Pediatric Neurology 95 (2019) 67–72

Contents lists available at ScienceDirect

Pediatric Neurology

journal homepage: www.elsevier.com/locate/pnu

Topical Review

Atypical Prodromal Symptoms Help to Distinguish Patients With Psychogenic Nonsyncopal Collapse Among Youth Referred for Fainting

Geoffrey L. Heyer, MD *

Department of Neurology, Dell Medical School, Austin, Texas

ARTICLE INFO

Article history:
Received 3 January 2019
Accepted 5 February 2019
Available online 13 February 2019

Keywords:
Conversion disorder
Functional
Pseudosyncope
Syncope
Psychogenic nonepileptic seizures
PNES
PNSC

ABSTRACT

Background: Distinguishing patients with psychogenic nonsyncopal collapse (PNSC), a conversion disorder that resembles syncope, can pose a difficult clinical challenge. Using the open-ended question "what does it feel like to faint?," the present study aimed to characterize how patients with PNSC perceive and communicate the prodromal symptoms associated with their attacks by comparing narratives between patients with PNSC and those with syncope.

Methods: During a 42-month database-type study of tilt-table diagnoses, all patients with a history of fainting were asked the open-ended question. Symptom descriptions were compared, qualitatively and quantitatively, between patients with PNSC and those with tilt-induced syncope (n = 121 in both cohorts).

Results: Twenty-nine patients (24%) diagnosed with PNSC and eight (6.6%) diagnosed with syncope either denied having any prodromal symptoms or could not recall any symptoms ($P < 0.001$). Among patients who reported prodromal symptoms, patient narratives led to the formation of 26 symptom categories. Symptom frequencies differed between cohorts in 19 of the symptom categories (each P value <0.05). Qualitative differences in the descriptions of symptoms were often present, even when symptom frequencies did not differ. More patients with PNSC described atypical symptoms than patients with syncope, 54 of 92 (58.7%) versus eight of 113 (7.1%), $P < 0.001$.

Conclusions: Symptom narratives can help to distinguish patients with PNSC from those with syncope. The use of a single, open-ended question as a screening tool for conversion disorder has immediate clinical relevance because it can be instituted easily in a busy clinical setting.

Introduction

Psychogenic nonsyncopal collapse (PNSC), also referred to as "psychogenic pseudosyncope," is defined as a transient alteration in behavior, movement, or arousal that resembles neurally mediated (reflex) syncope, but the attacks do not have the corresponding hypotension, bradycardia, or electroencephalographic (EEG) changes typical of syncope.[1-9] Instead, PNSC has a psychological origin. Parallels between PNSC and psychogenic nonepileptic seizures (PNES) have been demonstrated through the overlap in semiologic and clinical features[1-3,6,7,10,11] and the observation of dual diagnoses[4] in some patients. Accordingly, PNSC and PNES likely represent similar functional neurological symptom (conversion) disorders, with one resembling syncope and the other resembling seizures.[1,10]

The clinical differentiation of patients with a conversion disorder from those with syncope (or epilepsy) can be challenging. Among clinics specializing in the diagnosis and management of syncope, the published diagnostic rates for PNSC vary considerably, ranging from less than 1% to as high as 18.6%.[2,4,6,12-20] Given that up to 30% of patients with refractory epilepsy are diagnosed with PNES when referred to a tertiary care epilepsy center,[21] the variability in PNSC diagnostic rates probably represents insufficient recognition of the clinical features that are suggestive of a conversion disorder or an insufficient clinical approach to establish the definitive diagnosis.[2,4,7,14] Yet, establishing an accurate diagnosis is important as pediatric and adult studies have demonstrated that mere communication of the PNSC diagnosis can lead to improvements in attack frequency, in some cases leading to attack resolution, and

Funding source: None.
Conflicts of interest: None.
* Communications should be addressed to: Heyer; Department of Neurology; Dell Medical School; 1701 Trinity St.; Austin, TX 78712.
E-mail address: gheyer@austin.utexas.edu.

https://doi.org/10.1016/j.pediatrneurol.2019.02.006
0887-8994/

reduction in the utilization of health care resources, similar to published outcomes for PNES.[4,6,14]

Several "red flags" that represent clinical features considered atypical of syncope can be used to help differentiate patients with a conversion disorder from those referred for fainting. These atypical features include frequent fainting or numerous lifetime attacks, prolonged attacks, atypical triggers, absence of prodromal symptoms, rare or absent presyncope, eye closure with attacks, and tearfulness with attacks.[1,2,6-8,11,17,22,23] In contrast, when patients were queried about symptoms that are considered typical of syncope—lightheadedness, nausea, warmth, and sweating—the symptom ratings did not differ between cohorts with PNSC and with syncope.[5] Only ratings of visual changes (specifically responses to the statement "my vision goes white, gray, or black before I faint") differed, with significantly lower ratings among patients with PNSC.[5] Although assessments of atypical features of syncope might better discriminate patients with PNSC than assessments of typical features, differences in patient narratives about symptoms have not been explored. How patients describe the quality of their symptoms might further enhance our ability to detect patients with a conversion disorder that appears like syncope.

Accordingly, the aim of the present study was to characterize the symptoms leading up to PNSC based on responses to the open-ended question, "what does it feel like to faint?," by comparing responses, qualitatively and quantitatively, between patients with PNSC and patients with syncope. As the diagnosis of a conversion disorder might lead to feelings of shame, guilt, or embarrassment that could affect how patients report symptoms, all responses were obtained before testing or diagnosis. The term "fainting" in this article refers to all episodes (syncopal and nonsyncopal) that patients endorsed as syncope before diagnosis. The symptoms leading up to attacks are referred to as "prodromal," although it was not always clear whether a patient perceived a symptom as a trigger that caused fainting or as a component of the fainting process.

Patients and methods

Standard protocol approvals

The study was approved by the Institutional Review Board at the Nationwide Children's Hospital. Informed consent and assent (subjects aged 10 to 17 years) were obtained before any testing.

Subjects

Between January 2015 and June 2018, a database-type study was conducted of all patients referred to a pediatric neurology subspecialty clinic for tilt-table evaluation. Reasons for referral included atypical features of fainting, frequent or refractory fainting, transient loss of consciousness of unclear etiology, and frequent symptoms of orthostatic intolerance. Four of the 648 (0.6%) referred patients declined study participation, none of whom were ultimately diagnosed with PNSC. Of the remaining 644 database patients, 121 were diagnosed with PNSC. All patients diagnosed with PNSC acknowledged previous attacks that were similar to the attack(s) captured during testing where they perceived loss of consciousness, "blacking out," or "passing out," although some patients reported some form of sensory awareness during attacks (e.g., "I couldn't move or see, but I could hear [witnesses speaking]").

To compare prodromal symptoms between PNSC and syncope, a reference group of 121 sequential patients with tilt-induced syncope was established from the database. All patients in the syncope cohort confirmed that symptoms during tilt-induced syncope were similar to symptoms during the fainting episodes that prompted clinical referral. Patients who met the diagnostic criteria for postural tachycardia syndrome before syncope were excluded to minimize the contribution of symptoms attributable to orthostatic intolerance rather than the syncope prodrome. Other exclusion criteria were (1) age less than 10 or greater than 25 years; (2) use of medicine provocation to induce syncope during testing; (3) any form of psychosis; (4) use of antipsychotic medications for any reason; and (5) pregnancy.

Definitions

Tilt-induced syncope was defined as a transient loss of consciousness with hypotension, with or without bradycardia or asystole, and with corresponding EEG changes that are typical of neurally mediated syncope.[24,25] PNSC was defined as a period of apparent loss of consciousness during orthostatic challenge that the patient identified as fainting, but without corresponding hypotension, bradycardia or asystole, or EEG changes.

Patient interview

As previously published,[2,4] a standardized interview was used to obtain clinical information from all database participants, before testing or diagnosis. When patients reported a history of previous fainting, the open-ended question followed: "What does it feel like to faint?" When symptom descriptions were vague or unclear, further detail was requested. After each response, the patient was asked: "Were there any other symptoms?" The open-ended questioning continued until patients denied further symptoms. When patients denied any symptoms associated with fainting, they were asked to clarify whether they could recall the attacks and no symptoms were present or they could not recall if symptoms were present or not. If patients acknowledged at least one symptom, they were asked when the symptoms began in relation to perceived loss of consciousness, blacking out, or passing out. All responses were recorded; many were recorded verbatim when the descriptions of symptoms seemed atypical for syncope. The symptoms analyzed in the present study (1) were described in response to the open-ended question; (2) began before the fainting episode; and (3) had a direct relationship with fainting, meaning the patient recognized a strict or common association between the symptom(s) and the attacks. As the prodromal symptoms typical of syncope (e.g., lightheadedness, visual changes, nausea, warmth, sweating, etc.) were each queried separately during the standardized interview, fewer prodromal symptoms were reported from open-ended questioning than from the completed interview (data not presented). Although this methodology underestimated overall symptom reporting, it highlighted the prodromal symptoms that patients recalled without specific prompting, which has clinical relevance.

Protocol

The tilt-table protocol has been described previously.[2-5,26] The pre-tilt evaluation included a normal 12-lead electrocardiogram, a normal cardiac examination, and a negative urine pregnancy test for appropriately aged females. All medicines that could affect orthostatic tolerance were discontinued $\geq$5 half-lives before testing. Video-EEG (Comet AS-40, GRASS systems, Warwick, Rhode Island, USA) and continuous heart rate and blood pressure (Portapres, Finapres Medical Systems, Amsterdam, The Netherlands) were synchronized and monitored. After at least 30 minutes of recumbency, patients were tilted upright to 70°. Testing was

completed (table lowered) with syncope, apparent loss of consciousness consistent with PNSC, or after at least 45 minutes of tilt.

To optimize the diagnostic rate, additional testing was performed whenever the PNSC diagnosis was highly suspected.[2-5]

1. If syncope occurred during the initial tilt-table test, but (A) the patient reported that the event seemed different from prior attacks, (B) apparent loss of consciousness or head drop occurred before EEG changes, or (C) apparent loss of consciousness continued after EEG normalization, the tilt test was repeated with abdominal and lower extremity compression.[27] The compression garment limits orthostatic blood pooling, lowering the risk of tilt-induced syncope, without altering the suggestibility that fainting will occur among compression-naive patients. All patients with syncope and PNSC during testing were analyzed in the PNSC group.
2. If the tilt-table test was normal at 45 minutes, but the patient reported multiple or severe orthostatic symptoms or believed that fainting was imminent, then standing tests were performed. Patients were asked to stand rapidly from the seated position for up to 120 seconds, supported upright by two examiners. Up to six consecutive standing tests were conducted, with and without deliberate hyperventilation while seated. Patients were instructed to relax their breathing upon standing.
3. Two patients reported that their attacks only occurred with exercise. Both had thorough cardiac evaluations, which were normal. After normal tilt-table testing, repeating the exercise in the laboratory (jogging in place and "mountain climbers," respectively) led to typical PNSC in both patients. EEG was monitored without continuous blood pressure monitoring during exercise.

Statistical analysis

Symptom descriptions were considered atypical of syncope when none of the patients in the syncope cohort also described the symptom (e.g., bilateral leg weakness) or when the symptom did not match what would be expected from known physiology (e.g., "rainbow"-colored visual changes). Data were compared between patients diagnosed with PNSC and those diagnosed with syncope. The chi-square test or Fisher's exact test was used to compare categorical variables. Student's t test was used to compare continuous variables. All analyses were two-tailed where appropriate. Data are presented as mean ± standard deviation. All statistical analyses were performed using SPSS Version 25 (SPSS Inc, Chicago, IL, USA). The significance threshold was set at 5%.

Results

During the 42-month study period, 121 patients were diagnosed with PNSC. Patients with PNSC did not differ from matched patients with syncope (n = 121) by age (15.6 ± 2.3 vs 15.4 ± 2.5 years, $P = 0.4$) or sex (84.3% vs 79.3% female, $P = 0.32$).

Patients from both cohorts described multiple prodromal symptoms during open-ended questioning. Symptoms are listed by category in Table 1. Twenty-nine patients (24%) diagnosed with PNSC and eight (6.6%) diagnosed with syncope either denied having any prodromal symptoms or could not recall any symptoms ($P < 0.001$). Among the 92 patients with PNSC who reported prodromal symptoms, 12 (13%) reported one symptom, 28 (30.4%) reported two, 34 (37%) reported three, and 18 (19.6%) reported four or more. Among the 113 patients with syncope who reported prodromal symptoms, 12 (10.6%) reported one symptom, 50 (44.2%) reported two, 43 (38.1%) reported three, and eight (7.1%) reported four or more.

Table 1 lists the symptom categories and the differences in reported symptom frequencies between cohorts: 19 symptom categories differed with P values ≤ 0.05; seven of the 19 symptom categories differed with P values ≤ 0.001. Qualitative differences in the descriptions of symptoms were often present, even when symptom frequencies did not differ. For example, of the 70 patients with syncope who reported prodromal vision changes, 64 (91.4%) described visual dimming before loss of consciousness and the remaining six (8.6%) described "graying out" of vision or dimming of only the peripheral visual fields. In contrast, only 12 of the 57 (21.1%) patients with PNSC who reported vision changes described visual dimming, usually as an abrupt onset of "blackness," "blacking out," or inability to see. The remaining patients with PNSC and visual symptoms described various vision changes including bright colors (commonly red or "rainbow"), "blobs," static, or well-formed visual hallucinations. Similarly, patients with PNSC described qualitatively different types of muscle weakness, including the inability to move one or several muscles. The inability to stand—causing collapse—was attributed by some to muscle weakness of both legs. In contrast, the four patients with syncope who described muscle weakness later clarified during the interview or following tilt testing that the sensation of weakness matched a sensation of "feeling drained" or feeling generally weak, rather than an inability to move muscles. As muscle weakness was verbalized in the open-ended question, however, it was retained in the analysis. In all cases, the symptom(s) began while the patient was alert. For example, a patient with PNSC reported a full-body "seizure," described as head and bilateral limb shaking that always preceded perceived loss of consciousness and loss of tone. Notably, it was the loss of tone and lack of movement following collapse that prompted the neurology referral for refractory syncope rather than referral for seizures. Table 2 provides additional examples of symptom narratives from patients with PNSC.

Eleven symptom categories comprised symptoms only described by patients with PNSC (see Table 1). Commonly reported symptoms within these categories included the sensation of imminent sleep with fainting or sleepiness as the cause of collapse (inconsistent with features of narcolepsy or cataplexy), severe confusion or surprise immediately before fainting, stuttering or inability to find words, headache (typically described as severe) that only occurred with fainting or was notably worse with fainting, exhaustion or severe fatigue that only occurred with fainting or was notably worse with fainting, the sensation of being pulled to the floor, immobility or the description of muscle "freezing," and crying before fainting. Notably, the description of crying with PNSC was largely under-reported during open-ended questioning when compared with the observation of tearfulness preceding PNSC during diagnostic testing (3.3% vs 36.3%, $P < 0.001$).

Among patients who acknowledged prodromal symptoms, 54 of the 92 (58.7%) patients with PNSC and eight of the 113 (7.1%) patients with syncope described at least one atypical symptom, $P < 0.001$. Eighty-eight of the 92 (95.7%) symptomatic patients with PNSC also confirmed the presence of matching atypical symptom(s) during the laboratory-captured attack(s). Those patients without matching symptoms commonly reported that the attack(s) captured during diagnostic testing was "milder" than the attacks that prompted clinical referral. Patients with syncope described atypical symptoms related to shaking or tremor, body pain, weakness, numbness, and panic. In each case, further clarification of the symptom description(s) during the interview or following tilt

TABLE 1.
Prodromal Symptoms Reported by Patients With PNSC and Patients With Syncope

Prodromal Symptoms	PNSC (n = 121)	Syncope (n = 121)	*P* value
Lightheadedness	59 (48.8%)	98 (81%)	<0.001*
Vision change	57 (47.1%)	70 (57.9%)	0.09
Vertigo	49 (40.5%)	31 (25.6%)	0.014†
Sweating	28 (23.1%)	34 (28.1%)	0.38
Temperature change (warmth or cold)	13 (10.7%)	39 (32.2%)	<0.001*
Nausea	13 (10.7%)	36 (29.8%)	<0.001*
Dyspnea or hyperventilation	23 (19%)	10 (8.3%)	0.02†
Muscle weakness	18 (14.9%)	4 (3.3%)	0.002†
Numbness or tingling	15 (12.4%)	5 (4.1%)	0.02†
Shaking or tremor	14 (11.6%)	3 (2.5%)	0.006†
Change in hearing (includes hearing sounds or voices)	12 (9.9%)	6 (5%)	0.14
Heart racing	3 (2.5%)	9 (7.4%)	0.08
Body pain (other than headache)	9 (7.4%)	2 (1.7%)	0.03†
Chest tightness	3 (2.5%)	6 (5%)	0.31
Panic	5 (4.1%)	1 (0.8%)	0.1
Somnolence or sensation of imminent sleep	13 (10.7%)	0	<0.001*
Confusion or the sense of surprise	12 (9.9%)	0	<0.001*
Change in speech or language	11 (9.1%)	0	0.001*
Balance changes, sensation of falling or a downward force	8 (6.6%)	0	0.004†
Headache or head pressure	7 (5.8%)	0	0.007†
Fatigue, malaise, or exhaustion	6 (5%)	0	0.013†
Unable to move, but without muscle weakness	6 (5%)	0	0.013†
Tearfulness or crying	4 (3.3%)	0	0.04†
Anger or irritability	4 (3.3%)	0	0.04†
Depressed mood	2 (1.7%)	0	0.16
Odd taste(s) or change in taste sensation	2 (1.7%)	0	0.16
No prodromal symptoms with some or all past episodes	26 (21.5%)	6 (5%)	<0.001†
Unable to recall prodromal symptoms	3 (2.5%)	2 (1.7%)	0.65

Abbreviation:
PNSC = Psychogenic nonsyncopal collapse
* *P* value ≤0.001.
† *P* value ≤0.05.

testing matched what would be expected physiologically with syncope (e.g., panic occurring during routine venipuncture and followed by warmth, nausea, lightheadedness, and then loss of consciousness). None of the patients with syncope and atypical symptoms acknowledged other atypical features ("red flags") suggestive of PNSC.

Discussion

Distinguishing patients with a conversion disorder that resembles syncope from among those presenting with fainting can pose a difficult clinical challenge. The presence of atypical clinical features ("red flags") for syncope can be helpful, but only when

TABLE 2.
Descriptions of Prodromal Symptoms Reported by Patients With PNSC

Prodromal Symptom	Specific Examples From Patient Narratives
Vision change	"My eyes close so I can't see." Other vision changes reported include "rainbow-colored blobs," "zigzagging" lines, "dark fuzzies," "all red," and "red and staticky"
Vertigo	"I feel like the world is spinning, and I can't open my eyes"
Dyspnea or hyperventilation	"I can't breathe, so I pass out." "I can't catch my breath"
Muscle weakness	"Everything gives out." "My body goes limp."
Numbness or tingling	"I can't feel my legs"
Shaking or tremor	"I shake like I'm having a seizure." "I know I'm going to [faint because I cannot] stop shaking"
Change in hearing (includes hearing sounds or voices)	"Noises [sound] louder." "I hear the ocean." "I hear my parents screaming." [although parents were not present]
Body pain (other than headache)	It "hurts all over." "My chest starts to hurt [and then] my legs hurt and I can't stand."
Panic or anxiety	I feel "overwhelmed." "I get panicky." "[Fainting is] caused by stress."
Somnolence or sensation of imminent sleep	"My brain wants to sleep." "It feels like I am falling asleep."
Confusion or the sense of surprise	I feel "airy." I do not "remember if I lose consciousness or not; I get so confused." "My memory [drifts] in and out."
Change in speech or language	People tell me that "I stutter." "I can't come up with words." "I can't talk."
Headache or head pressure	It feels like "my head [will] explode." I feel "extreme pressure" in my head.
Balance changes, sensation of falling or a downward force	"Something pulls me down." "I have to fall down." "I feel 'tipsy,' [further described as off-balance]."
Fatigue, malaise, or exhaustion	I am "too exhausted to move."
Unable to move, but without muscle weakness	"Suddenly I can't move." "My arms and legs feel frozen, [further described as immobile]."
Tearfulness or crying	"Now, every time I cry I [feel as if I will] pass out."
Anger or irritability	"I get so mad."
Depressed mood	"It always happens when I am [sad]."
Odd taste(s) or change in taste sensation	I have a "weird taste."
No prodromal symptoms with some or all past episodes	"I never know it's coming." "Suddenly I am on the ground and [witnesses] tell me I fainted."

Abbreviation:
PNSC = Psychogenic nonsyncopal collapse

queried directly by the clinician or reported spontaneously by the patient. The present study demonstrated that symptom narratives also have value in distinguishing patients with PNSC. Responses to the open-ended question, "what does it feel like to faint?," varied among both cohorts, but patients with PNSC were more likely to describe atypical symptoms, even within categories of symptoms such as visual changes, which are considered typical during the syncope response. The use of a single, open-ended question as a potential screening tool for conversion disorder has immediate clinical relevance because it can be instituted easily in a busy clinic setting.

Patient narratives that contain bizarre symptoms or symptoms that do not conform with known neurological localization (e.g., bilateral convulsions with retained consciousness) will quickly evoke suspicions of conversion disorder. Subtler features that seem atypical, however, can be more difficult to interpret. As earlier published characterizations of prodromal symptoms with syncope lacked methodologies that systematically identified and excluded patients with PNSC, the possibility exists that rarer symptoms have been incorrectly attributed to syncope. The present study defined a symptom as "atypical" if it was not reported by any patients in the syncope cohort or if it could not be reasonably explained by known physiology. Although this definition is also susceptible to error, as was demonstrated by the presence of atypical symptoms among eight patients with syncope, the PNSC diagnosis was rigorously pursued. A 10-item screening tool for PNSC—the Fainting Assessment Inventory (FAI-10)—has recently been validated, and it demonstrated strong accuracy in distinguishing patients with a conversion disorder from otherwise healthy youth presenting with fainting.[5] Use of the FAI-10 in future research will better resolve which symptoms are typical of syncope and which symptoms are not typical. In addition, its clinical use will improve PNSC recognition. However, because the FAI-10 may not be readily at hand in clinics where presentations for fainting are rare or inconsistent, patient narratives from a simple, open-ended question remain clinically relevant.

Certain symptoms such as lightheadedness, vision changes, warmth, sweating, nausea, and hearing changes are reported commonly with tilt-induced syncope and were included among the typical symptoms in the present study. Vision changes during the evolution of syncope are thought to be related to the higher intraocular pressure compared with intracranial pressure.[28] Based on centrifuge data,[29,30] as cerebral perfusion becomes impaired, the pressure differential between the eye and brain causes greater impedance in perfusion to retinal cells than to cerebral cells, leading to visual dimming and other visual symptoms before loss of consciousness.[28] Applying the centrifuge model to neurally mediated syncope, patients with a gradual progression of hypotension tend to recall visual symptoms with syncope provided that they are alert enough during the symptom to encode the memory.[28] Hearing changes, commonly described as "muffled" hearing or hearing loss, can also occur if the blood pressure drops gradually.[28] In contrast, brightly colored vision changes, transient or fixed visual changes that do not progress with fainting (or occur independently of perceived loss of consciousness), or formed visual or auditory hallucinations represent atypical symptoms only reported by patients with PNSC and without a clear physiological correlation.

Three categories of symptoms atypical of syncope (somnolence or the sensation of imminent sleep, confusion or the sense of surprise, and a change in speech or language) were reported by more than 10% of patients with PNSC and none of the patients with syncope. Among the specific symptoms described the association of fainting as a feeling of sleep or sleepiness and the onset of stuttering (in individuals without a history of stuttering) each stand out as indicative of conversion disorder. Ictal stuttering has been documented among patients with PNES,[31,32] albeit with similar findings as a rare but highly specific feature. Previous studies of sleep and PNES or PNSC have focused either on signs of apparent sleep, sometimes referred to as pseudosleep, preceding attacks or on the patient reports of abnormal sleep patterns.[3,32-36] The present study demonstrates that a patient's description of sleepiness or imminent sleep with fainting should raise suspicion for PNSC. Additional atypical symptoms that suggest PNSC include isolated (or escalated) prodromal symptoms of headache, body pain, or fatigue; each has been described as associated with episodic conversion disorder, but rarely implicated as a specific symptom during the evolution of attacks.[3,37-42]

Limitations

There were several study limitations. First, as described above, symptoms were considered atypical of syncope if they were not reported by any patient with syncope or they did not conform to known physiology. The circularity of this definition and its proneness to error are acknowledged. However, it serves as a starting point in a field that lacks robust recognition of the conversion disorder that resembles syncope. Second, not all symptom reports were transcribed verbatim, which could have introduced error in the subsequent data collation and analyses. Third, the analyses excluded patients without PNSC or syncope during testing, which may have introduced an ascertainment bias in both groups. Fourth, patients were highly selected by their referral to a pediatric neurology, subspecialty clinic, which might have biased symptom reporting as individuals with simple reflex syncope rarely seek medical attention or receive subspecialty referral. Fifth, the use of an open-ended question underestimated symptom reporting in both cohorts, narrowing the scope of the data. However, although potentially limiting in terms of scope, this methodology highlighted the symptoms that patients could recall with minimal prompting, which is a strength of the study. Last, patients were categorized as PNSC even when they had both syncope and PNSC during testing. It is possible that patients with PNSC plus syncope differ in ways yet to be determined from those with PNSC only.

Conclusion

Although distinguishing patients with a conversion disorder from those presenting with fainting can pose a difficult clinical challenge, patient narratives can help to detect patients with PNSC. The use of a single, open-ended question as a screening tool for conversion disorder has immediate clinical relevance because it can be instituted easily in a busy clinical setting.

References

1. Heyer GL, Albert DV, Weber A, Gedela S, Vidaurre J. Comparison of semiologies between tilt-induced psychogenic nonsyncopal collapse and psychogenic nonepileptic seizures. Epilepsy Behav. 2016;62:171–175.
2. Heyer GL, Harvey RA, Islam MP. Comparison of specific fainting characteristics between youth with tilt-induced psychogenic nonsyncopal collapse versus reflex syncope. Am J Cardiol. 2017;119:1116–1120.
3. Heyer GL. Youth with psychogenic non-syncopal collapse have more somatic and psychiatric symptoms and lower perceptions of peer relationships than youth with syncope. Pediatr Neurol. 2018;79:34–39.
4. Heyer GL, Pabst LM, Kaucic BN, Coley TA. Early outcomes in youth with psychogenic nonsyncopal collapse. Neurology. 2018;91:e850–e858.
5. Heyer GL. The Fainting Assessment Inventory: a 10-item instrument developed to screen for psychogenic non-syncopal collapse among youth referred for syncope. J Nerv Ment Dis. 2019;207:255–263.
6. Tannemaat MR, van NJ, Reijntjes RH, Thijs RD, Sutton R, van Dijk JG. The semiology of tilt-induced psychogenic pseudosyncope. Neurology. 2013;81:752–758.
7. Benbadis SR, Chichkova R. Psychogenic pseudosyncope: an underestimated and provable diagnosis. Epilepsy Behav. 2006;9:106–110.

8. Raj V, Rowe AA, Fleisch SB, Paranjape SY, Arain AM, Nicolson SE. Psychogenic pseudosyncope: diagnosis and management. Auton Neurosci. 2014;184:66–72.
9. Saal DP, Overdijk MJ, Thijs RD, van Vliet IM, van Dijk JG. Long-term follow-up of psychogenic pseudosyncope. Neurology. 2016;87:2214–2219.
10. Hubsch C, Baumann C, Hingray C, et al. Clinical classification of psychogenic non-epileptic seizures based on video-EEG analysis and automatic clustering. J Neurol Neurosurg Psychiatry. 2011;82:955–960.
11. Grubb BP, Gerard G, Wolfe DA, et al. Syncope and seizures of psychogenic origin: identification with head-upright tilt table testing. Clin Cardiol. 1992;15:839–842.
12. Iglesias JF, Graf D, Forclaz A, Schlaepfer J, Fromer M, Pruvot E. Stepwise evaluation of unexplained syncope in a large ambulatory population. Pacing Clin Electrophysiol. 2009;32 Suppl 1:S202–S206.
13. Walsh KE, Baneck T, Page RL, Brignole M, Hamdan MH. Psychogenic pseudosyncope: Not always a diagnosis of exclusion. Pacing Clin Electrophysiol. 2018;41:480–486.
14. Tannemaat MR, Thijs RD, van Dijk JG. Managing psychogenic pseudosyncope: Facts and experiences. Cardiol J. 2014;21:658–664.
15. Strano S, Colosimo C, Sparagna A, et al. Multidisciplinary approach for diagnosing syncope: a retrospective study on 521 outpatients. J Neurol Neurosurg Psychiatry. 2005;76:1597–1600.
16. Luzza F, Di RS, Pugliatti P, Ando G, Carerj S, Rizzo F. Syncope of psychiatric origin. Clin Auton Res. 2004;14:26–29.
17. Petersen ME, Williams TR, Sutton R. Psychogenic syncope diagnosed by prolonged head-up tilt testing. QJM. 1995;88:209–213.
18. Novak P. Cerebral blood flow, heart rate, and blood pressure patterns during the tilt test in common orthostatic syndromes. Neurosci J. 2016;2016:6127340.
19. Robinson JA, Shivapour JK, Snyder CS. Tilt table testing to diagnose pseudosyncope in the pediatric population. Congenit Heart Dis. 2017;12:411–416.
20. Mathias CJ, Deguchi K, Schatz I. Observations on recurrent syncope and presyncope in 641 patients. Lancet. 2001;357:348–353.
21. Benbadis SR, O'Neill E, Tatum WO, Heriaud L. Outcome of prolonged video-EEG monitoring at a typical referral epilepsy center. Epilepsia. 2004;45:1150–1153.
22. Blad H, Lamberts RJ, van Dijk GJ, Thijs RD. Tilt-induced vasovagal syncope and psychogenic pseudosyncope: Overlapping clinical entities. Neurology. 2015;85:2006–2010.
23. Engel CL, Romano J. Studies of syncope; biologic interpretation of vasodepressor syncope. Psychosom Med. 1947;9:288–294.
24. Heyer GL, Schmittauer C, Islam MP. The clinical and electroencephalographic spectrum of tilt-induced syncope and “Near Syncope” in youth. Pediatr Neurol. 2016;62:27–33.
25. van Dijk JG, Thijs RD, van ZE, et al. The semiology of tilt-induced reflex syncope in relation to electroencephalographic changes. Brain. 2014;137:576–585.
26. Heyer GL, Harvey RA, Islam MP. Signs of autonomic arousal precede tilt-induced psychogenic nonsyncopal collapse among youth. Epilepsy Behav. 2018;86:166–172.
27. Heyer GL. Abdominal and lower-extremity compression decreases symptoms of postural tachycardia syndrome in youth during tilt table testing. J Pediatr. 2014;165:395–397.
28. Wieling W, Thijs RD, van DN, Wilde AA, Benditt DG, van Dijk JG. Symptoms and signs of syncope: a review of the link between physiology and clinical clues. Brain. 2009;132:2630–2642.
29. Lambert EH, Wood EH. The problem of blackout and unconsciousness in aviators. Med Clin North Am. 1946;30:833–844.
30. Wood EH, Lambert EH. The effect of anti-blackout suits on blood pressure changes produced on the human centrifuge. Fed Proc. 1946;5:115.
31. Vossler DG, Haltiner AM, Schepp SK, et al. Ictal stuttering: a sign suggestive of psychogenic nonepileptic seizures. Neurology. 2004;63:516–519.
32. Hoerth MT, Wellik KE, Demaerschalk BM, et al. Clinical predictors of psychogenic nonepileptic seizures: a critically appraised topic. Neurologist. 2008;14:266–270.
33. Duncan R, Oto M, Russell AJ, Conway P. Pseudosleep events in patients with psychogenic non-epileptic seizures: prevalence and associations. J Neurol Neurosurg Psychiatry. 2004;75:1009–1012.
34. Reuber M, Jamnadas-Khoda J, Broadhurst M, et al. Psychogenic nonepileptic seizure manifestations reported by patients and witnesses. Epilepsia. 2011;52:2028–2035.
35. Pavlova MK, Allen RM, Dworetzky BA. Sleep in psychogenic nonepileptic seizures and related disorders. Clin EEG Neurosci. 2015;46:34–41.
36. Latreille V, Baslet G, Sarkis R, Pavlova M, Dworetzky BA. Sleep in psychogenic nonepileptic seizures: Time to raise a red flag. Epilepsy Behav. 2018;86:6–8.
37. Asadi-Pooya AA, Asadollahi M, Sperling MR. Ictal pain: occurrence, clinical features, and underlying etiologies. Epilepsy Behav. 2016;61:59–62.
38. Elliott JO, Charyton C. Biopsychosocial predictors of psychogenic non-epileptic seizures. Epilepsy Res. 2014;108:1543–1553.
39. Shepard MA, Silva A, Starling AJ, et al. Patients with psychogenic nonepileptic seizures report more severe migraine than patients with epilepsy. Seizure. 2016;34:78–82.
40. Kerr WT, Janio EA, Braesch CT, et al. Identifying psychogenic seizures through comorbidities and medication history. Epilepsia. 2017;58:1852–1860.
41. Gazzola DM, Carlson C, Rugino A, Hirsch S, Starner K, Devinsky O. Psychogenic nonepileptic seizures and chronic pain: a retrospective case-controlled study. Epilepsy Behav. 2012;25:662–665.
42. Benbadis SR. A spell in the epilepsy clinic and a history of “chronic pain” or “fibromyalgia” independently predict a diagnosis of psychogenic seizures. Epilepsy Behav. 2005;6:264–265.