Evaluation of Visual Information Processing Speed in Depressed People

*Mohammad KHANAHMADI1, Maryam MALMIR2, Hosein ESKANDARI1, Tahereh ORANG3

1. Dept. of Clinical Psychology, Allame Tabataba’ee University, Tehran, Iran
2. Dept. of Exceptional Children Psychology, Science and Research Branch, Islamic Azad University, Tehran, Iran
3. Dept. of Psychology, Tarbiat Modares University, Tehran, Iran

*Corresponding Author: Email: m.khanahmadi80@gmail.com
(Received 07 July 2013; accepted 19 Sep 2013)

Abstract
Background: Depression disorders are associated with serious dysfunction and depressive symptoms. Cognitive slowing is a clear symptom observed in all depressed people. PVSAT is a measure of cognitive function that specifically assesses visual information processing speed and flexibility, as well as calculation ability. The aim of the present study was to determine whether the Paced Visual Serial Addition Test (PVSAT) might constitute a useful procedure for assessing cognitive functions in depressive disorder.
Method: Twenty–eight depressed patients, together with fourteen healthy control (HC) subjects participated in the study. All participants performed the PVSAT and a set of clinical tasks assessing information processing speed, working memory and executive functions.
Results: Compared with the HCs, the depressed patients were significantly impaired in their performance of the PVSAT. Significant impairment (compared with controls) was also evidenced by only one of the clinical tasks – the symbol coding task, which assesses information processing speed.
Conclusion: Our results demonstrate the high sensitivity of the PVSAT to cognitive impairment. However, correlation analyses showed that the main factor explaining the depressed patients PVSAT impairment was cognitive slowing.

Keywords: Paced visual serial addition test, Depression disorder, Information processing speed

Introduction

Findings from research examining the cognitive slowed in patients diagnosed with MD are so general. Based on reductionism, we studied information processing speed as a sign of cognitive function in depressed (treated/untreated) patients. Depression is a heterogeneous disorder and may be defined in different ways (1). In this study, we used depression diagnoses based on the criteria established in the Fourth Edition Text Revision of the Diagnostic and Statistical Manual of Mental Disorders (2).

Depression disorders are associated with serious dysfunction and depressive symptoms (3). Experimental research has shown that memory, learning, attention, motor function and problem solving may be affected in depressed patients (4, 5). Cognitive processing speed appears to be impaired in depression (6) and may contribute to neuropsychological impairment associated with unipolar major depression (7, 8). Patients suffering depression often report the subjective experience of a slowing in cognitive speed (9, 10).
Time plays a fundamental role in all perceptual processes (11). Some researchers have suggested that slowed information processing speed (IPS) was one characteristic of the cognitive impairment (12-16). However, others suggested that rather than being a separate deficit, slowed IPS underlies all other cognitive impairments (17, 18). IPS is reported to correlate with verbal fluency (19, 20). Similar to the findings with IPS and working memory, it has been suggested IPS is predictive of performance in executive function tasks that require effortful processing (20), and that IPS slowing is more evident with complex timed tasks.

Most measures of IPS rely on reaction time as the dependent variable. Measures of reaction time can often be confounded by changes to motor speed. Reaction time related somewhat to motor speed and accuracy.

Since information processing speed is impaired in depressed individuals, the use of paced visual serial addition test (PVSAT) could represent the difference and density of IPS in depressed and healthy individuals. PVSAT specifically assesses visual information processing speed and flexibility, as well as calculation ability (22-23). Sustained attention, working memory and executive functions appear to be involved, in addition to information processing speed (23). PVSAT is applied for both clinical and research purposes. This test had originally been developed for assessing the change of efficiency of brain during the recovery period of head injuries. Moreover, the test is used with various clinical diseases such as brain injuries (24), multiple sclerosis (25), and tuberculosis (26).

The aim of the present study was to determine whether the computerized PVSAT might constitute a useful procedure for assessing slowed IPS in depressed patients for both clinical and research purposes. Moreover, is there any significant difference between treated patients, untreated patients and control groups in cognitive performance?

Materials and Methods

This is a semi-experimental research that completed in 2011. Forty untreated depressed patients (20 females, 20 males) and forty treated depressed patients (20 females, 20 males) participated in the study. Depressed patients (treated and untreated) were selected as in access sample in Rahyab Psychological Clinic. Most of the patients advised from Ruzbeh Hospital to receive free services. Depression was defined according to the criteria of the DSM-IV-TR (4). Participants were required to show a Beck Depression Inventory (BDI) score higher than 15 (the recommended cut-off for major depression). Untreated patients were early in the clinic and had not received any medication, whereas treated patients receiving antidepressant medication and presented a stable clinical mood. Forty HC subjects (20 females and 20 males, chosen to match the patient group as closely as possible with respect to age and education) also participated in the study. They had no history of neurological or psychiatric illness, the BDI score lower than 7, and their family history was negative for depression disorder.

Visual acuity was screened using a Snellen chart in all participants. All participants had normal visual acuity. All participants were aware of the goal of research and took part in research consciously.

The characteristics of the patients and control subjects are shown in Table 1.

Table 1: Demographical characteristics of the participant groups

|                | Untreated patients | Treated patients | Healthy controls |
|----------------|--------------------|-----------------|------------------|
| **Number**     | 40                 | 40              | 40               |
| **Male/Female**| 20/20              | 20/20           | 20/20            |
| **Age (yr)**   | 35(6)              | 37(5)           | 34(6)            |
| **Years of Education** | 15(2)           | 15(2)           | 14(2)            |
| **BDI (13)**   | 19 (2)             | 13(5)           | 5(2)             |
| **Disease years** | 2(1)              | 4(2)            | 0                |
The Paced Visual Serial Addition Test (PVSAT)
The Paced Auditory Serial Addition Test (PASAT) (27) is a measure of attention and information processing speed sensitive to Mild Traumatic Brain Injury (MTBI). This study examines a computerized, simpler and less aversive visual analog of the PASAT (the Paced Visual Serial Addition Test); studies indicated that the PVSAT is correlated with and less difficult than the PASAT (28).

The test was designed according to the procedure described by Gronwall (27). Series of 61 numbers from 1 to 9 were randomly delivered at presentation rates of one number every 2.8 or 1.6 sec. Each series was preceded by a practice list of 10 numbers delivered at the same presentation rate. Each number from 1 to 9 was first stored as a visual file whose duration was .5 sec. Thereafter, two different series of 61 numbers were pseudo-randomly constituted. The presentation was controlled by software especially developed for the study.

Participants were instructed to add each number to the one immediately preceding it: the second had to be added to the first, the third to the second, and so on. Performance was assessed in terms of the percentage of correct additions. We record data by assistant researcher to minimize inaccuracy and motor skills. Also, subjects are instructed to focus on accuracy and answer as soon as they can.

Studies have reported that the reliability of PVSAT to be more than 0.90 (29) and internal consistency and test-retest reliability between .97 and .93(30). The reliability of the test, estimated through test-retest method, turned out to be .91(31).

The participants completed a set of clinical tasks (assessing processing speed, working memory and executive function) during the same session as the PVSAT task.

The Beck Depression Inventory (BDI)
The Beck Depression Inventory (BDI) is a 13-item (short form) test presented in multiple choice format which purports to measure present and degree of depression in adolescents and adults. Each of the 13-item of the BDI attempts to assess a specific symptom or attitude "which appears to be specific to depressed patients, and which are consistent with descriptions of the depression contained in the psychiatric literature. Internal consistency for the BDI ranges from 0.73 to 0.92 (32). The BDI demonstrates high internal consistency, with alpha coefficients of .86 and .81 for psychiatric and non-psychiatric population, respectively (33).

Symbol Coding
In this task, nine nonsense symbols were randomly presented in rows. A key was printed above these rows, showing each nonsense symbol paired with a number. The participant’s task was to say (as quickly as possible and without error) the number corresponding to each symbol. The task lasted 90 sec and performance was assessed in terms of the number of symbols correctly coded.

Digit Span
The WAIS-R digit span subtest yielded the participant’s forward and backward digit spans.

Digit Ordering Task
In this task, digit sequences were orally presented to the participants. The size of the sequences was adjusted to each participant’s forward span. They were instructed to repeat the sequence after ordering the digits in ascending order. The task comprised ten trials. Performance was assessed in terms of the percentage of correct responses.

Stroop Word - Colour Task
A 50-item version of the test was applied. The procedure (which has been fully described elsewhere (34)) comprised two trials – a baseline condition and an interference condition. Performance was assessed in terms of the time in seconds needed to complete each phase, together with an interference cost index.

Letter and Number Sequencing Task
This task (consisting of an oral version of the Trail Making Test) has been described fully elsewhere.
Performance was evaluated in terms of an alternation cost index.

**Crossed Tapping Test**
This test was designed by Godefroy et al. (1992) (36). Participants were given a stick and were instructed to listen to a sound recording. When they heard a single, brief sound, they had to tap twice on the table with the stick; when they heard two, consecutive, brief sounds, they had to tap once. Ten practice trials were run before starting the actual task, which comprised 40 trials. Performance was assessed in terms of the number of errors.

**Results**

**PVSAT**
Mean (SD) percentages of correct responses at each presentation rate are presented in Table 2. The ANCOVA revealed a significant main effect of presentation rate \[ F (1, 38) = 29.72, P < .001 \] and group \[ F (2, 38) = 8.34, P = .001 \]. There was a trend toward a significant group x presentation rate interaction \[ F (2, 38) = 3.03, P = .06 \]. Performance was always better at a low presentation rate. Post-hoc analyses revealed that treated depressed patients performed significantly worse than HCs \( (P = .02) \) with the 2.8 sec interstimulus interval. For untreated depressed patients, there was a trend toward performing worse than HCs \( (P = .058) \). There was no difference between the two patient groups \( (P = .87) \). With the 1.6 sec interval, both treated \( (P = .005) \) and untreated \( (P = .001) \) depressed patients performed significantly worse than HCs, and there was no difference between the two patient groups \( (P = .86) \).

**Clinical Tasks**
Performance (mean (SD)) is presented in Table 3. The MANCOVA yielded a trend toward a significant effect of group \[ F (18, 60) = 2.16, P = .014 \] on performance in the clinical tasks.

### Table 2: Percentage of correct additions in the PVSAT (mean (SD))

|                  | Untreated patients | Treated patients | Healthy controls |
|------------------|--------------------|------------------|------------------|
| **2.8 sec interval** |                    |                  |                  |
|                  | 78.81(17.49)       | 75.51(20.02)     | 93.44(8.74)      |
| **1.6 sec interval** |                    |                  |                  |
|                  | 57.17(17.21)       | 60.77(20.28)     | 83.11(13.05)     |

### Table 3: Performance scores (mean (SD)) for the clinical tasks

|                  | Untreated patients | Treated patients | Healthy controls | F(3,41) |
|------------------|--------------------|------------------|------------------|---------|
| **Symbol coding** |                    |                  |                  |         |
| Symbol correctly coded | 44.10(6.50)  | 43.08(8.92)      | 55.1(8.3)        | 6.55*   |
| Forward digit span   | 5.36(8.66)        | 5.92(1.15)      | 6.10(1.64)       | 1.33    |
| Backward digit span   | 3.78(0.98)        | 3.04(1)         | 3.70(1.33)       | 1.48    |
| **Digit ordering**   |                    |                  |                  |         |
| Correct responses   | 90.90(12.60)      | 83.92(13.30)     | 87.32(9.83)      | 2.17    |
| **Stroop word color test** |                |                  |                  |         |
| Time to complete phase I(sec) | 35.54(7.72) | 32.52(2.89)     | 31.60(5.65)      | 1.92    |
| Interference cost index | 26.18(16.72) | 20.10(6.80)     | 18.5(5.9)        | 1.33    |
| **Letter number sequencing task** |            |                  |                  |         |
| Alternation cost index | 20.26(9.52)  | 21.62(10.34)    | 15.47(4.66)      | 2.40    |
| **Crossed tapping test errors** | 1.36(5.24) | 2.53(2.78)      | .68(1.36)        | 2.14    |

*P<.01
Further analyses revealed a significant main effect of group on one parameter only: the number of symbols correctly coded in the symbol coding task \[F (3, 41) = 6.55, P = .001\]. There was no significant group effect on the other parameters (see Table 3). Post-hoc analyses revealed that both treated \((P = .001)\) and untreated \((P = .002)\) depressed patients performed significantly worse than HCs in the symbol coding task. There was no significant difference between the two patient groups \((P = .95)\).

**Correlation Analyses**

Since our analysis had not revealed any significant differences between the two depressed patient groups, correlations were calculated for the patient group as a whole (Table 4).

| Table 4: Correlation between performance in the PVSAT and in the clinical tasks (Spearman coefficients) |
|--------------------------------------------------|
| **Healthy controls** | **Depressed patients** |
|----------------------|-----------------------|
| **PVSAT 2.8** | **PVSAT 1.6** | **PVSAT 2.8** | **PVSAT 1.6** |
| Symbol coding | | | |
| Symbol correctly coded | .608* | .565* | .635** | .694** |
| Forward digit span | -.164 | .455 | .316 | .484* |
| Backward digit span | .232 | .156 | .098 | .372 |
| Digit ordering | | | |
| Correct responses | .505 | .380 | .281 | .160 |
| Stroop word color test | | | |
| Time to complete phase | -.593* | -.637* | -.821** | -.625** |
| I(sec) | -.373 | .027 | -.092 | -.145 |
| Interference cost index | | | |
| Letter number sequencing task | | | |
| Alternation cost index | .016 | -.123 | -.476* | -.578** |
| Crossed tapping test errors | -.079 | .118 | -.523** | -.349 |

*\(P<.05\), **\(P<.01\)

PVSAT performance by either patients or HCs did not correlate with the backward digit span, the percentage of correct responses in the digit ordering task or the interference index in the Stroop word-color test. In HCs, PVSAT performance merely tended to correlate with the time to complete the baseline condition of the Stroop word-colour test and the processing speed in the symbol coding task. In the patient group, PVSAT performance significantly correlated not only with these two latter parameters but also with the alternating cost index in the letter/number sequencing task and the number of errors in the crossed tapping task. Furthermore, there was a trend toward a significant correlation with the forward digit span. This suggests thus that PVSAT performance in depressed patients is related to both general information processing speed (as in HCs) and set-shifting abilities. However, this latter assumption appears difficult to justify, since our depressed patients did not show deficits in any of the tasks assessing executive function in general and set-shifting in particular (see Table 3). Since the only difference between both groups concerned the symbol coding task, it is possible that the correlation between PVSAT performance and the parameters assessing set-shifting abilities is due to cognitive slowing in depressed patients. Partial correlations (with processing speed in the symbol coding task held constant) were thus performed (Table 5). This analysis showed that PVSAT performance was correlated with the time to complete the baseline condition of the Stroop word-colour test but not with any of the other parameters.
Discussion

The present results demonstrated that speed of information processing, as measured by PVSAT, is impaired in untreated and treated major depressed patients and indicated the PVSAT’s high sensitivity to cognitive impairment. Indeed, with patients early in the course of depression, impairment in PVSAT performance was evident – even at a low presentation rate. In fact, the pattern of correlations between performance in the PVSAT and in the other clinical tasks reveals that PVSAT performance could mainly be explained by information processing speed: after controlling for the influence of this factor, no correlation with the other measures of cognitive function was observed. Moreover, besides the PVSAT, only symbol coding (a widely accepted measure of information processing speed) was significantly impaired in depressed patients. This finding agrees with the assumption of Gronwall (1977) (27) that PVSAT performance generally assesses rate of information processing, and thus suggests that the depressed patients’ decline in PVSAT performance is merely an index of their cognitive slowing. Consequently, despite its high sensitivity to deficit, the PVSAT also seems to provide more information than other tasks. Since it is easy to administer and does not require motor participation. It seems more suitable than other tasks for assessing cognitive slowing early in the course of depression. Alternatively, since depression and cognitive slowing are related together (37), it has been suggested that cognitive and executive slowing seen in those with major depression disorder could well be explained by general slowing of IPS.

Conclusion

The PVSAT does enable the assessment of specific cognitive dysfunction in depression. The depressed patients’ PVSAT impairment appears to be a consequence of their cognitive slowing. This slowing occurs in the course of the disease and is insensitive to antidepressant treatment, since no differences between treated and untreated patients were observed.

In clinical practice, the PVSAT is not used with depressed patients. Its use has been proposed by some research teams for the evaluation of attention, working memory and processing speed.

Ethical consideration

Ethical issues (Including Plagiarism, Informed consent, misconduct, data fabrication and/or fal-
sification, double publication and/ or submission, redundancy, etc) have been completely observed by the authors.

Acknowledgement

No fund was received for this study. The authors declare that there is no conflict of interest.

References

1. Airaksinen E (2006). Cognitive functions in depression and anxiety disorders. 3rd ed. Karolinska University Press, Karolina, pp. 28-43.
2. American Psychiatric Association (2000). Diagnostic and statistical manual of mental disorders. 4th ed. APA press, Washington DC, pp. 12-33.
3. Horowitz JL (2006). Preventing depression adolescents, A prospective trial of two universal prevention programs. Nashville, Tennessee.
4. Austin MP, RossM, Murray C, O’Carroll RE (1999). Cognitive function in depression: A distinct pattern of frontal impairment in melancholia. Psy Med, 29: 73-85.
5. Merens W, Booji L, Van der does AJW (2013). Residual cognitive impairments in remitted depressed patients. Dep & Anx. In press.
6. Caligiuri MP, Ellwanger J (2000). Motor and cognitive aspects of motor retardation in depression. Journal of Affective Disorders, 57: 83-93.
7. Brebion G, Amador X, Smith M, Malaspina D, Sharif Z, Gorman J (2000). Depression psychomotor retardation, negative symptoms, and memory in schizophrenia. Neuropsychiatry Neuropsychological Behavior, 13: 177–183.
8. Fann J, Uomoto J, Katon W (2001). Cognitive improvement with treatment of depression following mild traumatic brain injury. Psychosomatics, 42: 48-54.
9. O’Connor DW, Pollitt PA, Roth M, Roth M, Brook PB, Reiss BB (1990). Memory complaints and impairment in normal, depressed, and demented elderly persons identified in a community survey. Arch Gen Psychiatry, 47: 224-227.
10. Tsortos G, Thompson JD, Stough C (2002). Evidence of an early information processing speed deficit in unipolar major depression. Psychol Med, 32: 259–265.
11. Dorothea A, Poggel D, seasburger S (2004). Visual perception in space and time-mapping the visual field of temporal resolution. Acta Neurol, 64: 427-437.
12. Archibald CJ, Wei X, Scott JN, Wallace CJ, Zhang Y, Metz LM, et al. (2004). Posterior fossa lesion volume and slowed information processing in multiple sclerosis. Brain, 127: 1526-1534.
13. Mahler ME, Benson DF (1990). Cognitive dysfunction in multiple sclerosis: A sub cortical dementia. In: S.M. Rao (Ed). Neurobehavioral Aspects of Multiple Sclerosis, 6: 88-101.
14. Randolph JJ, Wishart HA, Saykin AJ, McDonald BC, Schuschi KR, MacDonald J W, et al. (2005). FLAIR lesion volume in MS: related to processing speed and verbal memory. J Int Neuropsychol Soc, 11: 205-209.
15. Rao S, Leo G, Haughton V, Aubin-Faubert P, Bernardin L (1989). Correlation of magnetic resonance imaging with neuropsychological testing in multiple sclerosis. Neurology, 39:161–166.
16. White RF, Nynhuis DS, Sax DS (1992). Multiple sclerosis clinical syndromes in adult neuropsychology: The practitioner’s hand book. Elsevier science publishers, Amsterdam, pp. 177-212
17. De Sonneville LMJ , Boringa JB , Reuling IEW , Lazeron RHC , Adèr HJ , Polman CH. (2002). Information processing characteristics in subtypes of multiple sclerosis. Neuropsychologia, 40: 1751-1765.
18. Mohr DC, Cox D (2001). Multiple sclerosis: Empirical literature for the clinical health psychologist, J Clin Psychol, 57: 479-499.
19. Berker- Collo SL (2006). Quality of life in multiple sclerosis: Does information processing speed have an independent effect? Arch of Clin Neuropsych, 21: 167-174.
20. Diamond BJ, Johanson SK, Kaufman M, Graves L (2008). Relationship between processing, Depression, Fatigue and Cognitive in multiple sclerosis. Arch of Clin Neuropsych, 23(2): 189-199.
21. Hamidi A, Roohi A (2012). Manufacture and Validation of new negative priming measurement for studying individual differences in working memory. EJOP, 8(2): 251-262.
22. Diehr MC, Heaton RK, Miller W, Grant I (1998). The paced auditory serial addition task (PASAT): Norma for age, education, and ethnicity. Assessment, 5(3): 375 – 387.
23. Rao SM, Leo GJ, Bernadin L, Underzagt F (1991). Cognitive dysfunction in multiple sclerosis: frequency, patterns and prediction. *Neurology*, 41: 685 – 691.

24. Brooks J, Fos LA, Greve KW, Hammond JS (1999). Assessment of executive function in patients with mild traumatic brain injury. *The Journal of Trauma*, 46: 159-163.

25. DeLuca J, Chelune GJ, Tulsky DS, Legenfelder J, Chiaravalloti ND (2004). Is speed of processing or working memory the primary information processing deficit in multiple sclerosis? *J Clin Exp Neuropsych*, 26(4): 550-562.

26. Shucard JL, Parrish J, Shucard DW, McCabe DC, Benedict RHB, Ambrus J (2004). Working memory and processing speed deficits in systemic lupus erythematosus as measured by the paced auditory serial addition test. *J Int Neuropsycho Soc*, 10: 35-45.

27. Gronwall DM (1977). Paced auditory serial addition task: a measure of recovery from concussion. *Percept Motor Skills*, 44: 367 – 373.

28. Fos AL, Greve WK, South BM, Mathias CBH (2000). Paced visual serial addition test. *App Neuropsych*, 7(3): 140-146.

29. Egan V (1988). PASAT: Observed correlations with IQ. *Personality and Individual Differences*, 9: 179-180.

30. McCaffrey RJ, Cousins JP, Westervelt HJ, Martynowicz M, Remick SC, Szbenyi S, et al (1995). Practice effects with the NIMH AIDS abbreviated neuropsychological battery. *Arch Clin Neuropsych*, 10: 241-250.

31. Hamidi F, Noorafkan N R (2011). Manufacture and validation of Paced Visual Serial Addition Test (PVSAT) for an Iranian population. *Procedia Computer Science*, 3: 138-143.

32. Groth-Marnat G (1990). *The handbook of psychological assessment*. 2nd ed. John Wiley and sons, New York.

33. Beck AT, Steer RA, Garbin MG (1988). Psychometric properties of the Beck Depression Inventory: Twenty five years of evaluation. *Clin Psycho Rev*, 8(1): 77-100.

34. Dujardin K, Blairy S, Defebvre L, Duhem S, Noel Y, Hess U, et al (2004). A deficit in decoding emotional facial expressions in Parkinson’s disease. *Neuropsychological*, 42: 239-250.

35. Dujardin K, Krystkowlak P, Defebvre L, Blond S, Destee A (2000). A case of severe dysexecutive syndrome consecutive to chronic bilateral pallidal stimulation. *Neuropsychological*, 38: 1305-1315.

36. Godefroy O, Rousseaux M, Leys D, Destée A, Scheltens P, Pruvo JP (1992). Frontal lobe dysfunction in unilateral lenticulostriate infarcts: Prominent role of cortical lesions. *Arch Neurol*, 49: 1285-1289.

37. Rogers D, Lees AJ, Smith E, Trimble M, Stern GM (1987). Bradyphrenia in Parkinson's disease and psychomotor retardation in depressive illness. *Brain*, 110(11): 761 – 776.