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Original article

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# Effect on post-stroke anxiety and depression of an early neuropsychological and behavioural treatment

## Summary

### Objectives

*Post-stroke depression and anxiety are common psychiatric symptoms that can interfere with the rehabilitation. This study aimed to examine the depression and anxiety levels in the sub-acute phase and at 2 months post-stroke evaluation in a sample of 50 inpatients. This study also evaluated the efficacy of an early neuropsychological and behavioral intervention on anxiety, depression and cognitive functioning. In addition the relationships between anxiety, depression and cognitive functioning were examined.*

### Methods

*A convenience sample of 50 post-stroke patients was enrolled from an inpatient rehabilitation study. They completed the Mini Mental State Examination (MMSE), the Raven's Colored Progressive Matrix (RCPM), the Verbal Memory Span and Visuospatial Memory Span, the Hamilton Rating Anxiety Scale (HAM-A), and the Hamilton Rating Scale for Depression (HAM-D) in the subacute phase (T1) and 2 months after stroke (T2). Medical information about the stroke and its characteristics was provided. The neuropsychological part of the treatment was aimed to develop compensating skills, awareness of limits and improvement of cognitive deficits. The behavioral part of the treatment was aimed to teach appropriate behaviors and prepare social reintegration. A control sample of 50 orthopaedic patients well-matched for age, gender, and level of education was enrolled. The two sample were compared for anxiety, depression, and cognitive functioning at T1 and T2.*

### Results

*Results showed significant differences between patients after stroke and control sample in anxiety and depression. These results persisted at two months after stroke evaluation. Moreover, the neuropsychological and behavioral treatment was efficacy to improve the cognitive functioning and reduce PSD and PSA.*

### Conclusions

*Findings highlight the importance of anxiety and depression in post-stroke patients in the early phases. Multidisciplinary approach are necessary for a better functional outcome.*

***Key words***

Anxiety • Cognitive impairment • Depression • Stroke • Treatment



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## Introduction

Post-stroke depression (PSD) occurs frequently in stroke rehabilitation worsening the functional outcome. Past studies have investigated the prevalence for major depression founding a rates of 19.3% among hospitalized patients and 23.3% among outpatient samples [1]. PSD is a relevant complication of stroke because is associated with greater disability as well as increased mortality [2]. Cognitive disorder is another factor associated with PSD at follow-up [3]. However the relation between depression and cognitive impairment is still unclear [4]. Results of a recent study have shown that the

severity of depressive symptoms assessed in the acute phase or 4 years after stroke is not a predictor of the length of survival at 18-year follow-up [5]. In addition, PSD and others mood disorder post-stroke growth in prevalence over the initial weeks post-stroke, suggesting their dynamic nature in the early stages [6]. It could reasonable to hypothesize that PSD is a multi-factorial and complex phenomenon. A more deeply knowledge of the mechanism of PSD may lead to tailored treatments. Hence, several studies have examined the underlying mechanisms with often contradictory results. Literature regarding therapy did not product reliable results and only a minority of patients are properly treated [7].

PSD has been identified in the clinical practice for more than 100 years and the most of past studies have focused on depressive symptoms. Nonetheless depression is not the only psychiatric symptom after stroke. Recently literature has begun to recognize and explored the post-stroke anxiety (PSA). The prevalence for any anxiety disorder in post-stroke patients is common with a rate of 22% and phobic disorder as the predominant diagnosis [8]. Anxiety and depression persist at 5-year follow-up in about a third of post-stroke patients [9]. However risk factors for anxiety and effects with depression remain still unclear. On one hand PSA has a negative effect on quality of life of post-stroke patients; on the other this negative effect is independent from depression [10].

Psychological interventions could help patients and clinicians to incorporate emotions into conversations and recognize decisional competences [11]. The relationship between clinicians, patients and their caregivers are often characterized by difficulties to recognize negative emotions [12]. Findings from other fields of research pointed out that the presence of a clinical psychologist as consultant can improve diagnostic and therapeutic practices [13]. Regarding non-pharmacological treatment, evidences from the literature have highlighted the efficacy of relaxation training to reduce anxiety after stroke [14]. Nonetheless there are few studies that have examine the efficacy of psychotherapy and pharmacotherapy treatments for PSA. A recent study has evaluated the clinical efficacy of a neuropsychological intervention for post-stroke patients and their relationship to cognitive functioning in the early stages after stroke [15]. Results pointed out the correlation between PSA and PSD. In addition the neuropsychological intervention was efficacy on anxiety but not on depression at 2 months post-stroke evaluation.

The first aim of this study was to examine the prevalence of PSA and PSD in a sample of patients following a stroke in the subacute phase and two months from the onset. The second aim of this study was to evaluate the efficacy of two months of early neuropsychological rehabilitation on anxiety, depression and cognitive functioning.

## Materials and methods

### Study design and participants

Participants with a primary diagnosis of stroke and admitted to the "Villa Sofia" Rehabilitation Institute of Acireale, Italy, were enrolled in this study. In addition, a control sample of orthopaedic patients admitted to the same rehabilitation unit for two months of treatment were enrolled. Participants of this study were selected to achieve group-wise matching on average chronological age, level of education, and balanced gender percentage. Inclusion criteria for stroke patients were first-ever stroke; sufficient comprehension assessed with neuropsychological tests; able to be assessed. Inclusion criteria for orthopaedic patients were no stroke in anamnesis; sufficient comprehension assessed with neuropsychological tests; able to be assessed. Exclusion criteria both for stroke sample and orthopaedic sample were a positive anamnesis for any psychiatric disorder included in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [16].

Ethical approval was obtained from the Ethics Committee of the hospital. Patients were included in this study from January 2018 to June 2018. An expert neuropsychologist explained that consent was voluntary and that participation in this study did not influence the rehabilitation program. Finally, informed written consent was obtained was obtained from participants.

At the time they were first approached, participants were about 10 days post-stroke. Patients were evaluated in the sub-acute phase and at 2 months post-stroke. This period was chosen because cognitive functioning enhanced significantly between 2 and 6 months. The baseline evaluation was made before the beginning of the neuropsychological intervention. Neuropsychological assessment was conducted according to standardized procedures. Clinical and radiological parameters were obtained from medical records. The assessment was administered by an expert neuropsychologist and took about 120 minutes to complete.

In total, 109 patients were contacted in this study. Fifty-five patients were admitted at the rehabilitation center with a primary diagnosis of stroke. Fifty-four patients were admitted with an orthopaedic diagnosis. Regarding post-stroke sample, 5 patients declined further participation in the study after the first assessment. Regarding orthopaedic sample, 4 patients declared that were not interest to participate at this study.

### Measures

The *Hamilton Rating Anxiety Scale (HAM-A)* [17] was used to assess anxiety symptoms during hospital stays. The HAM-A is administered by an interviewer who asks a semi-structured series of questions correlated to symptoms of anxiety. Seven of the items specifically assess

psychic anxiety and the others seven assess somatic anxiety. The interviewer rates the individual on a 5 point scale for each of the 14 items ranging from 0 (not present) to 4 (extreme symptoms). The interview and scoring takes about 15 minutes. Higher scores indicate high levels of anxiety.

The *Hamilton Rating Scale for Depression* (HAM-D) [18-19] was used to rate severity of depression. The HAM-D consists of 21 items and it is based on the clinician's interview with the patients and probes symptoms such as depressed mood, feelings of guilt, thoughts of suicide, sleep disturbances, and weight loss. The rater enters a number for each symptom that ranges from 0 (not present) to 4 (extreme symptoms). The interview and scoring takes about 15 minutes.

The *Mini-Mental State Examination* (MMSE) [20-21] was used to measure cognitive status. The *Raven's Colored Progressive Matrices task* (RCPM) [22-23] was used to assess abstract and logical reasoning. The RCPM consists of 36 items (three sets of 12) and involves completing a pattern or figure with a part missing by choosing the correct missing piece from among six alternatives. Patterns are arranged in order of increasing difficulty.

The *Verbal Span* and the *Visuospatial Span* (Corsi's block-tapping test, verbal span) [24] were used to assess the short-term memory. Specifically, the Verbal Span subtest is a short-term auditory memory task which requires the subject to repeat increasingly longer series of words. The series range in length from two to nine words. Two trials are presented for each series, and the subtest is discontinued after failure on both trials for a series. The Corsi block-tapping test was used to assess visuospatial memory and working memory. The examiner taps the blocks starting with sequences of two cubes. The subject has to reproduce a given sequence by tapping the blocks in the same sequence he see. If two sequences are correctly reproduced the sequence length increases by one item. The procedure ends when two sequences are reproduced erroneously. The Prose Memory task [24] was used to measure the long-term verbal memory. In this test, memory for details of a prose story is tested. The subject is asked to tell the story from memory and after 15 minutes to repeat the story to assess delayed prose recall.

### Statistics

Statistical analysis was performed using IBM SPSS Statistics version 22 (IBM Corporation, Armonk, New York, USA). Descriptive statistics, such as the mean and SD of the variables, are reported below. The data showed a good range of variance and there were no univariate outliers for the variables considered.

An independent t-test was used to compare the levels of anxiety and depression in the sub-acute phase and at 2 months post-stroke with a control sample. This period was taken because cognition improved significantly between 2 and 6 months post-stroke and patients could engage thought acute care and rehabilitation [25]. The standardized mean difference effect size statistic was used to compare measures of anxiety and depression between the two samples at T1 and T2. An effect size value of 0.20 is considered small, a value of 0.50 is considered medium, and a value of 0.80 is considered large [26].

A paired t-test was used to assess the efficacy of rehabilitation intervention for stroke patients at T1 (subacute phase of stroke) and T2 (after 3 months).

The Pearson correlation coefficients were calculated between the T1 and T2 evaluations. Correlation coefficients were also calculated between the baseline evaluation and the changes over time between T2 and T1 (T2 minus T1).

In addition, the standardized mean difference was used to compare the magnitude of treatment effect for post-stroke patients.

Comparisons were computed with Student's t-test and the Bonferroni correction. Significance levels were set at $p < 0.05$ and $p < 0.01$.

### Neuropsychological and behavioural treatment

Neurological patients underwent 45 minutes of neuropsychological rehabilitation four times weekly for two months. Neuropsychological treatment was aimed at developing compensating skills, recovery of cognitive deficit, awareness of limits and behavioral interventions. All cognitive exercises were planned after a first neuropsychological evaluation, where results defined different neuropsychological profiles. We proposed table exercises and cognitive rehabilitation software to stimulate selective language disorders, attention deficit disorders, apraxia, memory problems, and executive function disorders. To modify behavioural disorders demonstrated by patients, we also proposed behavioural interventions with the aim of teaching the patients to eliminate inappropriate behaviours and providing the means to prepare social reintegration designed to achieve a satisfactory degree of independence and the recovery of relational skills. After cognitive and behavioural treatment, the patients underwent a final neuropsychological evaluation in which changes in cognitive functions and any improvements achieved were recorded.

## Results

### Demographic and clinical characteristics

Socio-demographics and medical characteristics of the samples are presented in Table I. All patients were native Italian speakers and Italian nationals. The post-

**TABLE I.** *Baseline data of post-stroke patients and orthopaedic patients.*

| | Post-stroke patients (n = 50) (mean ± S.D.) (min-max) | Orthopaedic patients (n = 50) (mean ± S.D.) (min-max) | t (df = 98)* |
|---|---|---|---|
| Age, years | 60.14 ± 14.66 | 55.04 ± 17.07 | 1.60 |
| | (21-81) | (21-81) | |
| Education, years | 10.26 ± 3.83 | 10.26 ± 4.31 | 0.00 |
| | (5-17) | (5-17) | |
| **Gender** | | | |
| Male | 33 (66%) | 28 (56%) | |
| Female | 17 (34%) | 22 (44%) | |
| **Type of cerebral lesion** | | | |
| Ischemic | 32 (64%) | | |
| Hemorrhagic | 8 (16%) | | |
| Neoplastic | 5 (10%) | | |
| Vasculopathy | 3 (6%) | | |
| Head injury | 2 (4%) | | |
| **Hemispheric side of lesion** | | | |
| Right hemisphere | 34 (68%) | | |
| Left hemisphere | 14 (28%) | | |
| Bilateral | 2 (4%) | | |
| **Location of lesion** | | | |
| Internal/pons/capsule | 13 (26%) | | |
| Frontal-parietal cortex | 10 (20%) | | |
| Frontal lobe | 9 (18%) | | |
| Temporal lobe | 5 (10%) | | |
| Fronto-temporal cortex | 4 (8%) | | |
| Parietal lobe | 4 (8%) | | |
| Cerebellum | 3 (6%) | | |
| Parieto-occipital cortex | 1 (2%) | | |
| Temporal-parietal cortex | 1 (2%) | | |

** $p < 0.05$; ** $p < 0.01$*

stroke sample consisted of fifty post-stroke patients, 66% of the post-stroke sample was male, the mean age was 60.14 (SD = 14.66), and the level of education in years was 10.26 (SD = 3.83). Of the fifty post-stroke patients, 32 (64%) had an ischemic stroke and 8 (16%) an hemorrhagic stroke. Moreover, 5 (10%) patients had a neoplastic lesion, 3 (6%) a vasculopathy lesion and 2 (4%) an head injury. With respect to side of lesion, 34 (68%) patients had a right hemisphere lesion and 14 (28%) a left hemisphere lesion. Finally, 2 (4%) patients had a bilateral lesion. Regarding the location of lesion, 13 (26%) patients had an internal/pons/capsule lesion, 10 (20%) patients a frontal-parietal lesion, 9 (18%) a frontal lobe lesion, 5 (10%) a temporal lobe lesion, 4 (8%) a fronto-temporal cortex lesion, 4 (8%) parietal lobe lesion, 3 (6%) a cerebellar lesion, 1 (2%) a parieto-occipital cortex, and 1 (2%) a temporal-parietal cortex.

The control sample consisted of fifty orthopaedic patients, 56% was male, the mean age was 55.04 (SD = 17.07), and the level of education in years was 10.26 (SD = 4.31). No differences were found between post-stroke sample and control sample for the mean age and level of education.

**TABLE II.** *Results of t-test for independent samples for post-stroke sample at subacute phase (T1) and after 2 months (T2) and control sample of HRSA and HRSD.*

| Variable | Post-stroke sample (n = 50) M (SD) | Control sample (n = 50) M (SD) | Mean difference | Std. error difference | t (df = 98) | d |
|---|---|---|---|---|---|---|
| HAM-A T1 | 22.96 (1.77) | 12.64 (3.98) | 10.32 | 0.62 | 16.76** | 3.35 |
| HAM-D T1 | 22.20 (2.67) | 7.84 (4.64) | 14.36 | 0.76 | 18.98** | 3.79 |
| HAM-A T2 | 18.20 (2.13) | 10.16 (2.57) | 8.04 | 0.47 | 17.02** | 3.40 |
| HAM-D T2 | 18.20 (2.52) | 6.88 (4.07) | 11.32 | 0.68 | 16.70** | 3.42 |

*Notes: * p < 0.05**; p < 0.01; HAM-A: Hamilton Anxiety Rating Scale; HAM-D: Hamilton Depression Rating Scale*

### Comparisons between post-stroke sample and control sample at subacute phase and after 2 months

Table II shows the mean scores and results of the t-test for two independent samples. Results showed significant differences (with Bonferroni correction for four comparisons, the new critical alpha levels was 0.01) between post-stroke during subacute phase (T1) and control samples (p = 0.001). In addition the mean effect size between the two samples was large for anxiety and depression (respectively, d = 3.35 and d = 3.79).

As shown in Table II there were significant differences for the two samples after two months with post-stroke patients scoring higher than controls (p = 0.001). Moreover the mean effect size between the two samples was large both for anxiety and depression (respectively, d = 3.40 and d = 3.42).

Hence, post-stroke patients showed higher levels of anxiety and depression than control sample both at T1 and T2.

## Efficacy of neuropsychological and behavioural treatment for post-stroke sample

Table III shows results of the paired t-test, the correlation coefficients and the effect size for post-stroke sample among the subacute phase and after two months. Results of paired t-test (with Bonferroni correction for 7 comparisons, the new critical alpha level was 0.007) showed significant differences for all the variables considered. The post-stroke patients showed higher scores on cognitive functioning after two months of treatment. On the other hand, the anxiety and depression levels decreased after the treatment.

These results were confirmed by the mean effect size between the two evaluations for post-stroke sample. The largest effect size was for anxiety (d = 2.431) and the smallest for the MMSE score (d = -0.901). However all the variables considered showed large effect size.

Moreover the results showed high correlation coefficients between the subacute phase and after two months of treatment. The higher coefficient was for the MMSE (r = 0.70; p < 0.01) and the lowest was for the Prose Memory task (r = 0.35; p < 0.01). The anxiety and depression scores showed large correlation coefficients between the subacute phase and two months after stroke (respectively, r = 0.41, p < 0.01 and r = 0.51, p < 0.01).

Table III also shows correlation coefficients between the baseline evaluation and the changes after the neuropsychological intervention. All the evaluated variables at baseline were negatively correlated with the change over the time. Specifically, the higher coefficient was for the MMSE (r = -0.71; p < 0.01) and the lowest was for the Prose Memory task (r = -0.38; p < 0.01).

## Discussion

Depression and anxiety after stroke are associated with poorer quality of life and compliance to the rehabilitation. In fact, PSD and PSA are relevant symptoms in patients after stroke that can worse the functional outcome. Past studies have deeply examined PSD and the impact on rehabilitation (Robinson, 2003). Recently some researchers have also investigated PSA but there are still not clearly evidence about treatments. The first aim of this study was to examine the PSD and PSA levels in a sample of patients following a stroke. The first evaluation was made in the subacute phase comparing post-stroke patients with a control sample of orthopaedic patients. Results have shown that post-stroke patients have higher scores for both anxiety and depression comparing with control subjects. These results were confirmed at two months evaluation with post-stroke patients scoring higher than controls. In a previous study similar results were found, even if anxiety level was higher than depression level [15]. These results can be explained by the characteristics of the two sample influencing PSD and PSA. Nonetheless, PSD and PSA represent a relevant problem that must be addressed in the rehabilitation. A limit of the past studies was to consider separately anxiety and depression. Another limit of the scientific literature regards the presence of few studies

**TABLE III.** *Results of t-test, correlations at subacute phase of stroke (T1) and after two months (T2), and T1-T2 change of neuropsychological functioning, anxiety, and depression in post-stroke sample (n = 50).*

| Variable | T1 M (SD) | T2 M (SD) | Mean difference | Std. error difference | R | R T1-T2 change | t (df = 49) | d |
|---|---|---|---|---|---|---|---|---|
| MMSE | 23.68 (4.27) | 27.00 (2.99) | -3.32 | 0.43 | 0.70** | -0.71** | -7.69** | -0.901 |
| RCPM | 21.82 (7.16) | 28.24 (5.39) | -6.42 | 0.90 | 0.52** | -0.69** | -7.15** | -1.013 |
| CORSI | 1.66 (0.69) | 2.48 (0.61) | -0.82 | 0.10 | 0.44** | -0.60** | -8.39** | -1.259 |
| VERBAL SPAN | 1.76 (0.71) | 2.46 (0.68) | -0.70 | 0.10 | 0.53** | -0.53** | -7.30** | -1.007 |
| PROSE MEMORY | 1.42 (0.54) | 2.44 (0.73) | -1.02 | 0.11 | 0.35** | -0.38** | -9.72** | -1.589 |
| HAM-A | 22.96 (1.77) | 18.20 (2.13) | 4.76 | 0.30 | 0.41** | -0.42** | 15.70** | 2.431 |
| HAM-D | 22.20 (2.67) | 18.20 (2.52) | 4.00 | 0.36 | 0.51** | -0.54** | 11.00** | 1.541 |

*Notes: * p < 0.05**; p < 0.01; MMSE: Mini-Mental State Examination; RCPM: Raven's Colored Progressive Matrix Task; CORSI: Corsi block-tapping task; VERBAL SPAN: Verbal Span subtest; PROSE MEMORY: Prose Memory task; HAM-A: Hamilton Anxiety Rating Scale; HAM-D: Hamilton Depression Rating Scale*

on the efficacy of intervention. For this reason, the second aim of this was to evaluate the efficacy of a two months early neuropsychological and behavioral intervention to decrease anxiety and depression levels and to enhance cognitive functioning. The neuropsychological part of the treatment was aimed to develop compensating skills, awareness of limits and improvement of cognitive deficits. The behavioral part of the treatment was aimed to teach appropriate behaviors and prepare social reintegration. Results showed that the early intervention was efficacy to reduce PSA and PSD at two months evaluation. Results also showed that there was a significant improvement of the cognitive deficits evaluating at subacute phase. Moreover, the baseline evaluation of the variables is negatively correlated with the changes after the neuropsychological and behavioral intervention. At this regard the role of the values at baseline to predict the outcome should be investigated by the future research. Anyway the evaluation of cognitive functioning and emotional distress is needed for each patient following a stroke. As stated there are only few guidelines available for treatment of post-stroke patients with the most of patients that are not adequately treated [7]. Results of this study suggest that early neuropsychological and behavioral treatment could be useful to improve the rehabilitation outcome. However interventions of this kind must be considered only a part of a multidisciplinary rehabilitation program for post-stroke patients. If confirmed by future research, the results of this study could have implications in clinical practice. A supportive and time-limited group intervention [27-28] could helpful to decrease negative emotions and fostering mentalisation [29] after the early stages after stroke. Moreover time auxiliary therapies should be considered to enhance efficacy of multidisciplinary intervention [30].

In addition, future research must be addressed to examine psychological factors that can influence anxiety and depression levels after stroke. Findings from other fields of research have shown that metacognition is strictly associated with anxiety and depression and that can be considered as a vulnerability factor that pre-exist emotional disorders in patients [31-33], their caregivers [34], and clinicians [35]. The results of this study may be affected by a number of limitations that should be addressed by future research. First, the sample was small and all patients were consecutively recruited in a rehabilitation center. Hence the sample may not be representative of the clinical population of post-stroke patients. Second, the sample was heterogeneous for medical variables. We did not evaluate the influence of such variables as type and location of cerebral lesion. Third, there are some additional variables whose role we did not consider, such as social and family support, personality factors. For these reasons, this study needs replication to address the role of other variables and longitudinal data to examine the nature of the relationships found in this study.

## Conflict of interest

The Authors declare to have no conflict of interest.

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