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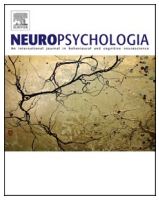

The association of brain injury severity with dream cessation and nightmares

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ABSTRACT

Mesial frontal and temporoparietal brain areas differ in activity between high and low dream recallers, and lesion studies have found cessation of dreaming in patients with damage to these areas. This study extends these findings by assessing the relationship of dream cessation, and dream and nightmare frequencies, to severity of injury in patients at a head trauma clinic. 52 participants (M = 36, F = 16; mean age = 39.69, SD = 13.72) completed a questionnaire assessing frequency of dreams, nightmares and night terrors, tests for depression and anxiety, and a neuropsychological examination and test battery. 34.6 % of patients reported that they do not dream, which is comparable to Solms' (1997) finding of 34.9 % in his brain injury sample. This contrasts with the rate of not reporting dreams in the general population of 6.1–7.1 %. Dream cessation participants had significantly greater severity of brain injury as measured by the Glasgow Coma Scale and post traumatic amnesia, but cessation was not related to neuropsychological scores. Compared to the general population there was a higher prevalence of weekly nightmares (13.5 %) and also of reporting never having nightmares (48.1 %), the former predicted by anxiety, the latter by brain injury severity. The data cannot distinguish between whether brain injury is halting the production of dreams or causes failure to encode and recall dreams on waking. Future studies using within-sleep behavioral or imaging methods may distinguish between these possibilities. Future studies should also determine if recovery of dream experience is predictive of more global recovery from brain injury.

1. Introduction

1.1. Brain lesions and cessation of dreaming

Solms (1997, p.137) found that 34.9 % of patients with diffuse head injuries and lesions with other etiologies reported the global cessation of dreaming. Specifically, bilateral white-matter lesions in the ventromesial frontal region or lesions of the inferior parietal region of either hemisphere were associated with a cessation of dreaming. Solms (2000) reported 110 cases, published over the previous century, of loss of dreaming due to focal forebrain pathology. Solms divided these cases into two anatomical groups: 1) in or near the region of the parieto-temporo-occipital (PTO) junction, and 2) the ventromesial quadrant of the frontal lobe. He states that the latter accords with a 70–90 % incidence of complete or nearly complete loss of dreaming found in several large series of prefrontal leukotomy from the 1940s–1970s. Solms (2000) concludes that these case studies and series increase to almost 1000 the number of reported cases of cessation of dreaming caused by focal forebrain lesions. Reports of cessation of dreaming have been confirmed by Yu's (2007) findings for 21 cases with lesions to the mesial frontal lobes, 17 of whom (81.0 %) experienced a global cessation of dreaming. The reports have also been confirmed by awakenings in the sleep laboratory. Murri et al. (1985) found that for nineteen patients with unilateral hemispheric lesions, 12 reported no dream recall across 10 days, and, when sleep was interrupted in the sleep laboratory during both N2 and REM sleep, eight reported still having no dream recall. Before illness all of the participants were experiencing dream recall at least once a week.

1.2. Non-lesion findings in accord with Solms (1997, 2000)

Various non-lesion studies have supported Solms' (1997, 2000)

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conclusions about brain areas important for the organization of dreaming. On a between-subject basis, Eichenlaub et al. (2014a) showed that brain reactivity, measured by evoked potentials, is greater for high than low dream recallers during sleep and wakefulness. Eichenlaub et al. (2014b) then found that, compared with low dream recallers (mean of .5 dream recall per week), high recallers (mean of 5.2 dreams per week) have higher regional cerebral blood flow (rCBF) in the temporoparietal junction (TPJ) during REM sleep, N3 (Slow Wave) sleep, and wakefulness, and in the medial prefrontal cortex (MPFC) during REM sleep and wakefulness. They conclude that the TPJ and MPFC might promote the mental imagery (which would include informational and emotional content) and/or memory encoding of dreams. Furthermore, Vallat et al. (2018) found that high dream recallers have a higher MPFC white-matter density than do low recallers, which they state is consistent with Solms' (1997, 2000) findings that lesions within the white matter of MPFC are associated with a partial or total cessation of dream reporting. They suggest that this region is likely to have a role in producing dreams.

The two areas identified by Solms might overlap with the frontal area and posterior 'hot zone' found by Siclari et al. (2017) to change activity during sleep to higher frequency EEG on a within-subject basis for participants who then report dreaming on waking, compared to reporting that they had not been dreaming. However, Domhoff (2022) cautions that the high-density EEG method of Siclari et al. cannot characterize and show the importance of the frontal areas found by lesion and brain imaging methods.

1.3. Dream cessation and neurocognitive insights into the neurological organization of dreaming

Solms (1997, 2000) hypothesizes that the frontal brain area provides the informational, emotional and motivational content of dreams, and the posterior area the visual and imaginal component. For Solms (1997, p.245), the dream process begins with 'the curiosity-interest-expectancy (appetitive) circuits in the mediobasal frontal region'. This appetitive process does not activate motor systems, as would occur during wake, and instead 'proceeds regressively in the direction of perceptual hallucination' through occipito-temporal mechanisms (p.246). The hallucination is taken as real due to the weakened frontal self-reflective system. Solms concludes that dreaming is based on dopaminergic forebrain mechanisms and the reactivation of reward- and emotion-associated memories during sleep. These proposed processes are cited by Tsunematsu (2023) as contrasting to the view that dreams are generated by random brainstem activity which periodically stimulates the cortex (Hobson and McCarley, 1977).

Domhoff and Fox (2015) state that Solms' (1997, 2000) findings accord with their proposal that dreaming is dependent on two default network subsystems. In a meta-analysis of all brain imaging studies that compare REM sleep to quiet wakefulness, Fox et al. (2013) conclude that dreaming is an intensified form of daydreaming. Domhoff (2022) extends this to claim that dreaming results when anterior and posterior regions of the default network become more active, but with the dorsal attention network and salience/ventral network deactivated so as to allow for the development of the embodied hallucination of the dream. Further support is shown by Vallat et al.'s (2022) finding that connectivity between right lateral parietal and medial prefrontal cortex is significantly higher in high than low dream recallers. Domhoff's (2022) neurocognitive model of dreaming builds upon the overlap between these imaging studies and the lesion and dream cessation work of Solms.

1.4. Replicating and extending the dream cessation findings

Given the above neuroanatomical, lesion, and imaging evidence of forebrain structures being involved in dream production and/or recall, the first aim of the current study was to ascertain the frequency of cessation of dreaming in a sample of patients attending a brain injury clinic. As many attendees of the clinic would have suffered some trauma, causing the injury, and may have sleep or mood disturbances, the frequency of nightmares and night terrors was also assessed. The second aim was to assess for the Traumatic Brain Injury (TBI) patients whether frequencies of dreaming, nightmares, and night terrors are related to sleep quality, anxiety, depression, PTSD and neuropsychological variables, and, as a novel research question, whether severity of brain injury is related to dream cessation and to nightmare frequency.

1.4.1. Sleep quality

Sleep was assessed because individual differences in dream recall can arise due to differences in amount of wakefulness during the sleep period (see review by Simor et al., 2023). For example, Vallat et al. (2017) presented high and low dream recallers with sounds during a whole night of sleep in the lab and recorded polysomnographic data to assess sleep microstructure. They found longer awakenings occurred during the night for high recallers and concluded that the minimum necessary duration of an awakening during sleep for the successful encoding of dreams into long-term memory is approximately 2 min. Eichenlaub et al. (2014a) similarly found a higher level of wakefulness during the sleep period for high recallers, as did van Wyk et al. (2019) in specifically NREM sleep. Indeed, the lack of wakefulness in recovery sleep after sleep deprivation almost completely abolishes dream recall (De Gennaro et al., 2010), in contrast to the increased visual vividness of dreams that results from higher levels of intra-sleep wakefulness (Scarpelli et al., 2020).

The patient sample tested here is expected to have problems with sleep. The prevalence of sleep disorders following TBI is between 50 and 73 % and daytime sleepiness and sleep disturbance are among the most universal symptoms of TBI (Castriotta and Lai, 2001). They are also expected to have nightmares as well as sleep problems (Faerman et al., 2024; Viola-Saltzman and Watson, 2012). However, the relationship of dream and nightmare recall with sleep quality is unclear, because whereas dream recall is greater when there are awakenings in sleep, sleep needs to be sufficient to allow dreams and nightmares to form. For example, Solms found that his dreaming patients had less disturbed sleep than did his non-dreaming patients (1997, p.165).

1.4.2. Neuropsychological variables

For the neuropsychological variables, no prediction is made for a relationship with dream cessation, as Yu (2007) studied this and did not report such associations. The main prediction is that deficits in performance on frontal/executive tasks will be associated with nightmare frequency. Simor et al. (2012) found that individuals with frequent nightmares are impaired on executive tasks involving the suppression of task-irrelevant semantic representations, and Carr et al. (2018) replicated the finding of nightmare frequency being associated with fluency perseveration errors. Related to this, Marquis et al. (2019) found negative correlations between rCBF activity in the medial prefrontal gyrus during negative picture viewing and dysphoric dream distress and dysphoric dream frequency. The frontal impairment or lower activity is proposed to cause a deficit in inhibition of highly negative imagery and emotions in dreams.

1.4.3. Anxiety and depression

Depression has been reported in between 30 % and 60 % of patients with TBI (Douglas and Spellacy, 2000), with roughly 40 % meeting the DSM-IV criteria for a major depressive episode (Kreutzer et al., 2001). In addition, anxiety (Dehbozorgi et al., 2024; Miles et al., 2021) and PTSD (Bryant, 2001) can occur following TBI. There is also growing evidence that some of these neuropsychiatric disorders may be related to sleep disruption (Krystal, 2020). Anxiety, depression, low wellbeing, and stress or trauma are related to nightmare frequency (Blagrove et al., 2004; Wood et al., 1992).

1.5. Hypotheses

1. Following Solms (1997, 2000), this TBI sample would show a higher level of reporting no dream recall than occurs for the general population.
2. Dream cessation will be associated with severity of brain injury, and with anxiety, depression and poor sleep quality.
3. Due to the relationship of low wellbeing and stress or trauma with nightmares, some participants will show a higher incidence of nightmares than does the general population, but, following hypothesis 1, for other patients, brain injury may halt nightmares as a consequence of the cessation of dream production in general.
4. Frequency of nightmares will be positively associated with anxiety and neuropsychologically assessed frontal dysfunction (Simor et al., 2012) and negatively associated with severity of brain injury.
5. Occurrence of night terrors will be associated with anxiety (Kales et al., 1980; Llorente et al., 1992; Szelenberger et al., 2005).

To address hypotheses 1 and 3 dream and nightmare frequencies are compared to the dream and nightmare general population survey findings of Schredl and Göritz (2020), which has n = 2492 with a mean age of 47.75 (SD = 14.41) years, as well as other surveys.

2. Method

2.1. Participants

52 patients who had suffered a head injury (M = 36, F = 16; mean age = 39.69, SD = 13.72, min = 20, max = 77) were recruited from referrals to a head trauma clinic. 35 (67.3 %) of the participants had sustained head injuries in a road traffic collision; 3 (5.8 %) during an assault; 4 (7.7 %) from a fall; 2 (3.8 %) from a blow to the head; 2 (3.8 %) from neurological reasons, and 6 had no cause recorded. They were all considered, by referring clinicians, to have suffered a moderate or severe head injury, based on levels of consciousness (recorded by Glasgow Coma Scale, GCS; Teasdale and Jennett, 1974). The majority of participants were considered by referring clinicians to show neuropsychological signs of frontal dysfunction. All patients had evidence of posttraumatic amnesia and loss of consciousness. Some had CT or MRI scan evidence of contusional injuries to one or both frontal lobes. Ten patients reported being on medication: four on anti-depressants, five for epilepsy, and one for HIV.

Participants were given an information sheet regarding the study and its procedures. Informed consent was obtained from all participants. The study, its procedures and participant documents were approved by the institutional Research Ethics Committee and by the Local National Health Service Research Ethics Committee.

2.2. Injury severity

The Glasgow Coma Scale (GCS; Teasdale and Jennett, 1974) and duration of Post Traumatic Amnesia (PTA; Greenwood, 1997) were used as indices of injury severity. The GCS is a measure of depth of coma and is assessed during admission to hospital, lower scores indicate greater severity. The duration of PTA is the time from injury to the start of continuous memories. It is the sum of any time spent in coma or in confusion when individuals lack the capacity to remember ongoing events.

2.3. Materials

2.3.1. Dream, nightmare, and night terror questionnaire

Participants used a Likert scale to report on their frequency of dreams, nightmares, and night terrors. The questions were 'How often do you wake up and remember a dream?', How often do you have a nightmare?', and 'How often do you have night terrors?' There was no instruction to participants to consider only the post-injury period in answering the questions, as such an instruction would have had only minor effect on responses, given mean time since injury of 49.35 months (see Table 4) and response options presented. The five response options given for each question were: 'at least once a week'; 'at least once a month'; 'at least once a year'; 'less than once a year'; and 'never'. The categories 'less than once a year' and 'never' were used so as to be sure that those reporting non-dreaming do not just dream very rarely. Nightmares were defined as 'a vivid dream that is frightening or disturbing, the events of which you can remember clearly and in detail on awakening.' A night terror was defined as 'a sudden awakening in fear, possibly accompanied by a scream, but where you do not remember any dream.' After the nightmare frequency question participants used a 'yes/no' format to answer two items on nightmare distress and repetitive nightmares: 'Is having a nightmare a problem or concern for you?' and 'Have you recently had the same nightmare more than once?'

2.3.2. Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983)

Anxiety was assessed using the 7 items forming the anxiety scale of the Hospital Anxiety and Depression Scale. Scores of 8 and 11 are indicative respectively of possible and probable clinical levels of anxiety (Crawford et al., 2001).

2.3.3. Beck Depression Inventory for Primary Care (BDI-PC-7: Beck et al., 1996)

This is a 7-item self-report instrument for symptoms of depression, with scores from 0 to 21. Scores of four and above indicate clinical depression (Beck et al., 1997).

2.3.4. Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989)

This 18-item self-report questionnaire examines sleep quality and sleep disturbances for the previous month. Participants provide information such as usual bedtime, usual time of getting up, time taken to fall asleep, and the length of actual sleep per night. The questionnaire yields seven component scores and one global sleep quality score. Higher scores indicate poorer sleep quality. An overall score of 21 on the PSQI indicates the worst sleep quality. Fichtenberg et al. (2000) found for a sample of TBI patients that those with clinician assessed insomnia had considerably higher PSQI scores than those without (means of PSQI were 13.9 (SD, 3.3) and 3.5 (SD, 2.3) respectively). The cut-off global score for poor sleepers is 5 and above (Buysse et al., 1989) and for insomnia 9 and above (Fichtenberg et al., 2000).

2.4. Cognitive neuropsychological assessment

A battery of 34 neuropsychological tests was included in the study to assess whether IQ, memory, frontal/executive functioning and other cognitive abilities are predictive of dream cessation, nightmares or night terrors. The instruments were: Wechsler Adult Intelligence Scale (3rd edition) (WAIS III; Wechsler, 1997a); Wechsler Memory Scale (WMS III; Wechsler, 1997b); the Hayling and Brixton Tests (Burgess and Shallice, 1997), and the Zoo Map, Key Search and Verbal Fluency from the Behavioural Assessment of Dysexecutive Syndrome battery (BADS; Wilson et al., 1996). The frontal/executive tasks predicted to be associated with nightmare frequency were: Haylings A, Haylings B, Haylings C, Brixton, Key search, and Zoo map 2. Test details are in the Supplementary material.

2.5. Procedure

Patients attending the head trauma clinic were invited to take part in the research. It was emphasized to patients both verbally and in written information sheets that participation was voluntary and that the research was entirely separate from their ongoing care at the head trauma clinic. The objectives of the study were explained to participants

as well as what would be required of them. After giving consent participants were administered the dream, nightmare, and night terror questionnaire, the Pittsburgh Sleep Quality Index, HADS, BDI-PC-7 and the neuropsychological tests described above. The presence of PTSD was assessed during a clinical interview with a neuropsychologist.

2.6. Statistical analyses

Number and percentage of participants in each of the categories for dream recall, nightmare, and night terror frequencies were computed. Pearson or Spearman correlations were conducted between predictor variables and the dreaming, nightmare, and night terror variables. Mann-Whitney U tests were used to compare dream cessation with dreaming participants on the predictor variables. Binary and ordinal logistic regressions were then used to assess predictors for dream cessation, nightmare frequency, and night terrors. Analyses were conducted using SPSS Statistics version 29.0.2.0 (15), IBM Corp.

2.7. Data statement

Data for the study are publicly available at: https://doi.org/10.5281/zenodo.14996394.

3. Results

3.1. Frequency of dream cessation, nightmares, and night terrors

The first aim of the study was to assess the frequency of dream cessation and of nightmares and night terrors after traumatic brain injury. Table 1 shows that 34.6 % of participants reported that they never dream, and that 48.1 % report never having a nightmare. These frequencies are higher than for the comparison general population data, shown in the table, of 7.1 % and 19.4 % respectively, from Schredl and Göritz's (2020, N = 2492) survey. In addition, 13.5 % reported experiencing at least one nightmare per week, which is higher than the general population figure of 8.7 %. The majority of participants reported never having had a night terror.

Table 1
Dream recall, nightmare, and night terror frequencies (percentages and ns in each frequency category) for TBI patients (N = 52) and dream and nightmare survey data from Schredl and Göritz (2020, N = 2492).

	Never	less than once a year	at least once a year	at least once a month	at least once a week
Dreams					
TBI	34.6 % (18)	5.8 % (3)	13.5 % (7)	23.1 % (12)	23.1 % (12)
Survey	7.1 %	15.2 %[a]		23.6 %	54.0 %
Nightmares					
TBI	48.1 % (25)	9.6 % (5)	11.5 % (6)	17.3 % (9)	13.5 % (7)
Survey	19.4 %	17.5 %	32.4 %	22.0 %	8.7 %
Night terrors					
TBI	59.6 % (31)	9.6 % (5)	9.6 % (5)	9.6 % (5)	11.5 % (6)

Notes: [a] Survey category here for dream recall frequency was for dreaming 'less than once per month', hence combining two categories for the current study. Of the 10 participants taking medication, only two reported never recalling dreams, these were taking antiseizure medication. All participants taking antidepressants did report dreaming. Of the 16 participants reporting at least one nightmare a month, 68.8 % reported the occurrence of repetitive nightmares, and 50 % stated that having a nightmare is a problem or concern for them. The only significant correlation for these two dichotomous variables was between nightmares being a problem or concern and anxiety, Pearson's biserial r = .60, p = .014.

3.2. Predictors of dream cessation, nightmare frequency, and night terrors

The second aim of the study was to investigate predictors of dream cessation, nightmare frequency, and night terrors.

3.2.1. Descriptive statistics for predictor variables

There were no significant sex differences for the dream, nightmare frequency, night terror, or predictor variables. Details of the Mann-Whitney and Chi square tests conducted to assess sex differences can be seen in the Supplementary material.

The Glasgow Coma Scale and length of posttraumatic amnesia were used as indices of injury severity. The percentage of participants in each of the categories of length of post traumatic amnesia are shown in Table 2.

Table 3 presents Glasgow Coma Scale score percentage (and n) of participants in each of the Glasgow Coma Scale severity categories (Mena et al., 2011).

Table 4 presents descriptive statistics for the sample.

34 participants (65.4 %) were classed as poor sleepers, using a PSQI cut-off of 5 (Buysse et al., 1989), and 17 (32.7 %) were classified as having insomnia, using a cut-off of 9 (Fichtenberg et al., 2000), but with a caveat for the insomnia estimate as a score of 9 can occur by accumulating a range of other symptoms that are not necessarily related to insomnia. Scores on HADS Anxiety was higher than for the general population. The sample mean exceeded the cut-off of 8 for 'possible' clinical levels of anxiety, 51.9 % met or exceeded the score of 11, indicative of a 'probable' diagnosis of anxiety (Crawford et al., 2001). Similarly, the mean score on the BDI-PC-7 indicates that the level of depression is elevated in this sample. 75.0 % of the sample scored at least 4, the criterion for depression (Beck et al., 1997). Six participants (11.54 %) were considered to have PTSD in their clinical interview with a neuropsychologist. Details of the neuropsychological battery and battery mean scores are in the Supplementary material.

Table 2
Percentage (and n) of participants in each of the length of posttraumatic amnesia categories (n = 50).

None	<1 h	<24 h	<1 week	<2 weeks	>2 weeks
4.0 % (2)	8.0 % (4)	14.0 % (7)	10.0 % (5)	24.0 % (12)	40.0 % (20)

Note: Length of PTA was not available for two participants.

Table 3
Percentage (and n) of participants in each of the Glasgow Coma Scale severity categories (n = 45).

Severe ≤8	Moderate 9–13	Minor 14–15
31.1 % (14)	28.9 % (13)	40 % (18)

Note: GCS score was not available for seven participants.

Table 4
Mean, standard deviation, maximum and minimum of Glasgow Coma Scale (GCS), age at injury, time since injury, sleep quality index (PSQI), HADS Anxiety, and BDI-PC-7 Depression variables for all participants (N = 52).

	Mean	SD	Min	Max
Glasgow Coma Scale (GCS)	10.91	4.24	3	15
Age at injury (years)	35.37	14.15	16	64
Time since injury (months)	49.35	36.13	9	177
Sleep Quality Index (PSQI)	8.29	5.22	2	21
HADS Anxiety	10.46	4.98	0	21
BDI-PC-7 Depression	7.15	4.73	0	21

3.2.2. Bivariate correlations between predictor variables and dream, nightmare, and night terror variables

PTA, GCS, PSQI, and HADS Anxiety had significant correlations with dream and nightmare variables, $|r_{sp}s|$ ranged from .30 to .53; PTA, PSQI, and HADS Anxiety had significant correlations with night terrors, $|r_{sp}s|$ ranged from .32 to .53. Details of these correlations are in the Supplementary material. Specifically, there were significant correlations between dream recall frequency and each of PTA (r_{sp} = -.42, p = .002, n = 50) and GCS (r_{sp} = .53, p < .001, n = 45), indicating that as injury severity increases dream recall frequency decreases. Depression, sex, age, time since injury, and all neuropsychological variables had no significant correlations with dream recall, nightmare frequency or night terror variables (summary of these correlation statistics is included in the Supplementary material).

3.3. Comparison of dream cessation and dreaming groups

As the primary interest of the study is the characteristics of the dream cessation group, means (SDs) of this group and of those who do report dreaming are presented in Table 5.

Table 5 shows that, compared to the dreaming group, the dream cessation group had significantly worse brain injury severity on both severity measures, and had lower anxiety and better sleep quality. There were no significant differences between dream cessation and dreaming groups on the neuropsychological measures, all zs < 2.11 and the majority of zs < 1.0.

3.4. Binary and ordinal logistic regressions for dream cessation, nightmare frequency, and night terrors

Binary and ordinal logistic regressions were conducted to determine what percentage of the variance in presence/absence of dreaming, nightmare frequency, and presence/absence of night terrors were accounted for by the predictor variables: GCS, PTA, PSQI, and anxiety. Depression, sex, age, time since injury and all neuropsychological variables were excluded as none of these had significant correlations with dream recall, nightmare frequency or night terror variables.

GCS and PTA were significantly correlated with $r_{sp} = -.80$, p < .001, and Variance Inflation Factor (VIF) = 2.02. Although the VIF does not meet common thresholds for multicollinearity the high correlation does evidence multicollinearity (Vatcheva et al., 2016). Regressions were thus calculated with GCS and PTA separately, which also avoids further data loss given that the PTA variable has n = 50 and the GCS variable has n = 45.

Table 6
Binary logistic regression predicting the likelihood of dream cessation after traumatic brain injury, with injury severity assessed by length of post trauma amnesia.

	B	SE	Wald	df	p	OR	95 % CI OR	
							LL	UL
PTA	−.50	.28	3.05	1	.081	.61	.35	1.06
Anxiety	.14	.08	3.19	1	.074	1.15	.99	1.34
PSQI	.06	.08	.68	1	.410	1.06	.92	1.23
Constant	.634	1.63	.15	1	.698	1.88		

Notes: PTA - Length of post trauma amnesia; PSQI – Pittsburgh Sleep Quality Inventory. Dream cessation coded 0, Dreaming coded 1.

Table 7
Binary logistic regression predicting the likelihood of dream cessation after traumatic brain injury, with injury severity assessed by the Glasgow Coma Scale.

	B	SE	Wald	df	p	OR	95 % CI OR	
							LL	UL
GCS	.25	.10	6.60	1	.010	1.28	1.06	1.54
Anxiety	.09	.09	1.06	1	.304	1.10	.92	1.30
PSQI	.08	.07	1.23	1	.268	1.09	.94	1.25
Constant	−3.58	1.36	6.95	1	.008	.03		

Notes: GCS – Glasgow Coma Scale; PSQI – Pittsburgh Sleep Quality Inventory.

3.4.1. Predicting dream cessation

The binary logistic regression model using length of PTA to predict dream cessation was statistically significant (Chi square (df = 3, n = 50) = 12.35, p = .006). The model explained between 21.9 % (Cox & Snell R square) and 30.3 % (Nagelkerke R square) of variance in dream cessation and correctly identified 76.0 % of cases. See Table 6 for regression statistics.

The binary logistic regression model using the Glasgow Coma Scale to predict dream cessation was statistically significant (Chi square (df = 3, n = 45) = 14.89, p = .002). The model explained between 28.2 % (Cox & Snell R square) and 38.7 % (Nagelkerke R square) of variance in dream cessation and correctly identified 80.0 % of cases. See Table 7 for regression statistics. Dream cessation was predicted only by GCS score.

3.4.2. Predicting nightmare frequency

As with the above binary regressions, ordinal regressions for nightmare frequency were conducted with length of post trauma amnesia and Glasgow Coma Scale separately.

The ordinal logistic regression model using length of post trauma amnesia to predict nightmare frequency was statistically significant (Chi square (df = 3, n = 50) = 24.869, p < .001), and with high goodness-of-

Table 5
Glasgow Coma Scale (GCS), post trauma amnesia (PTA), HADS Anxiety, BDI-PC-7 Depression, and Pittsburgh Sleep Quality Inventory (PSQI) scores for dream cessation and dreaming groups.

	Dream cessation (n = 18)		Dreaming (n = 34)		Mann-Whitney U, z	p
	Mean	SD	Mean	SD		
GCS[a]	8.063	3.94	12.48	3.57	100.00, 3.21	.001
PTA[b]	4.35	1.17	3.24	1.56	156.00, 2.66	.008
Anxiety	7.94	4.89	11.79	4.55	180.00, 2.43	.015
Depression	5.89	3.72	7.82	5.11	421.00, 1.08	.280
PSQI	6.22	4.75	9.38	5.19	351.50, 2.42	.015

Notes: [a] Dream cessation, n = 16, Dreaming, n = 29; [b] Dream cessation, n = 17, Dreaming, n = 33.

Table 8
Ordinal logistic regression predicting nightmare frequency after traumatic brain injury, with injury severity assessed by length of post trauma amnesia (PTA).

	B	SE	Wald	df	p	OR	95 % CI OR	
							LL	UL
PTA	−.618	.207	8.892	1	.003	.54	.36	.81
Anxiety	.246	.079	9.761	1	.002	1.28	1.10	1.49
PSQI	.015	.061	.059	1	.809	1.02	.90	1.14

Notes: PTA - Length of post trauma amnesia; PSQI – Pittsburgh Sleep Quality Inventory.

Table 9
Ordinal logistic regression predicting nightmare frequency after traumatic brain injury, with injury severity assessed by Glasgow Coma Scale.

	B	SE	Wald	df	p	OR	95 % CI OR	
							LL	UL
GCS	.251	.092	7.423	1	.006	1.29	1.07	1.54
Anxiety	.163	.081	4.084	1	.043	1.18	1.01	1.38
PSQI	.058	.061	.897	1	.344	1.06	.94	1.19

Notes: GCS – Glasgow Coma Scale; PSQI – Pittsburgh Sleep Quality Inventory.

fit (Pearson statistic = 153.396, df = 181, p = .933). The model explained between 39.2 % (Cox & Snell R square) and 41.7 % (Nagelkerke R square) of variance in nightmare frequency. See Table 8 for regression statistics. Nightmare frequency was predicted by length of PTA and anxiety.

The ordinal logistic regression model using the Glasgow Coma Scale to predict nightmare frequency was statistically significant (Chi square (df = 3, n = 45) = 20.922, p < .001), and with high goodness-of-fit (Pearson statistic = 177.339, df = 173, p = .395). The model explained between 37.2 % (Cox & Snell R square) and 39.7 % (Nagelkerke R square) of variance in nightmare frequency. See Table 9 for regression statistics. Nightmare frequency was predicted by GCS score and anxiety.

3.4.3. Predicting night terrors

Due to more than half of the participants reporting never having had a night terror, for the regression the sample was divided between those who reported never having had one, and those who had had at least one. The binary logistic regression model using length of post trauma amnesia to predict whether a night terror had ever been experienced was statistically significant (Chi square (df = 3, n = 50) = 18.29, p < .001). The model explained between 30.6 % (Cox & Snell R square) and 41.2 % (Nagelkerke R square) of variance in night terror incidence and correctly identified 72.0 % of cases. See Table 10 for regression statistics. Likelihood of a night terror was predicted only by anxiety.

The model using the Glasgow Coma Scale was statistically significant (Chi square (df = 3, n = 45) = 17.95, p < .001). The model explained between 32.9 % (Cox & Snell R square) and 44.0 % (Nagelkerke R square) of variance in night terror incidence and correctly identified 75.6 % of cases. See Table 11 for regression statistics. Likelihood of a night terror was predicted only by anxiety.

Table 10
Binary logistic regression predicting the likelihood of a night terror after traumatic brain injury, with injury severity assessed by length of post trauma amnesia.

	B	SE	Wald	df	p	OR	95 % CI OR	
							LL	UL
PTA	−.39	.25	2.55	1	.110	.68	.42	1.09
Anxiety	.26	.10	7.29	1	.007	1.30	1.07	1.57
PSQI	.08	.07	1.04	1	.309	1.08	.93	1.25
Constant	−2.53	1.51	2.80	1	.094	.08		

Notes: PTA - Length of post trauma amnesia; PSQI – Pittsburgh Sleep Quality Inventory.

Table 11
Binary logistic regression predicting the likelihood of a night terror after traumatic brain injury, with injury severity assessed by the Glasgow Coma Scale.

	B	SE	Wald	df	p	OR	95 % CI OR	
							LL	UL
GCS	.07	.10	.50	1	.480	1.07	.89	1.29
Anxiety	.32	.11	8.04	1	.005	1.37	1.10	1.71
PSQI	.08	.08	1.13	1	.287	1.08	.94	1.26
Constant	−5.24	1.76	8.87	1	.003	.01		

Notes: GCS – Glasgow Coma Scale; PSQI – Pittsburgh Sleep Quality Inventory.

4. Discussion

4.1. Dream cessation

34.6.% of patients reported that they did not dream, which is comparable to Solms' (1997, p.137) finding of 34.9 % for global cessation of dreaming in his lesions sample. This contrasts with the rate of not reporting dreams in general populations of 7.1 % in Schredl and Göritz (2020) and 6.1 % in Borbely (1984; cited in Strauch and Meier, 1996), and 6.5 % in a sample of sleep clinic patients (Pagel, 2003, study 1, mean age = 58.9). This finding confirms the first hypothesis, that the clinical sample would show a higher level of reporting no dream recall than occurs for the general population. On univariate analyses absence of dreaming was significantly related to severity of brain injury on both severity measures, GCS and PTA, and to anxiety and sleep quality. Absence of dreaming was not related to scores on frontal or cognitive tests, which accords with Yu (2007) relating dream cessation in 17 of his 21 patients to specific caudate lesion areas and not to executive function or memory tasks. Absence of dreaming was also not related to depression. Under binary regression, absence of dreaming was significantly associated with severity of injury as assessed by the Glasgow Coma Scale, with an odds ratio of 1.28, so that as injury is milder (GCS score higher) the likelihood of dreaming is higher. The relationship of dream cessation to length of amnesia after injury had an odds ratio of .61 (p = .081), the longer the amnesia the less likelihood of dreaming. Length of amnesia and Glasgow Coma Scale score were significantly related to dream recall frequency (r_{sp}s = -.42 and .53 respectively). This may indicate suboptimal mechanisms for dream production in some patients, which would suggest that reduced frequency of dreaming might be a subclinical form of global cessation of dreaming, a possibility noted by Solms (1997, p.149), but also a view that he argues against on the grounds that reduced frequency of dreaming has no localizing significance (1997, p.216). These findings partially confirm our second hypothesis, with dream cessation being associated on univariate analyses with GCS and PTA measures of severity of brain injury, and with anxiety and sleep quality, but only with GCS score under regression.

Solms (1997, p.145) reports that recovery from cessation of dreaming takes approximately a year for cases with posterior lesions, but that cessation persists if damage is elsewhere, including in the frontal and parietal lobes. All but four of our patients had had their injury over a year before assessment for this study, and so, given the level of cessation of dreaming, it may be that posterior damage was not prominent in our sample.

4.2. Nightmares

In the current study 14.5 % of participants reported having at least one nightmare per week. This is much higher than for the general population. For example, Sandman et al. (2013) found in a meta-analysis that approximately 2–5 % of the general adult population experience nightmares at least once per week. They also found in their

study of 69,813 participants that 3.5 % of men and 4.8 % of women report having nightmares at least once per week. A weekly nightmare prevalence of 8.7 % was found by Schredl and Göritz (2020), which is again less than for our sample.

In the current study 48.1 % of participants reported never having nightmares. For Levin (1994), 83 % of adults have at least one nightmare per year, and so 17 % have less than one nightmare per year, which is similar to the 19.4 % who never have nightmares in Schredl and Göritz (2020). The current sample thus had far higher incidences of weekly and of never reporting nightmares than for the general population. This confirms our third hypothesis, that some participants will show a higher incidence of nightmares than does the general population, but that for other participants, brain injury may halt nightmares as a consequence of the cessation of dream production in general.

Solms (1997, p.212) had found for a sample of 114 cases with brain injury or disorder that 9 had recurring nightmares and 17 non-recurring nightmares, these combining to 22.8 % of his sample. This is comparable to the 30.8 % having monthly or weekly nightmares in the current sample. Our finding that 68.8 % of those with at least monthly nightmares reported the occurrence of repetitive nightmares is, however, higher than for Solms, and may be due to the preponderance of head injuries due to traumatic events. Our sample had a higher mean level of anxiety than does the general population, which may explain the high incidence of frequent nightmares, given the extensive literature on associations of nightmares with anxiety and also with psychopathology and stressful or traumatic incidents (Blagrove et al., 2004; Wood et al., 1992). As said above, this high prevalence of frequent nightmares was coupled with many patients reporting never having nightmares, which may be because of the need for dream production still to be occurring if a nightmare is to ensue. In line with this, the ordinal regression results confirmed the fourth hypothesis that frequency of nightmares will be positively associated with anxiety and negatively associated with severity of brain injury.

However, the study did not confirm the hypothesized relationship of nightmare frequency with neuropsychologically assessed frontal dysfunction. The latter result thus does not accord with the findings of Simor et al. (2012) and conclusions of Marquis et al. (2019). However, in a conceptual replication of Marquis et al. (2019), Carr et al. (2020) found that frontal activity has a negative relationship with dysphoric dream distress but not dysphoric dream frequency, and Marquis et al. (2019) did find the relationship of medial prefrontal activity to be stronger with dysphoric dream distress than with frequency. Nightmare distress is a trait measure of various waking life negative reactions to having nightmares and can be a confound for apparent correlates of nightmare frequency, as cautioned by Blagrove et al. (2004). However, nightmare distress was assessed in Carr et al. (2018) and when partialled out did not remove the significant correlation between fluency perseverations and nightmare frequency. It may thus be that for non-brain injured participants such as in the above studies there is a relationship between lower frontal activity/impaired executive functions and nightmare frequency, but that this does not hold for participants with brain injury. Relevant to this is the finding that executive function was not a statistically significant predictor of treatment outcomes for nightmare frequency and distress in a sample with trauma-related nightmares (Rischard and Cromer, 2019). Nevertheless, Faerman et al. (2024) found, for mild brain injury participants, that nightmare frequency in the previous two weeks was associated with impulsivity although not self-reported executive dysfunction. Although the Behavioural Assessment of the Dysexecutive Syndrome (BADS) is a valid measure for distinguishing brain injured from non-brain injured samples, and for measuring executive dysfunction (Norris and Tate, 2000), the validity of executive tests may vary across different neurological populations and/or levels of severity (Wood and Liossi, 2006). Further replications are thus needed in brain injured samples of the relationship of nightmare frequency and nightmare distress to measurable executive impairment.

4.3. Night terrors

21.1 % of participants reported having night terrors at least once per month. This contrasts with the incidence of 3.5 % at least once monthly for adults in the general population (Hublin et al., 1999). Confirming hypothesis 5, only anxiety predicted incidence of night terrors, which accords with Llorente et al. (1992) and Kales et al. (1980), and with the conclusion of Leung et al. (2020) that whereas psychological factors are not frequent in children with night terrors they are common in adults with this disorder.

4.4. Deficits in dream production versus dream encoding and retrieval

There has been debate about whether the dream cessation findings of Solms mean that it is dream production or dream retrieval that is impaired by brain injury. Solms (1997, pp.34-35, 159–160) reviews the conflicting findings in previous studies and in his data on whether dream cessation might be related to short term memory deficits. Murri et al. (1985) and Solms (1997) found that dreamers and non-dreamers did not differ on spatial and verbal short-term memory tests and on a spatial long-term memory test. Furthermore, Bischof and Bassetti's (2004) case study of a 73-year old woman reporting cessation of dreaming showed that she had short-term and long-term memory scores in the normal range. Her dream loss lasted for over three months after a stroke, and in that time she had normal sleep architecture with preservation of REM sleep. She did not report dreaming after repeated awakenings from REM sleep. For the study here, there was no significant relationship between dream cessation and short or long term visual or verbal memory, which accords with Vallat et al.'s (2022) finding of no significant difference between high and low dream recallers in memory ability.

However, despite these findings, the results and methods here still cannot distinguish between whether this cessation is due to a deficit in dream production, or a memory or attention deficit which may leave dreams, although unrecalled, still occurring and even having a role in putative within-sleep emotional or memory processing functions. Nemeth (2023) distinguishes dream production (the generation of a conscious experience during sleep), dream encoding (storing a trace of this experience in memory) and dream retrieval (accessing the memory trace upon awakening). Nemeth argues that attention differences rather than production factors may cause some differences in dream recall between REM and NREM sleep, and between high and low dream recallers. For example, Blain et al. (2022) used a challenging auditory task during which participants were subjected to distraction. The study showed attentional differences between high and low dream recallers.

This question was also addressed by Vallat et al. (2020), who had participants in the sleep laboratory read a comic before having a nap, with no explicit instructions to memorize the comic. Dream(s) and comic recall were collected after waking. Functional connectivity at 5 min post-awakening showed enhanced connectivity in high dream recallers within the default mode network (DMN) and between regions of the DMN and regions involved in memory processes. High recallers differed from low recallers in dream recall, but not in comic recall. Vallet et al. state that this is the first evidence that brain functional connectivity just after awakening is associated with interindividual trait differences in dream recall and suggest that the brain connectivity of high recallers at awakening facilitates the maintenance of the short-term memory of the dream during the sleep-wake transition. There may thus be a DMN based memory difference between high and low dream recallers that does not extend to memory for material presented during wakefulness. It thus remains possible that the dream cessation findings in the current study, and in Solms and others, arise from an encoding and retrieval deficit when awakening, rather than a deficit in dream production.

4.5. Limitations

The main limitation of the current study is that the reporting of a

complete lack of dream recall was not confirmed by instrumental awakenings in the sleep laboratory. However, Pagel (2003, study 3) studied 16 individuals who reported never having experienced dreaming (termed non-dreamers), and 12 individuals for whom it had been longer than 1 year since their last experience of dreaming (termed rare-dreamers). Both groups underwent a night of clinical polysomnography with spontaneous and instrumental awakenings to elicit dream reports. The non-dreamers group had 36 awakenings in total. None of the non-dreamers reported an awareness of dreaming after any awakening. The rare-dreamers group had 32 awakenings, and three participants each reported one dream. The groups did not differ on polysomnographic variables. Pagel's sleep laboratory results thus confirm the lack of dreaming reported by his non-dreamer group and by most of the rare-dreamers, and suggest that the cessation of dreaming reported by questionnaire in the current study may be a true reflection of a complete lack of dream recall on waking for our participants.

The second limitation is that we did not have CT or MRI data for many of the patients and so have not been able to include locus of injury in the analyses.

The third limitation is that we did not collect data on dream recall frequency prior to brain injury, or data on whether patients consider that their dream recall has changed due to the injury. It may be that some of our sample were non-dreamers or rare-dreamers before their injury. Nevertheless, the incidence of non-dreaming found here after injury greatly exceeds the incidence that would be expected in a non-injured sample.

The fourth limitation is that the sample size precluded adding to the regressions some of the predictor variables that did not have significant bivariate relationships with the dream, nightmare and night terror variables. Even if not directly associated with the dependent variables, they may act as confounders or moderators, and their exclusion might have led to an oversimplified model or biased estimates.

The final limitation concerns ten of the participants reporting being on medication. However, this does not seem to have affected the overall dream cessation findings as only two of these ten reported never dreaming. These two were from the five participants taking antiseizure medications. From these data and the literature it is unlikely that our dream cessation results have been confounded by medication. For antidepressants, tricyclics can decrease dream recall but this effect is less clear for SSRIs/serotonin norepinephrine reuptake inhibitors (SNRIs) (Tribl et al., 2013). And for antiseizure medications, the literature does not report an effect on dream recall (Charpentier-Hélary et al., 2025).

4.6. Future research

There is ongoing debate about whether dreaming has an emotion or memory processing function for the sleeping brain (Blagrove et al., 2019; Bloxham and Horton, 2024; Hudachek and Wamsley, 2023) or no function at all (Flanagan, 2000). The possibility that dreaming has some function for the brain during sleep means that it is important to ascertain whether people who report a complete lack of dreaming are not producing dreams or are instead just failing to recall them. It is challenging currently to separate the measurement of the dreaming experience during sleep from the post-waking recall of that experience. Extensions of current behavioural (Konkoly et al., 2021) and imaging (Dresler et al., 2011) methods may be able to test for the presence of dream experience during sleep, separate from the recall of that experience. It would then be possible to test whether dream cessation in TBI individuals is due to a failure of dream production or of dream encoding and retrieval.

Changes in dream recall frequency may provide useful indicators of the course and quality of recovery from head trauma, reflecting changes in cerebral functioning, and hence future studies should address whether asking patients about dream recall frequency may be clinically useful in predicting psychological aspects of recovery.

CRediT authorship contribution statement

Sam Fisher-Hicks: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Data curation, Conceptualization. **Victoria Lovett:** Writing – review & editing, Writing – original draft, Validation, Formal analysis, Data curation. **Rodger Ll Wood:** Writing – review & editing, Supervision, Resources, Project administration, Methodology. **Mark Blagrove:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Formal analysis, Conceptualization.

Appendix A. Supplementary material

Supplementary material to this article can be found online at https://doi.org/10.1016/j.neuropsychologia.2025.109209.

Data availability

Data for the study are publicly available at: https://doi.org/10.5281/zenodo.14996394

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