**Title: Increased cognitive load reveals unilateral neglect and altitudinal extinction in chronic stroke**

Supplementary Material

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**Case 1**

 The first neuropsychological examination (T1) was conducted between one and three months after stroke onset (see Supplemental Table 1). On the Bells test (Gauthier et al., 1989), the patient omitted all of the 15 left-sided and only 1 of the right-sided bells (see Table 1). The Neglect subtest of the Test of Everyday Attention (TAP; Zimmermann & Fimm, 1994) was first performed without central fixation: the patient was slower to detect left-sided than right-sided targets and omitted 13 left-sided vs. 3 right-sided targets. On the Visual Scanning subtest of the TAP, the patient omitted only a few targets, but was very slow (> 3 sec when the target was present and > 10 sec when the target was absent). Response latencies also evidenced a deficit in the Alertness and Divided Attention subtests of the TAP. Executive functions were mostly preserved (protanopia prevented the realization of the Stroop test), except for minor difficulties of flexibility (Luria's graphic series: Luria, 1966; Tower of London: Coyette & Van der Linden, 1993). There was no short-term memory deficit, as indicated by a normal arithmetic span of 11 and a normal visuospatial span of 6. Verbal long-term memory was impaired, as evidenced by the learning and retrieval indices of the Buschke 15 (Rectem, Poitrenaud, Coyette, Kalafat, & Van der Linden, 2004). Speech was characterized by dysarthria. The patient received physiotherapy and neuropsychological rehabilitation for neglect, and was followed up because of a risk of developing subsequent epilepsy. A second neuropsychological examination (T2), conducted between five and six months after stroke onset, revealed no signs of left neglect. The patient made no omissions on the Bells test or on the Neglect subtest of the TAP. On the Visual Scanning subtest, his performance was within the norms, though he was slower to detect left (> 3 sec) than right targets (> 1 sec) and omitted more targets on the left than on the right side (14 vs. 0). A deficit was still observed in the Alertness and Divided Attention subtests but his verbal long-term memory had recovered.

**Case 2**

 The first neuropsychological examination occurred between one and three months (T1) following the stroke onset (see Supplemental Table 1). On the Bells test, the patient omitted 11 bells that were all located on the left side of the sheet (see Table 1). In the text-reading test, the patient omitted 9 words next to the left margin. In the Neglect subtest of the TAP (with central fixation), the patient failed to respond to 10 out of the 22 stimuli presented in his left visual field and 1 in his right visual field. The patient also demonstrated a general slowdown on all these tests. The Visual Scanning subtest of the TAP could not be performed because the patient was too tired, but his performance on the first trials showed several omissions and very long response latencies (> 3 sec when the target was present and > 18 sec when the target was absent). Response latencies and omissions also evidenced a deficit in the Alertness and Divided Attention subtests. Speech, verbal long-term memory (Buschke 15; Rectem et al., 2004) and executive functions were preserved (Stroop: Bruyer, Van der Linden, Rectem, & Galvez, 1995; Fluency test: Cardebat, Doyon, Puel, Goulet, & Joanette, 1990), except for abnormal response latencies in the Tower of London test (Coyette & Van der Linden, 1993). The arithmetic and visuospatial memory spans were equal to 7, revealing unimpaired short-term memory skills. The patient was administered a second neuropsychological assessment, between six and fifteen months after stroke onset (T2), on which his performance had improved on all tests previously used to assess neglect. He only omitted four left-sided targets in the Bells Test and detected all the flashed targets in the neglect subtest of the TAP. In the Visual Scanning subtest, he omitted one target but was still very slow (> 2.5 sec when the target was present and > 17 sec when the target was absent). Non-spatial attention was still impaired, in both the visual and the auditory modality, as evidenced by omissions and response latencies in the Divided Attention subtest of the TAP.

**Supplemental Table 1.** Neuropsychological examination. Patient #1 was examined between 1 and 3 months (T1) and between 5 and 6 months (T2) after stroke. Patient #2 was examined between 1 and 3 months (T1) and between 6 and 15 months (T2) after stroke. The scores in bold indicate that the performance of the patients deviated by more than 2 standard deviations from the norm.

|  |  |  |
| --- | --- | --- |
|  | **Patient #1** | **Patient #2** |
|  | **T1** | **T2** | **T1** | **T2** |
|

|  |  |  |
| --- | --- | --- |
| **Stroop Test** |  |  |

 |  |  |  |  |
| *Color naming (sec)**Word reading (sec)**Inhibition (sec)* | --- | --- | 70**56**118 | --- |
|

|  |  |  |
| --- | --- | --- |
| **Verbal fluency** |  |  |

 |  |  |  |  |
| *Semantic* | 27 | - | 19 | 38 |
| *Phonological* | 21 | - | 11 | 25 |
| **Short-term memory***Arithmetic span**Visuospatial span* | 116 | -- | 77 | -- |
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| --- |
| **Verbal long-term memory**  |

 |  |  |  |  |
| *Mean number of recalled words /15* | **9.2 \*** | 12.3 | 12 | - |
| *List learning %**Delayed recall /15* | **41.3 \*****9 \*** | 80.215 | 97.914 | -- |
| **Tower of London****(latency / total time / number of movements)** |  |  |  |  |
| *Level 3* | **-1.2**/**-2.5**/0.2 | 1.3/0.5/0.5 | **-4.1**/**-5.2**/0.5 | **-2**/**-3.3**/0.5 |
| *Level 4* | 1.2/0.9/0.9 | 1.5/-0.1/0.4 | 0.8/0.8/0.8 | **-1.4**/0.4/0.8 |
| *Level 5+* | 0.4/0.5/1 | 1.7/0.8/1 | **-1.1**/**-4.5**/-0.8 | -0.1/0.3/0.6 |
| *Level 5* | 0.6/-0.2/0.3 | 1.9/-0.4/-0.1 | 0.6/0.7/1 | -0.4/**-1.7**/-0.6 |
| *Level 5-* | 1.5/**-13**/**-4.2** | 1.7/-0.7/0 | 1.4/**-2.8**/**-1.4** | 0.3/**-9.9**/**-1.7** |
| *Level 6* | 0.7/-0.2/0.7 | 1/**-1.3**/-0.5 | -0.1/**-2.7**/0 | 0.1/**-5.7**/0.1 |
| **Luria Graphic Series** |  |  |  |  |
| Score /32 | **15** | 22 | - | - |

\* performed respectively 6 (T1) and 18 (T2) months after stroke

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