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Plasticity versus chronicity: Stable performance on category fluency 40 years post-onset

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What is the long-term trajectory of semantic memory deficits in patients who have suffered structural brain damage? Memory is, per definition, a changing faculty. The traditional view is that after an initial recovery period, the mature human brain has little capacity to repair or reorganize. More recently, it has been suggested that the central nervous system may be more plastic with the ability to change in neural structure, connectivity, and function. The latter observations are, however, largely based on normal learning in healthy subjects. Here, we report a patient who suffered bilateral ventro-medial damage after presumed herpes encephalitis in 1971. He was seen regularly in the eighties, and we recently had the opportunity to re-assess his semantic memory deficits. On semantic category fluency, he showed a very clear category-specific deficit performing better than control data on non-living categories and significantly worse on living items. Recent testing showed that his impairments have remained unchanged for more than 40 years. We suggest cautiousness when extrapolating the concept of brain plasticity, as observed during normal learning, to plasticity in the context of structural brain damage.

What happens in the long-term to patients who have suffered structural brain damage, more precisely what are the long-term recovery trajectories of the physical, cognitive and emotional deficits? The traditional view was that after an initial recovery period of 1–2 years, the mature human brain has little capacity to repair or reorganize. More recently, it has been suggested that the central nervous system is plastic with the ability to change in neural structure (e.g., Toda & Gage, 2018), connectivity (e.g., Mansvelder, Verhoog, & Goriounova, 2018) and function (e.g., Chen, Epstein, & Stern, 2010). Structural neuroimaging studies with normal subjects have shown effects of experience on the size of specific brain structures, such as the increased hippocampal volume in London taxi drivers (Maguire *et al.*, 2001). This finding has been corroborated by functional imaging studies showing that learning sequential finger movements is accompanied by

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changes in the motor cortex, cerebellum, and basal ganglia (e.g., Ungerleider, Doyon, & Karni, 2010). The clinical data appear to show a more mixed picture, suggesting that different aetiologies might provide for different patterns of recovery.

Patterns of recovery have been studied extensively after traumatic brain injury (e.g., deGuise *et al.*, 2008; Thomson, 1984). Overall, the majority of studies show good physical recovery but demonstrate persistent neuropsychological sequelae in terms of cognitive and behavioural disorders. Studies looking at very long-term outcome over decades confirm this observation with significant limitations for complex and instrumental activity in daily living tasks (e.g., Himanen *et al.*, 2006; Hoofien, Gilboa, Vakil, & Donovick, 2001). In a recent study looking at patterns of recovery of verbal fluency in patients with diffuse axonal injury, the first, short-term follow-up showed (mild) impairments in both letter and category fluency but after a year the difference between controls and patients was significant only on the letter fluency, suggesting an improvement of semantic memory.

Several longitudinal studies have shown that stroke patients improve in the first year after stroke (e.g., Douiri *et al.*, 2017; Nys, van Zandvoort, de Kort, Jansen *et al.*, 2005; Nys, van Zandvoort, de Kort, van der Worp *et al.*, 2005). There are few studies looking at the long-term recovery curves in stroke, mainly for the practical reason that stroke is largely an age-related disease. However, reports on the recovery of function in young stroke patients are now starting to appear (e.g., Synhaeve *et al.*, 2015). Overall, studies with stroke patients are hampered by a relatively low survival rate and high comorbidity after stroke. Crichton, Bray, McKevitt, Rudd, and Wolfe (2016) estimated the survival rate after 15 years post-stroke at 21%, and cognitive decline due to dementia and other diseases affecting the central nervous system is substantial.

Interestingly, as most patients with a brain tumour have a poor prognosis, a more positive view was published recently by Duffau (2014) on the plasticity after diffuse glioma resections. He suggests that neuropsychological assessments and functional neuroimaging before and after operation show that it is possible to carry out massive resections of 'critical' regions without permanent sequelae, thanks to reorganization of cerebral circuits. In addition, he suggests that repeated surgeries in cases of tumour relapse show functional remapping in the same patients over time. In his view, these observations demonstrate a huge plastic potential of the human central nervous system (CNS) in adults. Obviously, this position requires more rigorous testing of emotional and cognitive functioning.

A disease that may affect young people and that can cause chronic, stable damage to the brain is encephalitis (Kapur *et al.*, 1994). In their seminal clinical review of encephalitis, Kennard and Swash (1981) identified 29 patients with the diagnosis of 'presumed acute viral encephalitis', with a 10% mortality and 28% morbidity rate, including hemiparesis, bulbar palsy and epilepsy. EEG was abnormal in all but three cases and radiology showed gross abnormalities in only five cases. Mental change was observed in 17% of the cohort, and only one patient had a hemianopia. Post-mortem examination of deceased patients showed oedema, cytotoxic neural changes, microglial proliferation and perivascular cellular infiltration. Hokkanen and Launes (1997) carried out a follow-up study of the cognitive sequelae of 45 acute encephalitis patients. They were screened for difficulty in everyday life using the Blessed dementia scale some 4 years post-onset. A substantial number of patients still suffered from psychiatric/affective problems and 12.8% had cognitive impairments, such as a memory deficit. In eight of the 11 testable cases, cognitive performance had improved over the years, in two cases a decline was found and one patient with severe deficits showed no change. Stanhope & Kopelman (2000) report on the patient DJ who suffered unilateral left temporal damage after herpes

encephalitis. On the first examination, they observed a deficit in reading and (picture) naming and a severe verbal episodic memory problem. He was a professional artist and it appeared that his painting skills were relatively unaffected. They followed DJ for 7 years and during this period they observed little change on anterograde tests for episodic memory. The anomia and dyslexia remained virtually unchanged. Apart from his reading and naming problems, there were no other signs of a semantic memory impairment.

One cognitive ability in which plasticity is thought to play a central role is memory. Learning new facts and storing new events is based on synaptic plasticity (Mansvelder *et al.*, 2018) and neurogenesis (Toda & Gage, 2018). Therefore, we aimed to investigate the long-term effects of brain lesions on memory. Semantic memory deficits are a common problem after encephalitis, particularly in the form a category-specific impairment with the non-living items being relatively spared. It has been suggested that the anatomical substrate of living categories impairment is likely to involve the inferior and the basal regions of the temporal lobes (Laiacona, Capitani & Barbarotto, 1997). McCarthy, Kopelman, and Warrington (2005) report the patient RFR who suffered from a severe memory deficit including a semantic memory impairment following an attack of herpes simplex encephalitis. During the 16 year period in which his illness was followed up there had been no change in the extent of his semantic knowledge indicating that the loss of semantic knowledge was ‘stable and likely to have arisen at the time of his initial lesion’.

There are two further studies on the long-term effects on memory performance in the patient CW, an acknowledged musical expert, who suffered extensive bilateral damage to the hippocampi, amygdala, and parts of the temporal lobes after infection with the herpes simplex virus. He was profoundly amnesic, and as was the case with DJ (Stanhope & Kopelman, 2000), his procedural memory, most interestingly his ability to play the piano, was spared. His semantic memory was also impaired in the subacute phase (1985), and he was relatively worse on living than on non-living items. When he was retested in 1992 his semantic memory had improved somewhat (Wilson, Baddeley, & Kapur, 1995). Some thirteen years later, Wilson, Kopelman, and Kapur (2008) report that he is able to store new semantic information, such as for ‘mobile phone’ and ‘eurotunnel’. His category-specific semantic deficit was not reassessed.

We had the opportunity to investigate the long-term outcome with respect to semantic memory in the encephalitic patient MS, some four decades post-onset. We assessed his current semantic memory deficit with the fluency test paradigm using exactly the same categories with either living or non-living exemplars that were used in the eighties.

Materials and method

Case description

MS is a former police cadet who contracted a febrile illness in 1971, at the age of 23. A full case description has been given by Newcombe and Ratcliffe (1975) and Ratcliffe and Newcombe (1982), so we will only summarize the essential details here. The presumptive diagnosis was herpes encephalitis. Unfortunately, no brain biopsy was taken, and although blood serum was subsequently immunonegative for herpes, the presumptive diagnosis was herpes encephalitis. There are no further details in his case notes regarding the type of encephalitis. There is no known history of epilepsy. MS has a left homonymous hemianopia, but his visual acuity is normal (6/6; N5 or near vision). He also suffers from achromatopsia. More recently, Cowey and co-workers have provided evidence for perceptual blindsight (Alexander & Cowey, 2010; Pavan, Alexander,

Campana, & Cowey, 2011). He has a severe object agnosia (successfully identifying only 22% of line drawings of objects) and prosopagnosia (e.g., Newcombe, Young, & de Haan, 1989). During the early eighties, Freda Newcombe has attempted to work with him on improving his visual problems but to no avail. Interestingly, he remains able to read accurately. His comprehension of what he reads is, however, affected by an impairment of semantic memory which can also be seen in the fact that he could only successfully name 55% objects from verbal descriptions of their functions. This semantic memory impairment is more marked for living than for non-living things; MS could, for instance, define an anchor as 'a brake for ships', but was unable to say what a nightingale is (Ratcliffe & Newcombe, 1982). Apart from the semantic memory problem there are no aphasic symptoms; MS's speech is clearly enunciated and grammatically well-formed.

Here, we have reported the verbal descriptions of the neuropsychological assessment as carried out by Freda Newcombe in the nineteen eighties. Obviously, a more comprehensive reassessment of his mental abilities would have been useful but we were unable to retrieve the original test data. Therefore, it would have impossible to make a direct comparison of his test scores. We have found one more data-point, though, that makes a similar point regarding MS' achromatopsia. In their original paper, Mollon, Newcombe, Polden, and Ratcliff (1980) demonstrated a complete inability to perceive colours and they administered the full Farnsworth-Munsell 100-Hue test. His summed error score of 1,245 represented a performance no better than random responding. In 2017 his total summed error score on the Farnsworth-Munsell 100 Hue test was 1,268, again not better than random responding (Chadwick, Heywood, Smithson, and Kentridge (2017).

A long-term follow-up study of MS has been in progress for many years. Part of this follow-up has been aimed at his semantic memory deficit using the fluency test (Young, Newcombe, Hellowell, & de Haan, 1989). More specifically, his ability to generate examples of members of living and non-living semantic categories has been tested on a number of occasions. Table 1 shows the original table from the Young *et al.* (1989) study with the fluency data obtained for each of four living and four non-living semantic categories on four different occasions. These tests were carried out in 1980 and repeated in 1985, 1986, and 1988. Control data from Brown (1978) are presented for comparison. The data show a consistent impairment for 'living' categories. The same problem was evident when MS was asked (in 1988) to identify living and non-living objects (of matched word frequency) from brief descriptions. He successfully identified 38 of 48 non-living objects, but only nine of 48 living objects. Examples of descriptions MS failed to identify include 'it has eight legs and is usually black; it eats flies and spins webs to catch them in' (spider; MS could not think of any answer) and 'it is brownish coloured and has four legs; it lives in the desert and has big humps on its back' (camel; MS gave the incorrect answer 'giraffe'). Examples of descriptions he could identify include 'it is used in cooking for flattening out pastry; it is often made of wood' (rolling pin) and 'it is small, long and thin and made of metal; it has a point at one end and a hole in the other end; it is used for sewing' (needle).

Tests and procedure

We retested his semantic memory with the same fluency test that had been used in the past. Category fluency requires the retrieval of words within a specified semantic cluster. In this fluency test battery, MS was asked to produce as many exemplars as possible in one minute for eight different categories, four categories with living items (animals, fruit, birds, vegetables) and four with non-living items (occupations, clothes, sports, furniture). This task was repeated three times on consecutive days.

Table 1. MS's performance on the fluency tests in 1980 until 1988

| | 1980 | 1985 | 1986 | 1988 | Mean | Control mean |
|-------------|------|------|------|------|------|--------------|
| Living | | | | | | |
| Animals | 7 | 8 | 9 | 9 | 8.3 | 16.6 |
| Fruit | 7 | 7 | 6 | 7 | 6.8 | 10.9 |
| Birds | 5 | 4 | 4 | 5 | 4.5 | 13.7 |
| Vegetables | 6 | 6 | 9 | 9 | 7.5 | 10.8 |
| Mean | 6.3 | 6.3 | 7.0 | 7.7 | 6.8 | 13.0 |
| Non-living | | | | | | |
| Occupations | 18 | 17 | 24 | 16 | 18.8 | 12.6 |
| Clothes | 15 | 17 | 18 | 14 | 16.0 | 14.5 |
| Sports | 14 | 14 | 18 | 17 | 15.8 | 13.6 |
| Furniture | 11 | 10 | 8 | 8 | 9.3 | 10.1 |
| Mean | 14.5 | 14.5 | 17.0 | 13.8 | 15.0 | 12.7 |

Results

MS's performance on the three administrations of the fluency battery is presented in Table 2. The first observation is that his performance over the three consecutive administrations is very consistent. If we subsequently compare his average performance now with that of some 35 years ago per category, an extremely similar performance becomes apparent. Finally, we if compare the crucial difference between his average scores on the living (now: 6.8; then 6.8) versus the non-living (now 14.3; then 15.0) categories, we can only conclude that his category-specific semantic memory deficit has remained unchanged.

Discussion

In this case study, we were interested in the long-term trajectory of memory deficits after structural brain damage, more specifically the very long-term outcome of semantic

Table 2. MS's performance on the fluency tests in 2017 compared to his previous performance and controls

| | 2017 | | | | 1980–1988 MS mean | Controls |
|-------------|------|------|-----|------|----------------------|----------|
| | I | II | III | Mean | | |
| Living | | | | | | |
| Animals | 10 | 9 | 10 | 9.7 | 8.3 | 16.6 |
| Fruit | 7 | 5 | 6 | 6.0 | 6.8 | 10.9 |
| Birds | 3 | 2 | 2 | 2.3 | 4.5 | 13.7 |
| Vegetables | 9 | 7 | 6 | 7.3 | 7.5 | 10.8 |
| Mean | 7.3 | 5.8 | 6 | 6.3 | 6.8 | 13.0 |
| Non-living | | | | | | |
| Occupations | 17 | 17 | 15 | 16.3 | 18.8 | 12.6 |
| Clothes | 13 | 18 | 17 | 16.0 | 16.0 | 14.5 |
| Sports | 18 | 18 | 17 | 17.7 | 15.8 | 13.6 |
| Furniture | 5 | 10 | 7 | 7.3 | 9.3 | 12.7 |
| Mean | 13.3 | 15.8 | 14 | 14.3 | 15.0 | 12.7 |

memory impairment after encephalitis. There are two opposing views, one holds that after an initial recovery period, the mature human brain has little capacity to repair or reorganize while the other suggests that the brain is plastic and continues to recover. Structural neuroimaging studies have shown effects of experience on the size of specific brain structures (Maguire *et al.*, 2001), and functional imaging studies have shown that learning sequential finger movements is accompanied by changes in brain activations (Ungerleider *et al.*, 2010). The latter observations are, however, largely based on normal learning in normal subjects. In contrast, the clinical data appear to show a more mixed picture, suggesting that different aetiologies might provide for different patterns of recovery (e.g., Douiri *et al.*, 2017; Duffau, 2014; Himanen *et al.*, 2006). Long-term outcome studies are rare, probably because most common neurological conditions are confounded or are practically difficult. First, the situation is complicated by the fact that many neurological conditions are age-related providing for little opportunity to evaluate the long-term outcome over decades. In addition, many of these conditions involve age-related comorbidity, such dementia. Other common neurological conditions, such brain tumours and stroke, suffer from recidivism which is not always detected in clinical practice. Traumatic brain injury occurs often in young adults but the nature of the damage is different. Therefore, we suggest that less common conditions, such encephalitis, are well suited to investigate the very long-term effects of structural brain damage. Research on young stroke patients offer another possibility for future studies.

Here, we report a patient MS who suffered bilateral ventro-medial damage after presumed herpes encephalitis in 1971. He was seen regularly in the eighties, and we recently had the opportunity to re-assess his semantic memory deficits. Note that our first series of testing took place some 10 years post-onset and that he was well and truly in a chronic phase by then. This is corroborated by the study of Laiacona, Capitani & Barbarotto (1997) in which the recovery was monitored of two encephalitis patients. At a second examination, carried out 1–2 years later, their patient LF showed a good overall recovery, while the improvement of the second patient EA was restricted to the non-living categories, leaving the latter with a clear category-specific impairment.

Our recent testing showed that the category-specific semantic memory deficit, with relatively very poor knowledge of living (e.g., animals) compared to non-living (e.g., tools) things, has remained unchanged for more than 40 years. It could be argued that his unchanged performance actually reflects the sum total of two opposite effects, that is, improvement due to plasticity of the brain and deterioration due to ageing. However, his enduring, very good performance on the non-living categories of the fluency test argues against this idea. Therefore, the fact that there is no evidence for change in a patient who suffered structural brain damage 45 years ago but has remained healthy ever since, suggests to us that we need to be cautious when extrapolating the concept of brain plasticity, as observed during normal learning, to plasticity in the context of structural brain damage.

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