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Abstract

The effects of aging and brain damage on time perception are not well understood. In this study, we test the hypothesis that aging affects timing variability and that parietal damage affects timing magnitude. Young and healthy college-age students, older subjects, and a patient with bilateral parietal damage were assessed on their ability to estimate and produce time intervals ranging from four to fifteen seconds. Subjects completed two tasks: production of the duration of a stimulus in seconds and estimation of when the number of seconds presented as a stimulus had elapsed. Data was analyzed via power functions: $\psi = K\phi^\beta$. Aging and brain damage should theoretically result in either reduced exponents or increased variability in performance. Information processing models separate deficits in perception, decision-making, and memory, allowing more specific analysis of the locus of cognitive deficits. The results of this study show no effects of aging on time perception whereas parietal damage affects temporal processing in significant ways.

Keywords

aging, brain damage, time perception, timing, power functions, temporal, parietal, psychophysics, estimation, production, magnitude, clock, Biological Basis of Behavior, Natural Sciences, Anjan Chatterjee, Chatterjee, Anjan

The Effects of Aging and Brain Damage on Time Perception

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The effects of aging and brain damage on time perception are not well understood. In this study, we test the hypothesis that aging affects timing variability and that parietal damage affects timing magnitude. Young and healthy college-age students, older subjects, and a patient with bilateral parietal damage were assessed on their ability to estimate and produce time intervals ranging from four to fifteen seconds. Subjects completed two tasks: production of the duration of a stimulus in seconds and estimation of when the number of seconds presented as a stimulus had elapsed. Data was analyzed via power functions: $\psi = K\phi^\beta$. Aging and brain damage should theoretically result in either reduced exponents or increased variability in performance. Information processing models separate deficits in perception, decision-making, and memory, allowing more specific analysis of the locus of cognitive deficits. The results of this study show no effects of aging on time perception whereas parietal damage affects temporal processing in significant ways.

Introduction

While much evidence points to reductions in cognitive ability with aging, much of this research has avoided including sensory perception and its relationship to cognition in its explanation of the effects of aging on cognitive ability. In the same respect, perceptual researchers have postulated that aging affects visual and auditory perception, but they have failed to include cognitive functioning in their explanations. The age-related changes in both cognition and perception have prompted neuroscientists to begin researching the relationship between the two abilities (1).

Currently, much research has been performed on the effects of aging on cognition. It has been hypothesized and concluded that aging leads to reductions in processing abilities and processing speeds (5). The reductions in processing abilities associated with aging have been studied and empirically supported for a number of years, with the strongest evidence from studies by Hebb and by Horn. Hebb's study shows considerable age-related differences in Type A cognition. Hebb describes this

type of cognition as inherent intellectual ability which develops by neural maturation. Hebb distinguishes type A cognition from type B cognition, which represents intelligence that is acquired through experience (2). Horn's study shows age-related differences in fluid cognition (3). Fluid cognition is defined as functioning that requires an active effort to maintain information, whether verbal or visuospatial, in working memory so as to plan and execute goal-directed behavior. Fluid cognition is distinguishable, though not distinct, from crystallized knowledge, which represents information stored in long-term memory (4).

Furthermore, these reduced sensory processing speeds lead to deficits in cognitive functioning, as proposed by Salthouse in the processing-speed theory of cognitive aging phenomena (5). The main assumption in this theory is that age-related deficits in memory and cognition stem from the decreased speed with which many cognitive functions are executed with increased age (5). Research by Salthouse shows reduced processing speeds occurring with increased age in fluid cognitive functioning. The validity of these tests stems from the relatively undemanding tasks that the subjects were

required to perform, which controls for reductions in speed resulting from delayed sensory perception and sensory reaction. Thus, the reductions in speed occurred from reduced processing speeds of the central nervous system (6).

Despite the vast amount of research that has been performed on aging and cognition, relatively little is known about the link between the two, and how increased age may lead to mental degradation. In an attempt to broaden the understanding of the effects of aging on cognition, neuroscientists have begun delving into research on the neuropsychology of time perception.

The importance of temporal processing abilities provides further impetus for research on time perception. Interval timing in the seconds to minutes range is a skill necessary for motor control and making predictions about one's surroundings. In an evolutionary sense, interval timing is important for escape from predators and for attack on prey (7). In addition, general cognitive performance tests have been able to explain subjects' abilities in daily activities. Therefore, timing tasks may present additional factors that may be useful in predicting one's cognitive abilities in extra-laboratory settings (5).

Interval timing requires higher level cognitive functions such as attention, working memory, and decision-making ability. Since attention and working memory are known to degrade with increased age, it is hypothesized that interval timing abilities may also degrade with increased age. Moreover, interval timing, attention, and working memory are all associated with the same brain structures and neurotransmitter systems, and these structures are most sensitive to aging. Therefore, temporal tasks may provide very sensitive measurements of age-related differences in cognitive functioning (8). Essentially, reduced sensory processing abilities with increased age may be a factor of reduced temporal processing abilities with increased age.

Evidence points to the conclusion that age-related changes in cognition correlate with brain damage to the frontal lobes of the brain. Salthouse states that the frontal-deficit hypotheses that have abounded due to the relationship between aging and brain damage must be inspected through a double-dissociation pattern. Thus, certain cognitive deficits

should be shown to be affected by lesions to the frontal lobe while undergoing no effect by lesions to other anatomical regions of the brain. In addition, other cognitive deficits should be shown to be affected by lesions to certain regions of the brain while not being affected by lesions to the frontal lobes. Evidence currently shows a variety of brain regions involved in age-related changes in cognition, including the cerebellum (9).

One of these brain regions that lead to cognitive deficits may be the parietal lobes. Further, it is theorized that temporal processing occurs in the parietal lobes – in other words, the internal clock resides here. Numerous studies have shown that patients with parietal lesions suffer deprivations in magnitude estimations. Cohen et al. reported dyscalculia, difficulty in automatically processing numerical magnitudes, in patients suffering from parietal lesions as well as in healthy individuals that were subjected to transcranial magnetic stimulation (TMS) of the parietal cortex (10). Sack et al. report visuospatial deficits in subjects who underwent TMS to the parietal cortex (11).

Since patients with parietal lesions suffer deficits in magnitude judgments, it has been theorized that these patients would suffer deficits in timing magnitude (12). Critchley noted deficits in time, space, size, and number perceptions in patients with parietal damage. He also stated that it was much rarer to find patients who suffered timing deficits without suffering any spatial deficits (13). Alexander et al. performed a study on healthy individuals in which the subjects underwent TMS to the parietal cortex before and after both a timing task and a pitch discrimination task. The results showed significant effects of the TMS on time perception but no effects on pitch discrimination (12).

All these studies strongly suggest that some aspects of temporal processing occur in the parietal cortex. Moreover, the effects of aging on cognition may be a factor of reduced temporal processing abilities. Similarly, the effects of parietal lesions on cognition may be a factor of reduced temporal processing abilities. This research was conducted to uncover the effects of aging and parietal damage on time perception.

Certain studies have shown inconclusive evidence of the effects of unilateral spatial neglect on spatial

perception (14). However, reinterpretation of this data provided a clearer analysis of it (15). This data was reevaluated through the use of power functions, which take the form $\psi = K\phi^\beta$. In 1860, Gustav Fechner theorized a mathematical relationship between objective stimuli and the subjective perception of these stimuli. He extended the Weber fraction, which proposes that the just noticeable difference threshold for stimuli is a constant proportion of those stimuli, to apply to subjective sensory experiences of stimuli to the objective representation of these stimuli. In so doing, he introduced the discipline of psychophysics with his publication of *The Elements of Psychophysics*. Fechner's rendition of this mathematical relationship was logarithmic, which took the form $\psi = \log\phi + k$. Here, ψ represents the subjective experience, or the psychological value, and ϕ represents the objective stimuli, or the physical value. Stevens and colleagues identified the power function, $\psi = K\phi^\beta$, as a better descriptor of this psychophysical relationship (16). The exponent β and the constant K are both empirically determined.

Power functions represent the only mathematical model that has been successfully applied to all sense modalities: visual, auditory, kinesthetic, etc. Moreover, it has produced a characteristic exponent for each sense in terms of normal, non-brain-damaged subjects. This ability to apply across all senses strongly suggests that the central nervous system quantifies input from the external environment according to psychophysical principles (17).

The exponent of the power function captures heterogeneity in the variances of the subjective perception of stimuli. The constant of the power function captures the proportionate error in the subjective perception of stimuli (15). In this study, the exponent represents the variable processing errors evident in the subject while the constant represents the constant processing errors evident in the subject. The constant, thus, would capture delays in registering the presentation of stimuli, in this case, the presentation of the question mark in the estimation task or the number in the production task. It would also capture delays in reacting to the stimuli, such as pressing the space bar on the computer keyboard in the production task, etc. The exponent, capturing the variable processing errors, represents the cognitive ability of the subjects. Reduced

exponents (less than 1) would show reduced sensory processing at increasing levels of stimuli, while increased exponents (greater than 1) would show increased perception of stimuli as the stimuli levels were increased. Exponents with a value of 1 represent constant perception of stimuli at increasing levels of the stimuli.

Despite the robust nature of power functions, they have largely been applied to data gathered from normal, non-brain-damaged subjects. Recently, however, power functions have been employed to characterize the cognitive abilities of brain-damaged individuals. Chatterjee et al. conducted studies on patients suffering from unilateral spatial neglect and discovered that sensory processing in these patients is significantly affected. Their ability to perform simple spatial tasks, such as line bisection, was reduced when compared against the ability of normal, non-brain-damaged subjects (15, 18). Moreover, the performance of one patient improved over successive trials in these spatial tasks; however, the exponent of the power function formed from this patient's data remained unchanged. This represented a deficit in some critical area of the patient's cognitive processing (19). Further evidence for the exponent capturing the cognitive ability of subjects stems from research conducted by Chatterjee et al. (15), in which patients suffering from lesions to the central nervous system showed reduced exponents. If the exponent captured deficits in sensory transduction, in other words, transduction via the peripheral nervous system, no effect should be observed on the exponent. Thus, lesions to the central nervous system may influence the psychophysical power law in ways previously not anticipated (15).

In this experiment, two comparisons are conducted. To test age-related temporal deficits, the temporal processing abilities of older adults are compared against those of younger adults. To test the role of the parietal cortex in temporal processing, the temporal processing abilities of the patient with bilateral parietal lesions are compared against those of the age-matched older adults.

Power functions are used to perform these comparisons, and the exponent is the parameter of interest since, as stated earlier, it represents the cognitive ability of the subject. Moreover, the R^2 value, determined by the correlation between the data points and the power function curve derived

from these data points, is of interest since it represents the timing variability evident in the subject. An R^2 value near 1 implies consistency in the subject's temporal processing abilities at increasing stimuli levels. On the contrary, a significantly reduced R^2 value implies inconsistency in the subject's time perception across varying stimuli levels.

Methods

Subjects

Three groups were tested on their temporal processing abilities: a younger adult group, an older adult group, and a patient with bilateral parietal lesions. All the subjects consented to participate in this study, which the Institutional Review Board had approved.

Younger Adult Group

The younger adult group consisted of twenty college-age subjects from the University of Pennsylvania. Their average age was 18.85 ± 1.04 . Their ages ranged from 18 to 21. All of the subjects were right-handed, and none had any history of brain damage or consumption of psychoactive substances. Eleven of the subjects were male, and nine were female.

Older Adult Group

The older adult group consisted of twenty elder subjects from the Philadelphia area. Their average age was 66.35 ± 12.219 . Their ages ranged from 45 years old to 87 years old. All of the subjects were right-handed, and none had any history of brain damage or consumption of psychoactive substances. Eight of the subjects were male, and twelve were female.

Patient

The patient was a 38-year-old female who suffered from bilateral parietal-occipital infarctions with right internal artery occlusion (etiology unknown). The strokes are in a watershed distribution between the middle cerebral artery (MCA) and the posterior cerebral artery (PCA) territories.

Tasks

Two tasks were completed by each subject: an estimation task and a production task. Both the tasks were completed on a computer. These programs were created via E-prime software, and data collection was performed through this software. The tasks were presented in succession to the subject,

while the order in which the tasks were presented for each individual subject was randomized across the entire group. Thus, an equal number of subjects completed the estimation task first, followed by the production task, as the number of subjects who completed the production task first, followed by the estimation task.

Estimation Task

A question mark, the stimulus, appeared on the computer screen for a predetermined amount of time, and the subject was requested to estimate the number of seconds the question mark was presented. The stimuli durations ranged from four seconds to fifteen seconds, with each stimulus presented twice. Thus, a total of twenty-four stimuli were presented (twelve different time intervals with each interval presented twice). In addition, the stimuli were presented in random order, randomized for each subject. Thus, no two subjects were presented the same stimuli in the same order. So as to provide the subject with time to prepare between each stimulus presentation, an inter-trial-interval crosshair appeared on screen for a brief interval. Then, the question mark appeared, followed by these words: "How many?" At this point, the subject would verbally state the number of seconds that they perceived the question mark was presented, and their response was keyed in on a keyboard by the experimenter (V.R.D.). The subjects were permitted to use any strategy, including chronometric counting.

Production Task

A number, the stimulus, appeared on screen in this task. The numbers ranged from 4 to 15, and each number was presented twice. Twenty-four stimuli were presented (twelve numbers with each number presented twice). The numbers were presented in random order, randomized for each subject. Thus, no two subjects were presented the same stimuli in the same order. Once again, the inter-trial-interval fixation point appeared on screen for a brief amount of time in between stimuli presentations so that the subject would be prepared for each presentation. The subject was requested to press the space bar on the computer keyboard when they believed the number of seconds presented had elapsed. Thus, if a 6 were presented on screen, the subject would press the space bar when they believed six seconds had passed by. Again, the subjects were permitted to use any strategy, including chronometric counting.

Data Analysis

Data was analyzed via power functions, which take the form $\psi = K\phi^\beta$. The data collected from the younger adults should theoretically indicate an exponent around 1. In this case, it is expected that the younger adults, due to their young age and lack of both brain damage and consumption of psychotropic substances, show constant temporal processing abilities across varying time intervals.

It is hypothesized that the data collected from the older adults would show either increased timing variability or increased deficits in timing magnitude, or both. Similarly, the patient with parietal brain damage should hypothetically show increased timing variability and increased deficits in timing magnitude. Increased timing variability would be represented in a reduced R^2 value, calculated from the correlation between the data points at each stimulus level and the power function curve. Deficits in timing magnitude would be captured in the constant and the exponent, with either a reduced or inflated constant value and a reduced exponent value.

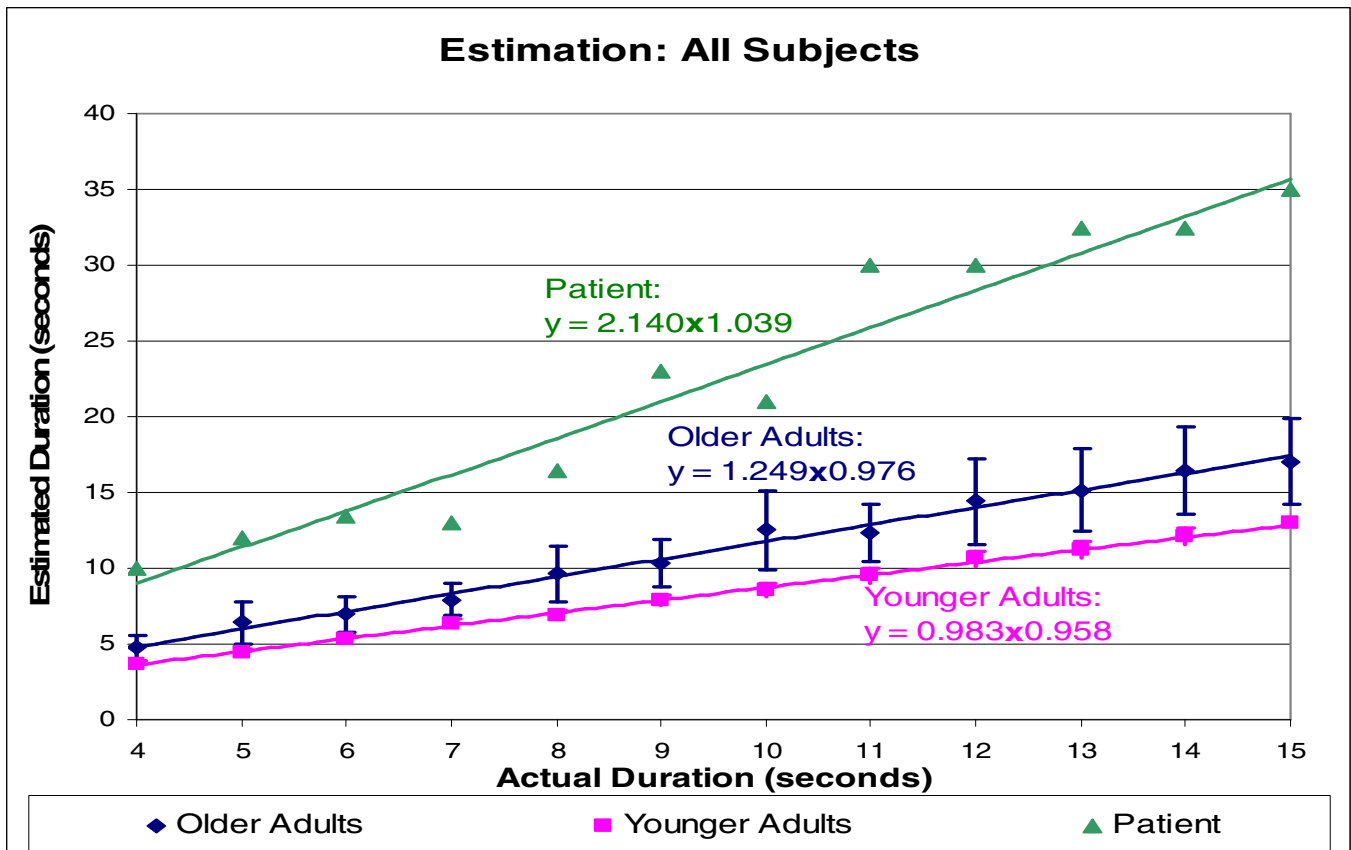
Results

The power functions were calculated for each of the three groups (younger adults, older adults, and the patient with bilateral parietal damage). These power functions were calculated by averaging the values for each parameter for each individual's power function. Thus, each subject's power function was derived, and the parameter values from this power function were averaged with the parameter values from the power functions of all the other subjects within their respective group.

Estimation Task

Figure 1 depicts the power functions for all three groups. The x-axis in this figure represents the actual duration of the stimulus in seconds. As mentioned earlier, the question marks in this task were presented for four to fifteen seconds, and thus, the x-axis ranges from four to fifteen. The y-axis in this figure represents the subjects' perceived duration of the presented stimuli in seconds. The data points are as shown in the figure, and the curves represent the best-fit power functions derived from these data points.

FIGURE 1:



The power functions for each group in the estimation task, along with their R^2 values, were as shown below:

Younger Adults: $y = 0.983X^{0.958}$; $R^2 = 0.9718$

Older Adults: $y = 1.249X^{0.976}$; $R^2 = 0.9712$

Patient: $y = 2.140X^{1.039}$; $R^2 = 0.9446$

The younger adults showed a constant value of 0.983 ± 0.277 , an exponent value of 0.958 ± 0.113 , and an R^2 value of 0.9718 ± 0.036 . The older adults showed a constant value of 1.249 ± 1.037 , an exponent value of 0.976 ± 0.072 , and an R^2 value of 0.9712 ± 0.035 .

From the data of the younger adults group and the older adults group, a t-statistic was calculated to compare their constant, exponent, and R^2 values, assuming unequal variances for each parameter. For the constant value, the t-statistic was -1.108 with a p-value of 0.14. The exponent value had a t-statistic of -0.5997 with a p-value of 0.2765. The R^2 value had a t-statistic of 0.0535 with a p-value of 0.4788. Therefore, neither the constant, nor the exponent, nor the R^2 values for the older adults were significantly different from those of the younger adults at the 95% confidence level.

95% confidence intervals were calculated for all three parameters for the older adults group for comparison against the patient's data. These intervals are as follows:

Constant: $0.653 < \mu < 1.845$

Exponent: $0.935 < \mu < 1.018$

R^2 value: $0.951 < \mu < 0.991$

All the patient's values fell outside of the 95% intervals for the older group (2.140 for the constant, 1.039 for the exponent, and 0.9446 for the R^2 value). Thus, all the parameters were significantly different for the patient when compared against the older adults group.

Production Task

Figure 2, on the following page, depicts the power function curves for all three groups in the production task. The x-axis represents the actual durations of the stimuli in seconds – in this case, the number that was presented on the screen during the task. The y-axis represents the subjective perception of these stimuli in seconds – in this case, the time in which the

subject pressed the space bar on the computer keyboard. The data points are as shown with the curves representing the best-fitting power function for each group's data points. The older adults' curve is the lowest one on the graph, followed by the younger adults' curve, and lastly, by the patient's curve at the top.

The power functions for each group in the production task, along with their R^2 values, were as follows:

Younger Adults: $y = 1.246X^{0.983}$; $R^2 = 0.9714$

Older Adults: $y = 1.022X^{1.001}$; $R^2 = 0.9675$

Patient: $y = 2.702X^{0.863}$; $R^2 = 0.8891$

The younger adults showed a constant value of 1.246 ± 0.464 , an exponent value of 0.983 ± 0.085 , and an R^2 value of 0.9714 ± 0.038 . The older adults showed a constant value of 1.022 ± 0.507 , an exponent value of 1.001 ± 0.118 , and an R^2 value of 0.9675 ± 0.040 .

Once again, a t-statistic was calculated to compare the average parameter values of the younger adults group with those of the older adults group, assuming unequal variances for each parameter. The constant value had a t-statistic of 1.454 with a p-value of 0.0771. The exponent value had a t-statistic of -0.5503 with a p-value of 0.2928. For the R^2 value, the t-statistic was 0.3180 with a p-value of 0.3761. Therefore, neither the constant, nor the exponent, nor the R^2 values for the older adults were significantly different from those of the younger adults at the 95% confidence level.

For the older adults group, 95% confidence intervals were calculated, with which to compare the patient's data for the production task. The following are these intervals for the constant, the exponent, and the R^2 values:

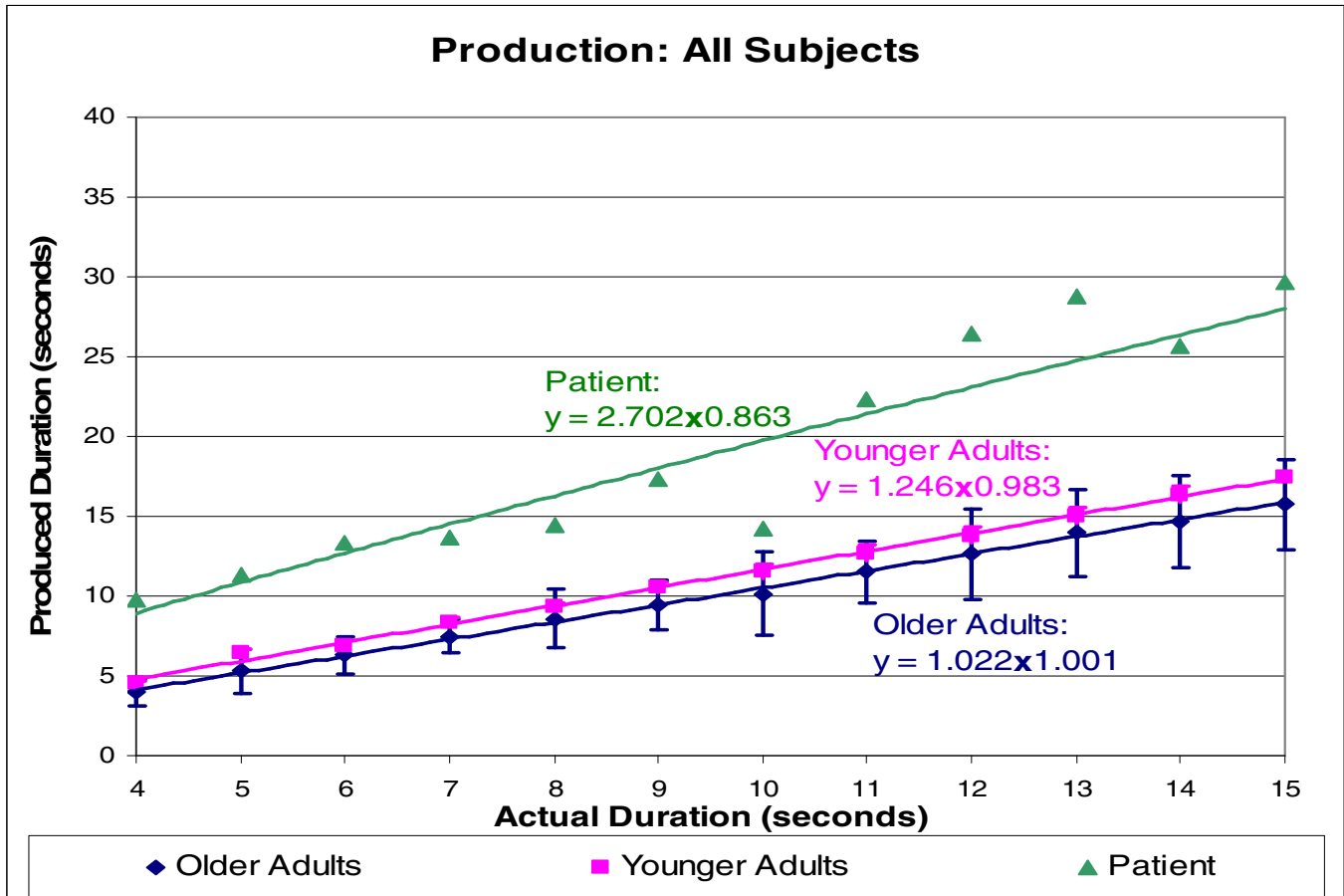
Constant: $0.731 < \mu < 1.314$

Exponent: $0.933 < \mu < 1.069$

R^2 value: $0.944 < \mu < 0.991$

All the patient's values fell outside of the 95% intervals for the older group (2.702 for the constant, 0.863 for the exponent and 0.8891 for the R^2 value). Thus, all the parameters were significantly different for the patient when compared against the older adults group.

FIGURE 2:



Discussion

The data collected in this study shows that the constant, exponent, and R^2 values for the older adults group were not significantly different from those of the younger adults group. This finding is inconsistent with our hypothesis that increased age would be correlated with decreased temporal processing abilities. More specifically, aging hypothetically would lead to increased timing variability, reflected in a decreased R^2 value, or increased deficits in timing magnitude, reflected in significantly affected constant and exponent values, or both. However, for both the estimation and the production task, none of the parameters were significantly different from the younger adults group. From this, we conclude that temporal estimation and production in this range are not compromised by aging.

However, there may be various reasons for this data showing no effects of aging on time perception. For one issue, the subjects in this study were allowed to perform chronometric counting – a method in which an individual subdivides time intervals according to the durations of verbal statements (for example, “one thousand *one*...one thousand *two*”). (Whether the subjects actually did perform chronometric counting is unknown.) Hinton et al. postulate that chronometric counting and not counting may have different psychophysical properties in timing (20). Essentially, chronometric counting may provide these older adults a method by which they can mask temporal processing deficits. This depends on the assumption that such counting would not be affected by cognitive decline with aging.

Hemmes et al. support this view that different psychophysical properties are evident based on

whether one counts or does not count. They state that different sensory systems govern time perception based on whether the individual performs chronometric counting or no counting and that this leads to differences in temporal processing. The attentional resources that must be distributed between the temporal task and the nontemporal task affect the performances of the subjects. Hemmes et al. showed that time perception with a concurrent nontemporal task, such as number counting, results in a flatter power function. This can be interpreted as reduced sensory processing at increasing stimuli levels (21).

Perbal et al. found that aging had no effect on time perception in a simple task, which allowed counting, whereas it had a significant effect in a concurrent task (22). Several other experiments have found similar results and have thus postulated that decreased temporal processing abilities result from reductions in attentional ability and/or memory ability (23). Perbal et al. explain their results by noting that in the simple task, older subjects may devote their full attentional resources to chronometric counting. If their internal clock was naturally slower than normal, then they were able to compensate for this fact through their use of chronometric counting. On the other hand, in the concurrent condition, division of attentional resources between the temporal task and the nontemporal task prevented subjects from counting chronometrically. This led to deficits in temporal processing (22).

Information processing models attempt to explain results such as these better. These models deconstruct the processing into three different stages of interval time perception. The first stage is the "internal clock" which consists of a pacemaker that emits pulses or ticks which are registered in an accumulator. The second stage is the "memory" stage which consists of two different memory stores: working memory and a longer-term reference memory. Information from the accumulator is transferred to the working memory. The reference memory holds information on time intervals based on previous experience. The third stage is the "decision-making" stage which compares information from the working memory to that in the reference memory and makes a decision (24).

Deficits in attention result from "central executive" deficits. The central executive function resides in working memory and divides attentional resources between tasks. Thus, aging could potentially show deficits in any or all of these three stages. With attention divided between two tasks, pulses or ticks from the pacemaker may not all be registered in the accumulator. This would result in an underestimation of time intervals, and many studies have shown that concurrent task conditions cause aged subjects to underestimate time intervals (22). The results of this experiment support the notion that aging may not affect the internal clock, unless the subjects compensated for variations in their clock speed through their use of chronometric counting.

The results of this experiment support the hypothesis that parietal lesions would affect time perception. The patient's data showed significantly different constant, exponent, and R^2 values when compared against the older group in both the estimation task and the production task. Presumably, the parietal lesions disrupted the temporal processing abilities of this patient.

The fact that the patient had bilateral parietal lesions indicates that temporal processing occurs in her region of damage in the two parietal lobes. Time perception may be determined by abilities of the left parietal lobe, the right parietal lobe, or both. Interestingly, experiments by Cohen et al. show that magnitude processing in numerical magnitude estimation tasks is disrupted by TMS directed at the right inferior parietal sulcus (IPS) but not by TMS directed at the left IPS (10).

Similarly, Sack et al. showed that visuospatial processing is disrupted when TMS is directed at the right IPS not when it is directed at the left IPS. Furthermore, both the right and left inferior parietal sulci show activity in visuospatial processing under normal circumstances (when no TMS is applied). This suggests that the right IPS potentially takes over functions typically presided over by the left IPS (11).

Alexander et al. stretch these findings to temporal processing abilities. In their experiment, in which TMS was directed at the parietal lobe before and after a timing task and pitch discrimination task,

different results were obtained when TMS was directed at different specific regions of the parietal lobe. TMS directed at the right inferior parietal cortex (IPC) disrupted temporal processing abilities in healthy subjects but not pitch discrimination abilities. On the other hand, TMS directed at the left IPC did not disrupt either temporal processing or pitch discrimination abilities (12).

These results summarized above indicate that the patient in this experiment would theoretically suffer time perception deficits due to bilateral parietal lesions. If the patient suffered from a unilateral lesion, the results of this study may not have been as conclusive. For instance, if the patient suffered a lesion to the left parietal lobe, though temporal processing in this patient may have been affected, the right parietal cortex may have been able to take over the functioning of the left IPC, just as had occurred in the visuospatial and magnitude processing tasks of the studies of Sack et al. and Cohen et al.

In addition, the patient in this study demonstrated an internal clock that speeds up at increasing stimuli levels. The estimation task shows an exponent value greater than 1. This indicates that at increasing time intervals in this task, the patient increasingly overestimated them. This equivalent to the following: suppose the question mark was presented for five seconds in the estimation task, and the subject stated that her perception of the duration of the question mark was five seconds. Then suppose that the same subject perceived a ten second presentation of the question mark as fifteen seconds. And lastly, suppose that this subject perceived a fifteen second time interval as thirty seconds. This subject would show increased perceptions of time intervals as the intervals increased. In other words, the internal clock of this subject would run faster at increasing time intervals. This is what the patient's data indicated in the estimation task.

The patient also showed a speeded clock at increasing time intervals in the production task. The reduced exponent indicated that the patient pressed the space bar at disproportionately earlier times at increasing stimuli levels. This is equivalent to the following: suppose a number five was displayed on the computer screen, and a subject pressed the space bar after ten seconds. Then suppose that a

number ten was displayed, and the subject pressed the space bar after twelve seconds. Lastly, suppose a number fifteen was presented, and the subject pressed the space bar after fifteen seconds. This indicates that the subject pressed the space bar at earlier times when the time intervals were increased. In other words, the subject counted faster and faster at increasing stimuli levels. The patient's data resembled this and shows a speeded up internal clock.

The constant values for the patient were intriguing. Typically, a subject whose clock ran too fast would show an increased constant in the estimation task (for instance, count ten seconds when the question mark appeared for only five seconds) and a decreased constant in the production task (press the space bar after three seconds when a number five appeared on the computer screen). Additionally, a subject who counted too slowly would show a decreased constant in the estimation task and an increased constant in the production task. This patient, on the other hand, showed an increased constant in both tasks, representing counting that is too fast in the estimation task and too slow in the production task. This data is perplexing, but what may be the cause of the slowing in the production task is the physical requirement of pressing the space bar. Perhaps the patient's internal clock did not actually change speeds in the tasks. Rather, the clock may have remained speeded up, while the patient's physical response to stimuli presentations was slow.

Further studies may show what specifically may be affecting this patient's clock. If the production task does indeed cause a slowing in responses in subjects who have difficulty or show slowness in physical responses, then the estimation task may be a better indication of the internal clocks of these subjects.

Moreover, further studies may better explain the stage of temporal processing – clock, memory, or decision-making – that suffers deficits with increased age. Currently, several researchers have devised experimental methods of testing a subject's temporal processing speed after circumventing one of the three stages listed above. For instance, Treisman et al. (25) performed an experiment which manipulated the pacemaker of the clock

stage by presenting a succession of clicks or flashes to the subject, resulting in an increased speed of the internal clock while maintaining constancy in the memory and decision-making stages (26). Experiments by Wearden et al. (27) and by Allan et al. (28) circumvented the use of reference memory by presenting subjects with two tones and requesting them to state whether the second tone was longer in duration than the first one. Thus, the subjects were required to compare information in the working memory to information that had been in working memory moments before information concerning the second tone arrived.

Isolation of the decision stage has received relatively little attention, despite the fact that many scientists believe temporal processing errors occur in the decision-making stage. Nevertheless, Wearden et al. (29) have devised a clever method of isolating this stage. Their task awards four different types of responses: hits, misses, false positives, and correct rejections. The task presented a tone of 400ms to subjects at the start, and then, successive tones ranged from 100ms to 700ms. The subjects were required to state whether successive tones were of the same duration as the tone in the beginning (the 400ms tone) or not. When the successive tone was 400ms, a response of "yes" (indicating that the subject perceived the successive tone to be of equal duration to the tone in the beginning) was classified as a "hit". Any other response was classified as a "miss". When the presented tone was not 400ms (in other words, if it was 100ms, 200ms, 300ms, 500ms, 600ms, or 700ms), a response of "no" was classified as a "correct rejection". Any other response was classified as a "false positive". Different classifications of the responses were given different payoffs. When "hits" were given higher payoffs than "correct rejections", subjects responded by increasing their responses of "yes" and decreasing their responses of "no". When "correct rejections" were given higher payoffs than "hits", subjects responded by increasing their responses of "no" and decreasing their responses of "yes". Wearden et al. constructed a mathematical model to describe this data. What they found was that when "hits" were given higher payoffs than "correct rejections", the successive tone could have greater variance from the original tone and still be classified as having the same duration. The opposite was the case when the payoffs were reversed (29).

Studies that isolate the different levels of information processing models during timing tasks may allow greater specificity in determining what may cause temporal processing deficits with increased age.

References

1. Schneider, B.A. & Pichora-Fuller, M.K. Implications of perceptual deterioration for cognitive aging research. *The Handbook of Aging and Cognition*. 2nd edition. Mahwah, N.J.: Lawrence Erlbaum Associates. (2000).
2. Hebb, D.O. 1942. The effect of early and late brain injury upon test scores and the nature of normal adult intelligence. *Proceedings of the American Philosophical Society*, **85**, 275-292.
3. Horn, J. L. 1982. The theory of fluid and crystallized intelligence in relation to concepts of cognitive psychology and aging in adulthood. In F. I. M. Craik & S. Trehub (Eds.), *Aging and cognitive processes*, 237–278. New York: Plenum.
4. Blair, C. 2006. How similar are fluid cognition and general intelligence? A developmental neuroscience perspective on fluid cognition as an aspect of human cognitive ability. *Behavioral and Brain Sciences*, **29**, 109-125.
5. Salthouse, T.A. July 1996. The processing-speed theory of adult age differences in cognition. *Psychology Revolution*, **103 (3)**, 403-428.
6. Salthouse, T.A. 1993. Speed mediation of adult age differences in cognition. *Developmental Psychology*, **29 (4)**, 722-738.
7. Meck, W.H. 2005. Neuropsychology of timing and time perception. *Brain and Cognition*, **58 (1)**, 1-8.
8. Rakitin, B.C., Stern, Y., & Malapani, C. 2005. The effects of aging on time reproduction in delayed free-recall. *Brain and Cognition*, **58 (1)**, 17-34.
9. Salthouse, T.A. & Craik, F.I.M. Closing comments. *The Handbook of Aging and Cognition*. 2nd edition. (2000).
10. Cohen, K.R., Cohen, K.K., Schuhmann, T., Kaas, A., Goebel, R., Henik, A., & Sack, A.T. April 2007. Virtual dyscalculia induced by parietal-lobe TMS impairs automatic magnitude processing. *Current Biology*, **17 (8)**, 689-693.
11. Sack, A.T., Sperling, J.M., Prvulovic, D., Formisano, E., Goebel, R., Di Salle, F., Dierks, T., & Linden, D.E.J. July 2002. Tracking the mind's image in the brain II: transcranial magnetic stimulation reveals parietal asymmetry in visuospatial imagery. *Neuron*, **35**, 195-204.
12. Alexander, I., Cowey, A., & Walsh, V. 2005. The right parietal cortex and time perception: back to critchley and the zeittraffer phenomenon. *Cognitive Neuropsychology*, **22 (3/4)**, 306-315.
13. Critchley, M. 1953. *The parietal lobes*, Hafner Press.
14. Bisiach, E., Bulgarelli, C., Sterzi, R., & Vallar, G. 1983. Line bisection and cognitive plasticity of unilateral neglect of space. *Brain and Cognition*, **2**, 32-38.
15. Chatterjee, A., Dajani, B. M., & Randall, J. G. 1994. Psychophysical constraints on behavior in unilateral spatial neglect. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, **7**, 267-274.
16. Stevens, S.S. 1959. The quantification of sensation. *Daedalus*, **88**, 613-618.
17. Stevens, S. S. 1970. Neural events and the psychophysical power law. *Science*, **170 (3962)**, 1043-1050.
18. Heilman, K. M., Watson, R.T., & Valenstein, E. 1985. Neglect and related disorders. In: K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology*. 2nd Ed. New York: Oxford University Press. 243-293.
19. Chatterjee, A., Mennemeier, M., & Heilman, K. M. 1992. A stimulus-response relationship in unilateral neglect: The power function. *Neuropsychologia*, **30**, 1101-1108.
20. Hinton, S.C. & Rao, S.M. 2004. "One-thousand one...one-thousand two...": chronometric counting violates the scalar property in interval timing. *Psychonomic Bulletin & Review*, **11 (1)**, 24-30.
21. Hemmes, N.S., Brown, B.L. & Kladopoulos C.N. February 2004. Time perception with and without a concurrent nontemporal task. *Perception & Psychophysics*, **66 (2)**, 328-341.
22. Perbal, S., Droit-Volet, S., Isingrini, M., & Pouthas, V. October 2002. Relationships between age-related changes in time estimation and age-related changes in processing speed, attention, and memory. *Aging, Neuropsychology, and Cognition*, **9 (3)**, 201-216.

23. Block, R.A., Zakay, D., & Hancock, P.A. 1998. Human aging and duration judgments: a meta-analytic review. *Psychology and Aging*, **13**, 584-596.
24. Wearden, J.H. 2004. Decision processes in models of timing. *Acta Neurobiologiae Experimentalis*, **64 (3)**, 303-317.
25. Treisman, M., Faulkner, A., Naish, P. L. N., & Brogan, D. 1990. The internal clock: evidence for a temporal oscillator underlying time perception with some estimates of its characteristic frequency. *Perception*, **19**, 705-748.
26. Wearden, J. H. & Grindrod, R. 2003. Manipulating decision processes in the human scalar timing system. *Behavioural Processes*, **61 (1-2)**, 47-56.
27. Wearden, J. H. & Bray, S. 2001. Scalar timing without reference memory: episodic temporal generalization and bisection in humans. *Quarterly Journal of Experimental Psychology*, **54B**, 289-310.
28. Allan, L. G. & Gerhardt, K. 2001. Temporal bisection with trial referents. *Perceptual Psychophysics*, **63**, 524-540.
29. Wearden, J. H. & Grindrod, R. 2003. Manipulating decision processes in the human scalar timing system. *Behavioural Processes*, **61 (1-2)**, 47-56.

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