Title
Self-Reported Severity and Causes of Traumatic Brain Injury in Patients With Epileptic or Functional Seizures

Journal
Neurology Clinical Practice, 12(6)

ISSN
2163-0402

Authors
Hickman, L Brian
Patel, Akash B
Dubey, Ishita
et al.

Publication Date
2022-12-01

DOI
10.1212/cpj.0000000000200098

Peer reviewed

RESEARCH ARTICLE

Self-Reported Severity and Causes of Traumatic Brain Injury in Patients With Epileptic or Functional Seizures

L. Brian Hickman, MD, MSc, Akash B. Patel, DO, Ishita Dubey, BS, Amir H. Karimi, BA, Xingruo Zhang, BS, BA, Emily A. Janio, MPH, Corinne H. Allas, BS, Siddhanka S. Sreenivasan, BA, Janar Bauirjan, RN, BSN, Shannon D'Ambrosio, BS, Mona Al Banna, MD, Andrew Y. Cho, MS, Jerome Engel, Jr., MD, PhD, John M. Stern, MD, and Wesley T. Kerr, MD, PhD

Correspondence
Dr. Kerr
wesleytk@ucla.edu

Neurology: Clinical Practice December 2022 vol. 12 no. 6 e189-e198 doi:10.1212/CPJ.0000000000200098

Abstract

Background and Objectives
Although moderate and severe traumatic brain injury (TBI) can cause posttraumatic epilepsy (PTE), many patients with functional seizures (FS) also report a history of mild TBI. To determine whether features of TBI history differ between patients with epileptic seizures (ES) and FS, we compared patient reports of TBI severity, symptoms, and causes of injury.

Methods
We recruited patients undergoing video-EEG evaluation for the diagnosis of ES, FS, mixed ES and FS, or physiologic seizure-like events at an academic, tertiary referral center. Patients and their caregivers were interviewed before final video-EEG diagnosis regarding their TBI histories, including concussive symptoms and causes of injury.

Results
Of 506 patients, a greater percentage of patients with FS reported a history of TBI than patients with ES (70% vs 59%, aOR = 1.75 [95% CI: 1.00–3.05], $p = 0.047$). TBI with loss of consciousness (LOC) lasting less than 30 minutes was more frequently reported among patients with FS than with ES (27% vs 13%, aOR = 2.38 [1.26–4.47], $p < 0.01$). The proportion of patients reporting other neurologic symptoms immediately after TBI was not significantly different between FS and ES (40% vs 29%, $p = 0.08$). Causes of TBI were found to differ, with TBIs caused by falls from a height (17% vs 10%, aOR = 2.24 [1.06–4.70], $p = 0.03$) or motor vehicle collisions (27% vs 11%, aOR = 2.96 [1.54–5.67], $p < 0.01$) reported more frequently in FS than ES.

Discussion
Our findings further the association of mild TBI with FS and prompt reconsideration of typical assumptions regarding the significance of a reported TBI history in patients with previously undifferentiated seizures. Although common in both groups, TBI with LOC less than 30 minutes and causes of injury that are commonly believed to be more severe were reported more frequently in FS than ES. This suggests that a patient or caregiver reporting of these features does not imply that PTE is a more probable diagnosis than FS. Although a history of TBI with LOC and presumed high-risk causes of injury intuitively raises suspicion for PTE, clinicians should be cautioned that these historical factors also were a frequent finding in patients with FS.

Department of Neurology (LBH, ID, AHK, XZ, CHA, SSS, JE, JMS, WTK), David Geffen School of Medicine at UCLA, Los Angeles, CA; Department of Internal Medicine (LBH), University of California at Irvine, Irvine, CA; Department of Psychiatry and Biobehavioral Sciences (ABP, EAJ, JB, SDA, MAB, AYC, JE, WTK), University of California Los Angeles, Los Angeles, CA; Department of Neurobiology (JE), David Geffen School of Medicine at UCLA, Los Angeles, CA; Brain Research Institute (JE), University of California Los Angeles, Los Angeles, CA; and Department of Neurology (WTK), University of Michigan, Ann Arbor, MI.

Funding information and disclosures are provided at the end of the article. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

Up to 30% of patients admitted for video-electroencephalographic (vEEG) monitoring were diagnosed with functional seizures,[1] otherwise known as psychogenic nonepileptic or dissociative seizures.[2,3] While epileptic seizures (ES) occur from electrophysiologic abnormalities, the pathophysiologic basis of FS involves alterations in motor and emotion-processing networks within the brain.[4] FS can present with involuntary dissociation and motor activity that may appear similar to an ES.[5] Factors associated with FS include comorbid medical and psychiatric disease, female sex, and psychological stressors, including a history of abuse.[6] The treatment of FS includes cognitive behavioral informed therapy.[7,8] Delays to diagnosis and treatment are associated with worse prognosis.[9] Therefore, a timely diagnostic distinction between functional and ES is paramount to improve outcomes.

Traumatic brain injury (TBI) is a common cause of new-onset epilepsy in adults. However, mild TBI is also associated with FS and other functional neurologic disorders.[10-14] Most TBIs in patients with FS are mild, as defined by the classification scheme for TBI by Annegers et al.,[15] which defines "mild" as a head injury resulting in limited neurologic sequela such as dizziness, nausea, amnesia, or loss of consciousness (LOC) lasting less than 30 minutes.[10] By contrast, LOC greater than 30 minutes, skull fractures, and intracranial hematomas/contusions are defined as moderate or severe using this classification method. Prior investigations have found that patients with FS may attribute head trauma as the underlying cause of their seizures.[12,16] Investigations of military veterans, an enriched population for the occurrence of TBI, likewise demonstrated that mild TBI had greater associations with FS, whereas moderate or severe TBI carried a greater association with epilepsy.[10] High-risk causes of injury such as motor vehicle collisions (MVCs) and falls from a height[17] were more likely to (but do not necessarily) cause severe TBI, resulting in structural damage and posttraumatic epilepsy (PTE).[18,19]

In this investigation, we evaluated whether specific patient-reported features of TBI could differentiate between epileptic and FS in a sample of patients admitted to a vEEG monitoring unit. We hypothesized that features associated with greater severity TBI—including LOC and high-velocity causes—would suggest PTE. By contrast, we hypothesized that mild TBIs, less injurious causes, and symptoms of postconcussive syndrome may be more associated with FS.

Methods

We investigated all patients admitted to the UCLA adult vEEG monitoring unit from May 2015 to March 2020. We required that diagnostic certainty meets the International League Against Epilepsy (ILAE) criteria for "documented" and used expert clinical assessment that integrated clinical history, physical examination, ictal and interictal vEEG, and neuroimaging.[20] We placed patients into one of 5 mutually exclusive categories: FS, physiologic seizure-like events (PSLEs), ES, mixed seizures, and inconclusive monitoring. PSLE was defined as events caused by identifiable physiologic factors including syncope, movement disorders, migraine with aura, dementia, and tremors. Mixed seizures referred to patients displaying comorbid FS and ES and represent an understudied subgroup.[21] Inconclusive monitoring was determined when patients did not experience all typical seizure types during monitoring and did not qualify for "documented" diagnostic certainty; inclusion of these patients reduces selection bias and improves modeling of confounding variables (e.g., age and sex) while not otherwise affecting conclusions regarding other diagnostic categories. When patients with inconclusive monitoring were readmitted for vEEG and met ILAE criteria for documented diagnostic certainty, we updated the categorization.

Standard Protocol Approvals, Registrations, and Patient Consents

Patients were prospectively recruited and consented for inclusion at the time of the first vEEG admission. The UCLA Institutional Review Board (IORG0005364-IRB#00006450) approved this investigation before patient recruitment. All patients provided written consent for participation, including the use of their records in research, and the investigation was conducted in accordance with the Declaration of Helsinki.

Standardized Interview

All data on TBI were acquired using standardized, in-person interviews conducted with trained research assistants or a neurology resident. Interviews were conducted within 48 hours of vEEG admission. Aside from age and sex, interviewers were blinded to the patients' medical records and evaluation, including results of vEEG monitoring. Although all patients were admitted to the adult vEEG unit, 3 patients were younger than 18 years (15, 17, and 17 years). Analysis of other portions of this interview has been published elsewhere.[6,22-24]

During patient interviews, we inquired about head injuries with open-ended questions. Based on patient/caregiver descriptions of head trauma, we graded severity using standardized definitions. Concussion was identified in cases of any immediate neurologic symptom or LOC, according to the American Academy of Neurology guidelines definition of concussion.[25] Mild TBI and concussion were defined as equivalent. LOC was further specified based on the Veterans Health Administration grading system of less than 30 minutes, between 30 minutes and 24 hours, or greater than 24 hours, with the latter inclusive of sedation used to maintain LOC because of the severity of the injury.[26] When patients did not know the exact duration of LOC, we used external cues to estimate the duration. If a patient regained consciousness before ambulance arrival, the duration of LOC was presumed to be less than 30 minutes. When patients regained awareness in the emergency department, a duration of 30 minutes to 24 hours was presumed. When patients reported regained awareness after multiple days, we estimated LOC greater than 24 hours. We also asked whether patients were informed of any objective trauma-associated brain injuries on neuroimaging. When the head injury was a consequence of a seizure that involved LOC (e.g., tonic-clonic seizure with a fall), we did not assess the duration or presence of LOC because we would be unable to distinguish LOC caused by the seizure or TBI.

To further delineate severity, we inquired about the cause of injury including fall from a standing height, fall from a height above standing, MVC, sports-induced, or violent trauma. We asked patients about symptoms persisting beyond 1 week after TBI to suggest postconcussive syndrome.[27] If patients had multiple TBIs of varying severity, each severity was recorded, resulting in, for example, some patients having a history of TBI with both less than 30 minutes and greater than 24 hours of LOC.

We also posed the open-ended question: "What was going on in your life around when your seizures started?" When patients attributed their seizures to a particular head injury or reported a head injury occurring within a year of seizure onset, we coded the head injury as precipitating seizure disorder onset. When considering the seizure disorder onset, we excluded early seizures occurring within 7 days of injury because these are less associated with the development of epilepsy.[15] Precise duration of time between TBI and onset of seizures was not otherwise determined. Patients were also asked whether they had ever received a diagnosis of depression, anxiety disorder, or posttraumatic stress disorder (PTSD) from a medical professional. Consistent with a typical neurologic interview, these reported psychiatric conditions were not separately confirmed using formal psychiatric interview or questionnaires. The remainder of the interview inquired about other factors related to FS and ES including psychological trauma (see Supplemental Material, links.lww.com/CPJ/A375).

Assessed Variables

Extracted variables used in both descriptive statistics and individual-level statistics encompassed sex, age, reported lifetime TBI, reported TBI precipitating seizures, LOC from reported TBI, duration of LOC from TBI (<30, >30 minutes to 24, >24 hours or coma), other neurologic symptoms from TBI (concussion symptoms), prolonged post-TBI symptoms, complex TBI (skull fracture or imaging abnormality), causes of TBI (fall from the ground level, elevated fall, MVC, sports-induced, or violent trauma), reported psychiatric diagnoses (depression, anxiety disorder, or PTSD), physical abuse, and military service.

Statistical Modeling

We analyzed the clinical information about head injuries using both population-level descriptive statistics and individual-level inferential statistics. Analyses were performed in MATLAB (R2021a). Descriptive statistics were performed to assess the association between a specific feature of head injury and a particular seizure etiology at a population level. By contrast, inferential statistics were performed to assess how informative features of head injuries are for predicting FS compared with ES (see Supplement for modeling equations, links.lww.com/CPJ/A375). We controlled for patient sex and age in all analyses.

Descriptive analyses were first completed assessing for main effects without interaction terms, then repeated while separately assessing for interactions with sex and psychiatric diagnoses. Unless otherwise specified, descriptive comparisons were made in reference to ES using indicator terms. The cutoff p-value for statistical significance was set at 0.05.

For individual-level inferential statistics, we used multivariate logistic regression to interpret whether the contribution of each individual historical factor was conditionally independent of other studied factors. After fitting the full model, we recursively removed the single feature with the least significant Wald statistic until all variables had Wald $p < 0.1$, also known as recursive feature elimination (RFE) or backward selection.[28] This data-driven feature selection was performed to generate a succinct model of the features of TBI that had an association with FS or ES, controlling for confounds for which we had sufficient data. We trained our model on patients with either FS alone or ES alone and assessed performance using leave-one-out cross-validation. Instead of reporting positive and negative predictive values, we report the predictive value of ES (compared with FS) because our sample lacks negative controls. When patients reported multiple TBIs of differing severity, those patients were included in analyses of both levels of severity. This matches logistic regression's assumption of conditional independence, which evaluates the influence of each finding when all others are kept constant. For patients reporting both mild and severe TBIs, their risk of ES was estimated based on the sum of the log odds associated with mild TBI and log odds associated with severe TBI.

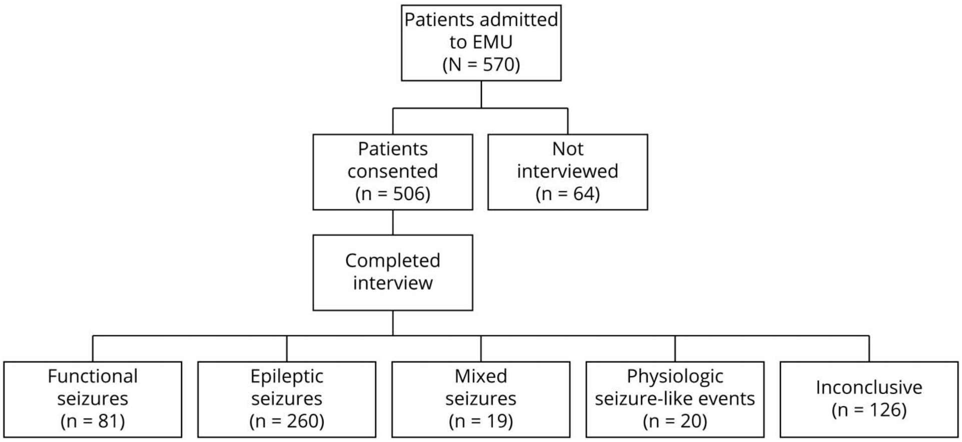
Data Availability

Deidentified raw data, code, and relevant standardized interview coding forms will be made available at SeizureDisorderCenterResearchGroup.org at the time of publication and will be maintained for the following one year. Additional documentation or information is available by request.

Results

Of 506 patients consented for inclusion, 81 were categorized as FS, 260 as ES, 19 as mixed seizures, 20 as PSLE, and 126 as inconclusive testing (Figure 1). There were no missing data points for any of the included variables.

Figure 1 Patient Enrollment and Categorization



During enrollment, 570 patients were admitted for video-EEG monitoring for diagnostic workup or presurgical planning. Of these, 506 patients consented and participated in the study. The remainder of patients declined enrollment or were not enrolled because of limitations in interviewer availability. All patients who consented completed the full interview.

Population-Level Differences

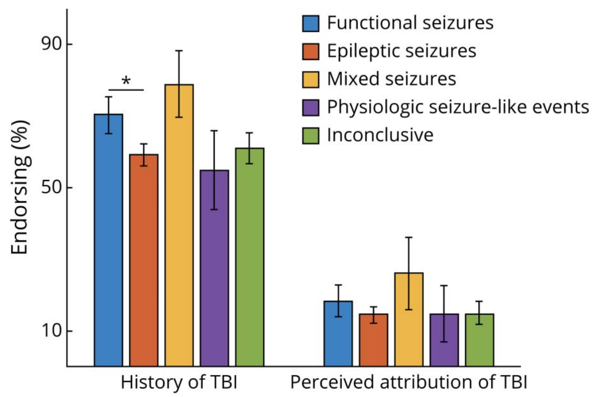
Patients with FS were older than patients with ES (mean 40.9 years vs 37.1 years, β = 4.13 [0.43–7.84], $p = 0.03$) and were more likely to be female (75% vs 50%, aOR = 3.19 [1.78–5.50], $p < 0.01$, eTable 1, links.lww.com/CPJ/A375). Patients with inconclusive testing were more likely to be older (mean 42.6 years, β = 5.94 [2.79–9.11], $p < 0.01$) and female (70%, aOR = 3.73 [1.20–11.55], $p < 0.01$) than patients diagnosed with epilepsy. Compared with patients with epilepsy, patients with mixed seizures were more likely to be female (79%, aOR = 3.73 [1.20–11.55], $p = 0.02$).

There was no significant difference between males and females in reporting a history of TBI (67% of males vs 59% of females, $p = 0.09$). Men were more likely than women to report a history of TBI as a precipitating cause of their seizures (20.4% vs 12.8%, OR = 1.74 [95% CI 1.07–2.81], Fisher exact $p = 0.03$) and to report a history of TBI from sports participation (17.4% vs 7.2%, OR = 2.70 [95% CI 1.53–4.76], Fisher exact $p < 0.01$). Other features of TBI history did not significantly differ between sexes.

More patients with FS reported a history of TBI compared with patients with ES (FS: 70%, ES: 59%, aOR = 1.75 [1.00–3.05], $p = 0.047$, Figure 2). Patients with FS or mixed seizures were not more likely to have a TBI precipitating seizure onset compared with patients with ES (FS: 19%, ES: 15%, $p = 0.36$; mixed: 26%, $p = 0.07$, respectively).

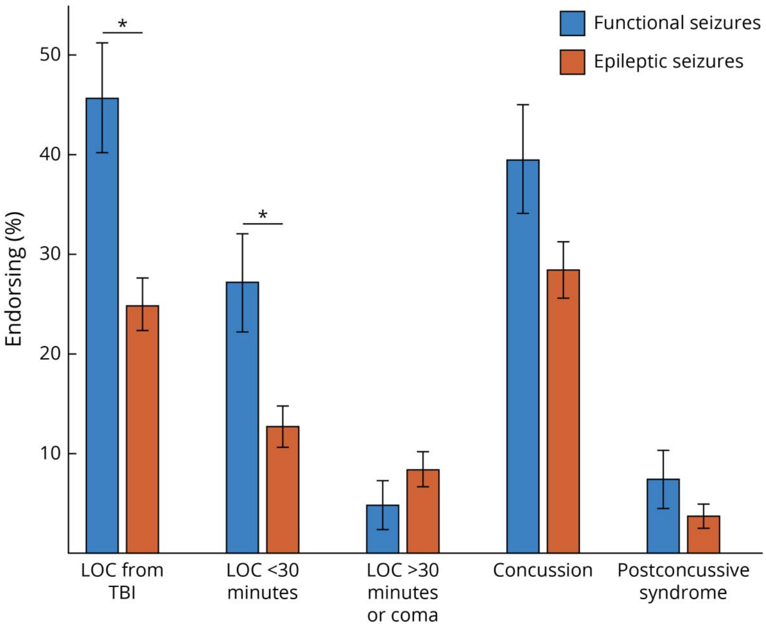
We next assessed the relationships between severity of TBI and type of seizure (Figure 3). Patients with FS were more likely to report LOC after TBI than patients with ES (46% vs 25%, aOR = 2.54 [1.48–4.34] $p < 0.01$). This remained significant when controlling for TBI precipitating seizure onset, which was measured including the onset of seizures within one year from TBI. When asked about duration of LOC, 27% of patients with FS reported LOC less than 30 minutes while 13% of patients with ES reported this (aOR = 2.38 [1.26–4.47], $p < 0.01$). There was no significant difference in reported LOC between 30 min to 24 hours, more than 24 hours, and in reporting coma between patients with FS and ES; this remained nonsignificant when considering coma or LOC greater than 30 minutes as one category (FS: 4.9%, ES: 8.5%, $p = 0.32$). However, the absolute number of patients reporting prolonged LOC was small in each group. The difference in the rate of immediate post-TBI concussive symptoms (i.e., confusion, amnesia, nausea, and/or dizziness) not including LOC in FS compared with ES was not statistically significant (40% vs 28%, $p = 0.08$). There was no significant difference in reporting persistent symptoms consistent with postconcussive syndrome (FS: 7.4% vs ES: 3.8%, $p = 0.27$).

Figure 2 Rates of Reported History of Traumatic Brain Injury (TBI) and Perceived Attribution of TBI as a Cause of Seizures



Standard error bars are displayed. Significant differences are denoted with asterisks. Patients diagnosed with functional seizures demonstrated significantly higher rates of TBI history compared with patients with epileptic seizures. Perceived attribution of TBI as a cause of seizures was defined as either an occurrence of TBI within one year preceding the onset of seizures or alternatively patients endorsing that their head trauma was the cause of their seizures. There was no significant difference between seizure types in rates of perceived attribution of TBI as the cause of seizures.

Figure 3 Rates of TBI Sequelae Between Patients With Functional Seizures and Patients With Epileptic Seizures



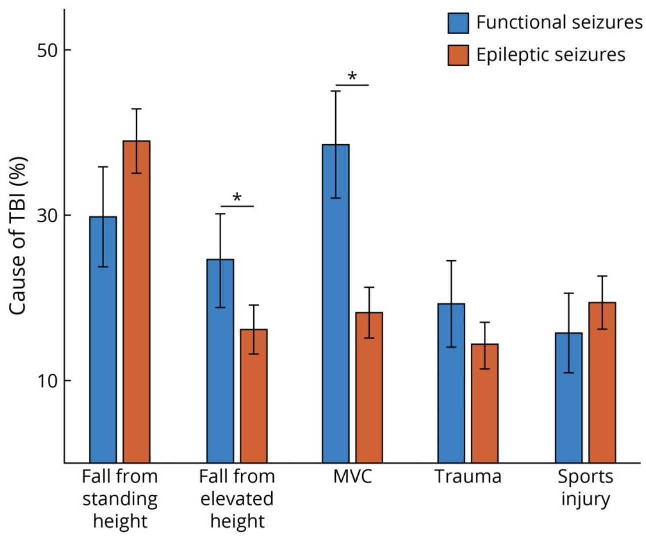
Asterisks denote significant differences. Standard error bars are shown. Patients with functional seizures were significantly more likely to report a history of TBI causing loss of consciousness (LOC) than patients with epileptic seizures. On separating this into duration of LOC, we found that patients with functional seizures were significantly more likely to report TBI with resulting LOC occurring less than 30 minutes, whereas there was no significant difference in the rates of LOC lasting greater than 30 minutes or producing coma. There was no significant difference between rates of TBI producing symptoms of concussion and postconcussive syndrome. TBI = traumatic brain injury.

There was a higher frequency of reported TBI from falls above the standing level in patients with FS than ES (17% vs 10%, aOR = 2.24 [1.06–4.70], $p = 0.03$). Patients with FS were more likely to report a TBI occurring because of MVC (27% vs 11%, aOR = 2.96 [1.54–5.67], $p < 0.01$) than patients with ES. This was not explained solely by higher rates of TBI in the FS group, as demonstrated by isolating rates among only patients who reported a past occurrence of TBI (Figure 4). By contrast, there was no significant difference between FS and ES in the number of participants who reported a traumatic fall from a standing height or lower (FS: 21% vs ES: 23%, $p = 0.64$) or TBI from violent trauma (FS: 14% vs ES: 8.5%, $p = 0.13$), despite physical abuse being more common in FS (FS: 35% vs ES: 18%, aOR = 2.02 [1.14–3.58], $p = 0.02$).

We also investigated relationships between vEEG diagnosis and reported psychiatric diagnosis. As also found in previous investigations,[16] patients with FS were significantly more likely than patients with ES to report having PTSD (15% vs 5%, aOR = 2.97 [1.26–6.93], $p = 0.01$); this also was more prevalent in patients with mixed seizure types (21%, aOR = 4.65 [1.32–16.32], $p = 0.02$ vs ES). Patients reported a history of depression more commonly in both FS (41%) and mixed seizures (53%) than in epilepsy (20%, aOR = 2.45 [1.41–4.24], $p < 0.01$ and aOR = 4.19 [1.60–10.96], $p < 0.01$, respectively). Reported anxiety disorders were also more common in FS (42%) and PSLE (42%) than in epilepsy (18%, aOR = 3.72 [2.10–6.58], $p < 0.01$ and aOR = 3.09 [1.13–8.46], $p = 0.03$, respectively).

We assessed for 2-way interactions between sex, psychiatric diagnoses, and seizure diagnosis with reported TBI history. A patient-reported history of PTSD, TBI because of a fall from a standing height, and diagnosis of FS displayed a significant interaction where the likelihood of FS was higher than expected in patients with both PTSD and TBI because of a

Figure 4 Comparisons of TBI Causes of Injury Between Patients With Epileptic Seizures and Functional Seizures



Displayed values are the percentage of patients endorsing a cause of injury among patients with a history of TBI. Significant differences are indicated with an asterisk. Error bars denote standard error. Patients with functional seizures had significantly greater rates of TBI due to motor vehicle collisions (MVC) compared with patients with epileptic seizures. Similarly, patients with functional seizures reported higher rates of TBI from a fall from height above the standing level. There was no difference in rates of TBI from falls at a standing height, violent trauma, and sports-associated injury. TBI = traumatic brain injury.

fall from a standing height, compared with the combined effect from each of these associations (aOR = 7.63 [1.34–43.19], $p = 0.02$). However, other causes of injury and features of TBI, including duration of LOC, did not show a significant interaction. There was a significant interaction between anxiety and FS in predicting a history of TBI (aOR = 2.37 [1.04–5.41], $p = 0.04$) and concussion with and without LOC (aOR = 2.44 [1.21–4.93] $p = 0.01$), where the combination of anxiety and FS made a history of TBI and concussion more likely than expected based on the combination of the individual associations. Patients with both depression and FS displayed higher rates of persistent postconcussive symptoms (aOR = 4.59 [1.34–15.7], $p = 0.02$ and aOR = 4.68 [1.27–17.20], $p = 0.02$), but a significant interaction demonstrated that the effects of each were not additive (aOR = 0.05 [0.01–0.67], $p = 0.03$).

Individual-Level Statistics

We created a multivariate logistic regression model with RFE selection for differentiating FS from ES using features of reported TBI history. This model is summarized in Table 1 (see eTable 2 for the pre-RFE model, links.lww.com/CPJ/A375). While the area under the receiver-operating characteristic curve was 71% (95% CI: 63–77%), the individual prediction of either FS or ES had a sensitivity of 99.5% and a specificity of 1.3% compared with gold standard vEEG documented diagnosis.[29] This suggests that the individual-level inferences did not differ significantly from the naïve prediction that all patients had ES. Despite lack of inferential ability, the RFE model accounted for a significant amount of variance in the likelihood of FS compared with ES (RFE model residual deviance 268 with $df = 12$, null deviance 352, $p < 10^{-12}$). The pre-RFE model also accounted for a significant amount of variance (pre-RFE deviance 236 with df = 43, $p < 10^{-7}$), but did not account for significantly more variance than the RFE model (deviance difference of 32, df = 31, $p = 0.59$).

Discussion

PTE is a well-known complication of TBI.[15] However, the relationship between TBI and FS is less often recognized by patients and clinicians. In our prospective investigation of patients admitted for video-EEG monitoring for the evaluation of seizures, a majority of patients reported a history of TBI, regardless of their final diagnosis. The prevalence of TBI in the general population is estimated as approximately 12%.[30] By contrast, 70% of patients with FS reported a history of TBI while 59% of patients diagnosed with ES reported prior TBI. Our results extend prior retrospective chart reviews of nonmilitary patients[12,31,32] and prospective studies of veterans,[10,16,33] which likewise report high rates of

Table 1 Multivariate Logistic Regression Model for Differentiation Between Functional Seizures and Epileptic Seizures

	OR	95% CI	*p*
Age (y)	0.98	0.96–1.00	0.02
Depression	0.40	0.21–0.78	0.01
PTSD	0.56	0.29–1.08	0.09
History of TBI	8.0	2.3–27.6	< 0.01
TBI with LOC	0.12	0.04–0.34	< 0.01
LOC >30 min	6.7	1.6–28.3	0.01
Fall from above the standing level	0.33	0.13–0.84	0.02
	Interactions with female sex		
History of TBI	0.17	0.05–0.52	<0.01
LOC <30 min	7.1	1.9–26.6	<0.01
Coma	6.8	0.7–67.2	0.10
MVC TBI	0.14	0.05–0.45	<0.01
Trauma/violence TBI	0.28	0.07–1.10	0.07
Sports TBI	0.28	0.07–1.17	0.08

Abbreviations: ES = epileptic seizure; FS = functional seizure; LOC = loss of consciousness; MVC = motor vehicle collision; PTSD = posttraumatic stress disorder; TBI = traumatic brain injury.
The variables displayed are those remaining after recursive feature elimination. Odds ratios (ORs) less than 1 signify predictive of FS, whereas OR higher than 1 signifies predictive of ES when controlling for the other variables in the model. When multiple variables are present, odds ratios multiply (e.g., a woman of an average age with a TBI with LOC <30 min: baseline female odds × 8.0 [main effect of a history of TBI] × 0.17 [interaction of female sex with a reported history of TBI] × 0.12 [main effect of TBI with LOC] × 7.1 [interaction of female sex with reported TBI with LOC <30 min]).The model demonstrated poor specificity for differentiating between FS and ES, signifying that studied features of TBI cannot be used to distinguish between seizure types alone.

TBI in patients with FS, by assessing which features of patient-reported TBI carry increased association with FS. Patients with FS were more likely to report TBI with LOC, including nearly twice as many patients with FS reporting LOC lasting less than 30 minutes than patients with ES. The presence of this immediate sequela of TBI was more predictive of FS than other reported neurologic symptoms including amnesia, confusion, nausea, and dizziness. Although these features are all compatible with mild TBI, the importance of brief LOC has not been previously emphasized. We also assessed the association between causes of reported TBI and eventual seizure diagnosis. Our finding that patients' reported TBIs from elevated falls and MVCs had greater association with FS than ES is unexpected because clinicians may suspect that these apparently more severe causes would portend greater risk of PTE than FS.

Although associations between mild TBI and FS have been demonstrated previously,[10-12] this investigation directly contrasts reports of TBI between seizure diagnoses in a civilian population. In military and other populations with a higher prevalence of severe TBI, there is a strong association between epilepsy and head trauma.[33-35] In these data sets, the risk of PTE substantially increases with the severity of injury.[33,35] In our civilian population, it was rare for patients to report severe TBI; therefore, this prospective sample did not reproduce this association. We previously replicated the association of severe TBI with epilepsy as compared with FS in a larger retrospective evaluation, although details on the severity of head trauma were inconsistently available in chart review.[6] Our findings on seizure attribution differ from studies of veteran populations, where patients frequently attributed TBI as the cause of both epilepsy and FS.[16] This may represent reduced awareness of TBI as a precipitating factor of seizures among nonmilitary patients. Alternatively, this may be because of higher rates of comorbid PTSD and service-associated injuries in military populations, which may result in these injuries carrying particularly high salience for patients with FS.

There are several considerations for explaining the association between TBI and FS. Prior investigations have theorized that patients with FS may experience heightened sensitivity or vigilance to physical symptoms occurring from injury,[16,22,36] with injuries of a similar magnitude holding greater salience for patients predisposed to developing FS. When searching for a possible explanation for FS, a past injury may then be recalled and considered a causal event. Supporting this, a prior investigation of veterans found that patients with FS reported a greater number of TBIs than patients with ES, despite similar combat exposure history.[16] Patients with FS had an elevated hypochondriasis subscore on the Minnesota Multiphasic Personality Inventory-2, which can signify a propensity toward somatic complaints including heightened sensitivity to injury.[37,38] However, this hypothesis was not supported by our data, where we did not find a difference in attribution of seizure cause between patients with FS and ES. Overall attribution of seizures to TBI was low across patient groups. We also did not observe an increased rate of TBI without LOC, as might be expected with over-reporting of minor head trauma or misattribution of head injury in patients with FS. Instead, we found that patients with FS reported a higher incidence of TBI *with* LOC. Given the degree of trauma needed to sustain LOC, this finding suggests that recall bias alone may not drive the difference in reported TBIs between these 2 patient groups. It is possible that some reported LOC after TBI could be due to a dissociative phenomenon immediately after an acute injury and, thus, distinct from LOC induced by structural damage. It is uncertain whether this is a potential contributor to reported rates of LOC after TBI.

Another possibility explaining the association between TBI and FS is that head injury itself may increase risk of developing FS in susceptible patients. This is supported by investigations linking TBI to other functional neurologic disorders.[14,39,40] Popkirov et al.[41] theorized the possibility of dissociogenic lesions that increase risk of functional experiences, including FS. Other research has shown that patients with FS are more likely to have experienced a severe head trauma compared with patients with idiopathic generalized (nontraumatic) epilepsy.[42] In addition to being physical stressors, TBIs may also act as a general psychological stressor that increases risk of FS. Thus, the well-replicated link between head injury and FS may be related to the stress response to trauma, heightened recall of traumatic events, and possible direct adverse effects of head injury in at-risk individuals.[43,44]

However, it is also worth considering if our results may be driven by our sample of patients admitted to an academic, tertiary referral vEEG monitoring unit. A history of TBI in a patient with clinical features otherwise fully consistent with probable FS may drive referrals for vEEG monitoring to establish a diagnosis. If so, our sample of patients with FS may be enriched for a reported history of TBI. Features of TBI that we found were associated with FS, such as LOC and high-velocity causes of injury, may further influence referral patterns. However, in a recent evaluation of factors that contributed to time to vEEG monitoring in this data set, a history of TBI was not associated with time to referral for FS, but it was associated with longer time to vEEG in patients with epilepsy.[24] Despite delays in referral, patients with suspected PTE also may be more likely to be referred to vEEG because of hypothesized surgical candidacy.[45,46]

We additionally considered what role psychiatric diagnoses may have in the relationship between FS and TBI. Salinsky et al.[11] showed a synergistic relationship between TBI and PTSD in veterans. In our investigation, civilian patients with FS were more likely to report a history of psychiatric diagnoses and patients with both TBI and psychiatric comorbidities were more likely to have FS. These findings may reflect comorbidities that predispose individuals to develop FS after TBI, or alternatively, forms of head trauma may increase risk of both FS and psychiatric disease. Distinguishing these possibilities is difficult without establishing temporality. We are further limited by differences in patient report vs confirmed diagnoses through chart history, with patients under-reporting psychiatric disease.[47,48] Although both FS and psychiatric disease are more common in women, we did not find that sex modified the relationship between reported TBI and seizure diagnosis.

Our findings hold particular relevance for neurologists performing the initial evaluation of patients presenting with seizures. The evaluation of most patients with seizures is dependent on patient-reported or caregiver-reported historical factors, including descriptions of ictal behavior, frequency, duration, and precipitating factors, in conjunction with physical examination, neurophysiologic testing, and neuroimaging.[23,49] While inquiring about TBI remains important, our results may signify that clinicians should not be overly swayed to diagnose epilepsy when a patient reports a history of TBI with LOC. This holds particularly true when LOC was estimated to be less than 30 minutes in duration. Similarly, causes of head injury that are putatively more severe, such as from a MVC or a fall from a height, did not suggest that epilepsy is more likely than FS. Even when patients report a history of head trauma with LOC, clinicians may continue to consider the possibility of FS or mixed FS with ES if suggested by other historical or objective clinical data.

These results should be interpreted with consideration of limitations. Although interviews were conducted before final diagnosis and interviewers were blinded to the inpatient workup, reports of TBI and sequelae were dependent on the patient or caregiver report rather than assessment at the time of injury. While this prevented us from verifying details, this level of evidence resembles the usual limitations in clinical practice. We included all patients admitted for video-EEG monitoring and were agnostic to suspected etiology of seizures (i.e., genetic vs acquired). Thus, our sample did not consist solely of patients with suspected PTE; comparisons of TBI characteristics between patients with FS and ES may differ in a sample consisting solely of patients with suspected PTE. Although we used a standardized interview process, we did not use a previously validated scale such as the Patient Seizure Etiology Questionnaire (PSEQ) because of patient enrollment preceding publication of the PSEQ.[50] Each individual item of the PSEQ was represented in our standardized interview, although coded as yes/no rather than using a Likert scale. Finally, our interviews did not measure the precise duration of time between events or establish temporal precedence; some injuries could have been caused by unrecognized or forgotten seizures, whereas other seizures around head injury may have been caused by acute TBI unrelated to the patient's underlying seizure disorder. In addition to confirming our findings beyond patients admitted for video-EEG monitoring, further research also may focus on further delineating how features of TBI contribute to seizure risk beyond the gradations of severity that we describe here. Objective clinical measures for assessing features of TBI at the time of injury may improve predictions of eventual risk of either ES or FS, particularly when used in combination with other neurodiagnostic tools.

In patients admitted for video-EEG monitoring, a history of TBI often is reported by both patients with FS and patients with ES. Among features of TBI, a reported history of LOC, particularly for less than 30 minutes, was observed more frequently among patients with FS than ES. We also found that LOC was more valuable for distinguishing between FS and ES than other concussive symptoms. Furthermore, patients reporting TBI with causes typically considered to be more severe (MVC or falls from heights) paradoxically demonstrated a higher likelihood of FS. Therefore, although reports of nonsevere TBI may raise concern for PTE, it does not exclude the possibility of FS. These insights provide clinically valuable information regarding interpretation of patient-provided and caregiver-provided neurologic history.

Acknowledgment
The authors thank Dr. Helena Hong for assistance with copyediting.

Study Funding
This work was supported by NIH R25 NS065723, NIH R25 NS089450, the UCLA-California Institute of Technology Medical Scientist Training Program (NIH T32 GM08042), the Neuroimaging Training Program (NIH T90 DA022768, R90 DA022768, and R90 DA023422), the William M. Keck Foundation, research grants to JE (NS03310 & NS080181), and the UCLA Departments of Psychiatry and Biobehavioral Sciences and Biomathematics.

Disclosure
L.B. Hickman, A.B. Patel, I. Dubey, A.H. Karimi, X. Zhang, E.A. Janio, C.H. Allas, S.S. Sreenivasan, J. Bauirjan, S. D'Ambrosio, M. Al Banna, A.Y. Cho, J. Engel, and J.M. Stern report no disclosures relevant to the manuscript. W.T. Kerr writes paid review articles on this topic for Medlink Neurology and has received personal compensation from SK Lifesciences unrelated to this manuscript. Drs. L.B Hickman, M. Al Banna, J. Engel, J.M. Stern, and W.T. Kerr have clinical responsibilities that include diagnosis/treatment of patients with epileptic and nonepileptic seizures. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

TAKE-HOME POINTS

- In a sample of patients admitted to an academic center for video-EEG monitoring, patients with functional seizures (FS) were more likely to report a history of TBI than patients with epileptic seizures (ES).
- Patients with FS were also more likely to report TBI with loss of consciousness (LOC) than patients with ES, particularly LOC lasting less than 30 minutes.
- TBI caused by motor vehicle collision and fall from above the standing height were more commonly reported by patients with FS than ES.
- Although reports of TBI with LOC and TBI due to presumed high-risk mechanisms intuitively raises suspicion for posttraumatic epilepsy, patients with FS frequently also reported these historical factors.

Publication History

Received by *Neurology: Clinical Practice* January 24, 2022. Accepted in final form September 19, 2022. Submitted and externally peer reviewed. The handling editor was Editor Luca Bartolini, MD.

Appendix Authors

Name	Location	Contribution
L. Brian Hickman, MD, MSc	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA; Department of Internal Medicine, University of California at Irvine, Irvine, CA	Drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data
Akash B. Patel, DO	Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design
Ishita Dubey, BS	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Amir H. Karimi, BA	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Xingruo Zhang, BS, BA	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Emily A. Janio, MPH	Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Corinne H. Allas, BS	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data

Appendix (continued)

Name	Location	Contribution
Siddhanka S. Sreenivasan, BA	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Janar Bauirjan, RN, BSN	Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Shannon D'Ambrosio, BS	Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Mona Al Banna, MD	Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Andrew Y. Cho, MS	Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data
Jerome Engel, Jr., MD, PhD	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA; Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, CA; Department of Neurobiology, David Geffen School of Medicine at UCLA, Los Angeles, CA; Brain Research Institute, University of California Los Angeles, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data
John M. Stern, MD	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA	Drafting/revision of the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data
Wesley T. Kerr, MD, PhD	Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA; Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA; Department of Neurology, University of Michigan, Ann Arbor, MI	Drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data

References

1. Benbadis SR, O'Neill E, Tatum WO, Heriaud L. Outcome of prolonged video-EEG monitoring at a typical referral epilepsy center. *Epilepsia*. 2004;45(9):1150-1153. doi:10.1111/j.0013-9580.2004.14504.x
2. Asadi-Pooya AA, Brigo F, Mildon B, Nicholson TR. Terminology for psychogenic nonepileptic seizures: making the case for "functional seizures." *Epilepsy Behav*. 2020;104(Pt A):106895. doi:10.1016/j.yebeh.2019.106895
3. Kerr WT, Stern JM. We need a functioning name for PNES: consider dissociative seizures. *Epilepsy Behav*. 2020;105:107002. doi:10.1016/j.yebeh.2020.107002
4. Foroughi AA, Nazeri M, Asadi-Pooya AA. Brain connectivity abnormalities in patients with functional (psychogenic nonepileptic) seizures: a systematic review. *Seizure*. 2020;81:269-275. doi:10.1016/j.seizure.2020.08.024
5. Dickinson P, Looper KJ. Psychogenic nonepileptic seizures: a current overview. *Epilepsia*. 2012;53(10):1679-1689. doi:10.1111/j.1528-1167.2012.03606.x
6. Kerr WT, Janio EA, Braesch CT, et al. An objective score to identify psychogenic seizures based on age of onset and history. *Epilepsy Behav*. 2018;80:75-83. doi:10.1016/j.yebeh.2017.11.035
7. LaFrance WC , Jr, Baird GL, Barry JJ, et al. Multicenter pilot treatment trial for psychogenic nonepileptic seizures: a randomized clinical trial. *JAMA Psychiatry*. 2014;71(9):997-1005. doi:10.1001/jamapsychiatry.2014.817
8. Goldstein LH, Robinson EJ, Mellers JDC, et al. Cognitive behavioural therapy for adults with dissociative seizures (CODES): a pragmatic, multicentre, randomised

controlled trial. *Lancet Psychiatry*. 2020;7(6):491-505. doi:10.1016/S2215-0366(20)30128-0

9. Walczak TS, Papacostas S, Williams DT, Scheuer ML, Lebowitz N, Notarfrancesco A. Outcome after diagnosis of psychogenic nonepileptic seizures. *Epilepsia*. 1995;36(11):1131-1137. doi:10.1111/j.1528-1157.1995.tb00472.x
10. Salinsky M, Spencer D, Boudreau E, Ferguson F. Psychogenic nonepileptic seizures in US veterans. *Neurology*. 2011;77(10):945-950. doi:10.1212/WNL.0b013e31822cfc46
11. Salinsky M, Storzbach D, Goy E, Evrard C. Traumatic brain injury and psychogenic seizures in veterans. *J Head Trauma Rehabil*. 2015;30(1):E65-E70. doi:10.1097/HTR.0000000000000057
12. Barry E, Krumholz A, Bergey GK, Chatha H, Alemayehu S, Grattan L. Nonepileptic posttraumatic seizures. *Epilepsia*. 1998;39(4):427-431. doi:10.1111/j.1528-1157.1998.tb01395.x
13. Trudeau DL, Anderson J, Hansen LM, et al. Findings of mild traumatic brain injury in combat veterans with PTSD and a history of blast concussion. *J Neuropsychiatry Clin Neurosci*. 1998;10(3):308-313. doi:10.1176/jnp.10.3.308
14. Mooney G, Speed J. The association between mild traumatic brain injury and psychiatric conditions. *Brain Inj*. 2001;15(10):865-877. doi:10.1080/02699050110065286
15. Annegers JF, Hauser WA, Coan SP, Rocca WA. A population-based study of seizures after traumatic brain injuries. *N Engl J Med*. 1998;338(1):20-24. doi:10.1056/NEJM199801013380104
16. Salinsky M, Rutecki P, Parko K, et al. Psychiatric comorbidity and traumatic brain injury attribution in patients with psychogenic nonepileptic or epileptic seizures: a multicenter study of US veterans. *Epilepsia*. 2018;59(10):1945-1953. doi:10.1111/epi.14542
17. Parreira JG, Rondini GZ, Below C, et al. Relação entre o mecanismo de trauma e lesões diagnosticadas em vítimas de trauma fechado. *Rev Col Bras Cir*. 2017;44(4):340-347. doi:10.1590/0100-69912017004007
18. Frey LC. Epidemiology of posttraumatic epilepsy: a critical review. *Epilepsia*. 2003;44(s10):11-17. doi:10.1046/j.1528-1157.44.s10.4.x
19. Temkin NR. Risk factors for posttraumatic seizures in adults. *Epilepsia*. 2003;44(s10):18-20. doi:10.1046/j.1528-1157.44.s10.6.x
20. LaFrance WC, Baker GA, Duncan R, Goldstein LH, Reuber M. Minimum requirements for the diagnosis of psychogenic nonepileptic seizures: a staged approach. *Epilepsia*. 2013;54(11):2005-2018. doi:10.1111/epi.12356
21. Baroni G, Piccinini V, Martins WA, et al. Variables associated with co-existing epileptic and psychogenic nonepileptic seizures: a systematic review. *Seizure*. 2016;37:35-40. doi:10.1016/j.seizure.2016.02.003
22. Kerr WT, Janio EA, Braesch CT, et al. Diagnostic implications of review-of-systems questionnaires to differentiate epileptic seizures from psychogenic seizures. *Epilepsy Behav*. 2017;69:69-74. doi:10.1016/j.yebeh.2016.11.002
23. Kerr WT, Janio EA, Chau AM, et al. Objective score from initial interview identifies patients with probable dissociative seizures. *Epilepsy Behav*. 2020;113:107525. doi:10.1016/j.yebeh.2020.107525
24. Kerr WT, Zhang X, Hill CE, et al. Epilepsy, dissociative seizures, and mixed: associations with time to video-EEG. *Seizure*. 2021;86:116-122. doi:10.1016/j.seizure.2021.02.002
25. Giza CC, Kutcher JS, Ashwal S, et al. Summary of evidence-based guideline update: evaluation and management of concussion in sports: report of the Guideline Development Subcommittee of the American Academy of Neurology. *Neurology*. 2013;80(24):2250-2257. doi:10.1212/WNL.0b013e31828d57dd
26. O'Neil ME, Carlson KF, Storzbach D, et al. Factors associated with mild traumatic brain injury in veterans and military personnel: a systematic review. *J Int Neuropsychol Soc*. 2014;20(3):249-261. doi:10.1017/s135561771300146x
27. Ryan LM, Warden DL. Post concussion syndrome. *Int Rev Psychiatry*. 2003;15(4):310-316. doi:10.1080/09540260310001606692
28. Guyon I, Weston J, Barnhill S, Vapnik V. Gene selection for cancer classification using support vector machines. *Machine Learn*. 2002;46(1/3):389-422. doi:10.1023/A:1012487302797
29. Kottas M, Kuss O, Zapf A. A modified Wald interval for the area under the ROC curve (AUC) in diagnostic case-control studies. *BMC Med Res Methodol*. 2014;14(1):26. doi:10.1186/1471-2288-14-26
30. Frost RB, Farrer TJ, Primosch M, Hedges DW. Prevalence of traumatic brain injury in the general adult population: a meta-analysis. *Neuroepidemiology*. 2013;40(3):154-159. doi:10.1159/000343275
31. Westbrook LE, Devinsky O, Geocadin R. Nonepileptic seizures after head injury. *Epilepsia*. 1998;39(9):978-982. doi:10.1111/j.1528-1157.1998.tb01447.x
32. LaFrance WC, Deluca M, Machan JT, Fava JL. Traumatic brain injury and psychogenic nonepileptic seizures yield worse outcomes. *Epilepsia*. 2013;54(4):718-725. doi:10.1111/epi.12053
33. Pugh MJV, Orman JA, Jaramillo CA, et al. The prevalence of epilepsy and association with traumatic brain injury in veterans of the Afghanistan and Iraq wars. *J Head Trauma Rehabil*. 2015;30(1):29-37. doi:10.1097/HTR.0000000000000045
34. Aarabi B, Taghipour M, Haghnegahdar A, Farokhi M, Mobley L. Prognostic factors in the occurrence of posttraumatic epilepsy after penetrating head injury suffered during military service. *Neurosurg Focus*. 2000;8(1):e1. 6, doi:10.3171/foc.2000.8.1.155
35. Englander J, Bushnik T, Duong TT, et al. Analyzing risk factors for late posttraumatic seizures: a prospective, multicenter investigation. *Arch Phys Med Rehabil*. 2003;84(3):365-373. doi:10.1053/apmr.2003.50022
36. Bowman ES, Markand ON. Psychodynamics and psychiatric diagnoses of pseudoseizure subjects. *Am J Psychiatry*. 1996;153(1):57-63. doi:10.1176/ajp.153.1.57
37. Bodde NMG, Bartelet DCJ, Ploegmakers M, Lazeron RHC, Aldenkamp AP, Boon PAJM. MMPI-II personality profiles of patients with psychogenic nonepileptic seizures. *Epilepsy Behav*. 2011;20(4):674-680. doi:10.1016/j.yebeh.2011.01.034
38. Del Bene VA, Arce Rentería M, Maiman M, et al. Increased odds and predictive rates of MMPI-2-RF scale elevations in patients with psychogenic non-epileptic seizures and observed sex differences. *Epilepsy Behav*. 2017;72:43-50. doi:10.1016/j.yebeh.2017.04.023
39. Matin N, Young SS, Williams B, et al. Neuropsychiatric associations with gender, illness duration, work disability, and motor subtype in a U.S. functional neurological disorders clinic population. *J Neuropsychiatry Clin Neurosci*. 2017;29(4):375-382. doi:10.1176/appi.neuropsych.16110302
40. Otallah SI. Psychogenic gait disorder complicating recovery after concussion: a case series. *Pediatr Neurol*. 2020;111:1-3. doi:10.1016/j.pediatrneurol.2020.05.005
41. Popkirov S, Carson AJ, Stone J. Scared or scarred: could "dissociogenic" lesions predispose to nonepileptic seizures after head trauma? *Seizure*. 2018;58:127-132. doi:10.1016/j.seizure.2018.04.009
42. Asadi-Pooya AA, Farazdaghi M. Is severe head injury associated with functional (psychogenic) seizures? *Seizure*. 2021;89:38-40. doi:10.1016/j.seizure.2021.04.018
43. Popkirov S, Asadi-Pooya AA, Duncan R, et al. The aetiology of psychogenic nonepileptic seizures: risk factors and comorbidities. *Epileptic Disord*. 2019;21(6):529-547. doi:10.1684/epd.2019.1107
44. Sharma AA, Goodman AM, Allendorfer JB, et al. Regional brain atrophy and aberrant cortical folding relate to anxiety and depression in patients with traumatic brain injury and psychogenic nonepileptic seizures. *Epilepsia*. 2022;63(1):222-236. doi:10.1111/epi.17109
45. Piccenna L, Shears G, O'Brien TJ. Management of post-traumatic epilepsy: an evidence review over the last 5 years and future directions. *Epilepsia Open*. 2017;2(2):123-144. doi:10.1002/epi4.12049
46. Hakimian S, Kershenovich A, Miller JW, et al. Long-term outcome of extratemporal resection in posttraumatic epilepsy. *Neurosurg Focus*. 2012;32(3):E10. doi:10.3171/2012.1.FOCUS11329
47. Hunt M, Auriemma J, Cashaw ACA. Self-report bias and underreporting of depression on the BDI-II. *J Personal Assess*. 2003;80(1):26-30. doi:10.1207/S15327752JPA8001_10
48. Johnson HP, Agius M. A Post-Traumatic Stress Disorder review: the prevalence of underreporting and the role of stigma in the Military. *Psychiatr Danub*. 2018;30(suppl 7):508-510.
49. Janocko NJ, Jing J, Fan Z, et al. DDESVSFS: a simple, rapid and comprehensive screening tool for the differential diagnosis of epileptic seizures vs functional seizures. *Epilepsy Res*. 2021;171:106563. doi:10.1016/j.eplepsyres.2021.106563
50. Salinsky M, Parko K, Rutecki P, Boudreau E, Storzbach D. Attributing seizures to TBI: validation of a brief patient questionnaire. *Epilepsy Behav*. 2016;57(Pt A):141-144. doi:10.1016/j.yebeh.2016.02.003