Rigorous health screening reduces age effect on memory scanning task

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Rigorous Health Screening Reduces Age Effect on Memory Scanning Task

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Eighty subjects participated in a study with five age groups (20, 30, 40, 50, and 60 years). Forty subjects showed evidence of factors related to brain dysfunction (risk factors). Their performance on a Sternberg-type memory scanning task was assessed. Age-related slowing of virtually all aspects of the memory scanning process was observed in the healthy group. However, the effect of the presence of risk factors was larger than that of biological age. The results of the present study make a reasonable case for the view that many age effects reported in the literature can be largely explained by suboptimal brain functioning, i.e., by other factors than aging per se.

INTRODUCTION

There is a decline in certain memory processes in many—if not all—healthy subjects during the later decades of adult life. This notion is widely accepted and supported by evidence from a growing number of empirical studies (Craik, 1977; Poon, 1985). Elderly subjects are characterized by an age-associated decline in nearly all cognitive functions (see Botwinick,

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1977, or Jolles, 1986, for review), which may be caused by a less active processing of information (Branconnier & DeVitt, 1984).

Although most studies on cognitive aging have been carried out with subjects over 65 years of age, there are indications that some performance decrement already exists in middle-age (e.g., Crook et al., 1986; Houx, Vreeling, & Jolles, 1989). It is not known whether the cognitive complaints and dysfunctions which arise in persons in their middle ages and older might have to do with biological aging per se, or some other factor, such as suboptimal health or exposure to risk factors for brain dysfunction. The present study is focused on the latter question.

The possibility that cognitive aging is influenced by factors other than biological aging per se was addressed by Rabbitt (1980). The prevailing notion until then had been that the age-associated cognitive decline was a continuous process; however, Rabbitt showed that the supposedly smooth decline over successive age groups could be a consequence of the fact that older experimental age groups merely contain more poorly performing subjects. We propose that suboptimal health and the presence of risk factors for brain dysfunction can be responsible for such a suboptimal cognitive performance.

This view is supported by several lines of evidence. First, numerous publications indicate that supposedly healthy individuals may have been exposed to some agent that may cause brain dysfunction, e.g., organic solvents or other neurotoxic factors (see Hartman, 1988). Second, closed head injuries are another serious hazard for cognitive dysfunctioning. Even mild head injuries can lead to persistent symptoms. Memory and concentration deficits are the most common postconcussive complaints (Binder, 1986). Third, mild complaints in the area of affective disorders are also associated with impairments of information processing (see, e.g., Koh & Wolpert, 1983).

In short, there are several factors related to brain dysfunction that can aggravate the often reported age-related decline in cognitive functioning. Thus, an age effect can be confounded by factors other than aging per se. Consequently, in research into the cognitive correlates of normal biological aging, one should exclude all factors known to harm the healthy functioning of the brain.

Haxby et al. (1986) described an experiment in which age-related changes in visual memory were investigated in subjects who had undergone "rigorous health screening." They found much smaller age-related changes than are usually reported for the general population. However, they did not test the performance of those who were excluded from their study. This approach is valid if used solely for purposes of aging research, but when the focus is also on the interactions between the effects of risk factors and aging, one should also test those affected by known risk factors and treat them as a separate experimental group. In a similar study Houx,

Vreeling & Jolles (1989) tested subjects who had been exposed to risk factors as well as those who had not. They found that exposure to one or more risk factors accentuated age-related decline in verbal learning capacity and delayed recall. Similar effects were found for response latency in a motor initiation test: not only did the older subjects show a reduced speed of movement initiation, especially with complex tasks, but this age-related slowing was also greatly aggravated in those affected by risk factors. It was concluded that risk factors can significantly contribute to the reduced verbal learning capacity and speed of motor initiation related to aging.

We now describe data on the effects of aging and risk factors on the speed of information processing, as measured with Sternberg's memory scanning task (Sternberg, 1975). This method is appropriate for research into age-related memory impairments as it enables a closer examination of (dys)functions of short term memory and of discrete stages of information processing.

The processing that occurs in the memory scanning task consists of several independent stages, the component latencies of which are thought to be additive (Sternberg, 1975). These stages can be identified as: (1) encoding of the stimulus; (2) serial memory scanning; (3) binary decision about the nature of the response; (4) response organization and execution. The time needed for the memory scanning stage can be inferred from the slope of the reaction times (RTs) as a function of memory load (set size). The three other stages are inferred from the intercept of this function. Theoretically, one can influence scanning and nonscanning stages differentially by varying different aspects of the task. During the last decade, this paradigm has become widely accepted, not only in experimental psychology, but also in other disciplines, such as neuropsychiatric research (e.g., Hart & Kwentus, 1987; Brand, 1987), drug research (e.g., Subhan & Hindmarch, 1984), and developmental psychology. For instance, Strayer, Wickens, & Cannon (1987) and Wickens, Braune, & Stokes (1987) found a substantial slowing with age of the nonscanning stages, and a less marked slowing of the speed of memory search in subjects aged 20–65.

We describe a multicohort study on the effects of aging on the performance of healthy subjects on a complex information processing task. Subjects of different age classes ranging from 17 to 63 years were recruited (See Table 1 for subject characteristics). Because the major question posed by the present paper is whether risk factors are of relevance to cognitive aging, we attempted to assess the intricate biological relationships between risk factors, aging, and cognitive functioning. Accordingly, we adopted a holistic perspective on brain dysfunction, such as proposed by Goldstein (1959; see Lezak, 1983, for discussion). This does not imply that various risk factors are additive, or even unidimensional, but for the purpose of

TABLE 1
DISTRIBUTION (RANGES, MEANS, AND STANDARD DEVIATIONS) OF AGE AND LEVEL OF EDUCATION

	±20 years		±30 years		±40 years		±50 years		±60 years	
	Age	Educ	Age	Educ	Age	Educ	Age	Educ	Age	Educ
No risk										
Range	18–23	3–7	27–31	4–6	37–42	3–6	47–53	3–6	59–63	3–5
M	20.75	4.88	29.75	4.75	39.75	4.63	49.75	4.50	61.38	4.38
SD	2.12	1.36	1.58	0.89	1.67	1.06	2.19	1.20	1.41	0.74
Risk										
Range	17–22	3–7	27–32	3–6	37–43	2–6	47–53	2–6	57–63	2–7
M	19.63	4.88	30.38	4.63	40.13	4.50	49.88	4.25	59.13	4.13
SD	1.77	1.55	1.92	1.06	2.64	1.31	2.42	1.28	2.10	1.73

Note. $N = 8$ for all cells; "age," age in years; "educ," level of education (Verhage, 1964).

the present study and in the absence of any more detailed information, this approach may suffice.

METHODS

Subjects. Volunteers were recruited from the normal population by means of advertisements in local newspapers. They were assigned to 5 age cohorts with mean ages from 20 to 60 years, centered around decades, with a maximum deviation of 2 years. Within each cell, subjects were balanced for sex and educational level. For this purpose, a Dutch scoring system developed by Verhage (1964) was used: a 7-point scale, ranging from "primary education not finished" (1) to master's degree (7). The advantage of this scoring system over the procedure of counting the years of scholastic education is that qualitative aspects of the education are also taken into account. Within each cell, 4 subjects had a Verhage-score of 1–4, and 4 subjects had a score of 5–7. There were two groups within each age cohort, i.e., those subjects who had been exposed to some risk factor (to be described in the next section), and subjects who had not. Each of the 10 cells consisted of 8 subjects. In order to prevent selection bias of the subjects, each cell was filled by the first 8 subjects who volunteered and met the entrance criteria for the study (see Table 1). All subjects were paid for their participation in the experiment.

Exclusion criteria and risk factors. All subjects were recruited by mean of advertisements in local newspapers or from a local brass band, sports club, or old people's home. Normal, healthy volunteers had explicitly been summoned. Subjects were preselected over the telephone: only those applicants were admitted who regarded themselves as being healthy, normal, and not in need of any help. Furthermore, those persons were excluded who, on being asked, reported major brain damage by trauma, stroke, disease, or poisoning, or who reported a major psychiatric illness known to be characterized by cognitive deficits. The subjects were then screened before the actual testing. Three additional subjects were excluded: two subjects appeared to have had a major head injury resulting in persisting cognitive dysfunctions in their medical history (available to the examiners), and one subject had been treated for brain tumor. Thus, we had a group of subjects without any a priori likelihood of brain dysfunction or cognitive dysfunctions attributable to a major neurological

TABLE 2

FACTORS POTENTIALLY ASSOCIATED WITH BRAIN DYSFUNCTIONS AND/OR COGNITIVE DECLINE THAT WERE USED AS CRITERIA FOR INCLUSION IN THE "RISK-FACTORS" GROUP

(1) Present or past treatment by a neurologist for brain disease (stroke, epilepsy, migraine, meningitis, encephalitis, brain trauma, etc.);
(2) Present or past treatment for diseases with possible repercussions on the brain;
(3) More than 3 concussions, or 1 with a PTA of more than 1 hr;
(4) More than 3 times a general anaesthesia, or 1 lasting more than 3 hr;
(5) Use of medication that affects driving ability or consciousness;
(6) Alcohol abuse (i.e., more than 35/21 glasses per week for men and women, resp.);
(7) Other neurotoxic factors, such as exposure to organic solvents or drug abuse;
(8) Psychiatric treatment less than 5 years ago;
(9) Birth complications or developmental problems of early childhood.

Note. See text for scoring and cutoffs (adapted from Houx, Vreeling, & Jolles, 1989).

or psychiatric illness. The average physical and mental condition of our subjects was at least as good as that of any experiment reported in the literature.

A neurologist administered a semistructured interview about the subject's medical history to assess the existence and severity of potential risk factors, summarized in Table 2. This took about 20 min. For three factors, it was possible to define quantitative scores and cut-off criteria. The other factors were scored as "present" when the subjects answered the questions positively. It was often possible to gather data provided by relatives to confirm the subject's statements. If necessary, medical files were consulted.

Factor 1, treatment by a neurologist concerning any brain disorder, was used to exclude all subjects with conclusive evidence of brain dysfunctioning or consequences thereof. Cut-off criterion was presence or absence of post or present treatment.

The same applied for factor 2, treatment for diseases that are supposed to have a negative effect on the normal functioning of the brain (e.g. diabetes, renal, liver, or thyroid disease; Knoefel & Albert, 1985).

Factor 3, closed head injuries, was scored present when a subject had sustained four or more traumas or at least one with a post-traumatic amnesia (PTA) of more than 1 hr, which is moderate, according to Jennett (1976). For quantitative analysis, weighted score was used: one point for every trauma, and in case of a PTA longer than 1 hr, four points.

There is increasing evidence that general anaesthesia (factor 4) has a negative influence on the central nervous system, although the exact nature and extent are still subject to discussion (Hartman, 1988). For the purpose of the present study the—admittedly—arbitrary criterion of anaesthesia on four or more occasions, or on one occasion lasting more than 3 hr, was applied. A similar quantitative score as used for traumas was applied: three points extra for each narcosis lasting longer than 3 hr.

For medication (factor 5), the criterion was at least one drug marked by the dispensing pharmacist with a yellow sticker indicating its possible influence on driving ability (Dutch Medical Pharmaceutic Committee, 1988). Subjects were asked to bring all drugs they regularly or incidentally used with them.

Scoring of the presence of alcohol abuse (factor 6) was based on data of a WHO report (1980): maximum average consumption of five glasses a day for men, and three for women (irrespective of the nature of the refreshment). To compare alcohol consumption for the sexes, the intake of the women was multiplied by 5/3.

There is still considerable dispute about the chronic neurotoxic effect of a number of other toxic factors (factor 7), such as organic solvents or aromatic carbohydroxins (Ganzevles & Jolles, 1989). The same goes for abuse of psychotropic drugs (Hartman, 1988). This

factor was scored present if the subject had (professionally) suffered intensive exposure for several years, or had sustained a single exposure to a very toxic substance (such as carbon monoxide inhalation with loss of consciousness).

Psychiatric disorders (factor 8) such as mood disorders are known to be associated with cognitive dysfunctions (e.g., see Weingartner, 1986). These disorders were scored as present when a subject had received psychiatric treatment within the past five years, or was still receiving it.

Perinatal complications or developmental problems in early childhood (factor 9) constitute a whole array of factors associated with cognitive disorders later in life. Among these factors are hypoxia at birth, M.B.D., and malnutrition during development (Kalverboer, 1988). These risk factors are the most difficult to score, especially in older adults. Unless positive information—anamnestic or of some other source—was available, the factor was scored "not present." Scored was either presence or absence.

If any of these factors was scored positive, a subject was assigned to the risk group. As a result, subjects could have been affected by more than one risk factor. It was not our aim to exhaustively cover all factors possibly associated with brain dysfunction.

Procedure. All subjects were tested individually by the same examiners. Apart from this test, the subjects had to perform several other tasks. The results of these will be discussed elsewhere. Parallel to neuropsychological testing, a complete neurological examination was carried out, with special focus on pathological and primitive reflexes (Vreeling, Verhey, Houx, & Jolles, 1988). The whole procedure took about 2 hr.

Apparatus. An Apple-IIe microcomputer was used for stimulus presentation and for recording reaction times. The stimuli were 0.6 cm in height and were presented by means of the green standard computer screen in ASCII-type characters and wre viewed from approx. 60 cm distance. Responses were given by two hand-held thumb-keys. The key for positive reactions was in the preferred hand.

Test description. A Sternberg-type memory scanning task was used, as described by Brand (1987). The method of target and nontarget selection was adapted from the method described by Logan (1978). The pool of possible stimuli consisted of the 21 consonants of the alphabet in capitals. The target set consisted of 9 different letters and there were 12 distractors or nontargets. Target items in one condition of the test never appeared as distractors in another condition.

Each stimulus was presented 1 sec after the previous response (i.e., self-paced) and was displayed for a maximum of 1 sec. Response timing started at stimulus onset and stopped at the moment that a key was pressed. The subjects were instructed to memorize the items of a memory set consisting of 1, 2, 3, or 4 letters presented for 5 sec. Thereafter, a series of letters was presented, one after another. The subject had to press the yes-button when the letter presented belonged to the memorized set and to press the no-button when it did not. Responses were to be as fast and as accurate as possible.

There were four equivalent conditions, corresponding to set sizes 1–4. Each consisted of at least 60 trials; there were 48 test trials, preceded by at least 12 practice trials. The test period started when there were no errors in the preceding 6 practice trials. There were 24 positive and 24 negative test trials, with equal probability of occurrence of the targets. The 12 nontargets were each presented twice in the rest series, and the same nontarget set was used throughout the whole test. Targets and nontargets were presented in the same semi-random order to each subject. No more than 3 targets or nontargets appeared in succession. The RT $\times$ set size function was calculated, with the median of RTs (y-axis) per set size condition plotted against the size of the memory set (x-axis). The most important parameters of this function were the slope (i.e., amount of extra time needed per item extra in memory) and the ($x=$)1-intercept (i.e., the response latency with a memory load of only one item.

Statistics. The medians of the RTs of both positive and negative responses, and the number

TABLE 3
PREVALENCE OF RISK FACTORS (SEE TABLE 2) IN THE RISK GROUP

Risk factor	Prevalence per age group ($N = 5 * 8$)					
	20 years	30 years	40 years	50 years	60 years	All
Neurology CNS	1	3	2	2	4	11
System disease	2	1	1	3	6	14
Concussions	0	1	2	2	4	9
Anesthesia	1	0	5	4	8	18
Medication	1	1	3	1	5	12
Alcohol abuse	1	2	4	1	4	12
Neurotoxins	1	3	1	0	2	7
Psychiatry	2	2	3	4	4	13
Dis. of birth/dev.	3	3	3	3	2	14
Total	12	16	24	20	39	

Number of risk factors per subject in age groups

	20 years	30 years	40 years	50 years	60 years	All
Mean	1.8	2.0	3.0	2.5	4.9	2.8
SD	0.66	1.00	1.41	1.22	1.54	1.64
Range	1–3	1–3	1–6	1–5	3–7	1–7

Note. Subjects can be characterized by the presence of more than one risk factor.

of errors per trial was analyzed. The main derivative variables were the 1-intercept, slope, and linearity of the RT × set size function. The effects of set size, age, type of response, and risk factors were tested in one design by means of MANOVAs, with set size and type of response as repeated measures and age and risk factors as between subject factors. As the positive and negative response times were two conditions of the same variable, they were treated as repeated measures, instead of being analyzed in a multivariate design. The effects of age and risk on the 1-intercept and slope were tested with two MANOVAs with type of response as within subject factors and age and risk as between subject factors. Confidence intervals of at least 95% were used to evaluate the effects.

RESULTS

Table 3 summarizes the prevalence of each risk factor and the distribution of the number of risk factors per subject in the "risk group." The number of risk factors in the risk groups increased with age ($r = .558$, $p < .001$). Especially in the older groups, some subjects were affected by a whole range of hazards (up to seven of nine), whereas only 4 subjects aged 20 or 30 had sustained a maximum of three risks. Within the risk group, there was a significant correlation between the number of sustained risks and age ($r = .560$, $p < .001$). The only substantial correlation between age and separate risk factors was that for the anesthesia score ($r = .522$, $p < .001$, Spearman's rank order correlation). The trauma

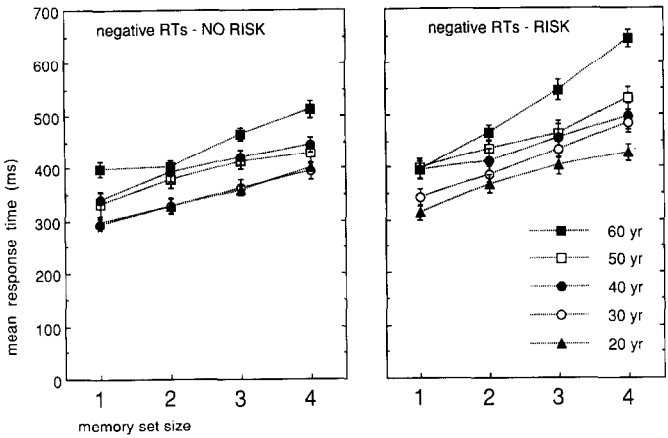

FIG. 1. Mean response latencies of the *negative* responses, as a function of the memory set size. Healthy (left) vs. "risk group" (right).

score correlated only weakly ($r = .269, p < .05$), and alcohol consumption not at all ($r = .096$). In all 80 subjects, trauma, anesthesia, and alcohol consumption were correlated with age ($r = .037$, $r = .351$ ($p < .05$), and $r = -.048$, respectively).

The test results are shown in Figs. 1 and 2. There was a significant overall within subjects effect for the type of response ($F(1, 70) = 235.3$, $p < .001$): regardless of age or risk factors, the mean 1-intercept of positive responses was 328.6 msec and 348.2 msec for the negative responses; the average slope of positive responses was less steep than that of the negative responses (35.8 msec and 42.5 msec, respectively; $F(1, 79) = 11.8$, $p = .001$). This can be taken as evidence that positive decisions take less time than negative ones. The other within subjects factor (size of the memory set) also affected the response time: $F(3, 210) = 213.8$, $p < .001$, which can be taken to indicate that the size of the memory set is indeed positively related to the response latencies in all conditions.

There was an overall effect of age on response time (see Figs. 1 and 2; between subjects effect $F(4, 70) = 13.3$, $p < .001$). The same was true for the occurrence of risk factors. The overall effect of risk factors on reponse times can readily be seen from the fan-shaped graphs and was statistically significant: $F(1, 70) = 21.1$, $p < .001$. As a group, and regardless of any task variable, risk-affected subjects (mean response time: 417.8 msec) were slower than healthy volunteers (373.9 msec). There was no interaction between age and risk factors on response time ($F < 1$).

Regarding the derivative parameters (slope and 1-intercept of the RT $\times$ set size functions), the slopes increased significantly with age (between subjects effect: $F(4, 70) = 7.6$, $p < .001$). Age also significantly affected

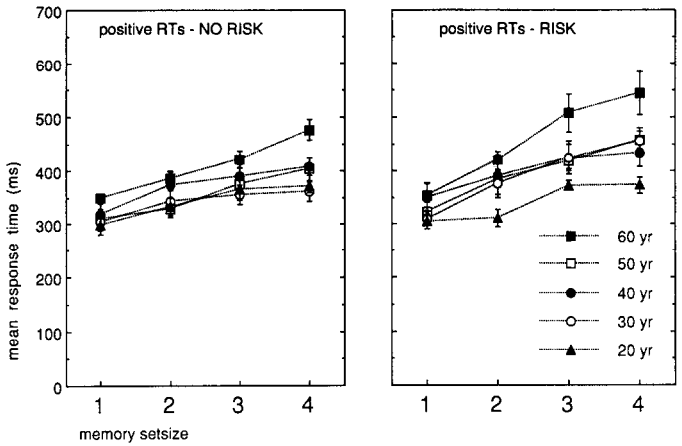

FIG. 2. Mean response latencies of the *positive* responses, as a function of the memory set size. Healthy (left) vs. "risk group" (right).

the 1-intercepts ($F(4, 70) = 4.8$, $p < .01$). For the memory scanning model, this suggests that both scanning and nonscanning stages are slower in the older age groups. Moreover, risk factors affected both slopes and 1-intercepts: $F(1, 70) = 13.7$, $p < .001$, and $F(1, 70) = 5.9$, $p < .05$. Apparently, risk factors can further delay memory processes. For the slopes, age and risk factors also showed significant statistical interaction, $F(4, 70) = 2.6$, $p < .05$, but not for the 1-intercept. This means that the effect of risk factors on memory scanning speed, but not on the nonscanning stages, is more pronounced in subjects aged 40–60 than in young adults. This, in turn, indicates that the speed of memory search is more vulnerable to the joint influences of age and risk factors than perceptual or motor processes.

The number of hazards the subjects had been exposed to and the test performance of these subjects correlated significantly with the slopes of the positive and negative responses ($r = .478$ and $.507$, respectively; $p < .001$), but regarding the 1-intercepts no significant correlation was found ($r = .073$ and $r = .121$, resp.)

The level of education of the subjects hardly affected any of the variables. The best correlation was that with the 1-intercept of the negative responses ($r = -.310$, $p < .05$). This means that less than 10% of the total variance on this parameter could be explained by the educational level of the subjects. There was no correlation between the other variables and the educational level of the subjects.

Both the slopes and 1-intercepts of the positive and negative responses were significantly correlated ($r = .696$ and $r = .671$, respectively, $p < .001$). This can be interpreted as evidence of a good internal consistency

of the task used. With very few exceptions, the linearity of response time × set size functions was quite high (overall mean $r = .855$).

The overall mean number of errors was seldom greater than 1 per trial × set size condition. There were far more false negative than false positive responses ($F(1, 79) = 47.67$, $p < .001$). The number of errors was not affected by age, risk factor, or education (see Table 4 for the mean number of errors per age and healthy/risk group).

DISCUSSION

The results indicate that the Sternberg task has a fairly high internal consistency, which confirms the findings of Brand (1987). Similarly, the almost invariably high linearity of the RT × set size functions further validates the use of the memory scanning paradigm in clinical settings. Furthermore, the lack of a significant correlation between any variable and educational level suggests that information processing, as measured by this task, is relatively independent of cultural factors.

There was a significant slowing with age of virtually all aspects of the memory scanning process. This was true for the actual memory search (reflected by the slopes of the RT × set size functions), as well as for perception and/or motor responses (reflected by the 1-intercept). Risk factors had similar effects in all age groups; thus, as far as the Sternberg task is concerned, it can be said that the existence of risk factors for brain dysfunction aggravate the effects of aging even for subjects younger than 65 years. There was no systematic relationship between these main factors and the educational level of the subjects; neither was there a variable that could predict the number of errors made in the task used in the present study.

The negative responses were consistently slower and had steeper slopes than the positive responses. This is compatible with the assumption of serial self-terminating search, as a positive match should—in the long run—occur earlier in the scanning process than the conclusion that an item does not belong to the memory set. Sternberg (1975) discussed some limitations of self-terminating search as an alternative model to that of serial exhaustive search. In spite of Sternberg's objections, it is unclear how the finding of clear-cut differences in positive and negative RTs by the present study and by Strayer, Wickens, & Cannon (1987) could be explained in terms of exhaustive search.

The present results indicate that aging already affects the speed of information processing in middle-aged subjects (aged 40 to 50) as contrasted to healthy young adults aged 20. Not only were the sensory and motor processes affected by age, as can be concluded from the increase in the intercept (which increased from 303 msec at age 20 to 330 msec at age 45), but in many instances memory scanning was also slower (the time needed per item extra in memory increased from 25.4 to 33.1 msec;

TABLE 4
MEAN NUMBER OF NEGATIVE AND POSITIVE ERRORS IN HEALTHY (H) AND RISK (R) COHORTS

	False positives								False negatives							
	Set size 1		2		3		4		1		2		3		4	
Age group	H	R	H	R	H	R	H	R	H	R	H	R	H	R	H	R
±20 years	1.00	0.75	0.75	0.63	1.00	0.13	0.38	0.88	1.13	0.25	0.38	0.00	0.25	0.38	0.13	0.25
SEM	0.45	0.26	0.33	0.28	0.40	0.13	0.28	0.42	0.58	0.17	0.28	0.00	0.26	0.28	0.13	0.17
±30 years	0.63	0.25	0.38	1.63	0.88	1.38	0.25	1.00	0.25	0.63	0.13	0.88	0.25	0.13	0.38	0.25
SEM	0.28	0.17	0.20	0.40	0.37	0.53	0.17	0.35	0.17	0.28	0.53	0.31	0.26	0.13	0.20	0.17
±40 years	0.38	1.00	1.00	0.88	0.63	0.50	0.25	0.38	0.88	0.38	0.25	0.50	0.13	0.25	0.50	0.00
SEM	0.28	0.29	0.53	0.37	0.35	0.29	0.17	0.20	0.55	0.28	0.17	0.31	0.13	0.26	0.29	0.00
±50 years	1.13	0.38	0.88	0.63	1.38	0.25	1.25	1.63	0.63	0.75	0.50	0.38	0.63	0.50	0.38	0.25
SEM	0.37	0.20	0.37	0.28	0.45	0.17	0.37	0.63	0.40	0.17	0.29	0.20	0.20	0.31	0.20	0.17
±60 years	1.13	0.13	1.13	0.63	0.63	0.75	1.25	0.50	0.38	0.25	0.13	0.13	0.25	0.38	0.75	0.00
SEM	0.32	0.13	0.13	0.35	0.28	0.39	0.78	0.40	0.20	0.17	0.13	0.13	0.17	0.20	0.26	0.00

see figures). The scanning speed is thought to be more central and therefore more relevant to the speed of overall cognitive functioning. These findings are in agreement with the data of Strayer, Wickens, & Cannon (1987) and Wickens, Braune, & Stokes (1987): they found a marked overall increase from subjects aged 20 to 60, and a lesser age effect on scanning speed. Furthermore, the RTs in their studies were in the same range as those found in the present study, although the overall RTs were somewhat longer, and the slopes of RT × set size functions tended to be steeper. This may be due to the fact that in their procedure, the stimulus presentation time was limited to 200 ms, whereas in our procedure, stimuli were displayed for a maximum of 1 sec.

The apparent decrease with age in the speed of information processing could explain why elderly people who do not experience any significant perceptual or intellectual loss often have difficulties with processing all information that is presented to them. Even without subjective memory complaints, older individuals report that they can no longer keep up with all the new impressions and issues that pertain to their personal life. These worries may well originate from a general reduction in the speed of information processing, which already exists in earlier decades of life, but which only becomes manifest in the seventh or eighth decade. To quote Jensen (1982) as far as intelligence is concerned, "Seemingly small differences in speed of information processing (..), when multiplied by months or years of interaction with the environment can in part account for the large differences observed between individuals in vocabulary, general information, and other developed skills assessed by IQ tests." Taking IQ as an index of cognitive functioning, one might infer that a reduced speed of processing in middle-aged may eventually result in disorders of higher cognitive functions in elderly individuals.

The finding of more false negative than false positive responses can probably be explained by the different sizes of the positive and negative sets: in all conditions, there were 12 distractors and a maximum of only 4 targets. No significant change in the speed–accuracy trade-off (Fitts & Radford, 1966) was observed. This is inconsistent with the notion that people become slower but more cautious (i.e., more accurate) as they grow older (Botwinick, 1977). A possible explanation for the absence of evidence for such a shift in the strategy used might be found in the small number of errors and the large individual differences in the number of errors in all age groups.

The observed effect of aging is greatly enhanced when risk factors are present. Although none of the factors mentioned in Table 2 can explain a substantial part of the individual differences in test performance, there were significant group differences. This confirms the data of Haxby et al. (1986) on differences between the visual memory of normal, healthy aging subjects and that of aging groups with health problems. Such differences

also give validity to their "rigorous health screening," since subjects who are discarded on health grounds indeed seem to perform significantly worse. Research of this kind may constitute an important step in gaining knowledge about aging in the normal population, especially when carried out on a prospective basis. Salthouse's suggestion (1988) to record a number of other background variables, such as "hours spent on reading per day and hobbies," and his data to support this, may form another important contribution to the validity of aging research.

The present findings may have serious implications for future cognitive aging research (Houx et al., 1989). The vast majority of studies on cognitive aging, did not screen the "normal subjects" explicitly for factors related to physical or mental health, other than dementia or severe brain damage. The present findings thus suggest that many of the effects of aging reported in the literature result from factors other than aging itself. We suggest that, in any future research on aging and cognitive functions, it is important to rigorously screen subjects for any known risk factor for brain dysfunction, or other aspect of suboptimal mental or physical health.

From the present study, no conclusions can be drawn about the frequency of the risk factors for age-associated cognitive decline in the normal populaton. However, we think that "normal aging" does not necessarily imply "healthy aging." For research into healthy cognitive aging, the true demographic distributions are irrelevant since all risk-affected subjects should be excluded, although it seems reasonable to assume that the probability of being exposed to the majority of the risk factors increases with age. A substantial part of the slowing of memory scanning observed in the risk groups of the present sample could be explained by the number of hazards the individuals had sustained (about 25%).

At present, it is impossible to assess the relative importance of any of the risk factors mentioned, as the groups were too small and the variance too high. Moreover, the severity of some factors could not be estimated adequately. In comparing a number of patients with subjective complaints following organic solvent intoxication or three other neurological conditions, Eskelinen, Luisto, Tenkanen, & Mattei (1986) found similar results: there were significant group differences. However, no specific rule for individual diagnostics could be given. A follow-up study with a larger number of subjects is being planned in order to address the notion that risk factors have differential interactions with cognitive aging.

The study of Amaducci, Fratiglioni, & Rocca (1986) is relevant in this respect: these authors found a significant relationship between clinically diagnosed Alzheimer's disease and a number of risk factors. This has motivated us to plan a 5-year follow-up study. This longitudinal study will test the hypothesis that the observed dysfunctions in information processing related to risk factors represent an enhanced effect of aging, in that the presence of risk factors portends a more rapid decline over time.

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