



Verbal memory deficits in relation to organization strategy in high- and low-functioning autistic children

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ABSTRACT

The present study examined the verbal memory profile and its relation to organizational strategies in high-functioning (Hi-AUT) and low-functioning (Lo-AUT) children with autism. Twenty-two Hi-AUT and 16 Lo-AUT, and 22 age-, gender- and handedness-matched normal children (NC) were required to remember a list of semantically related words for immediate and delayed recall and recognition. All autistic children showed impaired free recall, a reduced discrimination score and an elevated false alarm rate at recognition. While Hi-AUT children showed encoding and retrieval deficit, Lo-AUT children demonstrated more severe encoding problem and an additional retention difficulty. Lo-AUT, but not the Hi-AUT, children showed impaired semantic clustering. The recall performance in autistic children was not as strongly correlated with semantic clustering as in NC. The dual deficits of encoding and retrieval in autistic children, regardless of functioning level, implicate a frontal-lobe problem commonly observed in autism. The additional retention difficulty shown by low-functioning autistic children may be suggestive of pathological temporal-lobe involvement. The present findings may provide insights into future exploration of memory intervention for autistic children.

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1. Introduction

Autism is a neurodevelopmental disorder characterized by impairments in communication and social interaction, and restricted/stereotyped behaviors or interests (American Psychiatric Association, 2000). Variability in the degree of impairment was found in higher cortical functions such as memory. While some autistic individuals show severely impaired memory, some fall into the other extreme with 'savant' memory (O'Connor & Hermelin, 1989). One hypothesis suggests that autistic individuals with mental retardation (low-functioning) tended to have more severe memory impairment than those with normal intelligence (high-functioning) (see Shalom, 2003 for a review). Nevertheless, the exact memory profile and the underlying basis of memory processing in autism are uncertain.

According to the information processing model, memory processing comprises three stages: encoding (information acquired at learning trials), retention (storage of information over a delayed interval), and retrieval (recalling information from memory storage). Verbal memory deficits in different processing stages were documented in high- and low-functioning individuals with autism in previous studies (e.g., Bennetto, Pennington, & Rogers, 1996; Shalom, 2003; Williams,

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Goldstein, & Minshew, 2006). Some consistent findings were on the relatively intact retention ability of high-functioning autistic individuals, showing that they were capable of retaining as much acquired information as normal individuals at delayed recall (e.g., Bennetto et al., 1996; Minshew & Goldstein, 1993). However, studies on low-functioning autistic individuals have been rare, and initial experimental data (Boucher & Warrington, 1976) indicated that these individuals were impaired in recall at brief delays. Note, however, that their sample included autistic individuals with normal nonverbal intelligence.

Memory retrieval can be assessed by the difference in performance between free recall and recognition or by cued recall. Several studies showed that high-functioning autistic individuals who were impaired in free recall of word lists or stories were able to perform up to normal level when provided with recognition choices (Bennetto et al., 1996; Minshew & Goldstein, 1993) or semantic cues (Boucher & Warrington, 1976), suggesting that these individuals were not incapable of learning new verbal materials, but had difficulty in retrieving them from memory. In contrast, other studies reported intact free recall performance in high-functioning autistic individuals, which ran counter to the notion of retrieval deficit (Minshew, Goldstein, & Siegel, 1997; Toichi & Kamio, 2002, 2003). Furthermore, the finding that high-functioning autistic individuals demonstrated limited improvement in memory recall even when semantic cues or recognition choices were provided (Minshew & Goldstein, 2001; Williams et al., 2006) suggested that their memory problem may involve difficulties in encoding. Boucher and Warrington (1976) and Boucher (1981) showed that the memory profile of low-functioning autistic individuals was similar to that found in patients with temporal-lobe related amnesic syndrome which was characterized by impaired free recall and recognition. This suggests that the memory deficit of low-functioning individuals with autism was not simply a retrieval problem, but possibly involved a deficiency in learning and consolidating new information into long term memory. Unfortunately, their findings were not replicable in later studies. It was postulated that the poor recall and recognition performance might be the result of mental deficiency but not autism (Barth, Fein, & Waterhouse, 1995).

One possible reason for the inconsistent findings could be the different age of participants. The majority of the studies included autistic individuals with age-range spanning from teenage to adulthood (e.g., aged from 12 to 40 in Minshew & Goldstein, 1993, 2001; Minshew et al., 1997), or included only adult subjects (Bowler, Matthews, & Gardiner, 1997). The profile for autistic children may be different from autistic adults due to neural plasticity, that is, the adaptability of the developing autistic brain to accommodate inborn memory deficiency. Thus, the memory deficits of autism at a younger age may manifest as more severe than in adulthood.

Some researchers proposed that the verbal memory deficit in autistic individuals is secondary to these individuals' deficiency in utilizing effective strategies to monitor, organize and maintain the to-be-learned materials. The impairment is more prominent when learning materials are meaningful, semantically related and in vast amount (Minshew & Goldstein, 1993, 2001; Minshew et al., 1997). This executive control of memory processing was found to be mediated by the frontal-lobe, which matures up to early adulthood (Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). An increasing number of neuropsychological, neuroimaging and neuro-electrophysiological studies have reported abnormal frontal-lobe structure and functioning in autistic children and adolescents (Brocki & Bohlin, 2004; Chan, Sze, & Cheung, 2007; Courchesne & Pierce, 2005a, 2005b; Joseph, McGrath, & Tager-Flusberg, 2005; Kawasaki, Yokota, Shinomiya, Shimizu, & Niwa, 1997; Kleinmans, Akshoomoff, & Delis, 2005; O'Shea, Fein, Cillessen, Klin, & Schultz, 2005; Ohnishi et al., 2000). Given the postulations of frontal-lobe related memory deficit and pathological frontal development associated with autism, the memory profile of autistic individuals may vary across the life span as a function of frontal-lobe function. In addition, the immature frontal-lobe functioning in normal children may have reduced the discrepancy in memory ability from that of autistic children.

To the authors' knowledge, only two studies focused on verbal memory of autistic children (aged from 7 to 16) with intact intellectual functioning. While a recent study (Williams et al., 2006) supported an encoding deficit, an earlier study (Boucher & Warrington, 1976) supported a retrieval deficit given the autistic children's enhanced recall performance when prompted with semantic cues. However, the verbal materials used in the two studies were not directly comparable as Williams et al. (2006) used sentences and stories whereas Boucher and Warrington (1976) used word lists. Not only are the verbal memory profile of autistic children inconclusive, little is known about the underlying nature of the deficits.

The present study therefore aimed at investigating the verbal memory profile of high- and low-functioning children with autism using the information processing model. It was anticipated that high-functioning children with autism would exhibit encoding and/or retrieval deficits but intact retention, whereas low-functioning children would show either a similar deficit pattern at a more severe degree, or have additional difficulty in retention. The second aim was to explore the memory processes of autistic individuals in terms of strategies in organizing and storing to-be-learned materials. The memory problem of autistic children was anticipated to be associated with their ineffective semantic clustering strategy, regardless of functioning level. Findings from the present study may help shed light on the memory processing of autistic children as a function of the general intelligence level and memory strategy which may hopefully bring insights into future directions of research and clinical trials on possible effective memory intervention for autistic children.

2. Materials and methods

2.1. Participants

Twenty-two high-functioning (Hi-AUT) and 16 low-functioning (Lo-AUT) children with autism and 22 children with normal development (NC) participated voluntarily in the study with informed consent from their parents. The study was

Table 1

Demographic characteristics of the normal children (NC) and the high-functioning (Hi-AUT) and low-functioning (Lo-AUT) children with autism.

Variable	NC (n = 22)	Hi-AUT (n = 22)	Lo-AUT (n = 16)
Mean age, years	10.00 (2.09)	10.50 (1.87)	10.32 (3.31)
Gender, male/female	16/6	20/2	15/1
Handedness, right/left	18/3	17/1	13/3
TONI-III	106.68 (13.89)	101.68 (15.64)	72.13 (6.65)
CARS	–	31.94 (3.29)	33.91 (2.48)

Note: Standard deviations are in parentheses. Dashes indicate that the CARS was not administered to normal controls. TONI-III = deviation quotient of the test of nonverbal intelligence, 3rd edition; CARS = childhood autism rating scale.

approved by the Joint CUHK-NTEC Clinical Research Ethics Committee. The three groups were matched on age [$F(2,57) = .25$, $p = .78$], gender [$\chi^2 = 4.16$, $p = .13$], and handedness [$\chi^2 = 1.40$, $p = .50$] (see Table 1). High-functioning was defined as having an intelligence quotient above 80 on the Test of Nonverbal Intelligence-3rd Edition (TONI-III; Brown, Sherbenou, & Johnsen, 1992); otherwise, he/she was classified as low-functioning. While the Hi-AUT group was matched with NC group on nonverbal intelligence ($p = .76$), the Lo-AUT group had significantly lower intelligence level ranging from borderline to mild grade mental retardation ($p < .001$).

Normal children were recruited from local primary schools, and were healthy children without history of birth or developmental abnormalities, acquired brain injury, or neurological or psychiatric disorder reported by their parents. Autistic children were recruited from the Parents' Association of Pre-School Handicapped Children in Hong Kong and the participant database of the authors' laboratory. All autistic children received formal diagnosis of autism or autistic tendency by pediatricians of the Child Assessment Centres of the Department of Health in Hong Kong. Diagnosis was further confirmed by two clinical psychologists through standard clinical interviews with their parents (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). According to the DSM-IV-TR criteria, 30 children met the diagnosis of autistic disorder, and 8 met the criteria of pervasive developmental disorder not otherwise specified. Children comorbid with other forms of developmental, neurological, or psychiatric disorder, or those who were receiving psychiatric treatment for developmental problems other than autism, were excluded from the study. The Childhood Autism Rating Scale (CARS; Schopler, Reichler, & Renner, 1986) was administered to estimate the severity of autistic features. The Hi-AUT and Lo-AUT groups were matched on the total CARS score, $t(30) = 1.91$, $p > .05$ (Table 1).

2.2. Procedures and materials

The Hong Kong List Learning Test (HKLLT; Chan & Kwok, 1999; Chan, 2006), a well-established verbal learning test (Chan et al., 2009; Chan, Cheung, Law, & Chan, 2003; Chan, Ho, & Cheung, 1998; Chan et al., 2000; Cheung, Chan, Law, Chan, & Tse, 2000), was administered to each participant individually. A list of 16 Chinese words was read to each participant, who had to remember and immediately recall as many of the words as possible throughout three learning trials. The 16 words were randomly represented in four semantic categories: 2 concrete (furniture and vegetables) and 2 abstract (countries and relatives) categories. After the 3 learning trials with immediate recall, the participant was asked to recall the words from memory after 10- and 30-min delays. The examiner recorded the sequence of words recalled during each recall trial. Finally, the participant was given a recognition task, where 32 words (16 targets and 16 foils) were presented, and they were required to discriminate whether the words have been previously learnt.

2.3. Measures

The number of words correctly recalled across the three learning trials and at two delayed recall trials were recorded. At the recognition trial, apart from the correct hit (i.e., the correct identification of targets, CH) and false alarm (i.e., the false positive, FA) scores, a discrimination score was also calculated (i.e. $(CH - FA)/16 \times 100$). The rate of learning was reflected by the learning slope, which indicates the number of additional words recalled with more learning trials. The higher the value of the learning slope, the faster the learning rate, indicating a greater benefit of repetition on learning. The spontaneous organization strategy adopted to learn the word-list was measured by semantic clustering (SC). SC is the consecutive recall of words that belong to the same semantic category on the last learning trial. Given that the organization index would likely increase with a higher number of words recalled, the percentage of SC was calculated by dividing the total words recalled at the last learning trial and multiplied by 100.

3. Results

3.1. Encoding

A trial (three learning trials) by group (NC, Hi-AUT, Lo-AUT) repeated measures ANOVA was conducted to examine the number of words learnt across trials. Results showed a significant trial by group interaction effect [$F(4,112) = 4.59$, $p < .01$], main effect of trial [$F(2,56) = 52.16$, $p < .001$], and between-group difference [$F(2,57) = 8.94$, $p < .001$] (Fig. 1). Post hoc tests

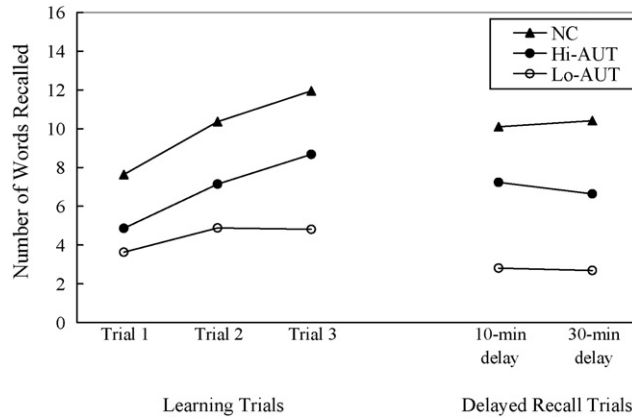


Fig. 1. Number of words recalled at three learning trials and two delayed recall trials by high-functioning (Hi-AUT) and low-functioning (Lo-AUT) children with autism and by normal children (NC).

revealed that autistic children, regardless of functioning level, recalled significantly fewer words than NC across the three trials [*F* values range from 21.1 to 27.42, $p < .001$]. The alpha level was adjusted to .017 to control for inflated Type I error as a result of multiple comparisons. The Lo-AUT group showed increasing discrepancy from the Hi-AUT group on later trials, beginning with comparable recall performance at the first trial ($p = .15$) to marginally fewer words recalled on the second trial ($p = .03$), and finally recalling significantly much fewer words on the last trial ($p = .001$). This trend can be seen in the Lo-AUT group’s flatter learning slope that reached a plateau on the last trial. ANOVA result showed a significant difference in mean rate of learning across three groups of children [$F(2,56) = 9.07, p < .001$]. Post hoc statistics indicated that the mean rate of learning of the Lo-AUT group ($M = 0.57$) was significantly lower ($p < .01$) than that of the Hi-AUT ($M = 1.91$) and NC ($M = 2.16$) groups, suggesting that the effect of repetition on Hi-AUT was comparable to NC, but that for Lo-AUT seemed to be less effective.

Given that previous studies reported that the impaired free recall of meaningful words was secondary to the deficient spontaneous use of memory strategies among individuals with autism, the percentage of semantic clustering (SC%) was thus compared between groups using ANOVA to explore how organization strategy was related to learning. It was found that the Hi-AUT group ($M = 25.7, SD = 15.41$) showed a comparable level of SC% as the NC group ($M = 27.40, SD = 13.60, p = .93$), but the Lo-AUT group ($M = 15.41, SD = 17.37$) showed a trend of less frequent semantic clustering than NC ($p = .05$), at a medium effect size of 0.31 (Fig. 2). The SC% was found to be highly correlated with the free recall performance in the NC group ($r = .67$,

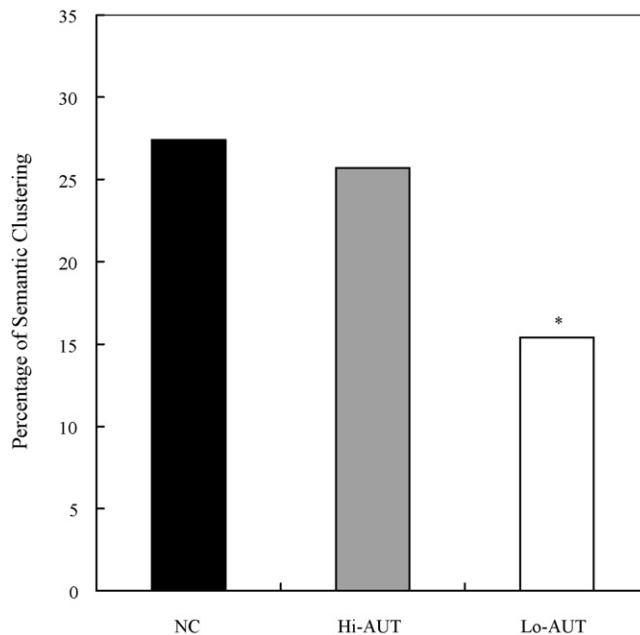


Fig. 2. Percentage of semantic clustering strategy adopted by high-functioning (Hi-AUT) and low-functioning (Lo-AUT) children with autism and by normal children (NC). * $p = .05$

$p < .01$), and moderately correlated with the Hi-AUT group ($r = .44, p = .04$). For the Lo-AUT group, the correlation became smaller ($r = .32, p = .22$) which may be due to the smaller sample size.

Some researchers found that autistic individuals did not show the typical pattern of superior recall of concrete over abstract words or the pattern of recalling more words from both the beginning (primacy effect) and end (recency effect) of a list as with normal individuals. Primacy effect was measured by the number of the first four items correctly recalled in the last learning trial, while recency effect was measured by the number of last four items correctly recalled in the last learning trial. It is therefore questionable whether their reduced free recall at learning trials is due to the atypical learning of specific types or positioning of words, or whether it is specific to their ineffective organization strategy. ANOVA results showed that all three groups recalled similar percentages of concrete [$F(2,57) = .80, p > .05$] and abstract [$F(2,57) = .85, p > .05$] words, and showed comparable primacy effect (from 26% to 30%) [$F(2,57) = .38, p > .05$]. For recency effect, however, the Lo-AUT group (42.55%) showed a stronger effect ($p < .01$) compared to NC (26.74%), while the Hi-AUT group (32.51%) was comparable to NC ($p > .05$). Surprisingly, the pattern of better recall of concrete over abstract words was not observed in all three groups, with all groups recalling approximately equal proportions of each type of words (NC: 51.61% concrete and 48.81% abstract; Hi-AUT: 50.69% concrete and 49.31% abstract; Lo-AUT: 57.94% concrete and 42.06% abstract).

3.2. Retention

Similar to the learning trials, ANOVA results showed that autistic children recalled significantly fewer words than NC in both short ($F(2,57) = 22.15, p < .001$) and long delayed trials ($F(2,57) = 26.60, p < .001$) (Fig. 1). The Lo-AUT group scored significantly lower than the Hi-AUT group at both delays, p 's $< .017$. We examined whether such differences were due to a higher forgetting rate or are due to carry-over effects of their impaired acquisition. The forgetting rate was calculated as follows: [(words recalled at delay – words recalled at learning)/words recalled at learning $\times 100$]. Fig. 3 shows the rate of forgetting of three groups of children at delayed recall trials. ANOVA results showed significant between-group differences at both delays ($F(2,57) = 11.12$ and $9.84, p$'s $< .001$). Post hoc statistics revealed substantial and rapid forgetting rate of the Lo-AUT group ($p < .01$).

Low-functioning autistic children have on average forgotten over 50% of the learnt words, and 9 of the children have forgotten all learnt words after 10 min. This showed that low-functioning autistic children were deficient in memory retention compared to high-functioning autistic and normal children whose mean forgetting rates were lower than 30%. The mean forgetting rate at 30-min delay of the Hi-AUT group (26%) was about double that of the NC group (13%), though the difference was not significant given the large standard deviation of the Hi-AUT group ($SD = 29\%$). The variability of the forgetting rate at the 10-min delay was substantially smaller ($SD = 16\%$). No NC lost more than one-third of the information learnt at the 30-min delay, whereas 32% of Hi-AUT children showed memory loss above that level, suggesting that some high-functioning autistic children may have subtle difficulties in retaining information for a prolonged period.

The effect of semantic clustering on memory was further examined at delayed trials. While the correlation between SC% at learning and the recall performances at the 10-min ($r = .60, p < .01$) and 30-min ($r = .56, p < .01$) delays remained significant in NC, those of Hi- and Lo-AUT children were non-significant (r 's ranging from .07 to .27, all p 's $> .05$). This

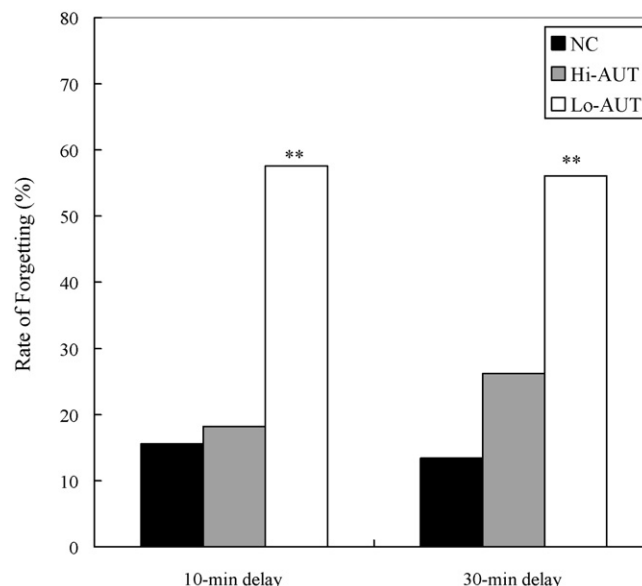


Fig. 3. Rate of forgetting at two delayed recall trials of high-functioning (Hi-AUT) and low-functioning (Lo-AUT) children with autism, and normal children (NC). *** $p \leq .01$

Table 2

Recognition performance among the normal children (NC) and the high-functioning (Hi-AUT) and low-functioning (Lo-AUT) children with autism.

Recognition	NC (n = 22)	Hi-AUT (n = 22)	Lo-AUT (n = 8)	F	Effect size
DS (%)	89.77 (12.87)	69.32 (26.79)	57.03 (32.47)	7.59***	0.48 (L)
Correct hit	14.59 (1.92)	12.91 (3.19)	11.63 (5.53)	2.99	0.33 (M)
False alarm	0.23 (0.43)	1.82 (2.81)	2.50 (1.51)	5.64**	0.43 (L)

Note: Standard deviations are in parentheses. DS = discrimination score; M = medium effect size; L = large effect size.

** $p < .01$.

*** $p \leq .001$.

suggested that normal children were able to make use of semantic clustering strategy to better retain the learnt information, whereas autistic children were less capable of sustaining the effect of semantic clustering on delayed recall.

3.3. Retrieval

To explore whether the impaired free recall of autistic children resulted from difficulty in retrieving the learnt materials, performance at recognition (where multiple choices were given as cues to ease the retrieval process) was compared with that of normal children. The discrimination score (DS) was calculated as the difference between correctly identified words (correct hits) and wrongly identified foils (false alarms) in proportion to the total number of target words. Within the Lo-AUT group, half of the children were excluded from the analyses due to their inability to comprehend the instruction of the recognition task.

As expected, both Hi-AUT and Lo-AUT children had significantly lower DS than NC ($F(2,49) = 7.59, p = .001$), with the Lo-AUT group at the bottom end and the Hi-AUT group in between (Table 2). Further inspection of the correct hit and false alarm rates found that the reduced DS in autistic children was largely related to their higher false alarm rate ($F(2,49) = 5.64, p < .01$). While normal children made either one or no false alarm error, autistic children made at most 9 errors. Increased false alarm rates have been commonly observed in autistic individuals, suggesting their vulnerability to interference generated from irrelevant information.

Despite the higher false alarm errors, the provision of recognition choices did benefit, to some extent, the autistic children. When comparing their 30-min delayed free recall with the correct hit at recognition using paired t -tests, both autistic groups demonstrated significant improvements at recognition ($t = 8.56$ and $5.08, p \leq .001$) (Table 2). A similar effect was observed in normal children ($t = 9.21, p < .001$). Improvement was greater in Lo- and Hi-AUT children (6–7 words on average) than in the NC children (4 words).

4. Discussion

The present study examined the verbal memory profile of a group of 6–17 years old high- and low-functioning children with autism. While Hi-AUT children exhibited encoding and retrieval difficulties, Lo-AUT children showed more severe encoding deficits with additional difficulty in retention as reflected by their rapid forgetting rate. Lo-AUT children learnt at least 50% fewer words than NC across the three learning trials and lost over 50% of the acquired words after a short delay. In contrast, Hi-AUT children learnt about 30% fewer words but had unimpaired forgetting rate relative to NC. These findings are consistent with Shalom (2003). This qualitative difference between high- and low-functioning autistic children in terms of memory processing stages was rarely reported in earlier studies (Boucher, 1981; Boucher & Warrington, 1976).

Although the memory performance of autistic children could be enhanced to some extent in the less effortful retrieval process, their subtly lower correct hit and elevated false alarm rates suggested that their memory impairment could not be simply attributed to retrieval deficit but also to their impaired encoding. The profile of dual deficit in encoding and retrieval, and increased vulnerability to interference (as indicated by increased false alarms) in our autistic children was similar to the profile of patients having frontal-lobe dysfunction, such as patients with depression (Bäckman & Forsell, 1994; Burt, Zembler, & Niederehe, 1995), schizophrenia (Chan et al., 2000), or frontal-lobe damage (e.g., Gershberg & Shimamura, 1995). The role of the frontal-lobe in mediating the process of memory encoding and retrieval has been supported by neuroimaging findings of increased activity in the prefrontal – particularly dorsolateral prefrontal – cortex during encoding and retrieval tasks (Fletcher & Henson, 2001). Evidence from clinical studies on patients with frontal-lobe pathology also showed impaired verbal encoding and retrieval and elevated intrusions or false alarms (Alexander, Stuss, & Fansabedian, 2003; Baldo & Shimamura, 2002; Gershberg & Shimamura, 1995).

More interestingly, the additional retention deficit in low-functioning autistic children suggested that the pathological involvement underlying their memory deficit maybe more than just frontal-lobe deficiency. Their rapid forgetting rate suggests mesial temporal-lobe involvement. Several studies have indeed reported the linkage of temporal-lobe pathology with low-functioning autism (e.g., Bauman & Kemper, 1985; DeLong & Heintz, 1997). Animal models of autism have also implicated that lesions in the amygdaloid complex and hippocampus were associated with more severe autistic features with mental retardation and memory impairment (Bachevalier, 1991, 1994). Yet, further investigation is needed as counter-evidence has also been proposed in other studies (e.g., Bailey et al., 1998; Piven, Bailey, Ranson, & Arndt, 1998).

In comparison with earlier studies on the memory of autistic individuals, the memory deficit pattern and its severity found in the present study were more extensive and robust. This may be explained by the different age of participants and methodological design. The majority of the studies finding normal or close to normal free recall performance in autistic individuals (e.g., Minshew et al., 1997; Toichi & Kamio, 2003) recruited participants from childhood to middle-age, whereas only children under 18 were recruited in the present study. The lower degree of impairment observed in those studies may be the outcome of the enhanced adaptability of the mature brain to compensate for inborn memory difficulty. Methodologically, some studies have employed only free recall (Boucher & Warrington, 1976; Minshew & Goldstein, 2001; Minshew et al., 1997) or recognition (Toichi & Kamio, 2002), making it difficult to compare between the two formats of memory retrieval. Even in studies that used both free recall and recognition trials, the false alarm rate was sometimes not taken into consideration (Boucher & Warrington, 1976; Williams et al., 2006). The present study therefore used the well-established Hong Kong List Learning Test, which has been validated to be sensitive to different memory profiles, and to provide a standardized platform for comparing different memory processes.

The differences in memory processing between high- and low-functioning autistic children were also found in the strategy that they employed. Relative to high-functioning autistic children, low-functioning autistic children showed deficient semantic clustering and increased rote-memory based recency effect. The reduced semantic clustering and reasoning ability in low-functioning autistic children was consistent with previous findings (Fein et al., 1996; Hermelin & O'Connor, 1970), suggesting that a higher level of intelligence is required to comprehend and make use of semantic characteristics to facilitate better learning and retention of information. For high-functioning autistic children, despite their unimpaired semantic clustering tendency, the lower correlation between semantic clustering and memory recall may indicate subtle inefficiency of clustering skills to aid memory compared to normal children. Such subtle inefficiency became more apparent at delayed recall, where the association became non-significant. It should also be noted that the normal level of semantic clustering shown by high-functioning autistic children may be due to developmental level. The normative data of the HKLLT shows that normal adults cluster on average up to 40–50% of the information. Yet, normal children in the present study were only able to cluster 27% of the information. This may be associated with the immaturely developed frontal-lobe which mediates the use of organization strategy in young normal children. Thus, the discrepancy in semantic clustering between normal and high-functioning autistic children was reduced at this younger age range. However, further studies are necessary to verify this hypothesis by examining the effect of age on semantic clustering in autism.

The compensation hypothesis for explaining the greater memory deficit of younger relative to older autistic individuals needs further verification. This could be done by examining a larger sample of autistic individuals stratified into different age groups, or by longitudinally following the current sample of autistic children in future studies. Thus, more solid evidence should be obtained using neuroimaging or electroencephalographic techniques to measure temporal-lobe activities of low-functioning compared with high-functioning autistic individuals during memory processing. It has been suggested that the memory deficit of low-functioning autistic individuals was a result from mental retardation but not autism (Barth et al., 1995). It is thus worthwhile to compare the memory profile of mentally retarded individuals with and without autism. Further studies may also compare children with other neurodevelopmental disorders with autism. These findings will help to shed light on the specificity of the verbal memory profile associated with autism.

5. Conclusions

In sum, the present study showed that verbal memory performance and strategy use in autistic children varied with the level of general intellectual functioning. Low-functioning autistic children showed more extensive and severe memory deficits. The dual deficits of encoding and retrieval in autistic children, regardless of functioning level, implicate a frontal-lobe problem commonly observed in autism. The additional retention difficulty shown by low-functioning autistic children may be suggestive of pathological temporal-lobe involvement. Low-functioning autistic children also demonstrated impaired spontaneous use of semantic clustering to aid memory. High-functioning autistic children showed subtle inefficiency in clustering skills to facilitate their memory as normal children did. These findings have provided some new understanding of the verbal memory profile of autistic children and its association with the underlying memory strategy. These may provide insights into future exploration of the effect of providing external semantic cues to enhance the verbal memory functioning of children with autism.

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References

- Alexander, M. P., Stuss, D. T., & Fansabedian, N. (2003). California Verbal Learning Test: Performance by patients with focal frontal and non-frontal lesions. *Brain*, 126, 1493–1503.
- American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders. 4th ed., text rev. Washington, DC: Author.
- Bachevalier, J. (1991). An animal model for childhood autism. In C. A. Tamminga & S. C. Schultz (Eds.), *Advances in neuropsychiatry and psychopharmacology*, Vol. 1 (pp. 129–140). New York: Raven Press.

- Bachevalier, J. (1994). Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia*, *32*, 627–648.
- Bäckman, L., & Forsell, Y. (1994). Episodic memory functioning in a community-based sample of old adults with major depression: Utilization of cognitive support. *Journal of Abnormal Psychology*, *103*, 361–370.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., et al. (1998). A clinicopathological study of autism. *Brain*, *121*, 889–905.
- Baldo, J. V., & Shimamura, A. P. (2002). Frontal lobes and memory. In A. Baddeley, B. Wilson, & M. Kopelman (Eds.), *Handbook of memory disorders* (2nd ed., pp. 363–379). London: John Wiley & Co.
- Barth, C., Fein, D., & Waterhouse, L. (1995). Delayed match-to-sample performance in autistic children. *Developmental Neuropsychology*, *11*, 53–69.
- Bauman, M. L., & Kemper, T. L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, *35*, 866–874.
- Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, *67*, 1816–1835.
- Boucher, J. (1981). Immediate free recall in early childhood autism: Another point of behavioral similarity with the amnesic syndrome. *British Journal of Psychology*, *72*, 211–215.
- Boucher, J., & Warrington, E. K. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology*, *67*, 73–87.
- Bowler, D. M., Matthews, N. J., & Gardiner, J. M. (1997). Asperger's syndrome and memory: Similarity to autism but not amnesia. *Neuropsychologia*, *35*, 65–70.
- Brocki, K. C., & Bohlin, G. (2004). Executive functions in children aged 6 to 13: A dimensional and developmental study. *Developmental Neuropsychology*, *26*, 571–593.
- Brown, L., Sherbenou, R. J., & Johnsen, S. K. (1992). *Test of nonverbal intelligence: A language-free measure of cognitive ability* (3rd ed.). Austin, TX: PRO-ED.
- Burt, D. B., Zembar, M. J., & Niederehe, G. (1995). Depression and memory impairment: A meta-analysis of the association, its pattern and specificity. *Psychological Bulletin*, *117*, 285–305.
- Chan, A. S. (2006). *Hong Kong List Learning Test* (2nd ed.). Hong Kong: Department of Psychology and Clinical Psychology Centre, The Chinese University of Hong Kong.
- Chan, A. S., Cheung, M. C., Han, Y. M. Y., Sze, S. L., Leung, W. W., Man, H. S., & To, C. Y. (2009). Executive function deficits and neural discordance in children with autism spectrum disorders. *Clinical Neurophysiology*, *120*, 1107–1115.
- Chan, A. S., Cheung, M. C., Law, S. C., & Chan, J. H. (2003). Phase II study of alpha-tocopherol in improving the cognitive function of patients with temporal lobe radionecrosis. *Cancer*, *100*, 398–404.
- Chan, A. S., Ho, Y. C., & Cheung, M. C. (1998). Music training improves verbal memory. *Nature*, *396*, 128.
- Chan, A. S., & Kwok, I. (1999). *Hong Kong List Learning Test: Manual and preliminary norm*. Hong Kong: Department of Psychology, The Chinese University of Hong Kong.
- Chan, A. S., Kwok, I. C., Chiu, H., Lam, L., Pang, A., & Chow, L. Y. (2000). Memory and organizational strategies in chronic and acute schizophrenic patients. *Schizophrenia Research*, *41*, 431–445.
- Chan, A. S., Sze, S. L., & Cheung, M. C. (2007). Quantitative electroencephalographic profiles for children with autistic spectrum disorder. *Neuropsychology*, *21*, 74–81.
- Cheung, M.-C., Chan, A. S., Law, S. C., Chan, J. H., & Tse, V. K. (2000). Cognitive function of patients with nasopharyngeal carcinoma with and without temporal lobe radionecrosis. *Archives of Neurology*, *57*, 1347–1352.
- Courchesne, E., & Pierce, K. (2005a). Brain overgrowth in autism during a critical time in development: Implications for frontal pyramidal neuron and interneuron development and connectivity. *International Journal of Developmental Neuroscience*, *23*, 153–170.
- Courchesne, E., & Pierce, K. (2005b). Why the frontal cortex in autism might be talking only to itself: Local over-connectivity but long-distance disconnection. *Current Opinion in Neurobiology*, *15*, 225–230.
- DeLong, G. R., & Heintz, E. R. (1997). The clinical syndrome of early-life bilateral hippocampal sclerosis. *Annals of Neurology*, *42*, 11–17.
- Fein, D., Dunn, M., Allen, D. A., Aram, D. M., Hall, N., Morris, R., et al. (1996). Language and neuropsychological findings. In I. Rapin & L. Wing (Eds.), *Clinics in developmental medicine: Preschool children with inadequate communication—Developmental language disorder, autism, low IQ* (pp. 123–154). London: Mac Keith Press.
- Fletcher, P. C., & Henson, R. N. A. (2001). Frontal lobes and human memory: Insights from functional neuroimaging. *Brain*, *124*, 849–881.
- Gershberg, F. B., & Shimamura, A. P. (1995). Impaired use of organizational strategies in free recall following frontal lobe damage. *Neuropsychologia*, *13*, 1305–1333.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Joseph, R. M., McGrath, L. M., & Tager-Flusberg, H. (2005). Executive dysfunction and its relation to language ability in verbal school-age children with autism. *Developmental Neuropsychology*, *27*(3), 361–378.
- Kawasaki, Y., Yokota, K., Shinomiya, M., Shimizu, Y., & Niwa, S.-I. (1997). Brief report: Electroencephalographic paroxysmal activities in the frontal area emerged in middle childhood and during adolescence in a follow-up study of autism. *Journal of Autism and Developmental Disorders*, *27*, 605–620.
- Kleinmans, N., Akshoomoff, N., & Delis, D. C. (2005). Executive functions in autism and Asperger's disorder: Flexibility, fluency, and inhibition. *Developmental Neuropsychology*, *27*, 379–401.
- Minshew, N. J., & Goldstein, G. (1993). Is autism an amnesic disorder? Evidence from the California Verbal Learning Test. *Neuropsychology*, *7*, 209–216.
- Minshew, N. J., & Goldstein, G. (2001). The pattern of intact and impaired memory functions in autism. *Journal of Child Psychology and Psychiatry*, *42*, 1095–1101.
- Minshew, N. J., Goldstein, G., & Siegel, D. J. (1997). Neuropsychological functioning in autism: Profile of a complex information processing disorder. *Journal of the International Neuropsychological Society*, *3*, 303–316.
- O'Connor, N., & Hermelin, B. (1989). The memory structure of autistic idiot-savant mnemonists. *British Journal of Psychology*, *80*, 97–111.
- Ohnishi, T., Matsuda, H., Hashimoto, T., Kunihiro, T., Nishikawa, M., Uema, T., et al. (2000). Abnormal regional cerebral blood flow in childhood autism. *Brain*, *123*, 1838–1844.
- O'Shea, A. G., Fein, D. A., Cillessen, A. H. N., Klin, A., & Schultz, R. T. (2005). Source memory in children with autism spectrum disorder. *Developmental Neuropsychology*, *27*, 337–360.
- Piven, J., Bailey, J., Ranson, B. J., & Arndt, S. (1998). No difference in hippocampus volume detected on magnetic resonance imaging in autistic individuals. *Journal of Autism and Developmental Disorders*, *28*, 105–110.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1986). *The childhood autism rating scale (CARS)*. New York: Irvington.
- Shalom, D. B. (2003). Memory in autism: Review and synthesis. *Cortex*, *39*, 1129–1138.
- Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, *2*, 859–861.
- Toichi, M., & Kamio, Y. (2002). Long-term memory and levels-of-processing in autism. *Neuropsychologia*, *40*, 964–969.
- Toichi, M., & Kamio, Y. (2003). Long-term memory in high-functioning autism: Controversy on episodic memory in autism reconsidered. *Journal of Autism and Developmental Disorders*, *33*, 151–161.
- Williams, D. L., Goldstein, G., & Minshew, N. J. (2006). The profile of memory function in children with autism. *Neuropsychology*, *20*, 21–29.