to have a higher hit rate than their younger counterparts. Below, we discuss the implications of our findings for research in this area.

Numerous ERP studies of visual processing demonstrate changes associated with normal aging. However, few provide clinical measures of visual acuity in their subjects. Most seem to have relied upon the self-report of participants indicating they had normal or corrected-to-normal vision. Our study and others (Friedman et al., 1999; Skeel et al., 2003) indicate that this kind of reporting may not be accurate. Because of absent documentation, it is impossible to ascertain the degree to which the visual acuity of subjects differed across studies, or contributed to the marked variability in reports of age-related differences in P1 and N1, especially in terms of amplitude (Celesia and Daly, 1977; Ceponiene et al., 2008; Curran et al., 2001; Czigler and Balazs, 2005; De Sanctis et al., 2008; Diaz and Amenedo, 1998; Dustman et al., 1981; Dustman and Beck, 1966; Dustman and Snyder, 1981; Falkenstein et al., 2006; Finnigan et al., 2011; Kolev et al., 2006; Kutas et al., 1994; Yordanova et al., 2004; Zanto et al., 2010).[6]

Age-associated differences in P1 and N1 have most often been understood in terms of the impact that aging has on early cortical visual processing (Ceponiene et al., 2008; De Sanctis et al., 2008; Diaz and Amenedo, 1998; Dustman and Beck, 1966; Dustman and Snyder, 1981; Finnigan et al., 2011; Zanto et al., 2010). In keeping with this practice, the results of the current study could be interpreted as indicating that older adults have preserved initial visual encoding (indexed by the P1 amplitude), degraded preliminary visual discrimination (indexed by the N1 amplitude), and slowed execution of both of these visual processing operations (indexed by P1 and N1 latencies).

Potential mechanisms cited in the literature to explain age-related differences in P1 and N1 include alterations in the neural structure or functioning of early visual processing areas, changes in top-down modulation of early sensory processing, a generalized reduction of processing speed, and loss of neurotransmitter availability (Ceponiene et al., 2008; Curran et al., 2001; De Sanctis et al., 2008; Diaz and Amenedo, 1998; Dustman and Snyder, 1981; Zanto et al., 2010). Age-associated reduction in the amplitude of the P1 or N1 have been interpreted as a reflection of the degradation of posterior brain areas (Ceponiene et al., 2008; Diaz and Amenedo, 1998), consistent with evidence of structural and physiological decline in posterior visual regions (Goh, 2011; Schmolesky et al., 2000; Spear, 1993; Yu et al., 2006), diminished activity on functional imaging studies (Davis et al., 2008; Dennis and Cabeza, 2008), and alteration in psychophysical markers of parvocellular and magnocellular pathways (Elliott and Werner, 2010). Alternatively, changes in the size of the posterior P1 and N1 components may represent the effects of impaired cortical-to-cortical facilitation, inhibition, or gating of early sensory operations (Ceponiene et al., 2008; Dustman et al., 1981; Dustman and Snyder, 1981; Finnigan et al., 2011). The prolongation of the P1 and N1 latencies has been interpreted as a function of age-related decline in the integrity of white matter tracts that slows the transmission of visual information between different nodes of the visual system, or a disruption of the cortical processing indexed by these components (Celesia and Daly, 1977; Diaz and Amenedo, 1998; Finnigan et al., 2011; Zanto et al., 2010).

The potential influence of visual acuity on findings of age-related differences in the P1 and N1 components has not been emphasized previously in the literature. This oversight may be problematic given the strong evidence that aging is associated with an increased likelihood of reduced visual acuity (Rodriguez et al., 2002; Rubin et al., 1997; Spear, 1993), an observation consistent with the results of the current study. Although changes in the central nervous system, such as alterations in the geniculo-striate pathway, may contribute to reduced visual acuity in older adults (Spear, 1993), most of the age-related decline is believed to be due to changes in the cornea or retina (Faubert, 2002; Kanthan et al., 2008; Klaver et al., 1998; Munoz et al., 2000; Rodriguez et al., 2002). For example, there are age-associated alterations in the thickness and color (yellowing) of the lens (Nguyen-Tri et al., 2003; Suzuki et al., 2006). One study (Kanthan et al., 2008) estimated that 72% of adults who were 49 years and older had cataracts or had had cataract surgery over a 10-year follow-up period. Moreover, the insidious development of cataracts often goes unrecognized (Rodriguez et al., 2002; Wood et al., 2010). Retinal changes leading to diminished visual acuity are associated with glaucoma, macular degeneration, diabetic retinopathy, and normal aging (Faubert, 2002; Klaver et al., 1998; Munoz et al., 2000; Rodriguez et al., 2002; Spear, 1993).

Support for a causal link between altered visual acuity due to peripheral factors and changes in visually-evoked responses come from studies that have experimentally manipulated visual acuity in healthy young adults through the use of external lenses. These studies have demonstrated that creating errors of refraction reduces the size of early visually-evoked responses (Harter and White, 1968; Millodot and Riggs, 1970). In keeping with these studies, the failure to account for refractive errors in investigations of cognitive aging could, for example, lead to incorrect inferences about age-related decline in the size of the P1 amplitude, or the inability to observe a true age-associated increase in P1 amplitude. Differences in visual acuity have also been shown to play a role in the apparent disruption of early visual ERP components in clinical populations other than aging (Friedman et al., 2012; Schechter et al., 2005). For example, in one study, differences between schizophrenic patients and normal subjects in N1 amplitude and latency, and in C1 latency did not remain significant after controlling for visual acuity, which was a particularly striking finding because subjects had been excluded if their vision or corrected vision was worse than 20/32 (Schechter et al., 2005).

A large body of research suggests a strong link between age-associated changes in non-cognitive variables, such as vision, and a variety of measures of cognitive aging (Baltes and Lindenberger, 1997; Lindenberger et al., 2001; Lindenberger and Baltes, 1994; Skeel et al., 2003). The classic version of the common cause hypothesis interprets this relationship as support for a common, biologically-based factor that is responsible for age-related deterioration at all levels of functioning, including ones that mediate peripheral sensory-motor operations (Baltes and Lindenberger, 1997; Christensen et al., 2001; Lindenberger et al., 2001; Lindenberger and Baltes, 1994). One could interpret our findings as corroborating this theory because after controlling for visual acuity age-related declines in early visual ERPs and performance on the experimental task did not survive. However, this explanation is countered by the observation that among older subjects, impairment of visual acuity was not associated with worse cognitive performance across a wide-range of neuropsychological tests, but was limited to ones particularly dependent on timed visual processing. Our findings seem more consistent with the idea that impaired vision has a negative impact on the ability to optimally see test or experimental material on tasks requiring vision (Anstey et al., 2001; Lindenberger and Baltes, 1994; Wood et al., 2010). Further research is necessary to confirm this hypothesis.

Several limitations to this study merit comment. Importantly, we do not know the cause(s) of diminished visual acuity in our subjects. The most benign possibility is that the subjects had inadequate refraction and needed an updated prescription for corrective lenses (Skeel et al., 2003), which is a common occurrence (Tielsch et al., 1990). No formal ophthalmologic examination was performed to evaluate abnormalities of the lens, or to determine if cataracts or retinal pathology were present, which are relatively common in older individuals (Faubert, 2002; Kanthan et al., 2008; Klaver et al., 1998; Munoz et al., 2000; Rodriguez et al., 2002). Presumably, in rare cases in which subjects with diminished vision have no ocular or retinal pathology, there is an increased chance that alteration in visual acuity is a reflection of more central factors. To the extent that worse visual acuity is the result of deterioration in white matter pathways or cortical visual areas, age-associated changes in visual acuity and the P1/N1 components may be serving as alternative ways to measure the decline in the same neuroanatomic structures. Since controlling for visual acuity in these instances may eliminate age-related differences in the measurement of P1 and N1 that are due to alteration in posterior cortical function, it may be reasonable to consider excluding such subjects from participating in ERP studies of visual processing.

Additional research is needed to demonstrate that the findings are not limited to the particular task demands and stimuli of our experiment, which involved selective attention to color, and presented letters subtending a visual angle of ~2.5° for 250 ms under a condition of high contrast. It would be interesting for future studies to present visual stimuli under conditions that manipulate luminance and color contrast to determine the extent to which visual acuity affects early visual ERP components that may be differentially influenced by magnocellular and parvocellular pathways mediating these visual functions (Elliott and Werner, 2010; Schechter et al., 2005).

Although our sample was a reasonable size (n = 62), before offering more definitive suggestions, the findings need to be replicated with a larger number of subjects. Also, approximately a third of our sample were 80 years and older, which probably influenced the outcome, as decline in visual acuity became more common as a function of age. The issues raised by this study are likely to become increasingly important as more attention is paid to the study of normal cognitive aging in adults over the age of 80, which constitutes the fastest growing sector of our population (Kinsella and He, 2009). We face the important challenge of developing a consensus about how to account for age-related differences in sensory fidelity in the study of neural markers of normal cognitive aging among the very old.

It also remains to be determined the extent to which age-related differences in visual acuity have an impact on later cognitive processing (e.g., as indexed, by the N2, P3, or slow wave components), or affect other ways to measure neural activity, such as fMRI or PET. For instance, there is an important line of research that has identified age-associated increases in anterior neural activity, as indexed by functional imaging, in response to various task demands. Some investigators have interpreted this finding as a compensatory mechanism for deterioration in early posterior visual processing (Cabeza et al., 2004; Davis et al., 2008; Park and Reuter-Lorenz, 2009; Reuter-Lorenz et al., 2000). Some of the studies (e.g., Reuter-Lorenz et al., 2000) indicate that subjects had normal or corrected-to-normal vision, whereas other studies (e.g., Cabeza et al., 2004; Davis et al., 2008) do not include information about visual acuity. To the best of our knowledge, these investigations have not tried to control for age-associated differences in visual acuity, which could impact neural markers of early visual processing.

In summary, the most important message of this study is that until the relationship between age-associated differences in visual acuity and early ERPs is clearly established, investigators may need to be more circumspect when interpreting the meaning of their

findings. Researchers using visual protocols to examine age-associated differences in neural activity during cognitive processing need to obtain information not only about conditions that may affect neurologic functioning, such as dementia, cerebrovascular disease, diabetes, and depression, but also risk factors that can impair vision, such as cataracts, glaucoma, and macular degeneration. It does not appear to be sufficient to simply ask subjects if they have normal or corrected-to-normal vision. Rather, it seems prudent to include a clinical measure of visual acuity. Additional investigation is necessary to determine the extent to which the issues addressed in this paper are critical to the study of age-related differences in neural activity in response to visual tasks and to establish guidelines for future research, especially of very old adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4. Washington, D.C: American Psychiatric Association; 1994.
2. Anstey KJ, Luszcz MA, Sanchez L. A reevaluation of the common factor theory of shared variance among age, sensory function, and cognitive function in older adults. J Gerontol B Psychol Sci Soc Sci. 2001; 56:3–11.
3. Baltes PB, Lindenberger U. Emergence of a powerful connection between sensory and cognitive functions across the adult life span: a new window to the study of cognitive aging? Psychol Aging. 1997; 12:12–21. [PubMed: 9100264]
4. Beck, AT.; Steer, RA. Beck Depression Inventory: Manual. San Antonio, TX: The Psychological Corporation; 1987.
5. Benton, AL.; Varney, NR.; DeSHamsher, K.; Spreen, O. Contributions to Neuropsychological Assessment. Oxford: Oxford University Press; 1983.
6. Cabeza R, Daselaar SM, Dolcos F, Prince SE, Budde M, Nyberg L. Task-independent and task-specific age effects on brain activity during working memory, visual attention and episodic retrieval. Cereb Cortex. 2004; 14:364–375. [PubMed: 15028641]
7. Celesia GG, Daly RF. Effects of aging on visual evoked responses. Arch Neurol. 1977; 34:403–407. [PubMed: 880065]
8. Ceponiene R, Westerfield M, Torki M, Townsend J. Modality-specificity of sensory aging in vision and audition: Evidence from event-related potentials. Brain Res. 2008; 1215:53–68. [PubMed: 18482717]
9. Christensen H, Mackinnon AJ, Korten A, Jorm AF. The “common cause hypothesis” of cognitive aging: evidence for not only a common factor but also specific associations of age with vision and grip strength in a cross-sectional analysis. Psychol Aging. 2001; 16:588–599. [PubMed: 11766914]
10. Curran T, Hills A, Patterson MB, Strauss ME. Effects of aging on visuospatial attention: an ERP study. Neuropsychologia. 2001; 39:288–301. [PubMed: 11163607]
11. Czigler I, Balazs L. Age-related effects of novel visual stimuli in a letter-matching task: an event-related potential study. Biol Psychol. 2005; 69:229–242. [PubMed: 15804549]
12. Daffner KR, Sun X, Tarbi E, Rentz DM, Holcomb PJ, Riis JL. Does compensatory neural activity survive old-old age? NeuroImage. 2011; 54:427–438. [PubMed: 20696255]

13. Daffner KR, Zhuravleva TY, Sun X, Tarbi EC, Haring AE, Rentz DM, Holcomb PJ. Does modulation of selective attention to features reflect enhancement or suppression of neural activity? Biol Psychol. 2012a; 89:398–407. [PubMed: 22178708]
14. Daffner KR, Tarbi EC, Haring AE, Zhuravleva TY, Sun X, Rentz DM, Holcomb PJ. The influence of executive capacity on selective attention and subsequent processing. Frontiers in Human Neuroscience. 2012b; 6:1–19.
15. Daselaar, SM.; Cabeza, R. Age-Related Changes in Hemispheric Organization. In: Cabeza, R.; Nyberg, L.; Park, D., editors. Cognitive Neuroscience of Aging. New York: Oxford University Press; 2005. p. 325-353.
16. Davis SW, Dennis NA, Daselaar SM, Fleck MS, Cabeza R. Que PASA? The Posterior Anterior Shift in Aging. Cereb Cortex. 2008; 18:1201–1209. [PubMed: 17925295]
17. De Sanctis P, Katz R, Wylie GR, Sehatpour P, Alexopoulos GS, Foxe JJ. Enhanced and bilateralized visual sensory processing in the ventral stream may be a feature of normal aging. Neurobiol Aging. 2008; 10:1576–1586. [PubMed: 17478011]
18. Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. Journal of Neuroscience Methods. 2004; 134:9–21. [PubMed: 15102499]
19. Dennis, NA.; Cabeza, R. Neuroimaging of healthy cognitive aging. In: Craik, FI.; Salthouse, TA., editors. The handbook of aging and cognition. 3. New York, NY, US: Psychology Press; 2008. p. 1-54.
20. Diaz F, Amenedo E. Ageing effects on flash visual evoked potentials (FVEP) recorded from parietal and occipital electrodes. Neurophysiologie Clinique/Clinical Neurophysiology. 1998; 28:399–412.
21. Dustman RE, Beck EC. Visually evoked potentials: amplitude changes with age. Science. 1966; 151:1013–1015. [PubMed: 5907289]
22. Dustman RE, Snyder EW. Life-span change in visually evoked potentials at central scalp. Neurobiol Aging. 1981; 2:303–308. [PubMed: 7335148]
23. Dustman RE, Snyder EW, Schlehuber CJ. Life-span alterations in visually evoked potentials and inhibitory function. Neurobiol Aging. 1981; 2:187–192. [PubMed: 7312096]
24. Elliott SL, Werner JS. Age-related changes in contrast gain related to the M and P pathways. J Vis. 2010; 10:4–15. [PubMed: 20465324]
25. Falkenstein M, Yordanova J, Kolev V. Effects of aging on slowing of motor-response generation. Int J Psychophysiol. 2006; 59:22–29. [PubMed: 16257076]
26. Faubert J. Visual perception and aging. Can J Exp Psychol. 2002; 56:164–176. [PubMed: 12271747]
27. Finnigan S, O'Connell RG, Cummins TD, Broughton M, Robertson IH. ERP measures indicate both attention and working memory encoding decrements in aging. Psychophysiology. 2011; 48:601–611. [PubMed: 21039584]
28. Folstein MF, Folstein SE, McHugh PR. "Mini-Mental State". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975; 12:189–198. [PubMed: 1202204]
29. Friedman SM, Munoz B, Rubin GS, West SK, Bandeen-Roche K, Fried LP. Characteristics of discrepancies between self-reported visual function and measured reading speed. Salisbury Eye Evaluation Project Team. Invest Ophthalmol Vis Sci. 1999; 40:858–864. [PubMed: 10102282]
30. Friedman T, Sehatpour P, Dias E, Perrin M, Javitt DC. Differential relationships of mismatch negativity and visual p1 deficits to premorbid characteristics and functional outcome in schizophrenia. Biol Psychiatry. 2012; 71:521–529. [PubMed: 22192361]
31. Goh JO. Functional Dedifferentiation and Altered Connectivity in Older Adults: Neural Accounts of Cognitive Aging. Aging and Disease. 2011; 2:30–48. [PubMed: 21461180]
32. Harter MR, White CT. Effects of contour sharpness and check-size on visually evoked cortical potentials. Vision Res. 1968; 8:701–711. [PubMed: 5729913]
33. Hillyard SA, Anllo-Vento L. Event-related brain potentials in the study of visual selective attention. Proc Natl Acad Sci USA. 1998; 95:781–787. [PubMed: 9448241]

34. Hillyard SA, Munte TF. Selective attention to color and location: an analysis with event-related brain potentials. Percept Psychophys. 1984; 36:185–198. [PubMed: 6514528]
35. Hillyard SA, Teder-Salejarvi WA, Munte TF. Temporal dynamics of early perceptual processing. Curr Opin Neurobiol. 1998a; 8:202–210. [PubMed: 9635203]
36. Hillyard SA, Vogel EK, Luck SJ. Sensory gain control (amplification) as a mechanism of selective attention: electrophysiological and neuroimaging evidence. Philos Trans R Soc Lond B Biol Sci. 1998b; 353:1257–1270. [PubMed: 9770220]
37. Ivnik RJ, Malec JF, Smith GE, Tangalos EG, Petersen RC. Neuropsychological tests' norms above age 55: COWAT, BNT, MAE Token, WRAT-R Reading, AMNART, STROOP, TMT, and JLO. Clin Neuropsychol. 1996; 10:262–278.
38. Johannes S, Munte TF, Heinze HJ, Mangun GR. Luminance and spatial attention effects on early visual processing. Brain Res Cogn Brain Res. 1995; 2:189–205. [PubMed: 7580401]
39. Kanthan GL, Wang JJ, Rochtchina E, Tan AG, Lee A, Chia EM, Mitchell P. Ten-year incidence of age-related cataract and cataract surgery in an older Australian population. The Blue Mountains Eye Study. Ophthalmology. 2008; 115:808–814. [PubMed: 17900695]
40. Kinsella, K.; He, W. International Population Report. Washington: US Government Printing Office; 2009. An Aging World: 2008 US Census Bureau.
41. Klaver CC, Wolfs RC, Vingerling JR, Hofman A, de Jong PT. Age-specific prevalence and causes of blindness and visual impairment in an older population: the Rotterdam Study. Arch Ophthalmol. 1998; 116:653–658. [PubMed: 9596502]
42. Kolev V, Falkenstein M, Yordanova J. Motor-response generation as a source of aging-related behavioural slowing in choice-reaction tasks. Neurobiol Aging. 2006; 27:1719–1730. [PubMed: 16246465]
43. Kutas M, Iragui V, Hillyard SA. Effects of aging on event-related brain potentials (ERPs) in a visual detection task. Electroencephalogr Clin Neurophysiol. 1994; 92:126–139. [PubMed: 7511510]
44. Lindenberger U, Baltes PB. Sensory functioning and intelligence in old age: a strong connection. Psychol Aging. 1994; 9:339–355. [PubMed: 7999320]
45. Lindenberger U, Scherer H, Baltes PB. The strong connection between sensory and cognitive performance in old age: not due to sensory acuity reductions operating during cognitive assessment. Psychol Aging. 2001; 16:196–205. [PubMed: 11405308]
46. Luck, SJ. An Introduction to the Event-Related Potential Technique. Cambridge: The MIT Press; 2005.
47. Mangun, GR.; Hillyard, SA.; Luck, SJ. Electrocortical Substrates of Visual Selective Attention. In: Meyers, DE.; Kornblum, S., editors. Attention and Performance XIV, Synergies in Experimental Psychology, Artificial Intelligence, and Cognitive Neuroscience. 1990. p. 219-243.
48. Martinovic J, Mordal J, Wuerger SM. Event-related potentials reveal an early advantage for luminance contours in the processing of objects. Journal of Vision. 2011; 11
49. Millodot M, Riggs LA. Refraction determined electrophysiologically. Responses to alternation of visual contours. Arch Ophthalmol. 1970; 84:272–278. [PubMed: 5457478]
50. Munoz B, West SK, Rubin GS, Schein OD, Quigley HA, Bressler SB, Bandeen-Roche K. Causes of blindness and visual impairment in a population of older Americans: The Salisbury Eye Evaluation Study. Arch Ophthalmol. 2000; 118:819–825. [PubMed: 10865321]
51. Nagamatsu LS, Carolan P, Liu-Ambrose TY, Handy TC. Age-related changes in the attentional control of visual cortex: a selective problem in the left visual hemifield. Neuropsychologia. 2011; 49:1670–1678. [PubMed: 21356222]
52. Natale E, Marzi CA, Girelli M, Pavone EF, Pollmann S. ERP and fMRI correlates of endogenous and exogenous focusing of visual-spatial attention. Eur J Neurosci. 2006; 23:2511–2521. [PubMed: 16706858]
53. Nguyen-Tri D, Overbury O, Faubert J. The role of lenticular senescence in age-related color vision changes. Invest Ophthalmol Vis Sci. 2003; 44:3698–3704. [PubMed: 12882826]
54. Park DC, Reuter-Lorenz P. The adaptive brain: aging and neurocognitive scaffolding. Annu Rev Psychol. 2009; 60:173–196. [PubMed: 19035823]

55. Reitan, R.; Wolfson, D. The Halstead-Reitan Neuropsychological Test Battery: Theory and Clinical Interpretation. Tucson: Neuropsychology Press; 1985.
56. Reuter-Lorenz PA, Jonides J, Smith EE, Hartley A, Miller A, Marshuetz C, Koeppe RA. Age differences in the frontal lateralization of verbal and spatial working memory revealed by PET. J Cogn Neurosci. 2000; 12:174–187. [PubMed: 10769314]
57. Riis JL, Chong H, Ryan KK, Wolk DA, Rentz DM, Holcomb PJ, Daffner KR. Compensatory neural activity distinguishes different patterns of normal cognitive aging. NeuroImage. 2008; 39:441–454. [PubMed: 17931892]
58. Rodriguez J, Sanchez R, Munoz B, West SK, Broman A, Snyder RW, Klein R, Quigley H. Causes of blindness and visual impairment in a population-based sample of U.S. Hispanics. Ophthalmology. 2002; 109:737–743. [PubMed: 11927431]
59. Rubin GS, West SK, Munoz B, Bandeen-Roche K, Zeger S, Schein O, Fried LP. A comprehensive assessment of visual impairment in a population of older Americans. The SEE Study. Salisbury Eye Evaluation Project. Invest Ophthalmol Vis Sci. 1997; 38:557–568. [PubMed: 9071208]
60. Ryan J, Paolo A. A screening procedure for estimating premorbid intelligence in the elderly. Clin Neuropsychol. 1992; 6:53–62.
61. Schechter I, Butler PD, Zemon VM, Revheim N, Saperstein AM, Jalbrzikowski M, Pasternak R, Silipo G, Javitt DC. Impairments in generation of early-stage transient visual evoked potentials to magno- and parvocellular-selective stimuli in schizophrenia. Clin Neurophysiol. 2005; 116:2204–2215. [PubMed: 16055375]
62. Schmolesky MT, Wang Y, Pu M, Leventhal AG. Degradation of stimulus selectivity of visual cortical cells in senescent rhesus monkeys. Nature Neuroscience. 2000; 3:384–390.
63. Skeel RL, Nagra A, VanVoorst W, Olson E. The relationship between performance-based visual acuity screening, self-reported visual acuity, and neuropsychological performance. Clin Neuropsychol. 2003; 17:129–136. [PubMed: 13680419]
64. Spear PD. Neural bases of visual deficits during aging. Vision Res. 1993; 33:2589–2609. [PubMed: 8296455]
65. Suzuki TA, Qiang Y, Sakuragawa S, Tamura H, Okajima K. Age-related changes of reaction time and p300 for low-contrast color stimuli: Effects of yellowing of the aging human lens. J Physiol Anthropol. 2006; 25:179–187. [PubMed: 16679715]
66. Tielsch JM, Sommer A, Witt K, Katz J, Royall RM. Blindness and visual impairment in an American urban population. The Baltimore Eye Survey. Arch Ophthalmol. 1990; 108:286–290. [PubMed: 2271016]
67. Tombaugh TN, Hubley AM. The 60-item Boston Naming test: norms for cognitively intact adults aged 25 to 88 years. J Clin Exp Neuropsychol. 1997; 19:922–932. [PubMed: 9524887]
68. Vogel EK, Luck SJ. The visual N1 component as an index of a discrimination process. Psychophysiology. 2000; 37:190–203. [PubMed: 10731769]
69. Wechsler, D. Wechsler Adult Intelligence Scale. WAIS-III. Administration and Scoring Manual. San Antonio, TX: The Psychological Corporation; 1997.
70. Wechsler, D. Wechsler Adult Intelligence Scale. 4. San Antonio, TX: Pearson; 2008.
71. Werkle-Bergner M, Shing YL, Muller V, Li SC, Lindenberger U. EEG gamma-band synchronization in visual coding from childhood to old age: evidence from evoked power and inter-trial phase locking. Clin Neurophysiol. 2009; 120:1291–1302. [PubMed: 19482545]
72. Woldorff MG, Fox PT, Matzke M, Lancaster JL, Veeraswamy S, Zamarripa F, Seabolt M, Glass T, Gao JH, Martin CC, Jerabek P. Retinotopic Organization of Early Visual Spatial Attention Effects as Revealed by PET and ERP's. Human Brain Mapping. 1997; 5:280–286. [PubMed: 20408229]
73. Wood J, Chaparro A, Anstey K, Lacherez P, Chidgey A, Eisemann J, Gaynor A, La P. Simulated visual impairment leads to cognitive slowing in older adults. Optom Vis Sci. 2010; 87:1037–1043. [PubMed: 21037492]
74. Yesavage JA, Brink TL, Rose TL, Lum O, Huang V, Adey M, Leirer VO. Development and validation of a geriatric depression screening scale: a preliminary report. J Psychiatr Res. 1982; 17:37–49. [PubMed: 7183759]

75. Yordanova J, Kolev V, Hohnsbein J, Falkenstein M. Sensorimotor slowing with ageing is mediated by a functional dysregulation of motor-generation processes: evidence from high-resolution event-related potentials. Brain. 2004; 127:351–362. [PubMed: 14607784]

76. Yu S, Wang Y, Li X, Zhou Y, Leventhal AG. Functional degradation of extrastriate visual cortex in senescent rhesus monkeys. Neuroscience. 2006; 140:1023–1029. [PubMed: 16678974]

77. Zanto TP, Toy B, Gazzaley A. Delays in neural processing during working memory encoding in normal aging. Neuropsychologia. 2010; 48:13–25. [PubMed: 19666036]

Highlights

- Does visual acuity impact markers of age-related changes in visual processing?
- Posterior P1 and N1 ERPs were measured across young, young-old and old-old subjects
- Controlling for visual acuity eliminated age-related increases in P1 and N1 latency
- Controlling for visual acuity resulted in age-related increases in P1 amplitude
- Measuring/controlling for visual acuity is needed in ERP/imaging studies of aging

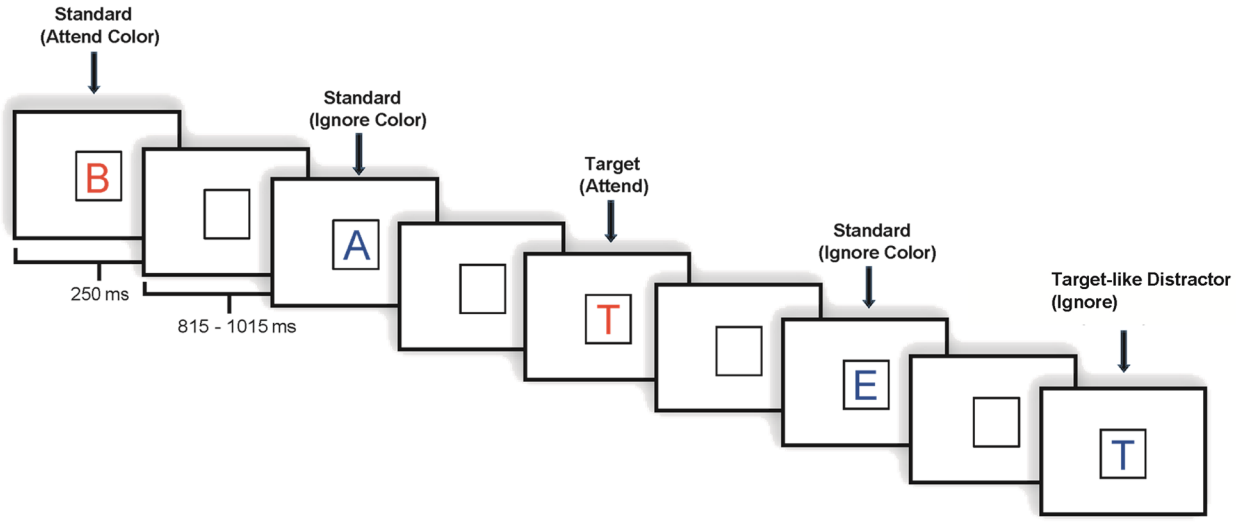

Figure 1.
Illustration of an experimental run.

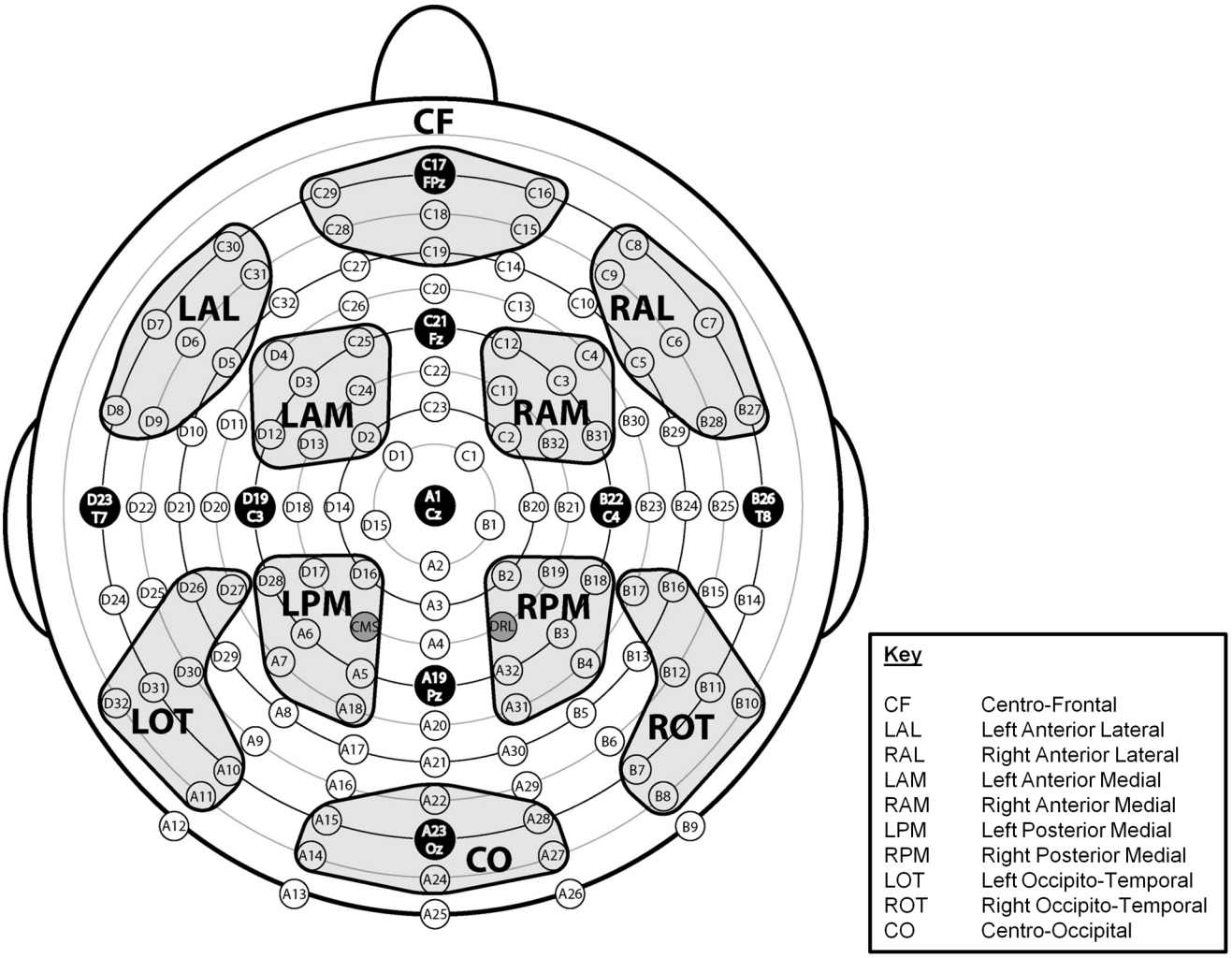

Figure 2.
Montage illustrating the location of 128 electrode sites and the designated regions of interest (ROIs).

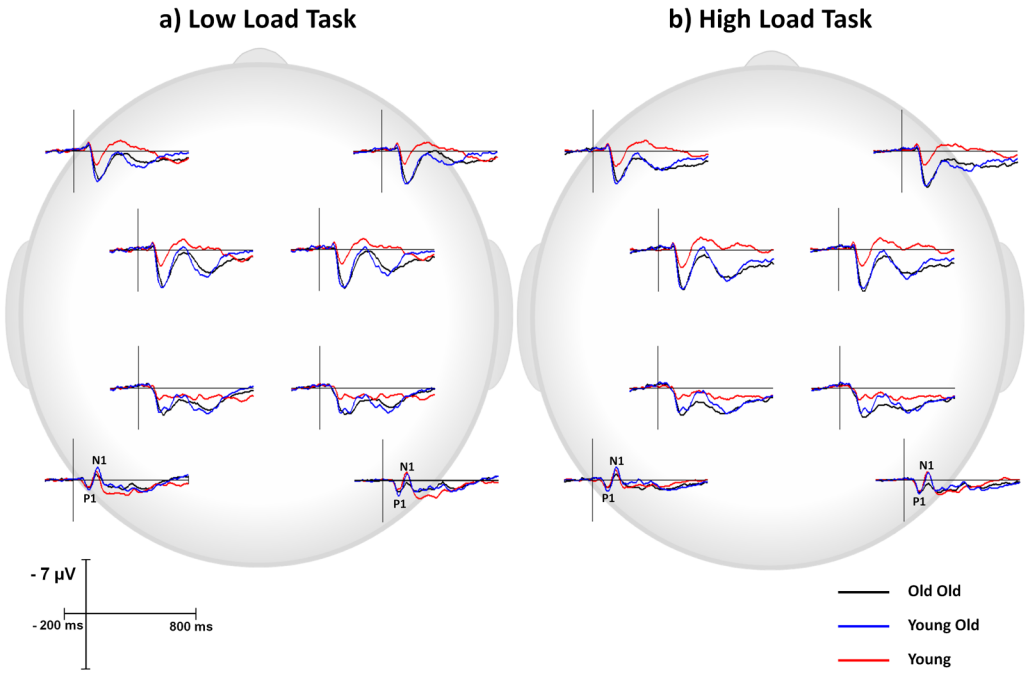

Figure 3.
Grand average ERPs for each age group under the attend condition at the 8 lateral ROIs in response to standard stimuli for the **a)** low load task and **b)** high load task. Note that the temporal window is from −200 to 800 ms to provide an overview of the ERP response.

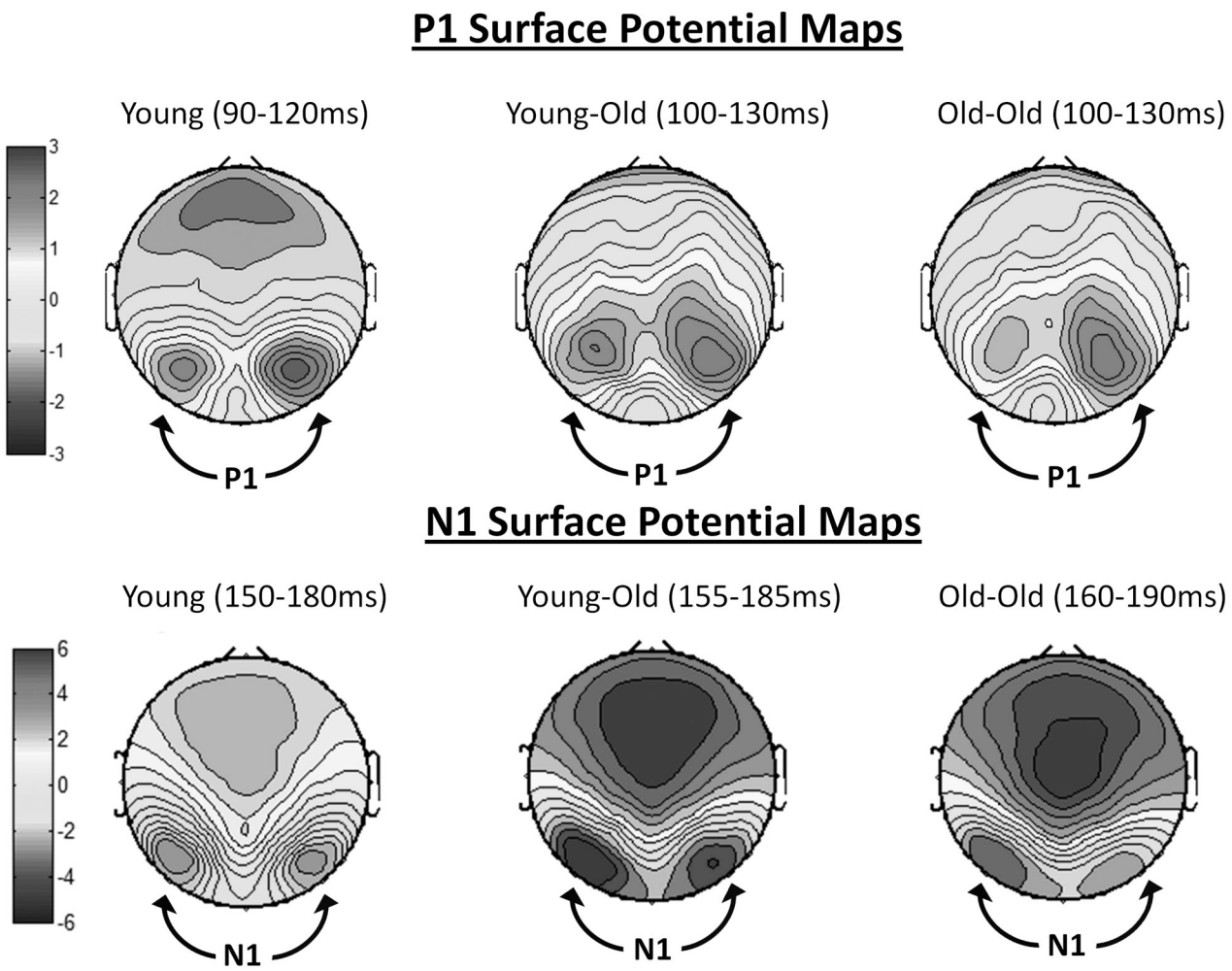

Figure 4.
Surface potential maps illustrating the scalp distribution of the posterior P1 and posterior N1 collapsed across memory load for each group. (Note that the scale for the P1 map is from −3 μV to 3 μV, and the scale for the N1 map is from −6 μV to 6 μV.)

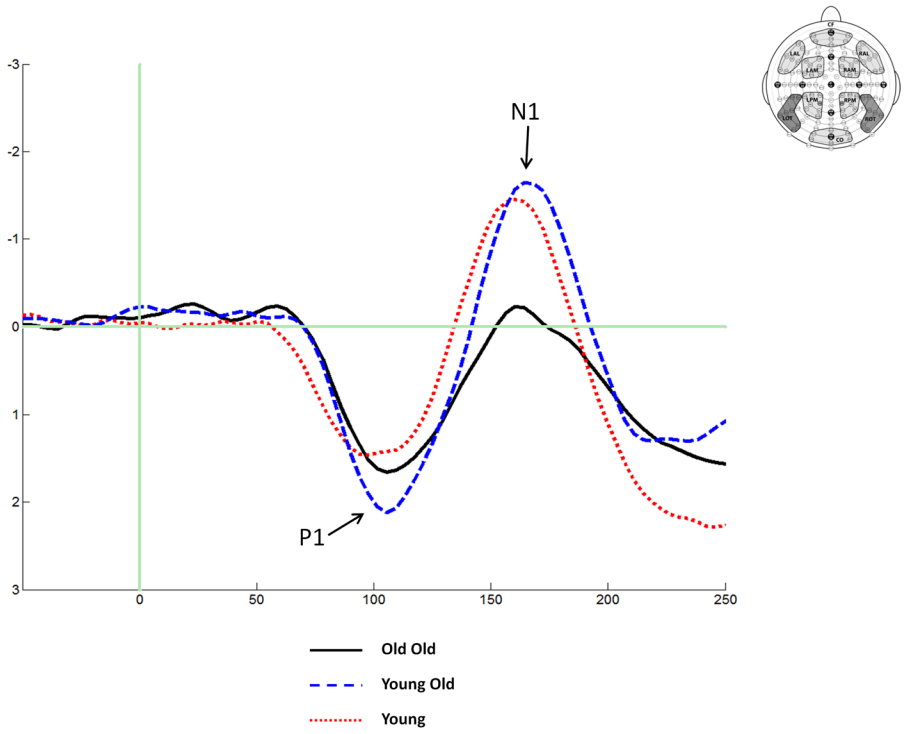

Figure 5.
Grand average ERPs collapsed across memory load and across the lateral posterior ROIs (ROT and LOT) for each age group. Note that the temporal window is from −50 to 250 ms to highlight the posterior P1 and N1components.

Table 1

Subject Characteristics (Mean (SD))

	Young	Young-Old	Old-Old
Number of Subjects	25	15	22
Gender (male:female)	12:13	6:9	7:15
Age[a]	22.6 (2.3)	73.9 (3.7)	84.2 (3.4)
Years of Education	15.9 (1.7)	16.5 (3.7)	16.1 (3.1)
AMNART IQ[b]	116 (6.8)	121.1 (8.5)	122.5 (7.2)
Composite z-score[c]	.21 (.40)	−.09 (.29)	−.18 (.40)
Visual Acuity[d]	1.05 (0.20) ≈ 20/20	0.75 (0.17) ≈ 20/27	0.63 (0.18) ≈ 20/32

[a] Effect of Age Group, $p < .001$

[b] Effect of Age Group, $p < .05$ (young ≤ y-old, $p < .07$; young < o-old, $p < .01$; y-old = o-old, $p > .5$)

[c] Effect of Age Group, $p < .005$ (young > y-old, $p < .05$; young > o-old, $p < .005$; y-old = o-old, $p > .4$)

[d] Effect of Age Group, $p < .001$ (young > y-old, $p < .001$; young > o-old, $p < .001$; y-old ≥ o-old, $p < .08$)

Table 2

Breakdown of Visual Acuities by Age Group

	20/16	20/20	20/25	20/30	20/40	20/50	20/80
Young	6	16	0	2	1	0	0
Young-Old	1	0	7	5	2	0	0
Old-Old	0	1	6	7	4	3	1