



Recall and Recognition in Huntington's Disease

Christoph J. G. Lang, Mirko Majer, and Paul Balan

Neurological Hospital, University of Erlangen-Nuremberg, Erlangen, Germany

Friedel M. Reischies

Psychiatric Hospital, Free University of Berlin, Berlin, Germany

We assessed 19 patients with Huntington's disease (HD) at early to moderately advanced stages of their disease using memory tests that investigated verbal and visual recall and recognition. In those tests where identical material was subject to recall and recognition the standardized results (z scores) were lower for recognition. Performance was better with pictorial than with verbal material. While recognition bias and savings scores did not differ significantly from controls, all other recognition parameters did so. This is in contrast to the claim that defective retrieval in HD is greatly enhanced by multiple choice recognition. One major reason for maintaining this assumption was apparently the disregard of false-positive responses. Our results indicate that verbal and visual recognition are impaired in HD, and the notion of a salient deficit of free recall is not supported. © 2000 National Academy of Neuropsychology. Published by Elsevier Science Ltd

Keywords: Huntington's disease, memory, recall, recognition, verbal, visual

Huntington's disease (HD) is an autosomal dominantly transmitted hereditary degenerative brain disease predominantly involving the striatum, caused by an extended trinucleotide (CAG) repeat on chromosome 4. Cognitive decline appears to start before the onset of motor signs and is correlated with the number of trinucleotide repeats (Jason et al., 1997).

Visuospatial and memory deficits are among the early signs of the disease (Josiassen, Curry, & Mancall, 1983) and worsen progressively. Although memory has been extensively studied in HD, little is known concerning immediate visual memory function (Derrix, 1994), and some of the published results are contradictory.

HD patients are said to have normal primary (short-term) memory, but impaired secondary (long-term) memory (Wilson et al., 1987). It has been held that the capacities for storage and recognition of verbal information are relatively unaffected (Moss, Albert, Butters, & Payne, 1986). Auditory verbal memory (acquisition and delayed recognition) has been considered defective, whereas savings scores and recognition have been

The help of Mr. Adam Lawrence with improving the English is gratefully acknowledged.

Address correspondence to Christoph J. G. Lang, Neurologische Universitätsklinik, Schwabachanlage 6, D-91054 Erlangen, Germany; E-mail: christoph.lang@neuro.med.uni-erlangen.de

deemed relatively normal. One study found the recognition abilities of HD patients to be impaired relative to controls, but superior to amnesics (i.e., mostly Korsakoff patients) (Butters, Wolfe, Martone, Granholm, & Cermak, 1985). Another study found the performance of HD and Alzheimer's disease (AD) patients equal for recall and recognition, the AD patients tending to have a more liberal ("yea-saying") response bias, being more easily enticed to false-positive responses by semantically related distractors (Brandt, Corwin, & Krafft, 1992). Correspondingly, Kramer et al. (1988) concluded that AD and HD patients matched for severity of dementia were equally impaired in discriminating between targets and distractors, although with AD there were more intrusions on recall tests.

In a study by Caine, Bamford, Schiffer, Shoulson, and Levy (1986), verbal (delayed) recall was two standard deviations (*SD*) lower than normal controls. Story recall was considerably impaired, most markedly in the last of a series of five word-list learning trials. HD patients were clearly impaired on verbal recall, similarly after a delay of 15 seconds as with a delay of 2 minutes. Since there was no marked decay between 15 seconds and 2 minutes, their performance appeared to be relatively better than that of Korsakoff (KS) or AD patients after the longer delay. The relative decay between the number of words recalled at 15 seconds and 2 minutes was even smaller than that of normals, indicating efficient recall.

Using the selective reminding procedure of Grober and Buschke, some learning progress was noted over three trials (Pillon, Deweer, Agid, & Dubois, 1993). When Weingartner, Caine, and Ebert (1979) had used the same paradigm, they had found out that HD patients cannot consistently retrieve responses that were recalled previously. They recalled fewer words than did normals, required far more trials to remember words, never recalled every word and did not discriminate between high and low imagery words. Since imageability of words as well as their release from proactive interference play a role in the encoding of the verbal material by HD patients to an extent comparable to normals, the importance of encoding deficits in the memory dysfunction associated with HD was questioned (Beatty & Butters, 1986).

The rate of forgetting—particularly on delayed visual recall—has been judged to be fairly normal, while findings with regard to visual recognition memory vary. Since memory impairment has been ascribed to a retrieval rather than an encoding or storage deficit (Butters, 1984, 1992), it is widely held that memory tasks aided by structured retrieval—such as the recognition mode as opposed to free recognition—are less influenced or even normal (Butters et al., 1985). The general claim is that (forced-choice) recognition is better preserved in HD than recall, since a great deal of the problems encountered seem to be due to ineffective and variable retrieval, while information already encoded and stored is not presumed to be lost rapidly (Butters, 1992; Martone, Butters, & Tranner, 1986; Wilson et al., 1987).

This study was designed to yield further information concerning visual and verbal recall and recognition in a group of HD patients at mild to moderate stages of their disease. Based on existent knowledge we wondered whether HD patients would display defective verbal learning. We also wondered whether there would be a difference between verbal and visual material. It was anticipated from previous work that nonverbal and verbal visual recognition would be impaired contrasting to the claims made by some of the above-mentioned researchers. On the other hand, savings scores were supposed to be normal or almost normal. Our study was particularly intended to address the interrelation between recall and recognition in HD. Since it is believed that in subcortical dementia (such as HD) patients are helped more by a recognition as opposed to a recall mode, we chose tasks requiring both free recall and recognition.

MATERIAL AND METHODS

As part of a larger study 19 patients, 5 women and 14 men, were assessed. In these patients the diagnosis of HD had been unequivocally established by choreic movements, personality, or neuropsychological changes compatible with developing or existent dementia and a positive family history, including at least one parent or the demonstration of a characteristically extended CAG repeat (>39) on chromosome 4 by molecular genetics. Controls were taken from the outpatient department presenting with diseases outside the central nervous system. They consisted of 19 persons, 9 women and 10 men, matched for age and education, who were either accompanying persons without any history of neurological disease ($n = 7$) or patients suffering from tension headache, psychogenic disorder, polyneuropathy ($n = 2$), facial palsy, cervical, or lumbar ($n = 2$) disk hernia, fibromyalgia, erectile dysfunction, mild hearing disorder, or Guillain-Barré syndrome. Patients' and controls' data are given in Table 1.

Staging was done according to the Senility Severity Rating System (SSRS; Berger, 1980). The scale is used for staging the severity of dementia and ranges from class (stage) I ("can function in any surroundings, but forgetfulness is often disruptive of daily activities") to stage VI ("bedridden or confined to a chair and responds only to tactile stimuli"). Stage II is defined as "can function without direction only in familiar surroundings," stage III as "needs direction to function even in familiar surroundings but can respond appropriately to instructions," and stage IV as "needs assistance to function; cannot respond to direction alone." There were no patients in either stage V or VI.

Verbal IQ was determined using the Multiple Choice Vocabulary Test, version B (MWT-B; Lehl, 1977), nonverbal IQ using Raven's Standard Progressive Matrices (RSPM; German version by Kratzmeier & Horn, 1979). As three patients refused to be tested with the RSPM, the number of results on this test was only 16.

The following memory tests were administered:

1. Syndrom-Kurztest (short syndrome test, SKT; Erzigkeit, 1986)
2. Rey Auditory Verbal Learning Test (RAVLT; Lezak, 1995) as modified by Reischies.

TABLE 1
Data: Patients and Their Controls^a

	Patients ^b		Controls ^c		<i>p</i> = .1786 ^a
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age (years)	48.42	7.78	51.05	16.66	.538 ^d
Years of schooling	9.00	1.37	8.68	1.57	.4368 ^c
Disease duration (years)	5.26	4.65	<i>na</i>	<i>na</i>	<i>na</i>
Disease stage (SSRS)	1.99	.92	<i>na</i>	<i>na</i>	<i>na</i>
MWT-B raw score	22.84	6.64	27.95	4.58	.0130 ^c
MWT-B IQ	94.21	10.25	105.63	13.84	<i>na</i>
RSPM raw score	21.95	11.50	42.50	9.81	.0001^c
RSPM IQ	83.47	14.33	114.25	13.84	<i>na</i>

Note. Significant results are in bold type. MWT-B = Multiple Choice Vocabulary Test, version B (Lehl, 1977), *na* = not applicable, RSPM = Raven's Standard Progressive Matrices (Kratzmeier & Horn, 1979), SSRS = Senility Severity Rating System (Berger, 1980); for an elaboration of the SSRS see text.

^aFourfold X² test.

^bFemale: $n = 5$, male: $n = 14$.

^cFemale: $n = 9$, male: $n = 10$.

^dStudent's *t* test.

^eMann-Whitney *U*-test—Wilcoxon rank sum *W* test.

The memory tests were chosen to combine different memory modalities and response modes.

The SKT is a test battery for the assessment of memory, attention, and perceptual speed. It contains three subtests, from a total of nine, which explicitly assess memory. After 12 pictures of common objects have been named, immediate recall of their verbal labels is required (subtest II, first memory trial). The display containing the pictures is then shown for an additional 5 seconds. Five intervening subtests later, delayed recall of the same words (subtest VIII, second memory trial) is followed by a multiple-choice visual recognition task of the 12 previously presented pictures interspersed with 36 new pictures presented simultaneously (subtest IX, third memory trial). Thus, pictures are first named, then the names recalled immediately and some minutes later, before multiple-choice (MC) visual recognition is tested. Since recognized items are not marked, a visual scanning component as part of the MC recognition results must be taken into consideration. Version D of five parallel versions (A–E) was used. For this study, we used the raw scores (recalled items), and within the recognition trial the number of correct hits (C), false alarms (F), difference of both scores ($D = C - F$), and d' -values according to the signal detection theory (Green & Swets, 1966). Based on the correct hit and false alarm rate, measures of accuracy (Pr) and bias (Br) according to the two-high threshold theory were also calculated as specified by Brandt et al. (1992). The savings score was determined as the retained items score on delayed recall divided by the score on immediate recall multiplied by 100 (Tröster et al., 1993).

For the RAVLT we used an abbreviated version by Reischies based on three recall trials of 15 items each, followed by a recognition trial comprising altogether 50 simultaneously presented items without delay. This version was chosen to shorten the test procedure knowing that improvement with recall on trials 4 and 5 is minor, and to ensure that the two tests had a reasonably similar number of trials. After auditory presentation, verbal material is first recalled immediately on three subsequent trials and then recognized visually in MC fashion. Again, recognized items were not marked. The word items were, with two exceptions, translations from the version by Geffen, Moar, O'Hanlon, Clark, and Geffen (1990). d' , Pr , and Br were calculated as with the SKT. Since there were no intervening tasks, a savings score was not calculated.

The tests were given in one session, the MWT-B first, RSPM last. The RAVLT and the SKT were administered in between in variable order.

Thus, the following aspects of visual (1.–2.) and verbal (3.–4.) memory were covered:

1. Immediate nonverbal visual recognition (SKT3)
2. Immediate visual verbal recognition (of auditorily presented words) (RAVLT4)
3. Immediate and delayed verbal recall (of visually presented nonverbal figures) (SKT1 and SKT2)
4. Immediate verbal recall (of auditorily presented words) (RAVLT1 through RAVLT3).

All scores were transformed into z scores of the respective controls. Nonparametric and parametric statistical analyses were performed using the SPSS program package (Norušis, 1993). The significance level was set at $p \leq .05$. In cases where more than one variable was tested at a time, an α -correction (p divided by the number of variables) was introduced. Outliers were determined according to Dixon (1953) (cf. Sachs, 1984, pp. 278–280).

RESULTS

The patients and their controls were well matched for gender, age, and education. However, on measures of verbal and nonverbal crystallized intelligence, the controls were superior.

Means, medians, standard deviations, ranges, and *z* scores of the test parameters are given in Table 2.

On the SKT all parameters differed significantly from the controls (Mann–Whitney *U*-test—Wilcoxon rank sum *W* test, $p < .0125$, Figure 1) except for the savings score (SKT2 compared to SKT1, same test, $p = .1629$), bias ($p = .0130$) and the gain from SKT2 to SKT3C (same test, $p = .5832$) or SKT3D (same test, $p = .9770$), respectively.

TABLE 2
Patients' Memory Performance, Raw Data, and Z Scores, Compared to Controls

Variable	<i>M</i>	Median	<i>SD</i>	Range	<i>p</i> =
SKT1 (immediate verbal recall)	4.42	4.00	1.98	2–8	.0011
<i>z</i> Score	–1.32	–1.56	1.13	–2.69–.72	
SKT2 (delayed verbal recall)	4.00	4.00	2.13	0–8	.0001
<i>z</i> Score	–1.42	–1.42	.85	–3.01–.17	
SKT2/1 savings score (%)	95.20	100.00	46.96	0–200	.1629
<i>z</i> Score	–.55	–.43	1.22	–3.03–2.17	
SKT3C (correct hits)	8.10	8.00	2.68	3–12	.0001
<i>z</i> Score	–2.50	–2.59	2.24	–6.76–.74	
SKT3F (false alarms)	.89	0.00	1.79	0–7	.1458
<i>z</i> Score	2.45	–.34	5.60	–.34–21.53	
SKT3D (<i>C</i> – <i>F</i>)	7.21	8.00	3.63	–1–12	.0000
<i>z</i> Score	–2.85	–2.26	2.73	–9.02–.75	
SKT3 <i>d'</i>	2.64	2.56	.99	.88–4.64	.0000
<i>z</i> Score	–1.79	–1.90	1.28	–4.08–.80	
SKT3 <i>Pr</i> (accuracy)	.62	.64	.23	.26–.95	.0001
<i>z</i> Score	–2.56	–2.40	2.25	–6.24–.68	
SKT3 <i>Br</i> (bias)	.11	.11	.09	.02–.29	.0130
<i>z</i> Score	–.89	–.95	.95	–1.91–1.09	
RAVLT1	3.21	3.00	1.58	1–7	.0000
<i>z</i> Score	–2.49	–2.63	1.10	–4.02–.15	
RAVLT2	4.42	4.00	1.64	2–9	.0000
<i>z</i> Score	–2.49	–2.19	1.10	–4.02–.15	
RAVLT3	4.68	4.00	1.95	2–10	.0000
<i>z</i> Score	–1.99	–2.26	.77	–3.05–.10	
RAVLT4C (correct hits)	8.58	9.00	3.10	3–13	.0000
<i>z</i> Score	–3.40	–3.13	1.96	–6.93–.60	
RAVLT4F (false alarms)	7.63	5.00	8.39	0–28	.0000
<i>z</i> Score	9.21	5.75	11.04	–.83–36.01	
RAVLT4D (<i>C</i> – <i>F</i>)	.95	2.00	7.52	–16–12	.0000
<i>z</i> Score	–6.01	–5.49	3.65	–14.23–.64	
RAVLT4 <i>d'</i>	1.24	1.10	.87	–.12–3.01	.0000
<i>z</i> Score	–2.92	–3.08	.99	–4.47–.91	
RAVLT4 <i>Pr</i> (accuracy)	.34	.32	.23	–.05–.80	.0000
<i>z</i> Score	–4.80	–5.04	2.05	–8.38–.62	
RAVLT4 <i>Br</i> (bias)	.31	.22	.24	.02–.78	.7729
<i>z</i> Score	.06	–.55	1.73	–2.02–3.45	

Note. Recognition trials are in italic. Significant results are in bold type.

C = correct hits; *F* = false alarms; *D* = differences score (*C* – *F*); SKT = Syndrome Short Test, Version D; SKT1 = immediate free recall of verbal labels for visually presented objects; SKT2 = delayed free recall; SKT3 = multiple choice nonverbal recognition of visual objects; *d'* = recognition performance as determined according to signal detection theory; RAVLT = Rey Auditory Verbal Learning Test; RAVLT1, RAVLT2, and RAVLT3 = trials 1 through 3, i.e., learning trials with verbal recall; RAVLT4 = recognition trial, MC = multiple choice. *Pr* = accuracy (*C* rate – *F* rate). *Br* = bias ($F \text{ rate}/[1 - Pr]$).

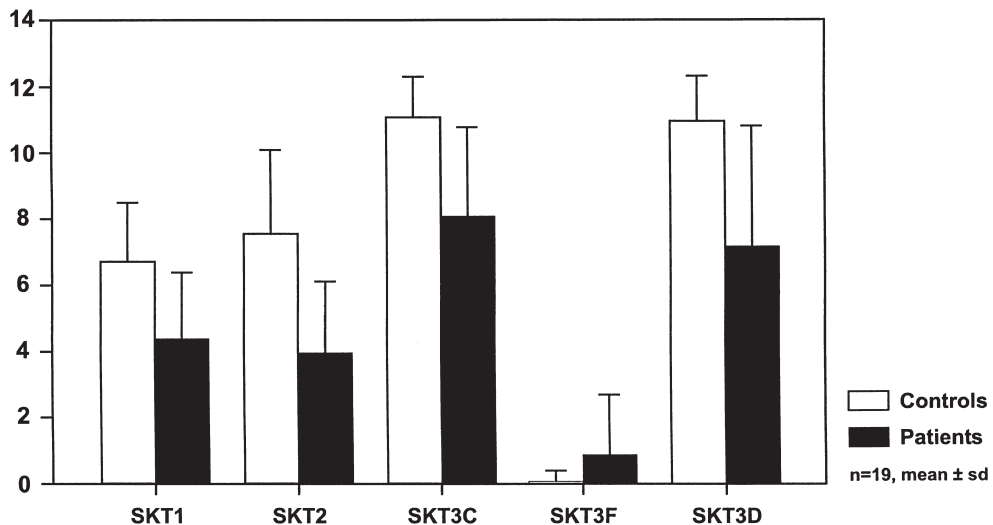


FIGURE 1. Patients' raw scores on the Short Syndrome Test (SKT) differ significantly from controls' on each of the three SKT subtests except for false alarms. SKT1 = immediate free recall of verbal labels for visually presented objects, SKT2 = delayed free recall, SKT3 = multiple-choice recognition of visual objects.

Remarkably, the number of errors on the SKT3 (SKT3F) did not differ between patients and their controls (same test, $p = .1458$). There were no outliers.

A nonparametric Friedman two-way analysis of variance (ANOVA) yielded significant differences between the three SKT subtests in patients ($df = 3$, $p = .0000$) being exclusively due to the differences between the recall and the recognition trials (Wilcoxon matched-pairs signed-ranks test, $p \leq .0034$, Table 3).

TABLE 3
Comparison of SKT and RAVLT Memory Scores in Patients

	$p =$			
	SKT2	SKT3C	SKT3D	
SKT1	.4441	.0001	.0034	
SKT2		.0001	.0021	
SKT3C			.0160	
	$p =$			
	RAVLT2	RAVLT3	RAVLT4C	RAVLT4D
RAVLT1	.0218	.0114	.0002	.3136
RAVLT2		.5195	.0002	.0733
RAVLT3			.0000	.0344
RAVLT4C				.0001

Note. SKT = Short Syndrome Test; RAVLT = Rey Auditory Verbal Learning Test; SKT1 = immediate free recall of verbal labels for visually presented objects; SKT2 = delayed free recall; SKT3 = multiple-choice recognition of visual objects; RAVLT1, RAVLT2, RAVLT3 = trials 1 through 3, i.e., learning trials with verbal recall; RAVLT4 = recognition trial; C = correct hits; F = false alarms; D = difference score ($C - F$). Significant results are in bold type. Pairwise comparisons between trials (Wilcoxon matched-pairs signed-ranks test, $n = 19$). Adjusted $\alpha = .0125$ for SKT and .01 for RAVLT.

All RAVLT variables differed significantly from controls (Mann–Whitney *U*-test—Wilcoxon rank sum *W* test, $p = .0000$), again with the exception of response bias (same test, $p = .7729$, Table 2, Figure 2).

A nonparametric Friedman two-way ANOVA yielded significant differences between the recall and recognition trials ($df = 4$, Wilcoxon matched-pairs signed-ranks test, $p = .0002$) when the number of correct hits (RAVLT4C) was considered; this difference disappeared (same test, $p \geq .0344$, corrected $\alpha = .01$) when the RAVLT4D score was used (Table 3).

Other than with the SKT, the number and variance of false positives was very high, spoiling the overall recognition performance as expressed in the d' and Pr scores (Table 2). Thus, while the gain from RAVLT3 to RAVLT4C was not significantly different ($p = .6650$, same test), the gain from RAVLT3 to RAVLT4D and false recognitions differed markedly ($p = .0000$, same test). There were no outliers.

When comparing the SKT3 and RAVLT4 recognition performance, significant differences were found for d' [signal detection theory] ($p = .0004$, 2-tailed, Wilcoxon matched-pairs signed-ranks test), Br [bias] ($p = .0038$), and Pr [accuracy] ($p = .0004$). Visual recognition (SKT) was better than verbal recognition (RAVLT).

DISCUSSION

The dementia severity of our patients was rather mild (mean stage close to II on the six-step SSRS scale). Fifteen of our patients were in stage II or less, and only 1 patient in stage IV. SSRS I to III is roughly comparable to Shoulson and Fahn's (1979) stage 1 to 3 being a 5-stage scale. In Butters, Wolfe, Granholm, and Martone (1986) study the HD patients rated 1 to 4, and in Butters et al. (1985) study 2 to 4 on the Shoulson and Fahn scale. Thus, severity of dementia was roughly equal among these samples.

Verbal and nonverbal intelligence of our patients differed significantly from the controls. Since the patients and their controls were comparable with regard to schooling, this finding probably reflects a deterioration induced by the disease itself.

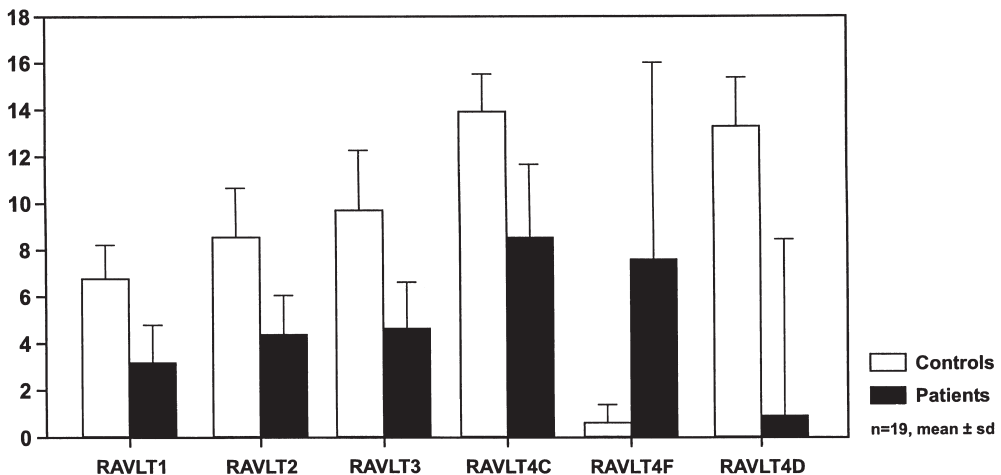


FIGURE 2. Patients' raw scores on the modified Rey Auditory Verbal Learning Test (RAVLT) differ significantly from controls' on each of the subtests. RAVLT1, RAVLT2, RAVLT3: first, second, and third recall trial. RAVLT4: recognition trial.

With regard to memory testing, the patients differed by more than -1 *SD* from the controls in almost all variables except response bias. This indicates that the tendency of patients to say “yes” when they were uncertain about the membership of an item was comparable to controls. The savings score (SKT) did not differ from that of the controls, either. However, in a study by Tröster et al. (1993) savings scores were useful in differentiating AD and HD patients from healthy controls and distinguishing between the two diseases in their early stages using the Wechsler Memory Scale (Wechsler, 1987) logical memory and visual recall subtests. The authors reported an overall classification accuracy of 74% or more.

In our study, only response bias and savings scores were normal; all other memory variables were subnormal. The patients were, therefore, clearly impaired in recall as well as in recognition. The improvement of overall memory performance from a recall to a subsequent recognition trial was not significant, thus not supporting the notion of a disproportionate aid by a structured response mode.

The most reliable measures of the RAVLT are apparently the total number of words learned over the learning trials and the performance on the retention trial (Geffen, Butterworth, & Geffen, 1994). However, it must be noted that we administered only three learning trials and no distractor list. The RAVLT scores in our study showed a gradual but only mild increase over three subsequent recall trials, from trial 1 to 2 more than from trial 2 to 3. Patients not only obtained results inferior to controls on the RAVLT1 through 3, they also exhibited a flatter learning curve. When the raw scores of the different trials were compared, significant differences appeared only for RAVLT1 versus RAVLT4C (i.e., disregarding false positives).

The performance of our patients on RAVLT1 was in a range comparable to the list A trial 1 performance of the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987) of another study (Massman, Delis, Butters, Levin, & Salmon, 1990). In this article, HD patients obtained significantly lower *z* scores for the discriminability index (a nonparametric signal detection measure that reflects the ability to discriminate between hits and false positives) than a control group. However, the authors omitted the scores of the normal group from the analysis because their high level of recall left little room for improvement on recognition testing. Also, 1 HD subject obtained minimum scores in both tests and was excluded from the analysis. Their HD group's mean difference score of 1.06 was significantly different from zero, $t_{(17)} = 3.12$, $p < .01$. This improvement of over a full *SD* (relative to the CVLT normative sample) on recognition testing indicated that HD patients benefitted substantially from aid with retrieval, but they obtained significantly higher *F* (false alarm) scores than controls. These authors analyzed the difference between long delay free recall (after 20 minutes) and recognition discriminability; we, however, assessed short delay free recall versus recognition ability. In their study verbal recognition (discriminability) was almost the same as free recall performance. In our study the verbal (RAVLT) recognition was even worse. It was remarkable that patients gave a high enough number of false alarms (a *z* score of 9.21 means very low performance in this case) to render the *D* score lower than any of the recall trials. Interestingly, a subset of persons at-risk for HD may also show increased false alarms on a word recognition memory test (Lanto, Riege, Mazziotta, Pahl, & Phelps, 1990).

When we compared our patients' verbal reproduction performance to Butters et al. (1985), they exhibited very similar learning curves from trial 1 to 3 (3.21/4.42/4.68 correctly recalled words vs. approximately 3.34/4.60/5.50 in Butters et al.) both being lower than in controls. Verbal recall, whether assessed by free story reproduction, selective reminding or paired associate learning has also been found to be impaired in HD in other

studies. This is at variance with the contention of Wilson et al. (1987) that primary (verbal, reproductive) short-term memory should be normal. Contrary to this view, in our study verbal memory was even worse than memory for visual material.

It appears from Figure 2 that the absolute improvement on free recall from trial 1 to trial 3 was greater in controls, whereas the leap from the recall trial 3 to the number of correct hits on the recognition trial 4 was about equal, although the high number of correct recalls by the controls on trial 3 left little room for improvement. The patients' greatly increased number and variance (Levene's test for equality of variances, $F = 25.803$, $p = .000$) of false positive responses, however, markedly spoiled their overall performance as expressed by respective compound scores (D , d' , Pr). Thus, it cannot be claimed that free (verbal) recall in HD is disproportionately worse than verbal recognition—at least when the number of false positives is also taken into account.

Martone, Butters, Payne, Becker, and Sax (1984), too, claimed that verbal recognition in HD was normal. They reported a $d' = 3.55$ for visual recognition of repeated 3- to 5-letter words (normals 4.54, difference not significant), and $d' = 1.46$ for unique words (normals .93, difference not significant), the first value being higher than any other comparable mean reported for HD patients. Contrary to this, our HD patients obtained results clearly inferior to normals on the RAVLT4D ($z = -6.01$), d' amounting to only 1.24 thus approaching the results for unique words (presented only once on 2 preceding days) obtained by Martone et al. (1984). The high d' values found by Butters et al. (1985) in normals as well as in patients (last recognition trial approximately 4.44 or 2.88 vs. only 1.24 in our study, mean for normals 6.12 to 6.76!) are obviously due to a systematic error. Their calculation differed considerably from others, since they assigned a value of $d' = 7.40$ for perfect recognition, whereas in our and other studies the maximum was 4.64. Regretfully, raw recognition scores were not given in Butters' et al. (1985) paper thus precluding direct comparison. This contention was attenuated for other reason, too (Kapur, 1988), because it has been objected that the apparently normal performance may be task-specific. In other recognition memory paradigms, such as those which follow multiple free recall trials, or those using less verbal material, patients have been found to be impaired. Further criticism stems from Miller and Morris (1993) who showed that some impairments in recognition memory do occur in HD.

The idea that the seemingly normal recognition performance is due to an artifact is further elucidated by the fact that in virtually every group of subjects memory testing via free recall is more sensitive to impairment and the recall scores lower than recognition scores if correct hits only are counted. In a study of HD patients by Caine et al. (1986) *all* memory parameters were lower and significantly different from normal controls (SD between -2.1 and -3.7), the delayed recognition measures especially *markedly* so (SD between -3.9 and -4.2 , $p < .001$ to $.0001$). Their delayed verbal recognition results were among only 5/27 variables, which differed from multiple sclerosis patients ($p < .01$). In another study by Caine, Ebert, and Weingartner (1977) recognition memory performance in HD was not half as efficient as that of normal controls or of Parkinson (PD) patients, although immediate and delayed free recall were equal. In our view, a most important drawback is that in many studies assessing recall versus recognition, false alarms are not taken into account and no d' or Pr measures are determined which are relatively independent of the response strategy. False alarms, however, may be critical and a very early sign of HD (Lanto et al., 1990).

Data on visual recall and recognition in HD are relatively sparse. While recall and recognition on the SKT were subnormal, the number of false alarms was not, nor were the response bias and savings scores. Thus, although the number of distractor items was almost equal for the SKT and the RAVLT, the false alarm rate was not elevated to the

same extent as with verbal material. While the subtraction of false alarms from correct hits on the RAVLT produced results inferior to the recall trial, there was still some gain on the SKT, which was not very different from normals. A comparison of parameters reflecting recognition performance yielded different results for pictures and words, pointing to some material-dependent heterogeneity within the recognition mode.

Finally, some possible shortcomings of our study must be addressed briefly. First, the two tests were not strictly parallelized. Second, the number of reproduction trials and the interval between these and the recognition trial was different. Third, while verbal recall is easily achieved by oral speech, free recall of pictures would require drawing which is difficult in HD. One possible way is the administration of the Rey-Osterrieth Figure, which, however, is not easily supplemented by a verbal counterpart.

CONCLUSION

According to our results, HD patients present with a memory deficit that is more verbal than visual and which is apparent in the reproduction *as well as* the recognition response mode. Response bias and the forgetting of visual material—responded to in a verbal recall mode—were not significantly different from controls. The virtual improvement of memory performance with recognition as compared to recall was apparent only if correct hits were considered and false alarms omitted. If parameters of global recognition performance such as D , Pr , or d' were regarded, patients performed significantly worse than controls. Based on these results it cannot be held that HD patients suffer from a prominent deficit of retrieval with an otherwise normal memory. The contention that—if helped by structured external cues—their memory performance is unremarkable is not supported by our data and is presumably a consequence of counting correct hits only while disregarding false positives. Since the distractor items were presented simultaneously rather than successively and recognized items were not marked, a visual search component cannot be excluded. Moreover, different algorithms for calculating d' are used in the literature, complicating matters further and precluding direct comparison in some instances. Since HD patients may commit a high number of false positive errors—especially with verbal material—their recognition memory performance may be even lower than their recall performance and might be used for the early detection of cognitive decline.

REFERENCES

- Beatty, W. W., & Butters, N. (1986). Further analysis of encoding in patients with Huntington's disease. *Brain and Cognition*, **5**, 387–398.
- Berger, E. Y. (1980). A system for rating the severity of senility. *Journal of the American Geriatric Society*, **28**, 234–236.
- Brandt, J., Corwin, J., & Krafft, L. (1992). Is verbal recognition memory really different in Huntington's and Alzheimer's disease? *Journal of Clinical and Experimental Neuropsychology*, **14**, 773–784.
- Butters, N. (1984). The clinical aspect of memory disorders: Contributions from experimental studies of amnesia and dementia. *Journal of Clinical Neuropsychology*, **6**, 17–36.
- Butters, N. (1992). Explicit and implicit memory disorders associated with dementia. In International School of Neuroscience (Ed.), *Proceedings of the course on neuropsychology: the neuronal basis of cognitive function* (pp. 68–79). New York: Thieme.
- Butters, N., Wolfe, J., Granholm, E., & Martone, M. (1986). An assessment of verbal recall, recognition and fluency abilities in patients with Huntington's disease. *Cortex*, **22**, 11–32.
- Butters, N., Wolfe, J., Martone, M., Granholm, E., & Cermak, L. S. (1985). Memory disorders associated with Huntington's disease: verbal recognition, verbal recognition and procedural memory. *Neuropsychologia*, **23**, 729–743.

- Caine, E. D., Bamford, K. A., Schiffer, R. B., Shoulson, I., & Levy, S. (1986). A controlled neuropsychological comparison of Huntington's disease and multiple sclerosis. *Archives of Neurology*, **43**, 249–254.
- Caine, E. D., Ebert, M. H., & Weingartner, H. (1977). An outline for the analysis of dementia. The memory disorder of Huntington's disease. *Neurology*, **27**, 1087–1092.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *California Verbal Learning Test: Research Edition*. New York: The Psychological Corporation.
- Derix, M. M. A. (1994). *Neuropsychological differentiation of dementia syndromes*. Lisse, Holland: Swets & Zeitlinger.
- Dixon, W. J. (1953). Processing data for outliers. *Biometrics*, **9**, 74–89.
- Erzigkeit, H. (1986). *Manual zum SKT. Formen A-E*. Weinheim, Germany: Beltz.
- Geffen, G. M., Butterworth, P., & Geffen, L. B. (1994). Test-retest reliability of a new form of the auditory verbal learning test (AVLT). *Archives of Clinical Neuropsychology*, **9**, 303–316.
- Geffen, G., Moar, K. J., O'Hanlon, A. P., Clark, C. R., & Geffen, I. B. (1990). Performance measures of 16- to 86-year-old males and females on the auditory verbal learning test. *Clinical Neuropsychologist*, **4**, 45–63.
- Green, D. M., & Swets, J. A. (1966). *Signal detection theory and psychophysics*. New York: John Wiley & Sons.
- Jason, G. W., Suchowersky, O., Pajurkova, E. M., Graham, L., Klimek, M. L., Garber, A. T., & Poirier-Heine, D. (1997). Cognitive manifestations of Huntington disease in relation to genetic structure and clinical onset. *Archives of Neurology*, **54**, 1081–1088.
- Josiassen, R. C., Curry, L. M., & Mancall, E. L. (1983). Development of neuropsychological deficits in Huntington's disease. *Archives of Neurology*, **40**, 791–796.
- Kapur, N. (1988). *Memory disorders in clinical practice*. London: Butterworth.
- Kramer, J. H., Delis, D. C., Blusewicz, M. J., Brandt, J., Ober, B. A., & Strauss, M. (1988). Verbal memory errors in Alzheimer's and Huntington's dementias. *Developmental Neuropsychology*, **4**, 1–15.
- Kratzmeier, H., & Horn, R. (1979). *SPM. RAVEN-Matrizen-Test. Standard Progressive Matrices manual*. Weinheim, Germany: Beltz.
- Lanto, A. B., Riege, W. H., Mazziotta, J. C., Pahl, J. J., & Phelps, M. E. (1990). Increased false alarms in a subset of persons at-risk for Huntington's disease. *Archives of Clinical Neuropsychology*, **5**, 393–404.
- Lehrl, S. (1977). *Mehrfachwahl-Wortschatz-Intelligenztest MWT-B*. Erlangen, Germany: Perimed.
- Lezak, M. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.
- Martone, M., Butters, N., Payne, M., Becker, J. T., & Sax, D. S. (1984). Dissociation between skill learning and verbal recognition in amnesia and dementia. *Archives of Neurology*, **41**, 965–970.
- Martone, M., Butters, N., & Tranner, D. (1986). Some analyses of forgetting of pictorial material in amnesic and demented patients. *Journal of Clinical and Experimental Neuropsychology*, **8**, 161–178.
- Massman, P. J., Delis, D. C., Butters, N., Levin, B., & Salmon, D. P. (1990). Are all subcortical dementias alike? Verbal learning and memory in Parkinson's and Huntington's disease patients. *Journal of Clinical and Experimental Neuropsychology*, **12**, 729–744.
- Miller, E., & Morris, R. (1993). *The psychology of dementia*. Chichester: John Wiley & Sons.
- Moss, M. B., Albert, M. S., Butters, N., & Payne, M. (1988). Differential patterns of memory loss among patients with Alzheimer's disease, Huntington's disease, and alcoholic Korsakoff's syndrome. *Archives of Neurology*, **43**, 239–246.
- Norusis, M. J. (1993). *SPSS for Windows. Base System user's guide. Release 6.0*. Chicago, IL: SPSS Inc.
- Pillon, B., Deweer, B., Agid, Y., & Dubois, B. (1993). Explicit memory in Alzheimer's, Huntington's, and Parkinson's disease. *Archives of Neurology*, **50**, 374–379.
- Sachs, L. (1984). *Applied statistics*. New York: Springer.
- Shoulson, I., & Fahn, S. (1979). Huntington disease: Clinical care and evaluation. *Neurology (Minneapolis)*, **29**, 1–3.
- Tröster, A. L., Butters, N., Salmon, D. P., Cullum, C. M., Jacobs, D., Brandt, J., & White, R. F. (1993). The diagnostic utility of savings scores: differentiating Alzheimer's and Huntington's disease with the logical memory and visual recall tests. *Journal of Clinical and Experimental Neuropsychology*, **15**, 773–788.
- Wechsler, D. (1987). *Wechsler Memory Scale-Revised*. New York: The Psychological Corporation.
- Weingartner, H., Caine, E., & Ebert, M. (1979). Imagery, encoding and retrieval of information from memory: some specific encoding-retrieval changes in Huntington's disease. *Journal of Abnormal Psychology*, **88**, 52–58.
- Wilson, R. S., Como, P. G., Garron, D. C., Klawans, H. L., Barr, A., & Klawans, D. (1987). Memory failure in Huntington's disease. *Journal of Clinical and Experimental Neuropsychology*, **9**, 147–154.