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CASE STUDY

Reorganization of verbal memory and language: A case of dissociation

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Abstract

Left-to-right reorganization of verbal memory following early left hemisphere damage has been reported in patients whose expressive language is governed by the right hemisphere. We present a case in which verbal memory performance was intact, despite severe left mesial temporal damage, and despite aphasia on left internal carotid sodium amytal ablation. The distribution and degree of left mesial temporal damage was assessed visually and quantitatively on MRI. These findings raise the possibility that verbal memory may shift to the language-nondominant hemisphere as a result of early left mesial temporal damage. (*JINS*, 1999, *5*, 69–74.)

Keywords: Memory, Verbal learning, Language, Cerebral dominance, Epilepsy, Temporal lobe

INTRODUCTION

Early left-sided focal epileptogenic insults have been implicated in shifts of language to the right hemisphere (RH; Duchowny et al., 1996; Rasmussen & Milner, 1977), as have extensive left-hemispheric developmental dysplasias (Calabrese et al., 1994; Reutens et al., 1993). A number of authors (Glosser et al., 1995; Helmstaedter et al., 1994; Rausch et al., 1991; Sass et al., 1995) have suggested that verbal memory function may also be subject to lateral (that is, left-to-right hemisphere) reorganization in the face of left-sided epileptogenic insult. Taken together, these reports raise two possibilities: (1) transfer of verbal memory may be facilitated by preexisting right hemisphere language dominance (Helmstaedter et al., 1994) or, (2) language and verbal memory necessarily shift in concert (Glosser et al., 1995; Rausch et al., 1991; Sass et al., 1995).

Loring et al. (1990) reported a case that appears to contradict the view that language and verbal memory are colateralized. Their patient had a transient aphasic disturbance after right temporal lobe resection for the control of complex partial seizures. There was a slight improvement in verbal selective reminding (vSRT) performance postoperatively, but a decline in nonverbal memory. Left, but not right, hippocampal stimulation interfered with verbal memory on the Brown–Peterson paradigm. Loring et al. (1990) concluded that "cerebral dominance for verbal memory and language are not necessarily linked" (p. 353).

Here, we present a case that also appears to run counter to the notion that co-lateralization is inevitable. Our patient, however, is left hemisphere language dominant with well documented evidence of severe and early *left* mesial temporal damage, and shows the neuropsychological features theoretically expected in the event of a shift of verbal memory to the right hemisphere.

CASE REPORT

K.G., a 19-year-old woman, experienced a prolonged febrile convulsion at the age of 2 years. At 7 years of age she experienced her first complex partial seizure. Six years of medication followed and K.G. was seizure free. After withdrawal of medication five seizures were recorded over the next 3 years. At 16 years, the frequency of her seizures increased significantly and proved refractory to antiepileptic medication.

K.G. was admitted to the Comprehensive Epilepsy Programme (CEP) at the Austin and Repatriation Medical Cen-

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tre for presurgical characterization. Continuous video–EEG monitoring using scalp and sphenoidal electrodes suggested left-sided epileptiform activity. Interictal PET (Ho et al., 1995) showed left mesial temporal hypometabolism (Figure 1).

Visual inspection (Jackson et al., 1993a) of magnetic resonance (MR) images revealed a markedly atrophic left hippocampus. T2-weighted images showed increased left hippocampal signal, and a loss of internal architecture on inversion recovery was observed (Figure 1). Single-slice T2 relaxometry (Jackson et al., 1993b) showed a normal right hippocampal relaxation time of 104 ms (normal $<$ 109 ms) while relaxation time for the left hippocampus was abnormal (146 ms). Multiple-slice T2 relaxation time measures of the hippocampus (Grünewald et al., 1994) and surrounding structures were made using oblique coronal and parasagittal T2 maps. Data for 50 normal controls were collected and normative T2 values for the amygdala, hippocampus (anterior, middle and posterior), entorhinal cortex, parahippocampal gyrus, and temporal lobe white matter were established (see Table 1). The measurements made in these regions for K.G. (Table 1) indicated abnormal (increased) signal in the left amygdala as well as the entire left hippocampus. Signal was within normal limits in the parahippocampal gyrus, the temporal lobe white matter and the entorhinal cortex bilaterally. However, there was signal change asymmetry between the left and right entorhinal cortices, and minor left entorhinal damage cannot be ruled out.

K.G. was referred to our centre with a previous neuropsychological characterization of superior scores on measures of verbal memory and a severely reduced score on recall of the Rey Complex Figure (RCF). We carried out a detailed neuropsychological assessment at the time of her admission to the CEP at our centre in September 1994. K.G. is a manifest left hander with no history of familial sinistrality. A Reynolds estimate (Reynolds et al., 1983) of the WAIS–R Full Scale IQ was 99. She achieved a total score of 34 on the Controlled Oral Word Association Test, 1.2 standard deviations (*SD*) below the norms of Yeudall et al. (1986), suggesting that the organization of retrieval in general was below expectation. Nevertheless, K.G. achieved a score of 17/18 on the related ("easy") pairs of the Paired Associate Learning (PAL) subtest of the Wechsler Memory Scale (WMS), comparing favourably with the Australian normative mean (*M*) of 16.78 (des Rosiers & Ivison, 1988). Her score on the unrelated ("hard") pairs was 9/12, above the normative mean of 7.74 (des Rosiers & Ivison, 1988). She obtained a score of 12.5 for immediate recall of prose, measured by means of the Logical Memory (LM) subtest of the WMS ($M = 12.16$; Ivison, 1986). Her delayed recall score (14.0) was also consistent with normative standards. On the acquisition phase (Trials 1–5) of the Rey Auditory Verbal Learning Test (RAVLT) she obtained a total score of 54, comparing favourably with the Australian age-appropriate normative standard ($M = 53.4$, $SD = 5.4$; Geffen et al., 1990). Postinterference recall (10/15) was less than 1 (-0.75) standard deviation below the normative mean, as was recognition (14/15; -0.44 *SD*). We collated her attempts to recall the RCF, which had been administered on four occasions (see Figure 2a–d) separated by at least 3 months, and with recall after delays ranging from 3 to 30 min. At one assessment (in September 1994) two delayed recalls were elicited at 7 and 30 min without representation of the RCF (see Figure 2c). On each occasion her copy of the RCF was near perfect. As Figure 2 shows, she was able to recall a little more of the overall configuration on successive occasions. At the most recent assessment (see Figure 2d), almost 4 years later, these gains had been lost. On this occasion we also administered the Warrington Recognition Memory Test (WRMT; Warrington, 1984). Word recognition was 50/50, well above ($+1.67$ *SD*) the mean of 45.6 ($SD = 3.3$) for 21

Table 1. T2 relaxation time measurements of mesial temporal structures

Region of interest	Normal*	Left[1]	Right[2]
Hippocampus:			
Anterior	$<$111	**144**	107
Middle	$<$106	**138**	103
Posterior	$<$105	**148**	104
Amygdala	$<$107	**126**	99
Temporal white matter	$<$87	83	84
Parahippocampal gyrus	$<$95	92	92
Entorhinal cortex	$<$109	103	83

*based on 50 normal controls
[1]K.G.: measurements in bold are abnormal
[2]K.G.

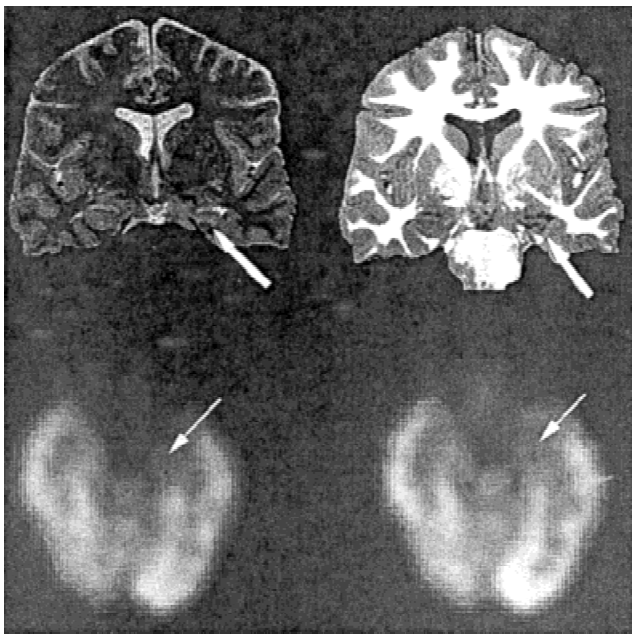

Fig. 1. Left hippocampal sclerosis (arrows) demonstrated by T2-weighted images (top, left) and an inversion recovery sequence (top, right) and decreased glucose metabolism in the left mesial temporal region (arrows) seen on PET (bottom) in K.G.

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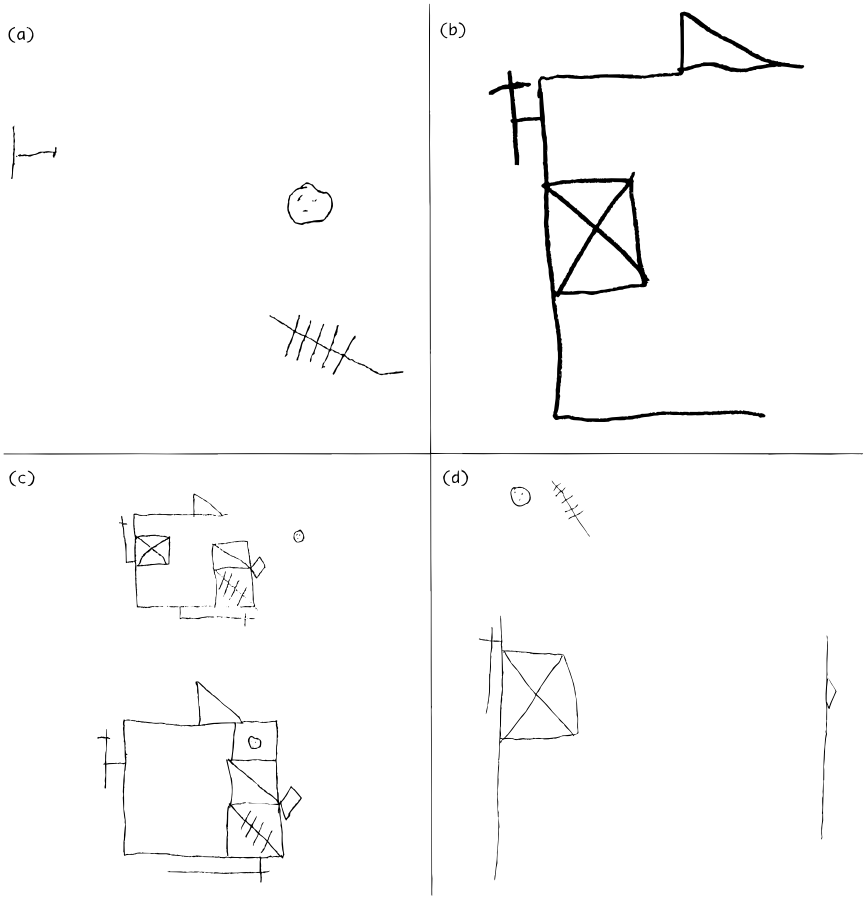

Fig. 2. Recall of the Rey Complex Figure by K.G. with delays of 3 (2a), 5 (2b), 7 (2c top), 30 (2c bottom) and 20 (2d) min on four occasions between September, 1993 (2a) and March, 1998 (2d).

left TLE patients (Saling, 1992). By contrast, her score for face recognition was 32/50 which is consistent with a scaled score of 5, and well below (-1.75 *SD*) the mean ($M = 40.6$, $SD = 5.7$) for 17 right temporal lobe patients (Saling, unpublished data).

In view of the nonconcordant memory findings, a left-sided Wada examination was performed. A pre-Wada angiogram did not show any interhemispheric cross-flow. Injection of 125 mg of sodium amytal at the same rate as the angiographic contrast resulted in a dense right hemiparesis and a prolonged aphasic arrest. K.G. was unable to name any of three line drawings of common objects, and was unable to read either of the two visually presented words. Resolution of language function was gradual and was characterised by prominent paraphasic errors on confrontation naming, repetition, and spontaneous discourse. Nevertheless, with the resolution of hemiparesis and aphasia, K.G. was able to recall the three pictorial and two verbal stimuli presented during the injection.

To date, K.G. has not proceeded to surgery for psychosocial reasons. She continues to have two or three seizures per day. Despite this, she is nearing the end of a paralegal training course, and has passed all examinations to date. She reports that she does not have difficulty with the demands that this role places on her memory.

DISCUSSION

The left mesial temporal region has been heavily implicated in the control of verbal memory (Hermann et al., 1987; Loring et al., 1991; Miller et al., 1993; Milner, 1967; Rausch & Babb, 1993; Saling et al., 1993; Sass et al., 1990). Despite severe damage to this region, K.G.'s verbal memory was preserved. In stark contrast, her performance on tests of nonverbal memory (RCF and WRMT) was severely impaired. This pattern may be the mnemonic analogue of the "crowding out" phenomenon (preserved language function but impaired spatial cognition) seen in patients with early left hemisphere insult and right hemisphere language (Helmstaedter et al., 1997; Satz et al., 1994; Teuber, 1974).

Does the integrity of verbal memory function in the face of left hippocampal damage always imply reorganization? McMillan et al. (1987) have argued previously that preserved LM scores in patients with left hippocampal sclerosis constitute evidence of verbal memory transfer, with the additional implication that lateral reorganization of this type is commonly encountered after early left mesial temporal neuronal loss. A similar view has been expressed by Seidenberg et al. (1997). These data, however, are not unambiguous since LM performance is more diffusely organized than other simpler measures of verbal memory (Baxendale, 1995;

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Saling et al., 1993), and is therefore less dependent on hippocampal integrity (Ojemann & Dodrill, 1985; Sass et al., 1992). On the other hand, our previous research (Saling et al., 1993) as well as work from other groups (Miller et al., 1993; Rausch & Babb, 1993) has shown that the acquisition of arbitrarily associated ("hard") word pairs, in particular, is critically dependent on the left mesial temporal region and is quantitatively related to the degree of hippocampal damage (Loftus et al., 1997). When normal performance on tasks such as LM and the RAVLT is accompanied by competent acquisition of "hard" paired associates, the argument in favour of lateral reorganization is strengthened. While Seidenberg et al. (1997) have used a wider variety of verbal memory measures than did McMillan et al. (1987), their data are also difficult to judge with respect to the reorganization issue for similar reasons. In particular, their "reorganized" and "nonreorganized" groups do not differ significantly on all aspects of verbal memory function.

The strength of the argument also depends on a neuroanatomical consideration, namely the extent of hippocampal damage. Quantitative T2 mapping, which has been shown to correlate with a reduced neuronal:glial ratio (Van Paesschen et al., 1997), demonstrated involvement of the entire left hippocampus and there was also increased signal in the left amygdaloid body. Functional neuroimaging of glucose uptake revealed extensive left mesial temporal hypometabolism. These findings indicate that considerable damage exists in the left mesial temporal lobe. Interestingly, the parahippocampal cortex appears to be intact in K.G.'s case, with the possibility of minor left entorhinal damage.

Selective lesions of the entorhinal cortex have been associated with mild forms of memory impairment (Meunier et al., 1993). Since the entorhinal cortex is a "gateway" to the hippocampus (Gloor, 1997) its involvement in memory function is not surprising. It would be unreasonable, however, to suppose that left entorhinal integrity or minor damage, against a background of severe ipsilateral hippocampal damage, allows for the preservation of verbal memory. Little has been written on the memory functions of the parahippocampal cortex. In the view of Eichenbaum et al. (1994) this area serves as an intermediate storage unit for isolated (i.e., unassociated or nonrelational) stimulus elements; relational learning is attributed to the hippocampus proper (Eichenbaum et al., 1994; Squire, 1992). On theoretical grounds, parahippocampal integrity is unlikely to account for K.G.'s PAL performance, and to the extent that other verbal memory tasks are partially dependent on relational learning, this argument must apply to her verbal memory in a more general sense.

Since K.G.'s left mesial temporal region is insufficiently preserved to account for normal verbal memory function, we propose that her verbal memory has been reorganized in the right hippocampal system. Support for this comes from the notion that poor visuospatial memory is a marker of "crowding out" of right mesial temporal function. In addition to the evidence of "crowding out" of visuospatial memory function, it seems unlikely that left neocortical systems developmentally deprived of an adequate interaction with the ipsilateral hippocampus could successfully mediate verbal memory on such a broad range of tasks (Eichenbaum, 1993; McClelland, 1994). On balance, then, we feel that the hypothesis of interhemispheric transfer in this case is an attractive one, and lends further impetus to the emerging view that co-lateralization of language dominance and verbal memory is not inevitable.

This view depends on the interpretation of K.G.'s Wada test performance. She demonstrated a prolonged aphasic arrest with a very gradual and progressive recovery of language. Her language status during the Wada examination was strongly reminiscent of the picture we encounter in the vast majority of left-sided injections.

Left language dominance, defined operationally in terms of the Wada findings, does not imply that the right hemisphere is devoid of rudimentary language processing capacity. Split-field research in normals suggests that the nondominant right hemisphere is capable of recognizing highly imageable and concrete nouns (Bub & Lewine, 1988; Ellis et al., 1988) of the type employed in Wada examinations and most list-learning tests of verbal memory. In essence, the right hemisphere may be equipped with rudimentary aspects of language. A difficulty that arises for the processing at a declarative level is the view that right hemisphere lexical processing does not proceed at a conscious level and that "further left hemisphere processing must take place for all word recognition tasks that demand an explicit judgment on the part of the subject" (Bub & Lewine, 1988, pp.182–183). This form of transcallosal interaction may also support hippocampal encoding and conscious *recall* of verbal material. Rudimentary preconscious language processing in right association neocortex may form a neurocognitive stepping stone between left hemisphere language mechanisms and the right hippocampus.

The pattern of neuropsychological function found in K.G. does not imply bitemporal damage, and should not represent a contraindication to left temporal resection for the relief of complex partial seizures. In all likelihood, verbal memory should be well preserved postoperatively in such cases. We emphasise, however, that this view is derived from a theoretically based interpretation. We have yet to build up experience with the postoperative outcome in cases of putative verbal memory transfer. In our own series, cases approximating the neurocognitive pattern exhibited by K.G. represent no more than 15% of those with left HS, making it unlikely that early left mesial temporal damage is a sufficient condition for transfer.

This case illustrates the importance of investigating the lateralization of language and memory separately for surgical purposes, since co-lateralization of these functions following interhemispheric functional reorganization can not be assumed.

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