Review

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Post-stroke emotionalism: Diagnosis, pathophysiology, and treatment

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Abstract

Background: Post-stroke emotionalism affects one in five stroke sufferers 6 months after their stroke, but despite its frequency remains a poorly understood stroke symptom. The literature is limited, especially compared to other frequently observed neurological conditions such as aphasia and visual neglect.

Aim and Methods: This narrative review presents a summary of the post-stroke emotionalism literature, to inform clinical practice and future research. We cover discussion of definitions, prevalence, neurobiology, predisposing and precipitating factors, and treatment.

Results: Increasing evidence suggests that damage to specific areas functionally linked to emotion expression or regulation processes, disruption to structural pathways and those related to serotonin production and modulation individually or in concert give rise to emotionalism-type presentations. A range of emotionalism measurement tools have been used in research contexts making between study comparisons difficult. Testing for Emotionalism after Recent Stroke–Questionnaire (TEARS-Q) has recently been developed to allow standardized assessment. Treatment options are limited, and there have been few adequately powered treatment trials. Antidepressants may reduce severity, but more trial data are required. There have been no randomized-controlled trials of non-pharmacological interventions.

Conclusions: More research is needed to improve recognition and treatment of this common and disabling symptom. We conclude with research priorities and recommendations for the field.

Keywords
Stroke, rehabilitation, neuropsychology, clinical psychology, emotionalism, neuropsychiatry

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Defining and diagnosing emotionalism

Emotionalism following stroke is a prevalent, widely acknowledged yet under-researched disorder of emotional expression typically involving recurrent uncontrollable episodes of crying and occasionally laughter.[1–3] Emotionalism is neurological in origin but has known secondary psychological and behavioral outcomes including anxiety, event-related distress, social avoidance, and negative self-beliefs.[4–8] Emotionalism is distinct from depression, but there is a central crying component to both.[3,5,9,10] Emotionalism can arise in Alzheimer's dementia, vascular dementia, motor neuron disease, traumatic brain injury, multiple sclerosis, stroke, and other conditions.[11]

Emotionalism may not be unitary in presentation or subtype, as reflected in the multiple terms used in the literature: pseudobulbar affect (PBA),[12] pathological laughter or crying (PLC),[13] emotional incontinence (EI),[14] involuntary emotional expression disorder (IEED),[15] and emotional lability (EL).[16] Carota and Calabrese[17] suggest conceptualizing EL and PLC/PBA as two conditions positioned on a "continuum" of emotionalism. They argue different presentations may have distinct but related neural causes, although the evidence is not established.[8,17] Although neurological in origin and with potential for unprovoked episodes, a proportion of emotionalism episodes seem to be triggered by emotional stimuli.[8] Categorization of emotionalism subtypes based on emotional salience of a person's triggers is plausible but has yet to be systematically explored. It is also

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Figure 1. Testing Emotionalism After Recent Stroke–Questionnaire (TEARS-Q). To download, go to https://expresslicensing.uea.ac.uk/product/testing-emotionalism-after-recent-stroke-questionnaire-tears-q.

Stroke can cause changes to emotional expression (how we show our emotions), in particular through crying. The following statements relate to your pattern of crying since your stroke. Please indicate by circling one response how true each statement is for you, **in the past two weeks**.

1. I feel more tearful **in the past two weeks** than before the stroke

Strongly Agree Agree Unsure Disagree Strongly disagree

2. I have actually cried more **in the past two weeks** than before the stroke

Strongly Agree Agree Unsure Disagree Strongly disagree

If response is disagree or strongly disagree on both items 1 and 2, discontinue the test

3. My crying comes on suddenly, with only a few seconds or no warning

Strongly Agree Agree Unsure Disagree Strongly disagree

4. My crying comes on when I am not expecting it

Strongly Agree Agree Unsure Disagree Strongly disagree

5. My crying comes on even if I do not feel sad at the time

Strongly Agree Agree Unsure Disagree Strongly disagree

6. When my crying comes on, I cannot control or stop it

Strongly Agree Agree Unsure Disagree Strongly disagree

7. I cry in situations I would not have cried in before the stroke

Strongly Agree Agree Unsure Disagree Strongly disagree

8. I cry in this way at least once per week or more often

Strongly Agree Agree Unsure Disagree Strongly disagree

[1] Broomfield NM, West R, House A, Munyombwe T, Barber M, Gracey F Gillespie DC & Walters, M. (2021). Psychometric evaluation of a newly developed measure of emotionalism after stroke (TEARS-Q). *Clinical Rehabilitation*, 35(6), 894 903. https://doi.org/10.1177/0269215520981727

[2] Broomfield N M, West R, Barber M, Gillespie DC, House A & Walters, M. (2021). Practical guidance on use of TEARS-Q to diagnose post-stroke emotionalism. *Clinical Rehabilitation*, 35(12), 1768-1772. https://doi.org/10.1177/02692155211024802

unknown where on any hypothesized emotionalism "continuum," uncontrolled laughter is best positioned. The interplay of neurological and psychological aspects here can also make differential diagnosis of post-stroke emotionalism (PSE) and depression difficult.

Accordingly, thorough screening and psychological assessment are necessary with attention to diagnostic criteria and psychological models, to which we now turn. Diagnostic criteria for emotionalism specifically following stroke were first delineated by House and colleagues as: (1) uncontrolled crying episodes lasting a few seconds to minutes not under usual social control, which (2) present frequently on a weekly or more often basis, (3) suddenly, with little or no warning, and (4) which represent a change from pre-stroke functioning.[3] Kim and Choi-Kwon[14] similarly refer to (1) excessive or inappropriate laughing or crying, or both, as compared with the premorbid state and (2) occurring according to the patient and the relative on at least two occasions, with inappropriate taken to mean "laughing or crying that occurs while talking, listening, meeting people, or watching television that is not particularly amusing or sad to ordinary people." (p. 1806).[14]

In stroke, growing consensus on PSE and convergence in diagnostic criteria has allowed development of a measure for tearful emotionalism (see Figure 1).[18,19] This offers hope for routine practice and research. However, there

remains diversity of terminology in the wider neurological field and diagnostic uncertainty regarding mental health symptoms and relevance of emotionally salient triggers. In our practice, we use PSE as an umbrella term covering the core diagnostic features while allowing for variation in presentation. In keeping with the James Lind Alliance Research Priorities for Stroke Survivors,[20] we call for an international consensus on emotionalism terminology, best practice in assessment and intervention, and identification of research priorities bridging the best neurological science with patient and family priorities.

Prevalence of PSE

Most PSE prevalence studies use either the "House" or "Kim" criteria for diagnosis. Pooled estimates from a single systematic review and recent single other longitudinal cohort study suggest PSE affects approximately one in five stroke survivors 6 months after stroke and at least one in 8 up to 12 months,[4,21] although recruitment biases overestimating prevalence are likely in those studies not deploying consecutive recruitment.[21] Only two studies offer longer term data, with rates of one in six at 15 months post-stroke[22] and just under one in 12 at 40 months.[23]

Following stroke, "laughter only" and "mixed crying and laughter" subtypes present more rarely than the common "crying only" variant.[5,21] The Testing Emotionalism After Recent Stroke (TEARS) cohort study noted 84% of the sample recruited through routine stroke services who had emotionalism showed the crying only sub-type, 11% laughter only, and 5% mixed presentation,[4] and previous studies have reported a similar pattern.[5] However, most studies do not differentiate subtypes, and the neurological etiologies of PSE subtypes remain unclear.[17]

We therefore argue that epidemiological research with more precise PSE measurement, including subtypes, conducted over longer follow-up periods post-stroke is required. With a consensus on assessment and diagnosis, there is potential (depending on local healthcare structures) for such data to be gathered through routine stroke service provision. This "big data" approach could significantly advance our understanding of the precise prevalence and trajectory of PSE over time.[1]

Neurobiology of PSE

Wilson[24] was the first to provide a neuroanatomical model of emotionalism. In keeping with a hierarchical approach to brain structure and function, Wilson hypothesized that involuntary emotional expression characteristic of emotionalism arose through lesions to inhibitory frontal and motor pathways that regulate lower-level emotional expression (laughter and crying) centers in the upper brain stem.

Wilson's seminal model predominated until modern imaging techniques enabled alternate theories to emerge. Combining clinical case work and magnetic resonance imaging (MRI) neuroimaging data, Parvizi et al.[25] postulated that emotionalism manifests when stroke lesions disrupt information flow between the brain stem, motor cortex, and cerebellum. They note that, in neurologically healthy individuals, the cerebellum modulates and adjusts emotional motoric responses (crying and laughter) based on information received from the cerebral cortex regarding the specific cognitive triggers and social context. Thus, loud laughter (motoric response) to a joke (cognitive trigger) might occur in one social context (e.g. at home and relaxed with family) but not in another (e.g. at work with senior colleagues). This adaptive emotional responding is ascribed to the normal functioning of the cerebellum and the associated cortico-ponto-cerebellar pathway. In emotionalism, a lesion could disrupt communication between the motor cortex, pons, and cerebellum. The cerebellum thus could initiate uncontrollable crying or laughing behaviors, disproportionate to, or in the absence of, a trigger and unmodulated by social context.[25]

Consistent with the Parvizi model, there is an established link of PSE to strokes which disrupt cortical (i.e. frontal, motor, temporal cortex) and descending subcortical (i.e. brain stem, cerebellum) neural locations and circuitry.[2,9,11,14,22,25–27] The possible role of the fronto striatal neural network has also been implicated in the pathophysiology of PSE.[28]

Disrupted serotonergic neurotransmission may also play a part.[11] Serotonin is an inhibitory neurotransmitter involved in mood regulation and which is produced exclusively in the Raphe nuclei of the brain stem, a neural location with links to PSE. Several serotonergic projection areas linked to cortical-subcortical regions are implicated in PSE neuroanatomy (cerebellum, striatum, frontal and motor cortices) and the diffuse neuroanatomical picture of PSE could be understood in terms of serotonin production and modulation. Genetic and imaging studies link serotonin to PSE[29–31] and Selective Serotonin Reuptake Inhibitors (SSRIs) which exclusively limit serotonergic uptake at the synapse have been shown to alleviate PSE in some patients,[23,32] although high-quality clinical trial data on safety and efficacy are needed.[1]

Therefore, a combined neurological model of PSE is emerging such that damage to specific areas functionally linked to specific emotion expression or regulation processes, disruption to structural pathways, and related serotonin production and modulation, might individually or in concert give rise to emotionalism-type presentations.

Assessing emotionalism after stroke

Systematic and robust clinical assessment of emotionalism remains challenging. A systematic review[11] of emotionalism

predictors and correlates across neurological conditions revealed a range of emotionalism measurement tools that have been used in research contexts, which compromises the validity of findings within, and across, studies. Moreover, while psychometric measures of emotionalism have been available for clinical and research use,[33,34] these have been generic and a tool developed and validated specifically for the stroke survivor population has been lacking. In the 2022 Cochrane review of pharmaceutical intervention for PSE,[1] the authors called for the development of a standardized method to diagnose emotionalism and determine symptom severity and change over time, based on a standard PSE definition. Broomfield et al.[18,19] have published the Testing for Emotionalism after Recent Stroke–Questionnaire (TEARS-Q; see Figure 1) and a semi-structured diagnostic interview schedule based on the House criteria.[4] As it stands, the diagnostic criteria outlined by House et al.[3] or Kim and Choi-Kwon[14] remain the clearest for routine clinical application.

Psychometric evaluation reveals that the TEARS-Q appears to be internally reliable (Cronbach's alpha 0.87) and diagnostically accurate, with a total TEARS-Q score of ⩾2 detecting PSE with 87% sensitivity and 90% specificity.[18] While further validation is required, TEARS-Q offers promise both for standardizing outcome measures in research studies and to assist clinicians in assessing emotionalism in practice,[19] including rapid screening. TEARS-Q comprises eight questions rated according to a 5-point Likert-type scale. The first two items serve as a screening tool, with negative responses indicating absence of PSE whereupon the measure can be discontinued. Validation work is currently ongoing on an informant version (TEARS-QI).

Formulating PSE

To complement the assessment of PSE outlined above, considerations to help develop a clinical case formulation of PSE based on the 5 Ps approach[35,36] are outlined. The 5 Ps are the presenting, predisposing, precipitating, perpetuating, and protective factors related to a clinical presentation. Figure 2 provides a visual summary of these factors and their hypothesized interactions.

It should be acknowledged that the mapping of underlying emotionalism constructs to this framework relies on individual judgment. For instance, neuroinflammation-induced exacerbation of emotionalism may be perceived as a perpetuating factor, insofar as it maintains the presence of emotionalism, or viewed as the worsening of a predisposing factor. Clinicians are, therefore, encouraged to be guided by allocations that are most meaningful to the patient or most informative for intervention.

Presenting

These are defined here as both the emotionalism symptoms, outlined earlier, and secondary psychological distress. Those with PSE are at greater risk of secondary psychological difficulties including depression, anxiety, event-related distress, embarrassment, social avoidance, and negative self-beliefs.[4–8,37]

Predisposing

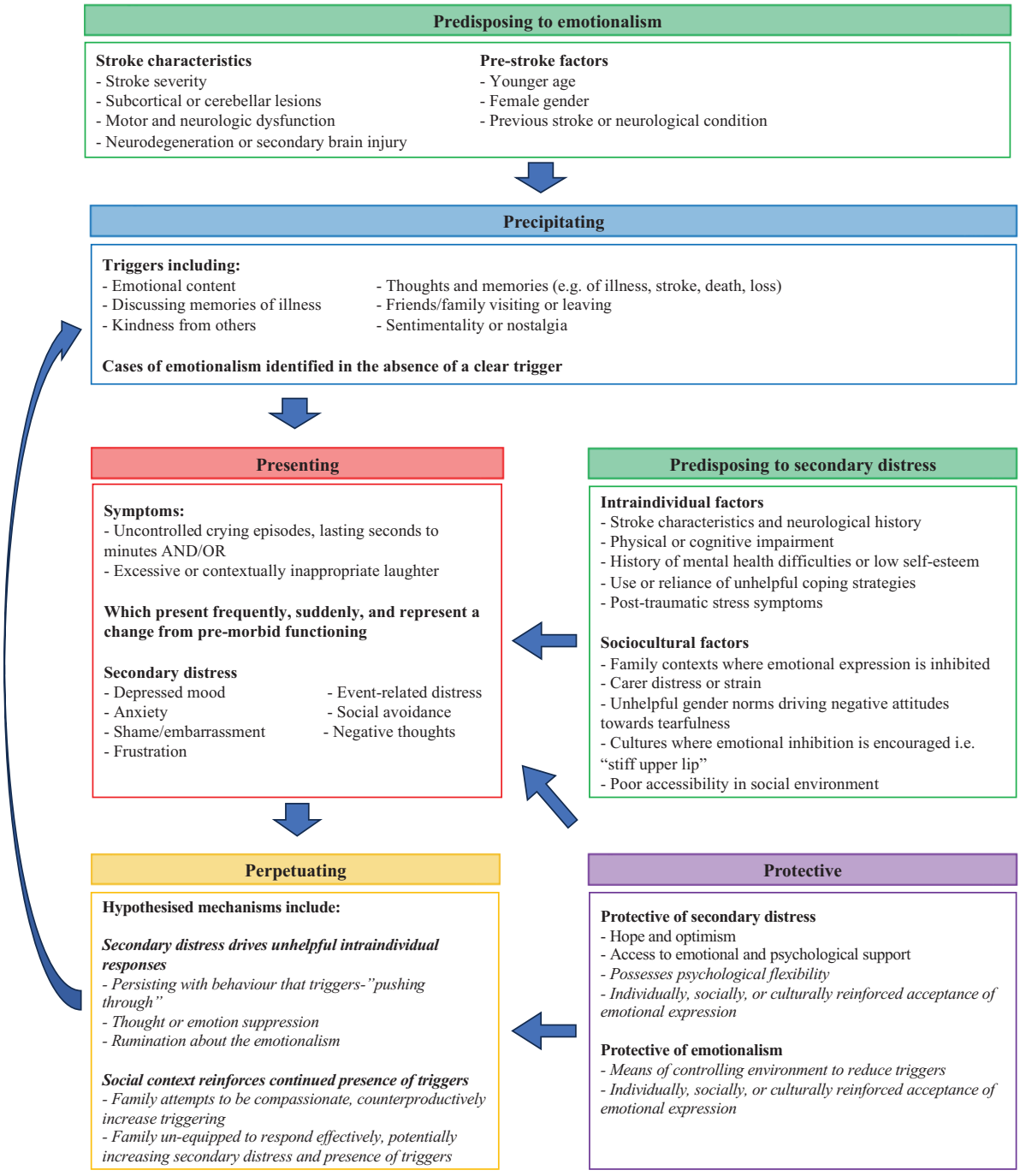
This refers to risk factors for both PSE itself and for secondary psychological distress. Factors predisposing emotionalism include female gender,[14] younger age,[9] and previous neurological condition.[2,9] Several stroke characteristics have been identified as risk factors for emotionalism, including stroke severity, subcortical or cerebellar lesions, and motor impairments.[2,14] These factors may be static, such as gender, or dynamic, such as the presence of neurodegeneration.[12] Importantly, while these variables have been shown to correlate with PSE, there may be differences in the degree to which each may be causal.

Risk factors for secondary distress span biological, psychological, and social domains. These include stroke characteristics,[38,39] neurobiological changes,[38,40] physical disability,[39,41] cognitive impairment,[42] history of mental health difficulties,[38,39,41] historic low self-esteem,[43] post-traumatic stress symptoms related or unrelated to the stroke,[6] low perceived social support,[38,39,44] carer distress,[43] and poor accessibility in one's environment.[45] Cognitive impairments, such as executive dysfunction, that bias information processing to reinforce dysfunctional appraisals are particularly understood as important drivers of mental health difficulties post-stroke.[46] Cultural factors have also been cited as important factors in vulnerability to secondary distress; beliefs about male expressions of emotion, such as "men should not cry" (p. 196), have appeared in qualitative investigations of PSE.[7]

Precipitating

These are defined here as triggers for episodes of PSE. Interviews of people with PSE[3,8] highlight precipitating triggers including emotional content on television, thoughts of illness or dying from stroke, discussion or memories of illness, death or losses, family visiting or leaving, and kindness from others. While these triggers have been broadly categorized into (1) thoughts and feelings associated with sadness/depression and (2) sentimentality,[3] these studies also identified the act of discussing the symptoms of PSE itself as a trigger. Episodes of PSE may also occur without any clear trigger,[7,8] which links to the conceptual differentiation between EL and PLC/PBA described above.

Figure 2. Visual representation of the causes and maintenance of PSE.



Hypothesised factors not directly evidenced but theoretically plausible are italicised. All other factors possess primary evidence from at least one source.

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Nonetheless, having an awareness of contextual triggers may be useful for patients and clinicians in managing PSE.

Perpetuating

These are defined as mechanisms by which the emotionalism symptoms or secondary psychological distress may be maintained or reinforced. Currently, there is no causal evidence that the emotionalism symptoms or their secondary outcomes directly result in a worsening of the condition itself. However, hypothesized mechanisms are outlined below.

Factors maintaining or increasing the presence of triggers/precipitating events may constitute one such mechanism. Intraindividually, psychological processes may result in inadvertent self-triggering of episodes. Qualitative studies have indicated that thoughts may indeed trigger emotionalism episodes[8] and attempts at suppression of mental activity have been shown to result in paradoxical activation of the suppressed content.[47] It is therefore plausible that attempts to suppress thoughts about triggering topics may increase their presence and thus episodes of emotionalism.[8] Rumination about one's emotionalism may also provide increased opportunities for triggering of episodes.

Interindividually, clinicians should consider social factors that may maintain the presence of triggers. For example, a case study exploring a behavioral conditioning intervention for emotionalism found that attempts to positively reinforce *not* being tearful via conditional increases in attention and interaction unintentionally *increased* emotionalism.[48] Thus, it is conceivable that misperception of emotionalism as depression may prompt loved ones to respond sympathetically, which might further trigger emotionalism and perpetuate this misinterpretation.[8] It must be noted that, even when compassion or sympathy are unambiguous triggers, such displays should not necessarily be avoided, given their important function in alleviating psychological distress after a stroke.[49]

Another possibility might be that behavioral responses to the secondary distress can cause a worsening of factors that predispose emotionalism. The detrimental effects of depressed mood on functional outcomes of stroke are well documented[50–52] and behavioral factors, such as disengagement from medical intervention or rehabilitation, may increase risks of neurological deterioration.[53] This could, in turn, worsen the severity of emotionalism.

Mechanisms by which secondary psychological difficulties may be maintained once precipitated by PSE have stronger experimental support and are outlined in cognitive-behavioral models of post-stroke depression and anxiety. These may include the reinforcement of negative or anxious cognitions via behavioral avoidance and consequential reduction in opportunities for these dysfunctional appraisals to be challenged or disconfirmed.[54]

Protective

Protective factors are here defined as psychological and environmental resources, which an individual may possess inherently or could be developed through intervention. Qualitative accounts indicate the importance of hope, optimism, normalization, and support from others in coping with PSE.[7,8,55]

While these factors have been shown to help people cope better, they say little about what may assist to reduce symptoms. Given the hypothesized perpetuating role of emotion suppression and rumination in maintaining emotionalism, those who possess greater psychological flexibility and emotional acceptance may be protected from more severe and enduring episodes of emotionalism.[8,56] Although not experimentally confirmed, qualitative studies have suggested that acceptance-based approaches have been helpful for some.[8]

Interventions

The evidence base for PSE treatment is limited and has received little attention, with no new studies published between the 2010 and 2022 Cochrane reviews of medication trials for PSE.[1,57] The literature on pharmacological and non-pharmacological interventions for PSE is summarized below, followed by clinical recommendations.

Medication

The most-recent Cochrane review analyzed results from five parallel randomized-controlled trials (RCTs), all investigating antidepressants, and concluded low-to-moderate certainty for the efficacy of medication in reducing PSE symptoms.[1]

The heterogeneity across included studies meant there was little scope for meaningful synthesis, requiring individual analysis of the included RCTs. However, many were underpowered and possessed high variation among participants; in one study, the time since stroke spanned 1 month to 13 years across just 28 participants.[31] In three studies, improvements were defined loosely as a reduction in tearfulness with high risk of bias due to lack of appropriate controls.[31,32,50] While high risk of bias compromises the basis to guide clinical treatment choice, preliminary evidence for the effectiveness of antidepressants in some individuals warrants future investigation.[1] Building on this, a new phase III trial evaluating the effectiveness of 50 mg sertraline daily for emotionalism has recently commenced (EASE; Evaluating Antidepressants for Emotionalism after Stroke; NIHR152423). When added to the existing total, the target sample size would more than double the pooled sample of all previous RCTs combined, from 200 to 510.

Non-pharmacological treatments

There have been no registered or published RCTs or quasi-experimental trials for non-pharmacological PSE interventions,[58] so their effectiveness is unknown.

One uncontrolled case-series for people with Locked-In Syndrome found apparent, but not statistically validated, improvements in tearful episode length after a 6-week intensive training intervention, involving movement of affected muscles once an episode was triggered.[59] A recent study using a single case experimental design in someone with a hemorrhagic stroke suggested breathing techniques may reduce episode length when applied.[48] This study also trialed a reinforcement/contingency-based intervention, which led to increased tearfulness. The authors in both articles argue for the potential for trained controllability but, alternatively, the use of these techniques may merely act as distractors, and it is yet to be understood if either is differentially effective over other distraction techniques.

The most used non-pharmacological approaches to emotionalism have been identified by two recent survey studies,[55,58] the former deploying Delphi methodology to reach emotionalism expert consensus, the latter an online exploration of UK National Health Service (NHS) stroke nursing, medicine and allied health clinician views. Taking the two studies together, education, normalization, acknowledgment and task continuation, discussion of goals, reassurance, distraction, and breathing techniques were considered most effective/helpful/accessible.[55,58] The definition of effectiveness/helpfulness is notably non-specific to the function (symptom reduction versus improved secondary psychological outcomes) in these and other PSE studies,[7,8] perhaps reflecting varied perspectives on the preferred outcome of any intervention.

Qualitative studies have highlighted variation between people in what they find helpful.[7,8] For those responding to PSE with self-criticism, reassurance and support from others are important. However, others found expressions of kindness or empathy a trigger for the PSE and, potentially, unhelpful. Some reported success in attempts to control PSE, or avoid triggers, while others emphasized the importance of acceptance. This indicates individual psychological formulation according to the processes outlined previously will be necessary to identify interventions or strategies that are effective. If secondary psychological outcomes and/or maladaptive coping strategies result in additional PSE symptom burden, then the above-listed techniques designed to alleviate secondary distress may also be effective in reducing PSE symptoms. However, this is yet to be determined.

Clinical recommendations

Clinicians may wish to consider a mixture of pharmacological and non-pharmacological interventions. Patients should be informed of the limited evidence base, and decisions about medication should be patient-led, with consideration of the options that maximize safety and minimize contraindications and side-effect profiles.

Clear communication of a suspected PSE diagnosis, with education and normalization, should be helpful in most cases to curb unhelpful or inaccurate beliefs about its cause or significance.[7,8] The apparent variation in patient perspectives as to which non-pharmacological strategies are most helpful suggests that selection of any intervention is best guided by clinician formulation and patient preference, rather than any person-general indicators of effectiveness.

Although psychological or behavioral PSE therapies are yet to be validated, they show potential in supporting people with mood disorders secondary to stroke and other physical health conditions.[55] These may be recommended for those whose PSE is formulated as a driver for their mood disorder. As stated above, the extent to which any effective psychological or behavioral PSE therapy could also alleviate core PSE symptoms remains unknown.

In considering interventions, what is a desirable outcome of a non-pharmacological PSE treatment? This will likely differ individual to individual. But should elimination of core neurological symptoms be aimed for? Non-pharmacological interventions aimed at alleviating secondary distress and negative psychosocial outcomes rather than eliminating core emotionalism symptoms would seem a more optimal approach to us. Adopting a third wave Acceptance and Commitment Therapy (ACT) theoretical framework to foster improved acceptance and mastery of core emotionalism symptoms (rather than their elimination) could prove fruitful, as has been shown in post-stroke depression[60] and is being tested in motor neurone disease.[61] In cases where emotionalism episodes cause physical discomfort, for example, strong diaphragm contractions during laughter episodes, symptom elimination may be targeted. However, efforts to prematurely end tearfulness episodes could be intrusive or unhelpful, particularly where the trigger is emotionally salient and reflective of underlying mood.

Furthermore, secondary distress from PSE could be promoted by an unsafe psychological environment around the patient. This could include family contexts where emotionalism is not responded to in a way that feels supportive for the patient or situations of bullying within the social network. Here, a focus on behavioral change to conform to the needs and expectations of others raises significant ethical concerns.

The right approach to emotionalism treatment is a personal one, and we recommend the treating clinician withhold any assumptions about the definition of a successful outcome. If symptom reduction is the goal, care must be taken not to inappropriately encourage strategies (e.g. deep breathing) where the tearfulness is a manifestation of

Table 1. Future research recommendations for emotionalism after stroke.

Future Research Domain	Potential future research questions
Conceptualization and terminology relating to emotionalism in stroke and across the neurological disorders	Can an international consensus on terminology for the study of emotionalism be reached? What are the preferred primary outcomes relating to PSE onset, frequency, maintenance over time, and secondary PSE-related psychological distress for use in future trials?
Routine and systematic collection of clinical "big data" on PSE	What are the measurement properties of tools used to diagnose PSE, measure PSE severity or evaluate PSE-related distress? How does measurement vary across cultural contexts? What is the precise prevalence of PSE and what increases vulnerability to PSE occurrence, maintenance over time and PSE-related distress? What is the trajectory of PSE over time and what variables are associated with different trajectories? What outcomes are achieved in services that have implemented PSE-specific treatment or management programs versus those that have not?
Development of neurological/ neuropsychological, psychological and social models of PSE episode frequency, PSE duration, or secondary PSE-related distress	What are the functions of serotonin, serotonin-mediated pathways, and specific brain areas in relation to PSE onset, type, and maintenance over time? Which psychological and social processes best account for individual variation in PSE presentation, maintenance and secondary PSE-related distress? Clinically, and from qualitative accounts, emotionalism may be triggered by emotionally charged events and content, or via neutral content or in a more unprovoked manner. At a population level, what proportion of episodes are precipitated by emotional versus neutral content, and are there individual differences? Furthermore, do population-level data on episode triggers support or refute current conceptualizations of emotionalism subtypes (PBA vs EL)? What is the role of the sociocultural context on the expression of PSE and secondary psychological outcomes?
Clinical Intervention development and evaluation for (1) Treatment of PSE onset and maintenance over time? (2) Management of PSE-related distress	Are SSRI antidepressants a safe and effective treatment for PSE? Is there potential for SSRI's to modify PSE onset, frequency, maintenance over time, or secondary PSE-related distress? Does education about PSE impact the expression of primary PSE (crying/laughter) and/or secondary psychological outcomes? What are the comparative effects of strategies aimed at controlling PSE versus acceptance strategies on the frequency of episodes and secondary psychological outcomes? What is the effect of Acceptance and Commitment Therapy adapted for PSE on PSE frequency and PSE related distress?
Improving service provision for PSE and PSE-related distress for stroke survivors and family caregivers	How can services best implement supportive interventions for people experiencing PSE and family caregivers at different phases of their stroke journey? What are the preferences and experiences of stroke survivors and family caregivers regarding how services respond to PSE?

PSE: post-stroke emotionalism; PBA: pseudobulbar affect; EL: emotional lability; SSRI: Selective Serotonin Reuptake Inhibitors.

underlying feelings and where gentle emotional support would be more beneficial. Because it is often difficult to differentiate causes of tearfulness, clinicians should set up an agreement with the patient on how such episodes will be approached.

Research recommendations

Based on the above, several research recommendations are proposed (see Table 1). Of note is the need for preliminary work on consensus definition of emotionalism to allow integration of work across neurological and psychological domains and clinical presentations and systematic collection of data to inform future theoretical and clinical developments. Given the diversity of presentation and intervention setting, treating professionals, and the biopsychosocial heterogeneity of stroke, clinical intervention research should follow the Medical Research Council (MRC) guidance for evaluating complex interventions.[56]

Conclusion

We offer a timely and much needed summary of current research and informed thinking regarding diagnosis, prevalence, neurobiology, assessment, psychological formulation, and intervention of PSE. There is now a pressing need

to build international consensus regarding terminology, conceptualization and treatment of PSE, and identification of priorities for clinical research. Our review, although brief and not systematic, draws on existing reviews of evidence and proposes hypotheses tentatively but with a view to driving the focus for future hypothesis-driven research. In addition, we can be confident in certain conclusions, notably regarding the absence of evidence for guiding interventions, especially important as a potentially helpful approach for one person may worsen the clinical picture in another. In summarizing the extant literature on PSE, we identify the need for multidisciplinary research addressing biopsychosocial processes pertinent to the development and maintenance of PSE and the likelihood of persisting secondary psychological distress. While we cannot draw firm, evidence-based conclusions to inform practice from the existing literature, we would confidently conclude that further research is needed, and we offer potentially fruitful lines of enquiry to better understand and treat this prevalent and neglected post-stroke condition.

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