# Depression Modulates Attentional Processing After Stroke

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**Abstract.** Depression is a common sequela after stroke and has severe implications on a patient's life. Post-stroke depression has been linked to cognitive impairment, but the mechanisms that lead to this deficit are not well understood. We tested 18 chronic stroke patients with depression in a psychophysical task to evaluate their attentional processing under varying cognitive loads. We found that the level of depression had no effect on the unconscious, bottom-up components of attentional processing but did influence the top-down ones. These results support the notion that depression might act like an additional cognitive load, impeding the conscious processes and responses although the information has been unconsciously processed.

## 1 Introduction

Stroke and depression co-occur frequently [1], with detrimental effects on quality of life. Post-stroke depression is linked to severity of impairment and poorer recovery results [2, 3], and affects roughly 31% of stroke patients [1]. As stroke becomes more prevalent [4], the detection and treatment of depression become important factors in therapy, especially for cognitive rehabilitation. The reason is that as a psychological disorder its strongest impact would be felt in the cognitive functioning of the patient [5], not on the motor impairment. Considering the known links between post-stroke depression and cognitive deficits [6], depression would likely hamper rehabilitation of cognitively impaired stroke patients. The dynamic of that interaction, however, remains an open question. A way to investigate cognitive dynamics is through psychophysical tasks, such as the validation gate (VG) task used here [7]. It is generally accepted that

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attention occurs through the interaction of two processes [8]—a bottom-up one, driven by the intrinsic characteristics of stimuli, and a top-down one, predicted by higher-order cognitive functions that include working memory (WM). Under certain conditions, they can be dissociated even in healthy subjects [7], showing that subjects have less cognitive resources to attend to a primary task when a secondary task increasingly demands WM capacity and reduces performance in behaviors that involve WM-dependent processing. However, bottom-up processes, being WM-independent, remain unaltered. Studies have shown that depression significantly influences WM [5] and processing speed [9], both of which are crucial in cognitive and conscious processing. Therefore, we hypothesized that depression might affect WM similarly to a cognitive load (CL) in the VG task. Cognitively impaired patients with depression performed the task, allowing us to analyze how depression severity influences cognitive processing alongside cognitive deficits. These results are the first in a larger study that investigates cognitive rehabilitation for stroke patients.

## 2 Methods

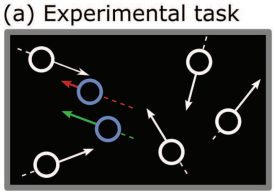
### 2.1 Patients

These preliminary results include data from 18 chronic stroke patients (8 female, 64.5 (7.2) years old, 1003.3 (923) days post-stroke, mean (SD)), recruited from Hospital de l'Esperança, Barcelona. All patients gave written consent before participation. The inclusion criteria were: (a) Cognitive impairment (Montreal Cognitive Assessment, MoCA < 26) due to a first-ever stroke over six months ago, and (b) aged between 45 and 75 years old. Patients specifically presenting hemianopia, spasticity, severe cognitive, physical or perceptual impairments that interfere with the execution of the experiment were excluded. This study was approved by the local Ethical Committee and registered at ClinicalTrials.gov (NCT02816008).

### 2.2 Set-up and Experimental Protocol

The patients performed one session of the VG task [7] while their eye movements were recorded (Tobii T120 eye tracker, Tobii Technology AB, Stockholm, Sweden). They were seated in front of a desktop computer (Sony Vaio All-in-One PC). The patients observed white circles moving linearly over a dark background (Fig. 1a). Every 2.5–4 s, one of the circles was displaced to a random position within a radius of 40 pixels from the previous location. After the displacement, it continued in its original trajectory. The patients reported detected displacements by pressing the spacebar on a keyboard. Concurrently, they were required to perform the Auditory Span Task (AST) [10] in the last 2 of 4 conditions: (1) 1 circle only (no CL), (2) 6 circles on-screen (low CL) (3) 6 circles on-screen while listening to short sentences (medium CL), and (4) 6 circles on-screen while listening to long sentences (high CL). Each condition is presented twice during the experiment pseudo-randomly, totaling in 8 blocks (Fig. 1b). The patients performed 2 training blocks at the start, one with and one without the AST.

(a) Experimental task



(b) Protocol

| Training | | Evaluation (2x) | | | |
|---|---|---|---|---|---|
| | | No CL | No CL | No CL | No CL |
| Fixed | | Fixed | Randomized | | |
| 5 trials | 5 trials | 30 trials | 30 trials | 30 trials | 30 trials |
| 4 circles | 4 circles LS | 1 circle | 6 circles | 6 circles SS | 6 circles LS |

**Fig. 1.** Task and protocol. (a) Circles move on a linear trajectory over a black screen. When hitting the edge of the screen, the circles "bounce" off and continue moving. Occasionally one of the circles (indicated in blue) jumps to a random location and continues moving (red arrow) in its original direction (green arrow). (b) The protocol consists of a training block and 2 evaluation blocks on which the analysis is based. LS: long sentences, SS: short sentences, Med: medium.

### 2.3 Outcome measures and analysis

At the start of the session, a neuropsychologist evaluated depression severity with the 17-item Hamilton Depression Scale and cognitive deficit with the MoCA. VG outcome variables are the proportion of correctly detected jumps by key presses (i.e. conscious detections) and by saccades (i.e. subconscious detentions). Correct key presses had to occur between 200 ms after the displacement to 3 s before the next displacement. For the saccade-detected displacements, following typical procedures with such data, the raw eye tracking data was first cleaned and further processed by interpolating missing values for both eyes. The interpolated data was passed through a low-pass filter (Butterworth) before angular displacement and angular velocity were calculated. A data point was labeled as a saccade when the angular velocity exceeded 30°/s, had a duration greater than 75 ms and an angular distance of more than 0.5°. We extracted the first valid saccade within 100 ms to 800 ms after a displacement. The proportion of key presses and saccades that were valid were averaged over all patients per condition. The resulting mean values for conscious and subconscious detections were correlated with the reversed Hamilton score using the Spearman's correlation coefficient. All data processing and analysis were performed using MATLAB 2017b.

## 3 Results

Correlation analysis revealed a significant positive relationship between depression levels and the ability to report stimuli for low, middle and high CL (Table 1). However, there is no correlation between depression severity and saccadic eye movements in either CL condition. This means, that while the stimuli are processed subconsciously equally well by depressed and non-depressed patients, the conscious detection and reporting is affected.

**Table 1.** Correlations with depression severity

| N = 18 | No CL | Low CL | Medium CL | High CL |
|---|---|---|---|---|
| Conscious detection | $r_s$ = .24, ns | $r_s$ = .57, $p < .05$ | $r_s$ = .65, $p < .01$ | $r_s$ = .60, $p < .01$ |
| Subconscious detection | $r_s$ = −.07, ns | $r_s$ =−.26, ns | $r_s$ = .13, ns | $r_s$ = .17, ns |

$r_s$ = Spearman's correlation coefficient

## 4 Discussion and Conclusion

We found that patients' bottom-up processing in a cognitive task was unaffected although performance for top-down related behaviors dropped with increasing levels of depression. It supports the idea that depression affects attentional processes similarly to a CL, burdening WM and thus the executive functioning of the patient. This implies several things. Firstly, depression should be considered for the evaluation and the treatment of cognitive function after stroke. Secondly, that cognitive diagnostics may be currently misinterpreted—if depression acts as a WM burden, compromising conscious processing, poor scores in neuropsychological tests might reflect a combination of cognitive deficits and post-stroke depression instead of cognitive deficits alone. In the future, we will not only study the causal directionality of this relationship, but also if cognitive rehabilitation modulates depression and whether this change augments the recovery of cognitive abilities.

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