Original research

TEARS: a longitudinal investigation of the prevalence, psychological associations and trajectory of poststroke emotionalism

Niall McIntyre Broomfield ,[1] Robert West,[2] Mark Barber,[3] Terence J Quinn ,[4] David Gillespie,[5] Matthew Walters,[4] Allan House[6]

[1]Clinical Psychology and Psychological Therapies, University of East Anglia, Norwich, UK
[2]University of Leeds Faculty of Medicine and Health, Leeds, UK
[3]Monklands Hospital, Airdrie, UK
[4]Institute of Cardiovascular and Medical Sciences, University of Glasgow, Glasgow, UK
[5]Department of Clinical Neurosciences (DCN), NHS Lothian, Edinburgh, UK
[6]University of Leeds, Leeds, UK

Correspondence to
Professor Niall McIntyre Broomfield, Clinical Psychology and Psychological Therapies, University of East Anglia, Norwich NR4 7TJ, UK; N.Broomfield@uea.ac.uk

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ABSTRACT

Objective There are few longitudinal studies of poststroke emotionalism (PSE) and our understanding of the psychological associations of PSE is limited, constraining assessment of existing interventions and the development of new therapies. This study aimed to assess the prevalence and course of PSE over the first year poststroke, and its psychological associations.

Methods Consenting stroke survivors who were physically and cognitively able to participate were assessed within 2 weeks, 6 and 12 months of stroke to determine PSE point prevalence using a diagnostic, semistructured PSE interview (Testing Emotionalism After Recent Stroke-Diagnostic Interview). At the same assessments, neuropsychological and disability status were determined using Hospital Anxiety and Depression Scale, Abbreviated Mental Test, National Institute of Health Stroke Scale, Barthel Index and Euro-Qol.

Results Two hundred and seventy seven stroke survivors were recruited between 1 October 2015 and 30 September 2018. Diagnostic data were available at baseline for 228 of 277 cohort participants. Point prevalence for PSE was 27.2% at 2 weeks; estimated prevalence at 6 months adjusted for baseline was 19.9% and at 12 months 22.3%. PSE was associated with symptoms of anxiety and event-related distress.

Interpretation PSE affects at least one in five stroke patients acutely following their stroke, and continues to affect one in eight longer term. PSE is associated with anxiety and event-related distress but is not simply a manifestation of mood disorder over time. Such psychological correlates may have implications for longer term social rehabilitation.

Key messages

What is already known on this topic

⇒ Poststroke emotionalism (PSE) is common and is experienced by at least some patients throughout the first year after stroke. However, high-quality longitudinal studies of the prevalence, trajectory and psychological associations of PSE are lacking.

What this study adds

⇒ Using statistical modelling to adjust for participant drop-out, the Testing Emotionalism After Recent Stroke (TEARS) study establishes PSE is present in nearly a quarter of stroke survivors, is not simply a manifestation of mood disorder and while some people show recovery over time, many do not.

How this study might affect research, practice and/or policy

⇒ Targeted PSE screening in stroke clinical settings and high-quality clinical trials of pharmacological and non-pharmacological interventions are needed. Both should use the TEARS study diagnostic measures.

INTRODUCTION

Poststroke emotionalism (PSE) is a common, socially debilitating, distressing and neglected stroke sequela. Spontaneous uncontrolled crying may occur as a result of external stimuli, or less commonly without triggers. Episodes of crying can happen frequently and are often disproportionate to events and thoughts.[1 2]

PSE typically arises following strokes which disrupt frontal lobe and descending corticobulbar-cerebellar brain circuitry. It is thought such lesions impede the ability of the cerebellum to modulate the normal motoric expression of emotion. Dysfunctional serotonergic and glutaminergic neurotransmission in the cerebellum may also play a mechanistic role by reducing volitional control over emotional expression. The precise pathophysiology and neuroanatomy of PSE has yet to be fully elucidated.[3–5]

High-quality longitudinal PSE prevalence studies are comparatively uncommon. In a recent synthesis of the evidence, Gillespie identified fifteen eligible studies involving 3391 stroke participants,[6] far fewer than reviews of poststroke depression and anxiety.[7 8] PSE prevalence in this meta-analysis was 20%, but only four studies sampled community participants beyond 6 months, only two followed participants to 1 year and none attempted statistical modelling to adjust for participant drop-out, leading to potentially confounding prevalence estimations.[3] This lack of longer-term follow-up data creates an evidence gap around the natural history of PSE and our understanding of the potential for improvement or relapse over time.

Current understanding of the specific association of PSE to psychological factors is also limited.

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Feelings of sadness often do not accompany crying episodes and many people with PSE do not have a diagnosable depressive disorder.[9–11] While clinically PSE is often characterised by irritability, mental intrusion and avoidant coping, existing data are cross-sectional with small case numbers so cause and effect cannot be concluded.[10 12] This lack of basic observational research inhibits thinking about causation and constrains the development of novel preventive measures and treatments. Survey data show that health professionals use cognitive behavioural techniques to help,[13] yet there are no proven non-pharmacological PSE interventions and high quality data on antidepressant treatments are lacking.[14]

This study was designed to: (1) determine PSE point prevalence at 2 weeks, 6 and 12 months poststroke, adjusting for attrition bias and (2) to explore the association of PSE with psychological and disability-related variables over time.

METHODS

The data underlying this article will be shared on reasonable request to the corresponding author and retired to the Virtual International Stroke Trials Archive.

Participants

Participants were recruited prospectively on 1 October 2015 to 30 September 2018, from acute stroke units. All participants were male or non-pregnant female, ≥18 years of age, with clinical stroke diagnosis. Individuals with subarachnoid haemorrhage, other extra-axial bleeds, transient ischaemic attack, severe concurrent medical conditions (metastatic cancer or cardiac failure with severe pulmonary odema), life expectancy ≤3 months, without spoken English or who had severe distressing behaviours secondary to stroke or dementia (hallucinations, delusions) were excluded.

Participants gave written informed consent. Individuals who lacked capacity or with aphasia on Frenchay Aphasia Screening Test (score <25)[15] were included in the wider Testing Emotionalism After Recent Stroke (TEARS) cohort but excluded from this prevalence study as TEARS-Diagnostic Interview (TEARS-IV)[16 17] was not completed (figure 1).

Measures

PSE diagnosis was made at 2 weeks, 6 and 12 months using TEARS-IV by pretrained research nurses[16 17] with TEARS- Questionnaire (TEARS-Q) as a supplementary PSE measure. Hospital Anxiety and Depression Scale (HADS)[18] evaluated mood symptoms, with Impact of Events Scale-Revised (IES-R)[19] and Social Ties Checklist (STC)[20] included at 6 and 12 months. Abbreviated Mental Test (AMT) determined cognition at baseline,[21] with disability-related measures of Barthel Index (BI),[22] National Institute of Health Stroke Scale (NIHSS)[23] and Euro-Qol (EQ-5D),[24] (figure 1).

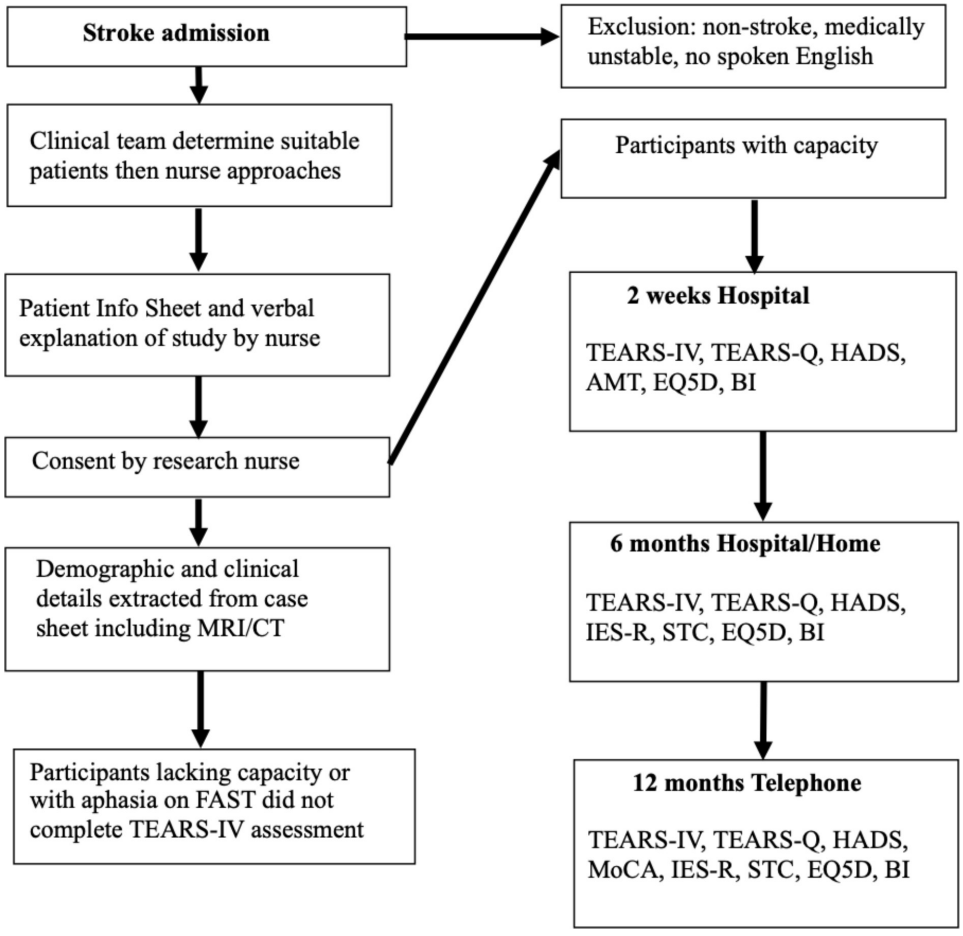

Figure 1 Study measures at each assessment time point. BI, Barthel Index; FAST, Frenchay Aphasia Screening Test; HADS, Hospital Anxiety and Depression Scale; IES-R, Impact of Events Scale-Revised; STC, Social Ties Checklist; TEARS-IV, Testing Emotionalism After Recent Stroke-Diagnostic Interview; TEARS-Q, TEARS-Questionnaire; MoCA, Montreal Cognitive Assessent; EQ5D, Euro-QoL.

Analysis plan

All statistical analyses were performed using R software.[25] Crude prevalence was the proportion of participants with PSE interviewed at each time point. Conditional prevalence (6 months) was the proportion of PSE cases at 6 months based on participant numbers with and without PSE at baseline, with prevalence estimated according to baseline prevalence to account for missing 6-month interviews. The same calculation method provided 12-month conditional PSE rates, based on 6-month observations (PSE present, absent or missing).

Cross-tabulation and follow-up statistics allowed comparison of participants with and without PSE on baseline sex, education, stroke type, stroke classification, BI and AMT. Associations between PSE and psychological and disability measures were determined using cross tabulation, equivalent to simple linear regression with t-test (for HADS-A, HADS-D, STC, BI, NIHSS), Mann-Whitney U test (IES-R components and total, AMT) or χ^2 test (EQ5D mobility, self care, usual activities, pain/discomfort, anxiety depression, overall health ; previous PSE state) for unadjusted statistics of association.

RESULTS

Sample size and characteristics

There were 277 participants in the TEARS cohort from nine stroke services. TEARS-IV interviews from 228 baseline participants were available, with 49 participants not receiving TEARS-IV. The final sample of 228 enabled prevalence estimation within 2.7% SE, assuming prevalence of 20% and the binomial distribution to establish CIs.

Baseline characteristics for the 228 included and 49 non-assessed baseline participants are in table 1. Median participant age at stroke onset was 67 years, 43.0% were female, 91.7% had sustained ischaemic stroke with mean NIHSS score of 3. Participants not receiving baseline TEARS-IV were older with more total and partial anterior strokes and higher disability.

Complete 6-month data were available from 159 participants with 118 not assessed, for 12 months 83 participants provided complete assessment data with 194 not assessed (figure 2). Of the 49 participants not assessed at baseline, 15 were assessed at 6 months while 4 participants not assessed at 6 months were assessed at 12 months. No participants were only assessed at 12 months.

Crude prevalence

Crude point prevalence rates were baseline 27.2% (62/228), 6 months 20.1% (32/159) and 12 months 14.4% (12/83) (figure 2).

Twenty participants had PSE acutely which remitted by 6 months, a further 10 remitted by 12 months. Thirteen participants with no PSE acutely developed it by 6 months, with six participants developing PSE by 12 months (table 2).

Conditional 6-month prevalence

Participants with PSE at baseline (40.3%) were less likely to receive interviews at 6 months than those without PSE (35.5%) (tables 2 and 3). We, therefore, calculated point prevalence at 6 months conditional on baseline PSE status.

For those who were assessed at baseline, the proportion without PSE at baseline who had PSE at 6 months was 12.2% (13/107) while for participants with PSE at baseline, the proportion with PSE at 6 months was 46% (17/37). For participants not assessed at baseline, PSE prevalence at 6 months was 13.3% (2/15).

Using these rates, we estimated the number of participants with PSE at 6 months who were not assessed. For the 59 participants without PSE at baseline not assessed at 6 months, this was $59\times0.1215=7.05$. For the 25 participants with PSE at baseline not assessed at 6 months, this was $25\times0.4595=11.49$. For the 34 participants not assessed at baseline and 6 months, this was $34\times0.1333=4.53$.

Conditional on baseline status, the number of participants with PSE at 6 months was therefore 32 (observed) plus $7.05+11.49+4.53=55.07$ of the 277 TEARS cohort, indicating 6 months conditional prevalence of PSE was 19.9%. Further refinement based on age, sex and deprivation (Scottish Index of Multiple Deprivation) had no impact on conditional 12-month prevalence estimate.

Conditional 12-month prevalence, on 6-month status

For participants without PSE at 6 months, PSE presence at 12 months was 7.8% (5/64). For participants with PSE at 6 months, PSE prevalence at 12 months was 40.0% (6/15). For participants not assessed at 6 months, PSE prevalence at 12 months was 33.3% (1/3).

Conditional on 6-month status and using the same method, 12-month conditional prevalence of PSE was 22.3%. Further refinement based on age, sex and deprivation had no impact on conditional 12-month prevalence estimate.

Sensitivity check

We estimated prevalence based on the assumption that all missing patients would have been PSE negative at assessment. Prevalences were 22.4% (baseline), 11.6% (6 months), 4.3% (12 months) and these represent the lower limits of our estimates of the frequency of PSE after stroke.

Baseline associations with disability-related and psychological variables

Using cross tabulation and follow-up statistics (table 4), no significant group differences were observed between participants with versus without PSE on sex, education, stroke type, stroke classification, BI and AMT, EQ-5D mobility, self-care, usual activities or pain.

The PSE group were significantly younger with more depression and anxiety on HADS, and on EQ5D, and poorer overall health on EQ5D (table 4).

Six-month associations with disability-related and psychological variables

No significant group differences in association were observed for 6-month BI or HADS-D across baseline PSE status, nor for EQ-5D mobility, self-care, pain or usual activities (table 5).

At 6 months, participants with baseline PSE reported significantly more anxiety on HADS-A, greater distress about the time they experienced the stroke on IES-R (IES-R total, IES-R Intrusion, IES-R Hyperarousal and IES-R Avoidance), more social ties on STC and more anxiety/depression on EQ-5D.

DISCUSSION

The TEARS cohort is typical of research stroke populations with 90% ischaemic stroke and a younger age group with milder stroke severity and disability. The data confirm PSE is common and while some people showed recovery over time, many did not. Diagnostic status fluctuated with some individuals having remission of PSE and others developing PSE.

Table 1 Characteristics of assessed (included) and non-assessed participants at baseline

Characteristic	Levels	Assessed	Not assessed	P value
N of participants	Total	228	49	
Age at stroke median (IQR)		67.0 (54.0–76.0)	74.00 (61.00–83.00)	0.004
Sex (%)	Female	98 (43)	24 (49)	0.543, NS
	Male	130 (57)	25 (51)	
Education (%)	Primary	5 (2.3)	1 (2.2)	0.032
	Secondary	148 (67.3)	31 (67.4)	
	University	34 (15.5)	8 (17.4)	
	Other	30 (13.6)	2 (4.3)	
	Unknown	3 (1.4)	4 (8.7)	
Stroke type	Infarct	209 (91.7)	40 (81.6)	0.085, NS
	Haemorrhage	18 (7.9)	8 (16.3)	
	Unknown	1 (0.4)	1 (2.0)	
Stroke classification (%)	Total Anterior Circulation Stroke (TACS)	10 (4.5)	12 (25.5)	<0.001
	Partial Anterior Circulation Stroke (PACS)	76 (34.5)	25 (53.2)	
	Lacunar Stroke (LACS)	79 (35.9)	5 (10.6)	
	Posterior Circulation Stroke (POCS)	54 (24.5)	5 (10.6)	
	Unknown	1 (0.5)	0 (0.0)	
NIHSS median (IQR)		3.00 (2.00–5.50)	6.00 (3.75–10.5)	0.016
Barthel median (IQR)		18.00 (14.00–20.00)	13.00 (5.00–18.00)	<0.001
AMT median (IQR)		9.00 (8.00–9.00)	9.00 (9.00–9.25)	0.252, NS
HADS Anx median (IQR)		5.00 (2.00–8.00)	4.00 (4.00–8.00)	0.692, NS
HADS Dep median (IQR)		3.00 (2.00–6.00)	2.00 (2.00–5.00)	0.599, NS
EQ5D mobility (%)	1	70 (31.1)	1 (16.7)	0.821, NS
	2	76 (33.8)	2 (33.3)	
	3	42 (18.7)	1 (16.7)	
	4	15 (6.7)	1 (16.7)	
	5	22 (9.8)	1 (16.7)	
EQ5D self care	1	107 (47.8)	4 (66.7)	0.667, NS
	2	71 (31.7)	1 (16.7)	
	3	23 (10.3)	0 (0.0)	
	4	16 (7.1)	1 (16.7)	
	5	7 (3.1)	0 (0.0)	
EQ5D usual activities	1	56 (24.9)	2 (33.3)	0.947, NS
	2	73 (32.4)	1 (16.7)	
	3	37 (16.4)	1 (16.7)	
	4	30 (13.3)	1 (16.7)	
	5	29 (12.9)	1 (16.7)	
EQ5D pain/discomfort	1	117 (52.0)	3 (50.0)	0.895, NS
	2	56 (24.9)	1 (16.7)	
	3	30 (13.3)	1 (16.7)	
	4	16 (7.1)	1 (16.7)	
	5	6 (2.7)	0 (0.0)	
EQ5D Anx/Dep	1	136 (60.4)	3 (50.0)	0.625, NS
	2	51 (22.7)	1 (16.7)	
	3	27 (12.0)	2 (33.3)	
	4	6 (2.7)	0 (0.0)	
	5	5 (2.2)	0 (0.0)	
EQ5D overall health median (IQR)		65.00 (50.00–80.00)	75.00 (72.50–84.00)	0.114, NS

AMT, Abbreviated Mental Test; HADS, Hospital Anxiety and Depression Scale; NIHSS, National Institute of Health Stroke Scale; NS, not significant.

We saw no consistent evidence PSE associated with disability measures over time but evidence of association with symptoms of anxiety and event related distress, with minimal clinically important difference on HADS-A (>1.7) and IES-R (>4.4).[26 27] Our sample was relatively mild stroke and as PSE associates with stroke severity[28] we may underestimate prevalence. Our baseline age difference between assessed and non-assessed participants and exclusion of aphasia might also contribute to this.

Our findings replicate Gillespie although our reported prevalence reduced smoothly over time. We used TEARS IV, which

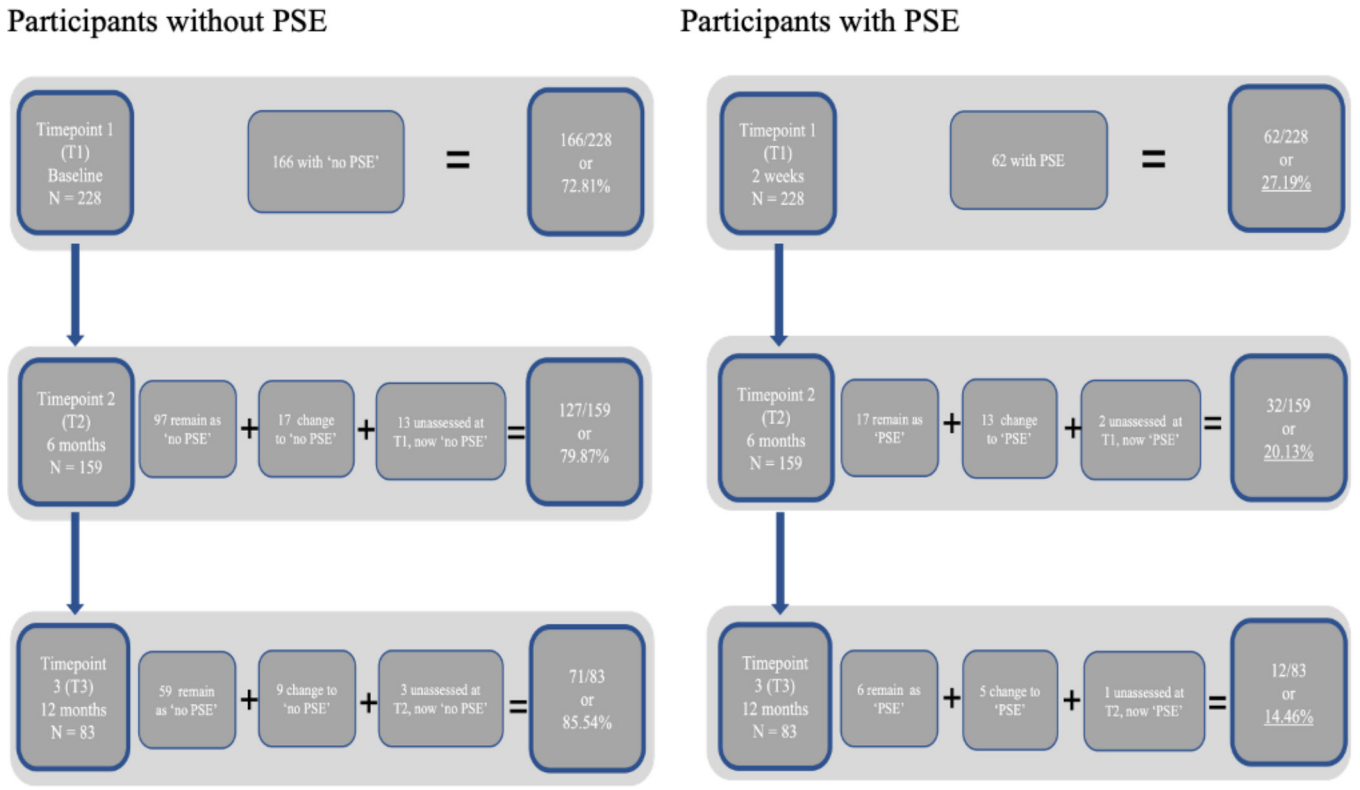

Figure 2 Flow of participants classified with and without PSE, across assessment points. PSE, poststroke emotionalism.

might explain this. Future longitudinal prevalence studies should use TEARS-IV. We opted to report crude and conditional prevalence and the difference in observed and calculated rates was influenced by the high drop-out rate which is not unusual in non-intervention cohort studies.[29] Our analyses reported elsewhere show drop out from TEARS was associated with older age and worse cognition but not emotionalism.[30]

Clinical implications of the prevalence data are obvious. Reliable, targeted screening is needed across acute and community stroke pathways although it will be important to show that emotionalism screening changes outcomes.[16 17] High-quality clinical trials must be prioritised to determine how to treat PSE effectively and safely. The current evidence is of very low quality, insufficient to definitively guide practice.[13 14] Our prevalence data on the natural history of PSE can inform trial design and analyses.

Our observation that PSE status is associated with anxiety and event related distress symptoms is interesting. It supports previous reports of avoidance and uncertainty in PSE[10 12 31 32] and suggests elevated anxiety may be a relevant psychological factor and potential treatment target to improve PSE psychosocial outcomes. It might also explain the rapid anxiolytic effect of antidepressant medicines to treat PSE in clinical practice.

Table 2 Transition chart for participants with and without PSE, baseline to 6 and 6–12 months

		6 months			
Baseline		No PSE	PSE	Not assessed	Total
	No PSE	94	13	59	166
	PSE	20	17	25	62
	Not assessed	13	2	34	49
		12 months			
6 months		No PSE	PSE	Not assessed	Total
	No PSE	59	5	63	127
	PSE	9	6	17	32
	Not assessed	3	1	114	118

PSE, poststroke emotionalism.

Table 3 Prevalence of participant PSE over time

Baseline	6 months	12 months	Frequency
PSE	PSE	PSE	3
PSE	PSE	No PSE	4
PSE	No PSE	PSE	1
PSE	No PSE	No PSE	3
PSE	Not assessed	No PSE	3
PSE	Not assessed	PSE	1
PSE	PSE	Not assessed	10
PSE	No PSE	Not assessed	16
PSE	Not assessed	Not assessed	21
No PSE	PSE	PSE	2
No PSE	PSE	No PSE	4
No PSE	No PSE	PSE	4
No PSE	No PSE	No PSE	47
No PSE	No PSE	Not assessed	43
No PSE	PSE	Not assessed	7
No PSE	Not assessed	Not assessed	59
Not assessed	PSE	PSE	1
Not assessed	PSE	No PSE	1
Not assessed	No PSE	No PSE	9
Not assessed	No PSE	Not assessed	4
Not assessed	Not assessed	Not assessed	34
Total N			277

PSE, poststroke emotionalism.

Table 4 Characteristics of baseline assessed participants, stratified by PSE diagnosis

Characteristic	Levels	Total responses by variable	No PSE	PSE	P value
N of participants			**166**	**62**	
Age at stroke median (IQR)		228	70.00 (59.00–77.75)	58.50 (50.25–68.75)	<0.001
Sex (%)	Female	228	65 (39.2)	33 (53.2)	0.079, NS
	Male		101 (60.8)	29 (46.8)	
Education (%)	Primary	220	3 (1.9)	2 (3.4)	0.322, NS
	Secondary		103 (64.0)	45 (76.3)	
	University		28 (17.4)	6 (10.2)	
	Other		24 (14.9)	6 (10.2)	
	Unknown		3 (1.9)	0 (0.0)	
Stroke type	Infarct	228	150 (90.4)	59 (95.2)	0.474, NS
	Haemorrhage		15 (9.0)	3 (4.8)	
	Unknown		1 (0.6)	0 (0.0)	
Stroke classification (%)	Total Anterior Circulation Stroke (TACS)	220	8 (5.0)	2 (3.4)	0.577, NS
	Partial Anterior Circulation Stroke (PACS)		59 (36.6)	17 (28.8)	
	Lacunar Stroke (LACS)		53 (32.9)	26 (44.1)	
	Posterior Circulation Stroke (POCS)		40 (24.8)	14 (23.7)	
	Unknown		1 (0.6)	0 (0.0)	
NIHSS median (IQR)		83	4.00 (1.00–5.00)	3.00 (2.75–6.25)	0.428, NS
Barthel median (IQR)		226	18.00 (15.00–20.00)	18.00 (13.00–20.00)	0.450, NS
AMT median (IQR)		224	9.00 (9.00–9.00)	9.00 (8.00–9.00)	0.076, NS
HADS Anx median (IQR)		225	4.00 (1.00–7.00)	8.00 (4.00–12.00)	<0.001
HADS Dep median (IQR)		225	3.00 (1.00–5.00)	5.00 (3.00–9.00)	<0.001
EQ5D mobility (%)	1	225	53 (32.3)	17 (27.9)	0.881, NS
	2		54 (32.9)	22 (36.1)	
	3		32 (19.5)	10 (16.4)	
	4		10 (6.1)	5 (8.2)	
	5		15 (9.1)	7 (11.5)	
EQ5D self care (%)	1	224	79 (48.5)	28 (45.9)	0.147, NS
	2		57 (35.0)	14 (23.0)	
	3		14 (8.6)	9 (14.8)	
	4		9 (5.5)	7 (11.5)	
	5		4 (2.5)	3 (4.9)	
EQ5D usual activities (%)	1	225	44 (26.8)	12 (19.7)	0.148
	2		57 (34.8)	16 (26.2)	
	3		26 (15.9)	11 (18.0)	
	4		21 (12.8)	9 (14.8)	
	5		16 (9.8)	13 (21.3)	
EQ5D pain/discom (%)	1	225	87 (53.0)	30 (49.2)	0.163
	2		41 (25.0)	15 (24.6)	
	3		24 (14.6)	6 (9.8)	
	4		10 (6.1)	6 (9.8)	
	5		2 (1.2)	4 (6.6)	
EQ5D Anx/Dep (%)	1	225	114 (69.5)	22 (36.1)	<0.001
	2		33 (20.1)	18 (29.5)	
	3		14 (8.5)	13 (21.3)	
	4		3 (1.8)	3 (4.9)	
	5		0 (0.0)	5 (8.2)	
EQ5D overall health median (IQR)		225	70.00 (53.75–80.00)	60.00 (50.00–70.00)	0.007

AMT, Abbreviated Mental Test; HADS, Hospital Anxiety and Depression Scale; NIHSS, National Institute of Health Stroke Scale; NS, not significant; PSE, poststroke emotionalism.

Interestingly, while PSE status was associated with depression at baseline, this was at subclinical levels and did not persist over time. More research is needed to determine which psychological factors influence recovery and why some individuals have poorer outcomes.

Table 5 Six-month participant characteristics, stratified by baseline PSE

Characteristic	Levels	Total responses by variable	No PSE=166	PSE=62	P value
6-month diagnostic status (%)	No PSE	144	94 (87.9)	20 (54.1)	<0.001
	PSE		13 (12.1)	17 (45.9)	
Barthel median (IQR)		154	20.00 (18.00–20.00)	20.00 (18.00–20.00)	0.648, NS
HADS-Dep median (IQR)		149	4.00 (2.00–7.00)	5.50 (3.00–7.00)	0.074, NS
HADS-Anx median (IQR)		149	4.00 (1.00–8.00)	7.00 (3.00–11.25)	0.007
IES-R total median (IQR)		140	1.00 (0.00–10.50)	12.00 (3.00–28.00)	<0.001
IES-R avoidance median (IQR)		140	0.00 (0.00–3.50)	4.00 (0.00–14.00)	0.001
IES-R intrusion median (IQR)		140	0.00 (0.00–3.50)	5.00 (1.00–9.00)	<0.001
IES-R hyperarousal median (IQR)		140	0.00 (0.00–3.00)	4.00 (1.00–7.00)	<0.001
Social ties checklist median (IQR)		145	4.50 (4.00–6.00)	4.00 (3.00–5.00)	0.008
Euro-Qol EQ5D mobility (%)	1	149	38 (34.9)	14 (35.0)	0.715, NS
	2		31 (28.4)	9 (22.5)	
	3		27 (24.8)	12 (30.0)	
	4		10 (9.2)	5 (12.5)	
	5		3 (2.8)	0 (0.0)	
Euro-Qol EQ5D self care (%)	1	149	66 (60.6)	24 (60.0)	0.690, NS
	2		29 (26.6)	8 (20.0)	
	3		10 (9.2)	6 (15.0)	
	4		3 (2.8)	2 (5.0)	
	5		1 (0.9)	0 (0.0)	
Euro-Qol EQ5D usual activities (%)	1	149	31 (28.4)	7 (17.5)	0.150, NS
	2		40 (36.7)	15 (37.5)	
	3		20 (18.3)	13 (32.5)	
	4		11 (10.1)	5 (12.5)	
	5		7 (6.4)	0 (0.0)	
Euro-Qol EQ5D pain/discom (%)	1	149	43 (39.4)	13 (32.5)	0.764, NS
	2		27 (24.8)	11 (27.5)	
	3		16 (14.7)	9 (22.5)	
	4		18 (16.5)	5 (12.5)	
	5		5 (4.6)	2 (5.0)	
Euro-Qol EQ5D Anx/Dep (%)	1	149	62 (56.9)	15 (37.5)	0.041
	2		26 (23.9)	11 (27.5)	
	3		17 (15.6)	7 (17.5)	
	4		2 (1.8)	3 (7.5)	
	5		2 (1.8)	4 (10.0)	

HADS, Hospital Anxiety and Depression Scale; IES-R, Impact of Events Scale-Revised; NS, not significant; PSE, poststroke emotionalism.

Systematic review from our group shows that emotionalism presents more commonly in younger people.[33] Our finding of PSE association with younger age in the TEARS cohort would support this. While future research will be required to account for why, clinicians should consider specific interventions to prevent or manage the heightened likelihood of PSE in younger people.

Interestingly, there was no association of PSE with cognition contrary to previous work.[34] Also, the PSE group showed higher social ties at 6 months. Perhaps they became more supported by family and friends although this could be a chance finding as STC is not stroke validated.[20] We saw no consistent evidence PSE was associated with disability-related measures, perhaps because we lacked a severe stroke sample.

There are study limitations, highlighted elsewhere.[16 17] In the absence of any standardised alternative we developed TEARS-IV based on expert consensus diagnostic criteria. We determined prevalence in three different 'settings' (hospital ward 2 weeks, ward or participant home 6 months, telephone 12 months), so some variation might be due to this. We only assessed PSE to 12 months and some people may continue to improve, thus our data are not the complete picture and we extended our study across nine hospital sites, so our data are not consecutive referrals to one stroke unit. The sample was relatively young, predominantly mild stroke and all gave informed consent so generalisability to the full stroke population and across other countries and cultures can be questioned. Nevertheless, the TEARS population were broadly similar to an unselected Scottish stroke cohort.[35] Finally, due to incomplete follow-up data and the high level of potential confounder variables, we could not reliably determine the effect of antidepressant prescribing on PSE. Drop out may associate with not being prescribed antidepressants at baseline.

A high proportion of participants (49 at baseline) did not receive interviews and there was a high number of non-assessed cases throughout, although due to patient reasons (worse neurological deficits, more disability) rather than site team factors. Completion rates of NIHSS measure were low and it is unknown if non-assessed individuals had PSE or did not, recovered, died or refused (although their non-assessment did not associate with emotionalism). Older age is a known predictor of worse stroke

outcome[36] and as non-assessed cases were more severe stroke and presumably less likely to be assessed, this probably accounts for the age difference between assessed and non-assessed cases observed. The difference in education status between assessed and non-assessed participants is more complex. This may be a chance finding but the established link between education status and poorer outcome after stroke[37] might have precluded assessment by an interview.

Despite our best efforts to meaningfully include people with aphasia and cognitive impairment, the numbers of such individuals were small as we had to balance inclusive recruitment against participant burden and feasibility of assessment. The true population prevalence of emotionalism is likely to be higher than we report and dedicated research will be needed to elucidate precise PSE prevalence in these important groups. Similarly, we opted not to report 12-month associations due to the low numbers and to reduce participant burden. We only collected IES-R and STC data at 6 and 12 months, precluding certain comparisons. Finally, we do not report PSE laughter prevalence. This is much less common[3] and clinically usually presents with pseudobulbar palsy (bilateral upper motor neuron weakness) in what is termed pseudobulbar affect. Importantly, PSE arises following strokes of many differing types including unilateral lesions and across a wider range of brain areas.[38]

Nevertheless, the data confirm that PSE affects at least one in five patients who had a stroke acutely and at least one in eight longer term. We observed PSE associations with anxiety and event-related distress, not depression, over time which could worsen the emotional and psychosocial consequences of PSE by driving avoidance and uncertainty. These factors should be a target for future research including novel non-pharmaceutical interventions for this common stroke sequela.[10]

Twitter Terence J Quinn @DrTerryQuinn

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ORCID iDs
Niall McIntyre Broomfield http://orcid.org/0000-0003-2599-3435
Terence J Quinn http://orcid.org/0000-0003-1401-0181

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