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Psychogenic non-epileptic seizures in children and adolescents: Part II – explanations to families, treatment, and group outcomes

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Abstract

Psychogenic non-epileptic seizures (PNES) – time-limited disturbances of consciousness and motor-sensory control, not accompanied by ictal activity on electroencephalogram (EEG) – are best conceptualized as atypical neurophysiological responses to emotional distress, physiological stressors and danger. Patients and families find the diagnosis of PNES difficult to understand; the transition from neurology (where the diagnosis is made) to mental health services (to which patients are referred for treatment) can be a bumpy one. This study reports how diagnostic formulations constructed for 60 consecutive children and adolescents with PNES were used to inform both the explanations about PNES that were given to them and their families and the clinical interventions that were used to help patients gain control over PNES. Families were able to accept the diagnosis of PNES and engage in treatment when it was explained how emotional distress, illness and states of high arousal could activate atypical defence responses in the body and brain – with PNES being an unwanted by-product of this process. Patients and their families made good use of therapeutic interventions. A total of 75% of children/adolescents (45/60) regained normal function and attained full-time return to school. Global Assessment of Functioning scores increased from 41 to 67 ($t(54) = 10.09$; $p < .001$). Outcomes were less favourable in children/adolescents who presented with chronic PNES and in those with a chronic, comorbid mental

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health disorder that failed to resolve with treatment. The study highlights that prompt diagnosis, followed by prompt multidisciplinary assessment, engagement, and treatment, achieves improved outcomes in children/adolescents with PNES.

Keywords
Psychogenic non-epileptic seizures, functional neurological symptom disorder, conversion disorder, dissociative convulsions, stress seizures, dissociation

Introduction

Psychogenic non-epileptic seizures (PNES) – time-limited disturbances of consciousness and motor-sensory control, not accompanied by ictal activity on electroencephalogram (EEG) – mimic epileptic seizures and are frightening for patients to experience and for parents and siblings to manage. PNES reflects an incidental/unwanted by-product of the body's response to threat and are triggered when a child or adolescent is in a highly aroused state. In Part I of this two-part article, we presented the diagnostic formulations – the clinical formulations about the probable neurophysiological mechanisms, known or hypothesized – that were constructed for 60 children and adolescents with PNES. Each formulation was our working hypothesis regarding the mechanisms by which distress, pain and states of high arousal were understood (or hypothesized) to cause the child/adolescent's PNES. The formulations clustered patients into six different PNES subgroups. This study, Part II, presents how we used the formulations to inform the explanations we gave to families – the lay versions of the diagnostic formulations – and to inform our choice of treatment interventions. Within each subgroup, we provide an example of a lay explanation and a brief summary of the interventions that were used to help patients in that subgroup to gain control over their PNES. We also report on the utility of training with a biofeedback device – MyCalmBeat – to help patients downregulate autonomic arousal and on the outcomes of the group as a whole.

Patients find the diagnosis of PNES difficult to understand. The transition from neurology to psychological medicine services can be a bumpy one. A common difficulty is that the patient and family perceive (and experience) PNES as a physical health problem – 'my child is having seizures' – and are often genuinely baffled (and sometimes angry and distressed) as to why they have been referred to psychological services. Explanations that the episodes are 'psychogenic' – that is, somehow caused by 'underlying psychological conflicts or stressors . . . often associated with depression, anxiety, trauma' (Reiter, Andrews, Reiter, & LaFrance, 2015, p. 3) – may only just add to the puzzlement. Moreover, the term *psychogenic* does little to enhance engagement. To a child or adolescent, it may seem that the doctor is suggesting that 'it's all in my head' or that 'I'm psycho', 'going off my head', or 'putting it on' – that is, suffering from a severe form of craziness or severe mental illness. To complicate matters, children/adolescents with PNES typically do not perceive themselves to be anxious, depressed or emotionally distressed (Kozlowska, Cruz, et al., 2016), so the implication of PNES as 'psychological' appears disconnected from the children/adolescents' personal stories and also from their somatic experiences of the 'seizures' (and the associated non-specific somatic symptoms) as a disturbance of the body. Not surprisingly, patients and their families are frequently offended and find the diagnosis difficult to accept.

Engagement with psychological services is enhanced when clinicians explain PNES in a way that makes sense to patients and families and that communicates effectively why a psychological, mind–body approach is likely to help the children/adolescents gain control of their non-epileptic seizures (Karterud, Risor, & Haavet, 2015; Kozlowska, 2013; Stone, 2014). A neurophysiological

perspective recognizes that a broad range of threat/stress stimuli, be they physiological and psychological, activate diverse brain–body responses. In patients whose neurophysiological responses lie on the extreme end of the normative curve, the body and brain may respond to the stressor in an excessively robust or exaggerated way, with PNES reflecting an incidental/unwanted by-product of the body's response to threat (Kozlowska, Catherine, et al., 2017). The neurophysiological perspective is also helpful in engaging patients and families: the clinician draws upon the patient's clinical history and presentation to construct a formulation and then explain how distress, pain and states of high arousal trigger the child/adolescent's PNES – via neurophysiological mechanisms that are known or hypothesized. This approach, the topic of this article, is the one we have adopted to work with children and adolescents with PNES and their families.

Because all brain–body defensive responses are preceded by changes in arousal, and because changes in arousal are part of the neurobiology of PNES and are the focus of clinical intervention, we provide the following brief summary of what is known about arousal in children/adolescents with PNES. Threats activate the body for action. When danger or potential danger is first identified, the body – or more accurately, the brain and body – responds with an increase in arousal, which is coupled with activation of the motor system. In our study with 57 children and adolescents with functional neurological symptoms – with more than half our participants experiencing PNES as one of their symptoms – we found that children/adolescents with PNES showed an increase in the baseline level of arousal. In the eyes-open, resting-state condition, our patients showed increased heart rate (reflecting increased sympathetic arousal) and lower heart rate variability (reflecting decreased vagal or restorative-parasympathetic tone) (Kozlowska et al., 2015). Under task conditions, they showed an increased cortical response to an auditory stimulus (suggesting activation of brain arousal systems) (Kozlowska, Melkonian, Spooner, Scher, & Meares, 2017) and increased motor readiness to emotion faces (suggesting activation of the motor system) (Kozlowska, Brown, Palmer, & Williams, 2013). In the current cohort of 60 children/adolescents with PNES, in the baseline eyes-open, resting state-condition, we also found that our patients (vs controls) showed increased heart rates and increased respiratory rates (Kozlowska, Rampersad, et al., 2017), consistent with the coupling between sympathetic arousal (which innervates the cardiac motor system) and the respiratory motor system. In one child in the sample, we also documented (via laryngoscopy) that the child activated an abnormally robust motor response in her larynx – adduction of the vocal chords – when she became frightened, distressed and highly aroused, resulting in cerebral hypoxia and a hypoxia-related non-epileptic seizure. The child's response is an extreme variant of the changes that occur in the body when the level of arousal increases: (skeleto)motor activation, which is part of the arousal response, causes an increase in the tone of proximal skeletal muscles of the body (including the laryngeal muscles), thereby raising the body and stabilizing it in preparation for action (and also, in the process, increasing the pitch of the voice). Taken together, these data suggest that patients with PNES show increases in brain–body arousal coupled with increases in activation of the motor system or specific components of it.

Finally, we want to mention the utility of diagnostic formulations for our work with children and adolescents with PNES. A formulation is a creative synthesis of a clinical case, drawing on elements from different system levels – biological, psychological, relational and social – expressed chronologically (Ross, 2000). The formulation tells the story of what has happened to the child/adolescent. When working with children/adolescents with PNES, the diagnostic formulation includes a working hypothesis about the probable neurophysiological mechanisms, known or hypothesized, that are understood (or hypothesized) to be the cause of the PNES. The diagnostic formulation – or, more specifically, the lay version of the diagnostic formulation that is communicated to the family – provides the clinical team and the family with a shared understanding of the PNES: how the PNES fit into the child/adolescents life story; 'how' emotional distress, illness and

states of high arousal have activated atypical defensive responses in the child/adolescent's body and brain; and 'how' the PNES are an unwanted by-product of this process.

The formulation also provides a roadmap for the journey of treatment (Gordon, Riess, & Waldinger, 2005). From the perspective of the Psychological Medicine team, the diagnostic formulation identifies areas of dysfunction that need to be addressed. From the perspective of the family, the formulation provides a rationale as to why certain mind–body, psychological, family and school interventions are being suggested by the Psychological Medicine team. The diagnostic formulation thus facilitates a problem-centred approach to therapy. In particular, it allows the Psychological Medicine team to collaborate with the family to implement treatment interventions that explicitly identify and target (and hopefully remedy) the underlying neurophysiological mechanisms – the area(s) of identified dysfunction – thereby helping the child/adolescent gain control of her PNES.

Aims of the study

This study has a number of aims. The first aim is to document the lay explanations of the diagnostic formulation, within each PNES cluster, that our Psychological Medicine team gave to children/adolescents referred for treatment of PNES and their families. The second aim is to summarize the treatment interventions that were used, within each PNES cluster, to help the child/adolescent gain control of the PNES. The third aim is to report on the utility of daily training with a biofeedback device – MyCalmBeat – to help patients downregulate their baseline levels of autonomic arousal/motor activation and to increase their capacity to experience states of calm and well-being. The fourth aim is to report on clinical outcomes of the group as a whole – tracked in terms of Global Assessment of Functioning scores, resolution of PNES, return to school and resolution of comorbid psychiatric disorders.

Methods

The methods are described in the companion article (Kozlowska, Catherine, et al., 2017). Only a very brief overview is provided here.

Participants

The study was approved by the Sydney Children's Hospital Network Ethics Committee. Participants and their legal guardians provided written informed consent in accordance with the National Health and Medical Research Council guidelines.

The participants of the study were 60 consecutive children and adolescents – 42 girls and 18 boys, aged 8–17.67 years (mean (M) = 13.45; standard deviation (SD) = 2.61) – who were referred to Psychological Medicine for treatment of PNES after assessment in the Department of Neurology during a 5-year period (April 2011–March 2016). The time from onset of PNES ranged from 1 day to 48 months (median = 2 months). In 28 cases (47%), the PNES presented alongside other functional neurological symptoms; in 10 cases (17%), the PNES presented alongside a chronic pain presentation; and in 22 cases (36.7%), the PNES were the primary presenting symptom. In 19 cases (32%), the PNES presented alongside a comorbid neurological condition (with developmental delay in two cases). In 42 cases (70%), the child/adolescent suffered from a comorbid mental health disorder, most commonly an anxiety disorder.

For the MyCalmBeat component of the study, 17 healthy controls from within the same age range – eight girls and nine boys, aged 9–17 years (M = 14.07; SD = 2.34), were recruited from the community

Procedure

All patients with PNES completed a comprehensive neurology assessment where the diagnosis of PNES was made and a referral to Psychological Medicine was arranged. The Psychological Medicine team conducted a comprehensive family assessment (Kozlowska, English, & Savage, 2013), at the end of which the team determined its diagnostic formulation (based on all available information) and presented it, in a lay version, to the child/adolescent and family. This family assessment was followed by an individual assessment with the child/adolescent. Updates to the formulation were communicated to families at the weekly family meetings that were a routine part of the hospital admission.

Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; DSM-IV-TR) diagnostic categories were used for identifying comorbid anxiety, depression and other mental health disorders. The Royal Alexandra Hospital for Children Global Assessment of Functioning (RAHC-GAF) – a modified DSM-IV-TR GAF (scores of 1–100) that includes functional impairment secondary to physical illness – was used to document functional impairment at presentation and on follow-up at a minimum of 12 months.

The first task in the individual assessment with the child was to determine, using MyCalmBeat, whether biofeedback could feasibly be used for daily training with the patient. The child version of the MyCalmBeat programme shows a picture of lungs – breathing in and breathing out – at an initial rate of up to 20 breaths/min.[1] The child/adolescent synchronizes her breathing with the picture and then decreases her breathing rate to the breathing rate associated with the highest heart rate variability – which is typically the lowest that she is capable of. This slow-breathing rate is called the resonant frequency rate because it increases the resonance between breathing, heart rate fluctuations and blood pressure fluctuations. Because breathing and autonomic arousal are tightly coupled (Dum, Levinthal, & Strick, 2016), breathing at the resonant frequency rate downregulates arousal via activation of the restorative vagus (reflected in increased heart rate variability and increased vagal tone) (Lehrer, Vaschillo, & Vaschillo, 2000). Resonant-frequency breathing rates are used for daily training with MyCalmBeat. The lowest resonant frequency rate attained by each child/adolescent with PNES by the end the training was used for the data analysis reported in this study. Healthy controls also completed the MyCalmBeat assessment during a single visit to the hospital.

The second task in the individual assessment was the completion of a body map on which the child/adolescent documented all her somatic symptoms on an outline of a human body. The body map was subsequently used in therapeutic work with the child/adolescent to identify warning signs of impending PNES and to monitor clinical progress (documented by changes in the symptoms depicted by the body map) as the child/adolescent got better.

The third task in the individual assessment was to repeat the explanation previously given to the family, to make sure that the child/adolescent understood the explanation and understood the rationale for the mind–body strategies used in the Mind–Body Programme.

The structure of the Mind–Body inpatient programme has been described previously (Kozlowska, English, Savage, & Chudleigh, 2012; Kozlowska, English, Savage, et al., 2013), and its specific application to children/adolescents with PNES is detailed in a number of published case histories (Chandra et al., 2017; Chudleigh et al., 2013; Kozlowska, Chudleigh, Elliott, & Landini, 2016). All patients enrolled in the Mind–Body Programme engage in daily individual therapy to learn how to manage their PNES, attend the hospital school to commence reintegration back to school and complete a physiotherapy exercise programme to increase their body's capacity to manage changes in body state and to increase their physical resilience. The standard inpatient programme for children/adolescents with PNES runs over a 2-week period. Admissions are typically followed by outpatient

treatment – both individual and family based – with community-based mental health services to address stressors that reside within the family and school systems and that function to trigger or perpetuate the patient's symptoms. The Psychological Medicine team continues, as needed, to support clinicians working in community-based services via telephone contact.

Follow-up with the family (and treating clinicians in the community) took place by telephone on a yearly basis. Children/adolescents from the first, second, third, fourth and fifth years of the study were followed up for 5, 4, 3, 2 and 1 years, respectively, making for a minimum of follow-up of 12 months.

Data analysis

The qualitative data – including clinical examples of lay explanations of PNES that were given to children/adolescents and their families, and brief summaries of the treatment interventions that were used to help children/adolescents gain control of their PNES – are presented below for each PNES subgroup. The quantitative data on patients' (vs healthy controls') ability to utilize MyCalmBeat and on clinical outcomes are presented for the PNES group as a whole using chi-square analyses and independent *t*-tests for categorical and continuous variables, respectively. Within the PNES group, a paired-samples *t*-test was used to look at the functional impairment (RAHC-GAF scores) at presentation and at follow-up, and Pearson's correlations were used to examine the relationship between outcome GAF scores and illness duration at presentation and outcome GAF scores and IQ. A cutoff of ≤3 months was chosen to delineate acute presentations. The adolescent who remained unwell due to postprandial and orthostatic syncope was excluded from the outcome analyses.

Results

Missing data

Resonant-frequency respiratory rates were available for 41/60 children/adolescents with PNES; data for 7 participants were missing, and the remaining 12 participants had not been able to decrease their respiratory rates to 20/min (the starting rate for the child version of MyCalmBeat).

Functional impairment at presentation

At presentation, RAHC-GAF scores for children/adolescents presenting with PNES ranged from 11 to 65 ($M=41$; median = 43). There was no difference in RAHC-GAF scores between patients with acute versus chronic presentations (≤3 months ($n=42$) and >3 months ($n=18$)) ($t(58)=.002$; $p=.998$), psychiatric comorbidities versus those without ($\chi^2=.74$; $p=.39$) and neurological comorbidity versus those without ($t(57)=.83$; $p=.41$). Correlations between GAF scores on presentation and duration of illness (PNES) ($r=-.05$; $p=.72$) and GAF scores on presentation and IQ ($r=.02$; $p=.89$) were not significant.

Service utilization

A total of 56 children/adolescents (93%) were admitted into the inpatient Mind–Body Programme, and 4 (7%) implemented a home programme. Of the former, the majority of patients (45/60, 75%) had one admission of 1 ($n=17$), 2 ($n=23$) or 3 weeks ($n=5$) duration. Six patients (10%) had very long admissions (range: 5–20 weeks; $M=8$ weeks) because comorbid functional neurological symptoms made earlier discharge impossible. Four patients had more than one admission for treatment of PNES (four had two admissions, and one had three admissions). Altogether, bed days for

the children/adolescents who participated in the inpatient programme ranged from 1 to 20 weeks, with a mean of 2.85 weeks and median of 2 weeks.

PNES Subgroup 1: lay explanation and treatment interventions for dissociative PNES

Our explanation for children/adolescents (and families) who were clustered into the dissociative PNES subgroup and their families was some version of the following script:

> You must be very relieved that Mary does not have epileptic seizures and that she does not have any nasty brain disease. Mary has non-epileptic seizures, which are caused by stress. There has been quite a lot of research into these types of seizures in the last 10 years. Now researchers have some pretty good hypotheses as to what might be going on. It seems that the brain is very sensitive to the effects of stress. Now when I say 'stress', I mean any event that the body finds stressful – illness, injury, or emotional distress caused by stressful life events or by trauma. Now, in Mary's case it seems that [restate stressors elicited in the family story] have activated her stress system – including stress systems in her brain. We know her stress system is activated because her body is signalling this in a variety of ways: [identify the stress-related symptoms suffered by the child such as pain, increased respiratory rate, increased heart rate]. It seems that Mary's stress system has been switched on but is not switching off like it is supposed to. Non-epileptic seizures are just another type of stress symptom. Mary is also a girl, and she is post-pubertal. All these stress-related disorders are more common in girls because female sex hormones also activate the stress system.

If the family wants to know more about the PNES (or comorbid functional neurological symptoms), a more detailed explanation about the probable neurobiology can be provided:

> The newer parts of the brain (the cortex) are very sensitive to stress and can be disrupted by stress and by stress hormones (catecholamines and endogenous opioids). When brain function is disrupted, the parts of the brain that process arousal and emotions – emotion-processing regions – become overactive, and they seem to disrupt motor-sensory programmes and to cause all sorts of weird motor and sensory symptoms. In Mary's case she has [describe all motor and sensory functional symptoms suffered by the child]. Sometimes, when there are sudden increases in arousal, the overstressed brain is pushed to the limit of its capacity. When this happens, the older parts of the brain (the brain stem), which are usually controlled by the cortex, can get out of control, in which case Mary can have a non-epileptic seizure.
>
> We know that you have never heard of non-epileptic seizures before, but we see children and adolescents with non-epileptic seizures all the time. We have seen quite a few kids who have presented just like Mary. There are lots of different names for these non-epileptic seizures, so I will write them all down for you. Please be careful if you look them up on the Internet, because adults with this condition don't do very well, whereas kids do extremely well – so you don't want to read things on the Internet that do not apply to Mary. To treat Mary, we will need to help her learn how to manage stress and how to better regulate her body and brain's response to the stress. This can be difficult at first, and even adults find it hard to do. But we actually have had a lot of success with kids, and we will also need to work with your family and Mary's school to try and manage the tricky issues you have raised.

Because our diagnostic formulations in the dissociative PNES subgroup were based on the hypothesis that the PNES are triggered by sudden increases in cortical arousal leading to a functional disruption (dissociation) of brain areas that typically work together, the interventions used with this subgroup targeted the arousal system. These interventions helped our patients both to identify states of increasing arousal and to downregulate the arousal before PNES were activated – thereby learning to avert their PNES. We also used generic interventions – daily training with MyCalmBeat and engagement

in the daily exercise programme – to help patients decrease baseline arousal and to enhance flexibility and resilience of autonomic (visceromotor) and skeletomotor systems.

The interventions were implemented as follows. Initially, patients engaged in daily training with MyCalmBeat (or another type of relaxation exercise if unable to use MyCalmBeat) to downregulate baseline arousal. The use of MyCalmBeat, combined with body-awareness training (Ogden & Fisher, 2015), enabled the child/adolescent to somatically experience what her body felt like when it was in a 'calm' versus a 'revved up' state. Patients practised their body-awareness skills on a daily basis and documented their somatic sensations on body maps (see Figure 1). In parallel, the child/adolescent undertook the task of identifying warning signs of their PNES and documenting them on a body map. Warning signs were subsequently integrated into the child/adolescent's management plan and were used as indicators that an arousal-decreasing intervention – and an immediate transfer to a safe sitting position – needed to be implemented to avert the PNES. Arousal-decreasing interventions that were used to attain non-epileptic seizure control included slow-breathing exercises, soothing imagery, mindfulness exercises, relaxation exercises, rhythmic tensing of large muscle groups, exercises involving attention and movement (e.g. attending to a ball and throwing the ball from one hand to another (or to a parent)), distraction and grounding exercises. Once the child/adolescent had achieved some control over the PNES – and could see that control was possible – the therapy (with the child and with the family) changed focus to address the psychological or physiological threats that triggered the PNES in the first place.

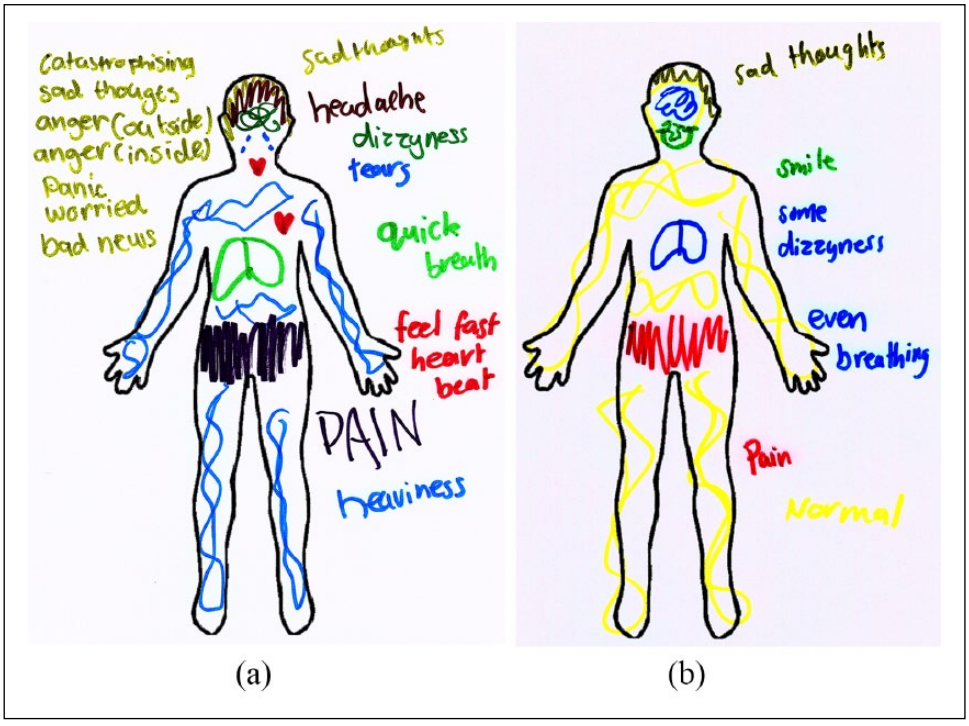

Figure 1. The body maps are the work of an adolescent girl. (a) The first body map depicts the girl's body state at the beginning of a therapy session, when she is in a highly aroused state. (b) The second map depicts the shift in the girl's body state at the end of the therapy session, after the girl has completed a MyCalmBeat training session followed by a relaxation exercise with soothing imagery.

PNES Subgroup 2: lay explanation and treatment interventions for dissociative PNES triggered by hyperventilation

Our explanation for children/adolescents (and families) who triggered their PNES by hyperventilation (HV) was some version of the following script:

> As we have been saying, the brain (the cortex) is very sensitive to stress and can be disrupted by stress. When we were talking during the session, I counted Martha's breathing rate – it was 35 breaths per minute. Do you know what her respiratory rate should be? It should be less than 20 breaths per minute. And actually, because Martha is an athlete, it should be much less than that. Martha's high breathing rate tells me that Martha's body is really revved up – really aroused. Martha's body is telling me that it is very aroused in lots of other ways [include all other signs and symptoms of physiological arousal]. I think that Martha's body has become so aroused because [list stressors elicited in family story; if stressors are distant, the idea of a primed stress system can be included]. Now, when Martha hyperventilates like that, she is actually changing her brain function. The brain hates hyperventilation. When Martha hyperventilates, she blows out too much carbon dioxide. The level of carbon dioxide in the blood drops too low. The brain does not like low levels of carbon dioxide, and it responds by secreting stress hormones and constricting arteries. These stress-related changes cause all sorts of neurological symptoms [altered consciousness, dizziness, head dropping, staring, visual changes, or eye rolling], just like you described for Martha. The first thing we can do to help Martha control her non-epileptic seizures is to help her notice when she starts to hyperventilate, and to help her control her breathing. If she can learn to read her body better, she should be able to settle her body and her brain, and to avert the non-epileptic seizures before they start. We shall also need to address [other problems identified at assessment]. But that part of the treatment will take much longer, and we shall need the involvement of the whole family.

Because our diagnostic formulations in the dissociative PNES triggered by HV subgroup were based on the clinical observation that the child/adolescents PNES were typically preceded by and triggered by HV, the interventions used with Subgroup 1 targeted the respiratory motor system (breathing). Using body-awareness training, we helped the children/adolescent identify warning signs of an impending PNES (typically somatic symptoms of sympathetic arousal coupled with increased breathing rate (activation of the respiratory motor system) and symptoms of HV). The child/adolescent documented the warning signs on a body map. Because it is possible to exert voluntary control over breathing, the patients were then taught to avert their PNES with breathing interventions (by slowing down breathing rate and changing body-brain state). For children who were unable to utilize breathing interventions, because attention to breathing had a paradoxical effect and made them breath faster, other arousal-decreasing interventions were used (see section above regarding dissociative PNES). When HV was triggered by anxious thoughts, catastrophizing or trauma-related flashbacks, the treatment intervention naturally expanded into therapies that addressed these issues via individual and family work. Wherever possible, the patients in this subgroup also engaged in MyCalmBeat training to downregulate their baseline arousal and to help them differentiate what their body felt like when it was calm and when it was aroused (and breathing fast). All patients also engaged in the physiotherapy exercise programme.

PNES Subgroup 3: lay explanation and treatment interventions for innate defence responses presenting as PNES

Our explanation for children/adolescents (and families) with PNES resulting from innate defence responses was some version of the following script:

> An important function of the brain is to activate brain-body defence responses that protect us from danger. The programmes for these defence responses are located in the older part of the brain – the brain stem [can show a picture of the brain]. The tricky part is that we don't have control over the on-off switch: the brain can activate the programmes automatically when we are exposed to danger or when body arousal is very high. For the brain, high arousal signals danger.
>
> You probably know about some of these responses already. [Discuss flight, flight and freezing and show the child pictures]. But there is also another type of defence response called [insert tonic immobility or collapsed immobility as relevant], which you probably have not heard about. In tonic immobility the animal or person goes into a state of unresponsive immobility [show video of an opossum going into an immobility state]. The body can be rigid, this is called tonic immobility, or the body can be floppy, this is called collapsed immobility. In collapsed immobility the person will just faint or collapse in response to the fear, so some people call this response fear-induced fainting. It seems that your brain thinks you are unsafe so your brain is activating tonic/collapsed immobility just like the possum we saw in the video.

Because the majority of patients in this subgroup had been subjected to maltreatment or exposed to significant loss or trauma, our first priority was to make sure that the child/adolescent was safe in the current family context. Next, we implemented body-awareness training to help the children/adolescent identify signs of increasing arousal – and with time – the trigger events (usually memories/thoughts of trauma-, loss-, or fear-related events) that functioned to activate the tonic/collapsed immobility response. In individual therapy, the child/adolescent then trialled a range of de-arousal and grounding interventions to establish which were most helpful in downregulating arousal and averting the activation of the innate defence response (PNES). If the child/adolescent was willing – and the family supportive – the intervention then progressed to a trauma-focused intervention whose aim was the processing of trauma-related memories. Wherever possible, the patients in this subgroup also engaged in MyCalmBeat training to downregulate their baseline arousal and to help them differentiate what their bodies felt like when calm and when aroused. All patients engaged in the physiotherapy exercise programme.

PNES Subgroup 4: lay explanation and treatment interventions for PNES associated with syncope triggered by vocal cord adduction in the context of distress

Our explanation for the child (and family) with vocal cord adduction involved an outline of the sequence of events – including the cord adduction – that led to the child's non-epileptic seizure:

> When Mika experiences a tight feeling in the chest, he gets worried that an asthma attack is about to occur. His worry then progresses to panic. When he panics, his body becomes very aroused (revved up). We know this because his face goes red, he cries, and he starts to breath faster and to take in large gulps of air. Sometimes he gets himself into such a state that he starts coughing. When the body becomes so panicked, the arousal system and the motor system are activated. All the muscles in the body activate and tense up. In Mika's case, the muscles in Mika's voice box – the part of his airway that houses the vocal chords so he can talk – also activate. When these vocal-chord muscles activate too much, they close Mika's vocal chords [draw a picture of the vocal chords closing if this is helpful]. This closing up of the vocal chords shuts off the airway and interferes with the flow of oxygen to the lungs. We know this because Mika goes blue around the lips. That means the oxygen in his blood is a bit low. This is called hypoxia. When brain oxygen is low, the child will faint and will sometimes also have a hypoxia-related non-epileptic seizure. A hypoxia-related non-epileptic seizure looks just like an epileptic seizure, but Mika does not have epilepsy. To avoid the non-epileptic seizures, we have to help Mika manage his panic better. We will teach him (and you) exercises to help manage this panic. At the same time, the speech therapist will teach Mika exercises that will help him open his vocal chords.

Because our diagnostic formulation for the child in Subgroup 3 was based on clinical findings of vocal cord adduction that occurred in the context of distress, the intervention with the child targeted the management of the motor respiratory system and anticipatory anxiety about having an asthma attack. We integrated 'voice' exercises that opened up the vocal cords into the anxiety-management strategies that were practised with the child (see Vignette 4 in companion study for details) (Kozlowska, Catherine, et al., 2017).

As a word of warning, in the case of patients with asthma, care needs to be taken when introducing slow-breathing interventions that aim to downregulate sympathetic activity and upregulate vagal activity. In a subset of patients, breathing interventions may trigger their asthma because vagal efferents are also involved in bronchoconstriction, in the production of mucous secretions, and in mast cell modulation in the lungs.

Subgroup 5: lay explanation and treatment interventions for non-epileptic seizures associated with syncope triggered by activation of the valsalva manoeuvre in the context of distress

Our explanation to the child (and her grandmother) whose PNES were associated with activation of the valsalva manoeuvre is outlined below:

> When Lizzy becomes distressed, she holds her breath. In the videos from her vEEG you can see her grimacing as she holds her mouth tightly closed. When Lizzy holds her breath for too long, the oxygen levels in the lungs and blood decrease, and Lizzy faints. Soon after she faints, the brain reboots itself and normal breathing is reestablished. Sometimes fainting is accompanied by jerking movements due to the lack of oxygen in the brain – which is called a hypoxia-related non-epileptic seizure. The jerking movements can look just like an epileptic seizure. We know that Lizzy does not have epilepsy because her EEG was normal. So to help Lizzy you will need to coach her to do a nice slow-breathing exercise when she becomes distressed. You will also need to find a therapist to work with Lizzy to help her manage her grief and her distress. We will talk to the therapist and explain to her what Lizzy is doing with her breathing.

Subgroup 6: lay explanation and treatment interventions for non-epileptic seizures associated with syncope triggered by reflex activation of the vagus

The explanation to the child/adolescent (and family) for PNES associated with reflex activation of the vagus is some version of the script written below:

> Thank you for giving such clear history of the non-epileptic seizures. It seems like Siew faints in response to the pain. Let me explain what I think might be happening. Siew has chronic pain. But there are days when she experiences acute surges of pain. Acute pain surges can activate a nerve called the defensive vagus. Activation of the defensive vagus causes a sudden decrease in heart rate and a sudden drop in blood pressure. The brain does not get enough oxygen so it becomes hypoxic and Siew faints. Sometimes the faint is followed by jerking movements that look just like an epileptic seizure. The jerking is a hypoxia-related non-epileptic seizure that occurs in some people when they faint. It can look just like an epileptic seizure. The term *vasovagal syncope* is also used to refer to this type of fainting. Convulsive syncope is used to refer to fainting episodes that are followed by hypoxia-related seizures.

Ability of children/adolescents with PNES to utilize the MyCalmBeat biofeedback tool

Of 53 patients with PNES who participated in the MyCalmBeat evaluation, 41 (77%) were able to utilize the biofeedback tool. With daily training, these patients were able to attain

resonant respiratory rates of 4.5–20 breaths/min ($M = 8.6$; median = 7; $SD = 3.24$), which were comparable to those of healthy controls ($n = 17$), who attained respiratory rates of 4.5–17 breaths/min ($M = 7$; median = 6.5; $SD = 2.92$) ($t(37.25) = 1.69$; $p = .1$). There were no significant differences between patients and controls in terms of age ($t(75) = -.89$; $p = .38$) and sex ($\chi^2 = 3.06$; $p = .08$).

A total of 12 patients with PNES were unable to utilize MyCalmBeat because they were unable to decrease the initial starting respiratory rates of 20 breaths/min. Most of these patients (10/12) were able to utilize a different breath intervention while lying down with a cup on the abdomen, typically coupled with imagery of a safe space. Comorbid neurological abnormality was overrepresented in this subgroup of children as compared to the rest of the sample ($\chi^2 = 4.93$; $p = .03$). There were no differences in the spread of IQs ($\chi^2 = 1.57$; $p = .67$).

The two remaining patients were unable to use any type of breathing intervention. Focusing on the breath would cause one 13-year-old boy to hyperventilate, sometimes triggering PNES or HV-induced chest pain. The other, an 11-year girl, presented to our team with long periods of unresponsiveness and did not engage with any interventions that required her cooperation.

Outcomes

A total of 75% of the children/adolescents (45/60) regained normal function and attained a full-time return to school (see Table 1). The majority of these children/adolescents attained full control over their PNES ($n = 40$), and only a handful relapsed for short periods in the context of subsequent stress ($n = 5$) (see Table 1). All these patients and their families made good use of the therapeutic intervention and got better at monitoring their stress levels and utilizing their mind–body strategies over time. Improvements in function were reflected in RAHC-GAF scores: mean GAF score increased from 41 to 67 ($t(54) = 10.09$; $p < .001$). Follow-up GAF scores were documented at a minimum of 12 months after presentation.

Post hoc examination of vulnerability and risk factors showed the following. Children/adolescents who presented with chronic PNES (>3 months' duration) had worse outcomes than children/adolescents who presented with acute PNES (≤3 months' duration) (mean outcome GAF = 58 vs 71; $t(53) = 2.77$; $p = .008$). Likewise, Pearson's correlation showed that the longer the illness duration (PNES) at the time of presentation, the worse the outcome GAF score ($r = -.33$; $p = .015$). The four patients who did not gain control of their PNES with intervention and who went on to have chronic PNES had already been ill for 12–34 months ($M = 19.5$ months) at enrolment into the Mind–Body Programme. A small group of children/adolescents ($n = 10$; 18%) who either presented with a severe chronic mental illness (that failed to resolve despite treatment) or whose functional neurological symptoms evolved into a severe chronic mental illness also had worse outcomes than the rest of the cohort (mean outcome GAF = 41 vs 73; $t(53) = 8.21$; $p < .001$) (see Table 1). By contrast, children/adolescents with neurological comorbidity ($n = 19$) – of whom only 2 had developmental delay – had comparable outcomes to those with PNES without any neurological comorbidity (mean outcome GAF = 68 vs 65; $t(52) = .64$; $p = .52$). Likewise, children/adolescents presenting with PNES and other functional neurological symptoms or with PNES and chronic pain disorder had comparable outcomes to the rest of the cohort (mean outcome GAF = 69 vs 66; $t(53) = .731$; $p = .46$/mean outcome GAF = 60 vs 69; $t(53) = 1.50$; $p = .14$, respectively). Examination of IQ showed that there was no correlation between IQ and outcome GAF ($r = .039$; $p = .776$. Likewise, comparison of outcome GAF between children/adolescents with higher IQ (superior and average) and lower IQ (borderline and delayed) was not significant ($t(53) = 1.04$; $p = .30$)

Table 1. Clinical outcomes of the 60 children and adolescents participating in the study (a minimum of 12 months' follow-up).

Outcome	Number	Percent	GAF score Range Mean Median
Full Recovery from PNES **Full-time return to school**			
PNES control and fully recovery from any comorbid mental health disorders	29	48.3	60–90 Mean 78 Median 80
PNES control, ongoing management of a comorbid mental health disorder by a mental health service, full-time return to school. Comorbid mental health problems included anxiety ($n = 3$), anxiety and behavioural difficulties including angry/aggressive outbursts ($n = 3$), anxiety and bipolar disorder ($n = 1$) and anxiety, behavioural problems and chronic pain ($n = 1$)	8	13.3	45–65 Mean 58 Median 60
PNES control. Initially suffered from relapses of other conversion symptoms. Subsequently fully recovered. Full-time return to school	3	5.0	45–85 Mean 62 Median 55
PNES relapses with subsequent stress **Full-time return to school**			
PNES relapses with stress, otherwise well with full-time school attendance	4	6.7	60–80 Mean 74 Median 78
PNES relapsed with stress, chronic mental health disorder (anxiety, depression and unresolved grief) and full-time return to school	1	1.7	GAF 55
Recovery from PNES **Part-time return to school**			
PNES control, ongoing management of a comorbid mental health disorder by a mental health service, part-time (slower pathway) return to school. Comorbid mental health problems included depression with recurrent self-harm and suicidal attempts ($n = 1$), chronic anxiety needed hospitalization ($n = 1$), chronic fluctuating anxiety and depression ($n = 1$) and chronic depression ($n = 1$)	4	6.7	30–45 Mean 39 Median 40
PNES relapses with subsequent stress **Part-time return to school**			
PNES relapses with stress, chronic mental health disorder (developmental delay with severe anxiety). Part-time (slower pathway) return to school	1	1.7	GAF 41
PNES relapses with stress, chronic comorbid mental health disorder (anxiety, recurrent depression and possible bipolar). Failure to return to school. Enrolment in homeschooling	1	1.7	GAF 35
Chronic PNES			
PNES chronic, chronic mental health disorder, failure to return to school. Comorbid mental health issues included anorexia nervosa with anxiety and depression; chronic anxiety and depression; chronic anxiety, depression and relapsed of conversion paralysis; and recurrent relapsed of depression, PTSD and conversion paralysis	4	6.7	35–45 Mean 40 Median 40

(Continued)

Table 1. (Continued)

Outcome	Number	Percent	GAF score Range Mean Median
Chronic NES (other)			
Chronic postprandial and orthostatic-related NES	1	1.7	GAF 41
Lost to follow-up			
Dropped out of treatment with the team and was lost to follow-up	2	3.3	–
Completed the intervention with the team, was referred to local mental health services and was subsequently lost to follow-up	2	3.3	–
	60	100.0	56

PNES: psychogenic non-epileptic seizures; NES: non-epileptic seizures; PTSD: post-traumatic stress disorder.

Discussion

This study tracked the assessment, engagement process and treatment outcomes of 60 children and adolescents with PNES who presented over a 5-year period, and who were treated in the Mind–Body Rehabilitation Programme with the Psychological Medicine team. We found that children/adolescents with PNES and their families were able to accept the diagnosis of PNES and to engage in treatment when we used a neurobiological framework to explain how emotional distress, illness and states of high arousal could activate atypical defence responses in the body and brain – with PNES being an unwanted by-product of this process. Families were able to tolerate the knowledge that our explanations were working hypotheses reflecting our best effort to understand their child/adolescent's story using current research findings and that scientists were still in the process of running research studies to better understand PNES. We also found that once patients and their families had accepted the formulations, they made good use of therapeutic interventions, with three quarters of children/adolescents regaining normal function and attaining a full-time return to school. Risk factors linked to poor outcomes included long illness (PNES) duration at presentation and the presence of severe chronic mental health disorder(s) that failed to resolve with treatment.

The neurophysiological framework also enabled us to use a problem-centred approach to therapy, one involving the use of targeted interventions to help the child/adolescent better regulate both the body and the mind (emotions and cognitions), thereby undercutting the processes generating PNES. Most importantly, this way of working – connecting the body and mind – produced promising clinical outcomes. Three quarters of our patients had a good functional outcome. These outcome data provide more reason for optimism than those cited by Yadav, Agarwal, and Park (2015), who found that only 50% of their sample of 90 adolescents had attained PNES control at 1 year (55% at 2 years) (Yadav et al., 2015). The difference in outcomes between the two studies suggests that targeted interventions delivered soon after diagnosis via an intensive inpatient programme delivered by a specialist multidisciplinary team – followed by outpatient care – provide better outcomes than routine outpatient care.

We also examined a range of vulnerability and risk factors and their impact on patient outcomes. Akin to findings by Yadav et al. (2015), we found that prompt diagnosis of PNES close to symptom onset – along with, in our work, prompt assessment engagement and treatment by the Psychological Medicine team – was associated with favourable outcomes. Conversely, children/adolescents who presented with PNES symptoms for more than 3 months' duration had less favourable outcomes. We also found that children/adolescents who suffered from or who subsequently developed a chronic

severe mental health disorder that failed to resolve with treatment had significantly worse outcomes even though their PNES may actually have resolved (see Table 1). Our data highlight the important role of mental health professionals in providing systemic interventions that address other comorbid mental health disorders – and relational risk factors – that commonly present alongside PNES.

In contrast to Yadav et al. (2015), outcomes for children/adolescents with comorbid neurological abnormality (including epilepsy) were comparable to the rest of the group. Theoretically, neurological comorbidity increases an individual's risk of PNES because brain abnormalities of any sort are likely to make the individual's cerebral cortex more sensitive, and less resilient, to the effects of hypoxia or arousal-related brain changes and less able to utilize cognitive strategies to manage psychological stress. Our clinical impression from working with this group of children/adolescents was that they required more support from the treatment team and the family, both during and after the mind–body intervention, to attain good outcomes (see, for example, Chandra et al, 2017). This impression was supported by our findings that neurological comorbidity was overrepresented in the subgroup of children who were unable to utilize MyCalmBeat. Therapists working with these children had to find alternate individualized interventions to help the child/adolescent downregulate arousal.

Limitations have been discussed in previous publications (Kozlowska, Rampersad, et al., 2017) (Kozlowska, Catherine, et al., 2017). An additional limitation in this study was the small number of patients ($n=2$) with intellectual disability. Although our clinical impression is that the challenges of working with patients with developmental delay are substantial and that the outcomes of children/adolescents with developmental delay and PNES are likely to be less favourable, the small number of children with developmental delay did not allow us to examine this issue. Another limitation is the lack of a formal control group that was randomized to treatment as usual. However, because the illness duration at time of presentation to our tertiary service varied enormously, the children/adolescents with long illness duration prior to enrolment into the Mind–Body Programme served as a de facto comparison group. Our results demonstrate that better outcomes can be achieved through prompt diagnosis, engagement and targeted treatment informed by recent advances in the understanding of neurophysiological mechanisms. Untreated PNES are likely to prime brain systems, enabling reactivation of the affected networks by relatively modest stress stimuli, thereby perpetuating the PNES and resulting in a chronic, difficult-to-treat clinical picture.

In conclusion, in this study, we describe our clinical approach to working with children and adolescents with PNES in our inpatient Mind–Body Rehabilitation Programme designed for the treatment of PNES and other functional neurological disorders. The study provides child and adolescent psychiatrists and other mental health clinicians with a neurophysiological framework for understanding and treating PNES. This framework can be used, in turn, to discuss PNES with patients and their families. Conceptualizing PNES from a neurophysiological perspective enables clinicians to talk about and explain probable underlying mechanisms in a straightforward way that decreases patient fear and anxiety, facilitates engagement, provides a rationale for treatment and increases the child/adolescent's and family's understanding of, and commitment to, treatment.

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Note

1. We had a child version of MyCalmBeat developed for our work with children and adolescents because we were not able to use the adult version, in which the highest respiratory rate allowed is 7 breaths/min, which most of our patients with psychogenic non-epileptic seizures (PNES) – and many healthy children/adolescents – are unable to attain.

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