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Link between post-stroke psychopathology and scope-of-action awareness

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Abstract

Background: Epidemiological research has failed to confirm laterality of lesion site as a neurobiological source of post-stroke psychopathology. However, acquired communication disorders have proved to be a key risk factor for depression, apart from established parameters such as pre-stroke psychopathology and physical immobility.

Objectives: The present work examines a new predictor of post-stroke psychopathology: psychological flexibility. This concept describes an accepting attitude toward irreversible loss following stroke while using remaining agency.

Design: Overall, 70 individuals engaged in a cross-sectional study conducted in the subacute stage after an ischemic or hemorrhagic event, a period with elevated prevalence of mental-health problems (2 weeks to 6 months after stroke).

Methods: Outcomes included standardized self-report and clinician-rated measures of depression, anxiety disorders, and general psychopathology (Beck Depression Inventory; Hospital Anxiety and Depression Scale; ICD-10 Symptom Rating; Hamilton Depression Rating Scale) alongside lack of psychological flexibility (Acceptance and Action Questionnaire II). The study design controlled for pre-stroke psychopathology and physical immobility (Barthel Index).

Results: Partial correlation analyses revealed a significant medium-to-large association between the entire set of clinical outcomes and lack of psychological flexibility ($r \leqslant 0.62$, $p < 0.001$). In moderator analyses, the magnitude of this association did not vary significantly with diagnosis of acquired communication disorders (i.e., aphasia, apraxia of speech or dysarthria; separately or combined).

Conclusion: The current results demonstrate a substantial link between post-stroke psychopathology and psychological flexibility. This finding opens new avenues for research on depression and other mental-health problems in stroke survivors with and without acquired communication disorders.

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Plain language summary

Sense of agency may predict mental-health problems after stroke

The present work examines a possible key to mental-health problems after stroke: psychological flexibility. By definition, this term describes an accepting attitude toward irreversible loss following stroke while using remaining agency. Showing a link between psychiatric syndromes and psychological flexibility, the current results motivate new behavioral forms of treatment beyond pharmacotherapy to alleviate mental-health problems after stroke.

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Introduction

Almost one-third of stroke survivors meet official criteria of mental-health problems[1] within 5 years of an ischemic or hemorrhagic event, with syndromes ranging from depression[2] to anxiety disorders.[3] During the same period of time, an estimated 3–4 in 1000 people with post-stroke psychopathology commit suicide,[4] a rate significantly increased compared to the general population.[5] As yet, the etiology of post-stroke depression remains not fully understood.

A longstanding controversy surrounds laterality of lesion site, traditionally thought to account for post-stroke depression in the case of damage to the left rather than the right hemisphere—a view not consistently supported in meta-analyses[6] and based on data likely to be confounded by acquired communication disorders, including aphasia.[7] Indeed, epidemiological data suggest that social isolation due to aphasia raises the risk of post-stroke depression by a factor of 7.4.[8] This finding converges with the antidepressant outcome of interventions compensating for social isolation in individuals with aphasia and subclinical "low mood"[9] or above-threshold psychopathology.[10] Critically, acquired communication disorders tend to be underrepresented in epidemiological research on post-stroke psychopathology and will therefore receive closer attention below. Aside from aphasia, meta-analyses have identified history of nonvascular psychopathology and degree of physical immobility as independent predictors of depression immediately after stroke, while lack of social support appears to play a more detrimental role in later stages.[11]

The current work examines a potential predictor of post-stroke psychopathology previously established in psychiatry but little known in neurology: psychological flexibility. By definition, psychological flexibility manifests in the ability to (1) accept circumstances that are beyond deliberate control (e.g., coming to terms with loss of communication skills after stroke), and (2) actively shape life within the bounds of what is still possible (e.g., attending intensive speech-language therapy). Decades of research have shown an association between nonvascular psychopathology and reduced psychological flexibility in individuals with depression,[12] anxiety disorders,[13] eating disorders,[14] obsessive-compulsive disorders,[15] and chronic pain.[16] Drawing on this association, Acceptance and Commitment Therapy[17] aims to improve psychological flexibility to relieve mental-health problems, with evidence of efficacy from numerous clinical trials.[18]

To date, only a few studies have investigated the association between mental-health problems and psychological flexibility in neurological conditions. These studies point to a link between severity of depression and reduced psychological flexibility in individuals with mild traumatic brain injury,[19,20] spinal cord injury[21] as well as in mixed samples with stroke survivors and their relatives[22] or a wide spectrum of neurological syndromes such as hypoxia.[23] No research has so far explored the implications of reduced psychological flexibility for the pathogenesis of mental-health problems in a sample limited to stroke survivors. Moroever, no research has considered if, and to what extent, the association between post-stroke psychopathlogy and psychological flexibility varies with diagnosis of acquired communication disorders as a moderator. Common—and frequently concurrent—syndromes of acquired communication disorders after stroke are aphasia,[24] apraxia of speech,[25] and dysarthria.[26]

The present work seeks to determine the association between mental-health problems and psychological flexibility in an adequately powered sample of stroke survivors with and without acquired communication disorders. All individuals will be within the first few months after stroke where risk of mental-health problems is especially high.[27] To address independent predictors of depression immediately after stroke,[11] the study design will rule out history of nonvascular psychopathology (as per exclusion criteria) and adjust for degree of physical immobility (as a confounder in statistical analyses). Although distinct hypotheses may be premature in light of available epidemiological

evidence, as summarized above, we expect a significant positive association between post-stroke psychopathology and lack of psychological flexibility in line with previous work on nonvascular neurological conditions[19–21] or mixed samples.[22,23] We anticipate that the magnitude of this association does not differ statisticially with acquired communication disorders as a moderator, as speech and language skills do not seem to interfere with psychological flexibility in a unique way.

Methods

Study design and setting

We here present a cross-sectional study conducted in an in- and outpatient rehabilitation center in Berlin, Germany, between March and April 2023 (study acronym: PSYFLEX).

Study sample and power analysis

After routine referral to the study team, we invited possible participants to a screening session to check their eligibility. Inclusion criteria were: first-time cortical or subcortical ischemic or hemorrhagic event, as validated by MRI or CT scans before admission to the rehabilitation center; and subacute stage (2 weeks to 6 months after stroke). Exclusion criteria were: history of nonvascular mental-health problems; clinical record of concomitant severe chronic somatic disease that may challenge the interpretation of data as attributable to stroke, as was the case for one person with Morbus Wegener who did not enter the study sample; extremely impaired verbal comprehension that may prevent understanding of basic instructions, as indicated by less than 10 correct responses on the request subscale of the Aachen Aphasia Bedside Test[28]; and serious nonverbal cognitive deficits that may lead to unreliable findings, as demonstrated by performance more than two standard deviations below the norm average on the Corsi Block-Tapping Task.[29]

A total of 70 individuals participated in the present study (see Table 1). This sample size was calculated in an a-priori power analysis (one-tailed $\alpha = 0.05$; $1-\beta = 0.99$; $\rho = 0.47$; resulting $n = 63$; expected dropout rate during testing = 10%; final $n = 70$).[30] Our effect size estimate derives from published data on our primary clinical outcome for screening of depression and anxiety disorders, as specified below, in the context of acquired brain injury.[23] On average, individuals were aged 68.4 years (standard deviation: 11.8 years) and 6.7 weeks post-onset of stroke (standard deviation: 3.4 weeks). Of the entire sample, no individuals had a diagnosis of post-stroke delirium, with documented prevalence reaching its peak in the first 24 h after an ischemic

Table 1. Sample characteristics.

Parameter	*M* ± SD (range) or *n* (%)
Age	68.4 ± 11.7 (40–90)
Gender	
Female	27 (38.6%)
Male	43 (61.4%)
Years of education	13.2 ± 2.7 (8–23)
Corsi Block-Tapping Task[29]	6.4 ± 2.1 (4–12)
Barthel Index	85.7 ± 12.0
Stroke etiology (as per MRI or CT scan)	
Ischemia	65 (92.9%)
Hemorrhage	5 (7.1%)
Laterality of lesion site	
Right	35 (50.0%)
Left	35 (50.0%)
Lesion location (as per MRI or CT scan)	
Cortical	6 (8.6%)
Subcortical	24 (34.3%)
Cortical and subcortical	40 (57.1%)
Time after onset of stroke (in weeks)	6.7 ± 3.4 (2–21)
Antidepressant medication	10 (14.3%)
Neuroleptic medication	16 (22.9%)
Acquired communication disorder (at least one syndrome)	27 (38.6%)
Language disorder (aphasia)	11 (15.7%)
Speech disorder (apraxia of speech and / or dysarthria)	21 (30.0%)

Sample characteristics documented during eligibility screening or retrieved from clinical records (see Methods section).

or hemorrhagic event and declining to a very low level in the subacute stage of our testing period.[31] A subgroup of 11 individuals met the criteria of aphasia, as confirmed by the Aachen Aphasia Test.[32] Moreover, 21 individuals had apraxia of speech (difficulty translating target words into corresponding speech-motor planning units; key symptoms: slowed verbal utterances with search behavior; diagnosis in analogy to previous work[33]) or dysarthria (difficulty executing articulatory movements; key symptoms: unclear, slurred verbal utterances; diagnosis consistent with published typological classifications[34]). Overall, 27 individuals suffered from aphasia, apraxia of speech or dysarthria either as a single or combined diagnosis.

Outcomes

As co-primary clinical outcomes, we administered the simplified 20-item version of the Beck Depression Inventory (BDI)[35] and the 14-item version of the Hospital Anxiety and Depression Scale (HADS).[36] Both the BDI and HADS are self-report measures known for their robust psychometric properties, including reliability and validity, in individuals without aphasia. Additional evidence supports the construct validity of the BDI in individuals with aphasia.[10] Designed for screening of depression and anxiety disorders in the context of a nonpsychiatric hospital, the HADS accounts for the specificity of an in- and outpatient setting by omitting physical symptoms that may equally arise from somatic disease.

To complete the BDI and HADS, we used the 7-item version of the Hamilton Rating Scale for Depression (HAM-D)[37] and the 29-item version of the ICD-10 Symptom Rating as secondary clinical outcomes (ISR).[38] Both diagnostic instruments have excellent psychometric properties in individuals without aphasia. As a clinician-rated measure of depression, the HAM-D, was also successfully piloted in individuals with aphasia.[10] Assessing general post-stroke psychopathology, the ISR reflects symptoms of depression, anxiety disorders, obsessive-compulsive disorders, somatoform disorders, eating disorders and other mental-health problems. As a self-report measure, the ISR exhibits good psychometric properties in individuals without aphasia.

To quantify psychological flexibility, we employed the German version of the Acceptance and Action Questionnaire II (AAQ-II).[39] This self-report measure shows very good reliability and construct validity in individuals without aphasia, with higher scores indicating greater lack of psychological flexibility. We wish to note that a recent AAQ-II version developed for individuals with acquired brain injury has not yet been validated in German.[23] AAQ-II items focus mainly on mindful handling of negative emotions and painful memories that may result from critical life events, such as a stroke (e.g., "I'm afraid of my feelings" or "I worry about not being able to control my worries [. . .]").

As a possible confounder in partial correlation analyses, we included a clinician-rated measure of physical immobility with strong psychometric properties in German, the Barthel Index.[40] As a moderator in regression analyses, we considered diagnosis of acquired communication disorders (i.e., aphasia, apraxia of speech and dysarthria; separately or combined). For diagnosis of aphasia, we used the corresponding subscales on the Aachen Aphasia Test.[28] Despite superior psychometric properties in German, the Aachen Aphasia Test as a whole tends to be time-consuming. To ensure feasibility, we expressed diagnosis of aphasia as a dichotomous parameter by applying the required subpart of the Token Test (up to at least 6–7 errors to meet the threshold criteria) and Written Comprehension subscales (up to at least 77–78 points). For each participant, clinical linguists examined the criteria for apraxia of speech and dysarthria, as referred to above, to represent these conditions as a dichotomous parameter in analogy to aphasia.

Statistical analyses

We performed a-priori partial correlation analyses separately for each clinical outcome and the AAQ-II, with physical immobility as a potential confounder. We refrained from addressing acquired communication disorders as a simple second confounder in our partial correlation analyses, given the underestimated role of aphasia in the epidemiological literature. Instead, we operationalized the influence of acquired communication disorders as a possible moderator of the association between post-stroke psychopathology and psychological flexibility. For this purpose, we conducted a-priori linear regression analyses separately for each clinical outcome, with two covariates: AAQ-II scores as a continuous parameter,

and diagnosis of acquired communication disorders as a dichotomous parameter (in the case of at least one syndrome: yes; in the absence of any syndrome: no). Moderator effects in our regression model were operationalized as interaction between AAQ-II scores and diagnosis of acquired communication disorders. All a-priori and post-hoc evaluations complied with formal prerequisites for regression analyses, including normal distribution and homoscedasticity, as confirmed by Shapiro-Wilk and Levene tests. Based on alpha levels of 0.05, we report two-tailed p values and used the Bonferroni-Holm correction for post-hoc multiple comparisons.

Results

The a-priori partial correlation analyses yielded a significant medium-to-large association between each clinical outcome and lack of psychological flexibility ($r \leqslant 0.62$, $p \leqslant 0.001$, $n = 70$; for details, see Figure 1 and Table 2). This association was responsible for almost 39% of the variance observed on the BDI. As expected, the a-priori regression analyses did not yield a significant effect for diagnosis of acquired communication disorders as a moderator of the association between post-stroke psychopathology and psychological flexibility on any clinical outcome ($|t| \leqslant 1.21$, not significant).

Subsequent post-hoc evaluations consolidated the significant association between each clinical outcome and lack of psychological flexibility in partial correlation analyses separately for individuals with ($r \leqslant 0.56$, $p \leqslant 0.021$, $n = 27$) and without acquired communication disorders ($r \leqslant 0.70$, $p \leqslant 0.041$, $n = 43$). Additional post-hoc evaluations consistently verified the absence of a significant moderator for the association between post-stroke psychopathology and psychological flexibility when incorporating aphasia as a sole parameter in our regression model. Likewise, apraxia of speech and dysarthria remained nonsignificant as a moderator when viewed separately from aphasia.

Final post-hoc evaluations focused on the HADS and ISR subscales. Again, the association between clinical outcomes and lack of psychological flexibility was significant for depression ($r = 0.41$, $p = 0.001$) and anxiety disorders on the HADS ($r = 0.47$, $p < 0.001$) as well as for depression ($r = 0.47$, $p < 0.001$), anxiety disorders ($r = 0.34$, $p = 0.005$), obsessive-compulsive disorders ($r = 0.35$, $p = 0.003$), somatoform disorders ($r = 0.35$, $p = 0.003$), eating disorders ($r = 0.36$, $p = 0.003$) and other mental-health problems on the ISR ($r = 0.48$, $p < 0.001$, each $n = 70$).

Discussion

The current study aimed to determine the association between post-stroke psychopathology and psychological flexibility. To further explore this association, the study design was sensitive to diagnosis of acquired communication disorders as a potential moderator, motivated by recent epidemiological data[8] and evidence on treatment of subclinical[9] and clinical depression in aphasia.[10] Moreover, the design controlled for independent predictors of depression immediately after stroke: history of nonvascular psychopathology (ruled out during eligibility screening) and degree of physical immobility (reflected as a confounder in statistical analyses).[11] Based on 70 individuals in the subacute stage after stroke with increased risk of psychopathology,[27] partial correlation analyses revealed a significant medium-to-large association between the entire set of clinical outcomes and lack of psychological flexibility (for details, see Figure 1 and Table 2). In moderator analyses, the strength of this association did not vary significantly with diagnosis of acquired communication disorders (i.e., aphasia, apraxia of speech or dysarthria; separately or combined). Importantly, partial correlation analyses yielded significant results across subscales on all clinical outcomes, covering a broad range of syndromes beyond depression and anxiety disorders, such as obsessive-compulsive disorders, somatoform disorders or eating disorders. Taken together, our data suggest a strong interplay between post-stroke psychopathology and psychological flexibility, regardless of whether individuals meet the criteria for diagnosis of acquired communication disorders.

Limitations

A critical point touches on causality, given the nonprospective correlative design of the present study. At first glance, our data may imply that lack of psychological flexibility contributes to post-stroke psychopathology, consistent with the premise of prominent treatment programs[17] and ample evidence of efficacy.[18] However, the overall picture may be more nuanced and merits a closer look. Although embracing inevitable change in

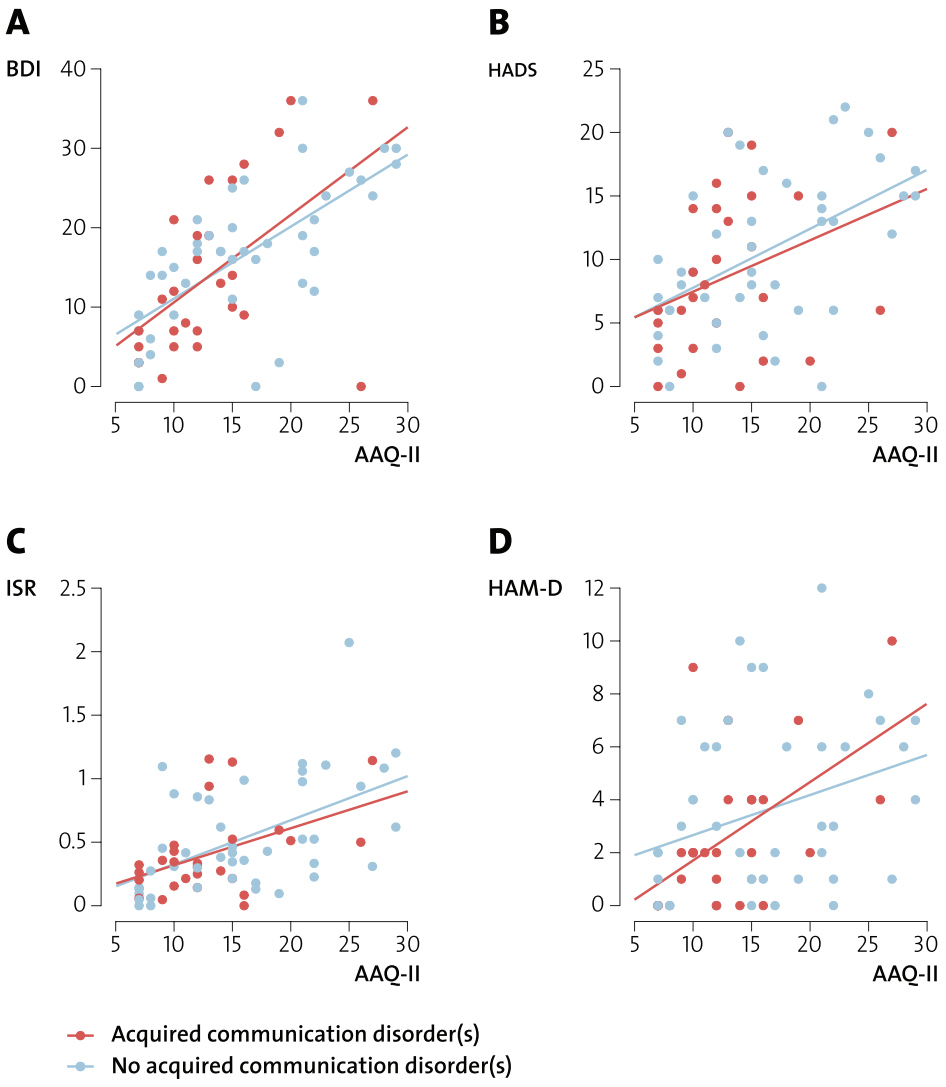

Figure 1. Results. Partial correlation analyses for the association between the AAQ-II and several clinical outcomes: BDI (A), HADS (B), ISR (C) and HAM-D (D), visualized for individuals with and without diagnosis of acquired communication disorders (i.e., aphasia, apraxia of speech or dysarthria). Partial correlation analyses account for physical immobility as a potential confounder (see Methods section).
AAQ-II, Acceptance and Action Questionnaire II; BDI, Beck Depression Inventory; HADS, Hospital Anxiety and Depression Scale; HAM-D, Hamilton Rating Scale for Depression; ISR, ICD-10 Symptom Rating.

life and using remaining agency arguably diminishes the probability of post-stroke psychopathology, negative bias typically inherent in depression may equally lead to inappropriately low self-rating of psychological flexibility. In other words, it may be challenging to obtain an objective measure of psychological flexibility during an episode of depression. Only a prospective study design may eventually account for issues of causality by addressing lack of psychological flexibility as a premorbid predictor that manifests in post-stroke psychopathology at a later point in time. An alternative, more pragmatic approach to this problem may be to note that, while desirable from an epidemiological angle, an objective measure of psychological flexibility and certainty about causality is neither possible nor imperative as long as tailored treatment proves helpful. In clinical practice, such tailored treatment seeks to promote psychological flexibility in stroke survivors[41] by

Table 2. Results.

Clinical outcome	Correlation between AAQ-II and clinical outcome	*p* value of correlation analysis	*p* value of moderator analysis
BDI	0.62	<0.001	0.557
HADS	0.49	<0.001	0.782
Depression	0.41	<0.001	0.452
Anxiety disorders	0.47	<0.001	0.236
ISR	0.54	<0.001	0.720
Depression	0.47	<0.001	0.452
Anxiety disorders	0.34	0.005	0.791
Obsessive-compulsive disorders	0.35	0.003	0.702
Somatoform disorders	0.35	0.003	0.232
Eating disorders	0.36	0.003	0.465
Other mental disorders	0.48	<0.001	0.832
HAM-D	0.39	<0.001	0.259

Partial correlation coefficient and *p* value for the association between the AAQ-II and several clinical outcomes: BDI, HADS, ISR, and HAM-D. Partial correlation analyses account for physical immobility as a potential confounder (see Methods section). Moderator analyses refer to the influence of acquired communication disorders on the association between the AAQ-II and clinical outcomes.
AAQ-II, Acceptance and Action Questionnaire II; BDI, Beck Depression Inventory; HADS, Hospital Anxiety and Depression Scale; HAM-D, Hamilton Rating Scale for Depression; ISR, ICD-10 Symptom Rating.

adopting various methods proposed in the literature (e.g., setting new goals congruent with personal values).[17] This pragmatic view may allow epidemiological claims about causality to retain a hypothetical character, yet without identifying mechanisms of efficacy in the context of psychological flexibility.

A related issue concerns the psychometric properties of our clinical outcomes (BDI, HADS, ISR, HAM-D) and our measure of psychological flexibility (AAQ-II). All instruments are self-report questionnaires (BDI, HADS, ISR, AAQ-II), apart from one clinician-rated outcome (HAM-D). Most self-report questionnaires still await formal testing in individuals with aphasia, except for our co-primary outcome, a simplified BDI version.[35] Indeed, the BDI shows promising signs of discriminant validity in individuals with post-stroke depression and aphasia, as confirmed by previously published correlation analyses with self-efficacy as an external criterion ($r=-0.49$; $p<0.001$, $n=60$).[10] In the present sample ($n=70$), accessible language of the simplified BDI may be one reason for the especially high association between this clinical outcome and the AAQ-II ($r=0.62$; see Figure 1A and Table 2) compared to other measures of psychopathology ($r\leqslant 0.55$; see Figure 1B–D and Table 2). Notably, the magnitude of this association did not differ depending on diagnosis of acquired communication disorders as a moderator in a-priori regression analyses. While deriving conclusions from a nonsignificant moderator is methodologically debatable in light of reduced statistical power achieved with smaller subgroups, the results do demonstrate that barrier-free outcomes such as the simplified BDI may be similarly suitable for both individuals with and without acquired communication disorders after stroke. In post-hoc regression analyses, aphasia, apraxia of speech and dysarthria continued to be nonsignificant as a

moderator when considered separately from one another. Despite the mentioned caveat of statistical power with smaller subgroups, this finding adds weight to the idea that speech and language skills do not influence psychological flexibility in a unique way.

As for content validity, our measure of psychological flexibility, the AAQ-II, focuses primarily on the ability to take advantage of negative emotions (e.g., "I'm afraid of my feelings" or "I worry about not being able to control my worries [. . .]").[39] In contrast, this outcome does not explicitly distinguish between accepting inescapable setbacks (while concentrating on remaining scope for action) or falling into despair over the unalterable (in an unproductive state of resignation). Although exercising control over emotions rather than trusting them may indicate some difficulty facing adversity in life, the AAQ-II only indirectly reflects the existential choice between embracing fate and struggling with it. Recognizing this choice, however, may be particularly pertinent in stroke survivors with profound loss of function. For example, anecdotal evidence from psychotherapy sessions for individuals with aphasia and concomitant psychopathology suggests that, prior to dealing with negative emotions, many stroke survivors first need to become aware that they have a choice (e.g., devoting all efforts to effective speech-language therapy or, alternatively, loosing precious resources by holding on to false expectations of prompt and complete recovery). Being conscious about this choice may, in a next step, facilitate mindful handling of negative emotions that ultimately has the potential to alleviate post-stroke psychopathology.[42] Obviously, it is challenging to conceive a psychometrically sound *and* barrier-free instrument that operationalizes existential choice. Nonetheless, it may be worthwhile to replicate the current results with such an instrument in a sample of stroke survivors with and without acquired communication disorders (Stahl and Romanzcuk-Seiferth, in preparation).

Conclusion

The present study is the first to consolidate the substantial link between post-stroke psychopathology and psychological flexibility. This finding opens new avenues for clinical research on depression and other mental-health problems in stroke survivors with and without acquired communication disorders.

Declarations

Ethics approval and consent to participate

This study received approval from the ethics review board at the Medical School Berlin, Germany (file number: MSB-2022/91). A team of neuropsychologists obtained written informed consent from participants or their legal representatives, with sufficient room for questions and reflection to account for potential aphasia-immanent verbal comprehension issues.

Consent for publication

Not applicable.

Author contributions

Benjamin Stahl: Conceptualization; Formal analysis; Methodology; Supervision; Visualization; Writing – original draft; Writing – review & editing.

Kristina Becker: Investigation; Supervision; Writing – review & editing.

Kevser Kocyigit: Investigation; Supervision; Writing – review & editing.

Petra Denzler: Investigation; Supervision; Writing – review & editing.

Paula Röder: Conceptualization; Formal analysis; Investigation; Methodology; Supervision; Writing – review & editing.

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Competing interests

The authors declare that there is no conflict of interest.

Availability of data and materials

To access our original data upon reasonable request, please contact the corresponding author.

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