



**HAL**  
open science

# From epileptic hysteria to psychogenic non epileptic seizure: Continuity or discontinuity for contemporary psychiatry?

J. Bruno, J. Machado, Y Auxéméry

## ► To cite this version:

J. Bruno, J. Machado, Y Auxéméry. From epileptic hysteria to psychogenic non epileptic seizure: Continuity or discontinuity for contemporary psychiatry?. *European Journal of Trauma and Dissociation*, 2020, 5 (1), pp.100190. 10.1016/j.ejtd.2020.100190 . hal-03095620

**HAL Id: hal-03095620**

**<https://hal.science/hal-03095620>**

Submitted on 3 Feb 2023

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial 4.0 International License

**De l'hystéro-épilepsie à la crise psychogène non épileptique :  
Continuité ou discontinuité pour la psychiatrie contemporaine ?**

**Bruno, J.<sup>2,3</sup>, Machado, J.<sup>2,3</sup> & Auxéméry, Y<sup>1</sup>.**

<sup>1</sup>Service de Psychiatrie et de Psychologie Clinique, Hôpital d'Instruction des Armées Legouest

27 Avenue de Plantières BP90001, 57070 METZ CEDEX 3, France.

<sup>2</sup>Université de Lorraine, EA 4360 EPSAM-APEMAC, 57000 Metz, France <sup>3</sup>Centre

Pierre Janet – Université de Lorraine, 57000 Metz, France

Corresponding author : Julien BRUNO – [Julien.bruno@univ-lorraine.fr](mailto:Julien.bruno@univ-lorraine.fr) – Centre Pierre Janet,

Université de Lorraine, 57000 Metz, France

## **From epileptic hysteria to psychogenic non epileptic seizure:**

### **Continuity or discontinuity for contemporary psychiatry?**

#### **Abstract**

In the wake of Charcot, the École de la Salpêtrière became passionate about major crises or hysterical attacks: at the time, hysteria was not just about this great crisis, even if it represented an ideal heuristic model for a patient presentation that left a lasting impression on people's minds. The great attack is a framework for the study of hystero-epilepsy that opens the way to psychopathological explanation. The heirs of the Salpêtrière School's knowledge protesters each boast of imposing a universal model of hysteria seen from the perspective of its causes. If Babinski considers hysteria to be a purely psychogenic mechanism, it is of the subconscious mind or self-suggestion, the subject abandoning himself to his symptom. Unlike pithiatism, Freud and Breuer understand crisis as the cleavage of the content of consciousness that testifies to the return of a forgotten traumatic memory. These questions, partly eclipsed by modern psychiatry, are resurfacing with the description of a new clinical entity: the nonepileptic psychogenic seizure.

Non-epileptic psychogenic seizures (PNES) can be defined as paroxysmal manifestations clinically suggestive of epileptic seizures but related to unconscious psychogenic processes. Clinical diagnosis is particularly difficult. Most patients with PNES incorrectly use anticonvulsant therapy, which is ineffective and has frequent side effects. To establish a diagnosis that discriminates between PNES and epilepsy, the electroencephalographic recording coupled with a video recording is the paraclinical examination of choice. The treatment of PNESs is complex: despite a diagnosis made by the patient and joint follow-up by a specialized team of neurologists and psychiatrists, the prognosis remains uncertain.

We sought a phenomenal continuity between a major hysterical attack, hystero-epilepsy and a non-epileptic psychogenic seizure. From one time to another, the diagnosis of epileptic seizures is a diagnosis of clinical and paraclinical elimination. A reference to hysteria is found over time, currently confusing into dissociative and somatoform disorders. Consistent with ancient theories, the psychotraumatic dimension of the epileptoid phenomenon is found in recent epidemiological studies.

Since hysteria was not just about major attacks, the entity called PNES is a group of clinical manifestations with heterogeneous etiologies. Some research teams consider PNESs to be the result of non-epileptic, normal electroencephalogram neuronal hyperexcitability. Other authors have adopted the etiological hypothesis of recurrent dissociative disorders and equivalent to a particular form of post-traumatic stress disorder.

**KEYWORDS :**

Psychogenic non epileptic seizure, hysterical seizure, differential diagnosis, epilepsia, psychic traumatism, dissociative disorder.

## **Introduction**

Already mentioned by Louyer-Villermay in *Dictionnaire des sciences médicales* (Louyer-Villermay, 1818), epileptic hysteria found an aura in Paris in the end of the 19<sup>th</sup> century where clinicians were passionate about hysteria. “L’École de la Salpêtrière” describes the “big crisis” or “hysterical big attack”. Several students of Charcot will remain faithful to the Master by repeating his descriptions and theoretical approaches. When he died, others will emancipate themselves from models by testing an etiopathogenical approach of what is then called and described as “*hysteria*”. But, thereafter, the phenomenon of epileptic hysteria is overshadowed.

This phenomenon might come back today by the term of *psychogenic non epileptic seizure* (PNES). After an historical approach of big epileptoid seizures, we will define the modern concept of psychogenic seizure. These two constructs present many similarities.

### **I. Descriptions of “L’école de la Salpêtrière”: The “big hysterical attack” from the 19<sup>th</sup> century.**

#### **1) The great four-phase hysterical attack**

The description of the big hysterical attack takes shape in the work of Charcot (Charcot, 1892). For the articles of the leader as for his *Leçons*, the place left to hysteric phenomena is not much important compared to the huge number of works purely neurological. The several chapters given to hysteria evoke the big seizures which settle in the nebula of hysteria, which may express itself via many other clinical forms. The famous descriptions of “L’école de la Salpêtrière” interested some hysteric-epileptic patients whom we find the same clinical pictures from one author to another. If the ambition of each member of

Charcot's team was written by a treaty on hysteria, the concept itself remains elusive despite the size of the volumes that merge in the semeiological descriptions.

In his « *Traité complet de l'hystérie* » published in 1846, Landouzy defined for the first time the idea of epileptic-hysteria (Landouzy, 1846). The issue is discussed in the chapter on the complications of hysteria, including complicated epilepsy hysteria. The author was based on Dr. Beau's study conducted in 273 convulsive subjects at the Salpêtrière and who found 209 epileptic etiologies, 19 cases of hysteria, 32 cases of hystero-epilepsy and 13 cases without a definite diagnosis among these observations. For Landouzy, hystero-epilepsy is divided into two clinical forms of different temporal expressions: hystero-epilepsy with distinct seizures and hystero-epilepsy with compound seizures. In the first description, hysterical attacks and epileptic seizures occur in separate seizures over time, separated by symptomatic pauci free intervals. In the second presentation of hystero-epilepsy, the same seizure combines hysterical and epileptic symptoms to create a composite form. The frequency of made up crises would be much lower than that of separate one. Landouzy reports that in the same patient, distinct and compound seizures can coexist, although this alternative is rare. The author articulates the course of hystero-epileptic seizures according to the subject's biographical trajectory. Primary epilepsy is more often complicated by post-pubertal hysterical seizures than the opposite. In this usual case, it is exclusively a hystero-epilepsy with distinct seizures. On the other hand, a fright or violent emotion experiencing a nervous subject would favor a combined seizure hystero-epilepsy. Landouzy devotes himself mainly to a semiological description of the seizures enriched with some etiopathogenic considerations but without theory of the causes or mechanisms involved. This lack of precise etiological reflection is the rule among Charcot's students who apply themselves to accurately transcribing the master's Lessons. In his famous clinical studies on great hysteria or hystero-epilepsy (Richer, 1885), Richer mentions only a

few psychopathological considerations to limit himself exclusively to description. Briquet described various attacks and ensured the frequent mixing of the paintings by always reaching Charcot's position (Briquet, 1859). Finally, in the second part of his clinical and therapeutic treatise on hysteria (De la Tourette, 1895), De La Tourette scrupulously repeats the description of the great hysterical attack, still called a great convulsive attack, whose first epileptoid period includes successive tonic and clonic phases.

Thus, hysteria is not limited to the great crisis, even if the latter represents an ideal heuristic model for a patient presentation that left a lasting impression on people's minds. At La Salpêtrière, the great attack was an archetype intended for the study of the declinations of hysteria: the epileptoid symptoms are only one phase of the great attack, itself a fragmented symptomatic element of hysteria which takes a moderate place in Charcot's immense medical work. However, these epileptoid phenomena have questioned and even amazed many clinicians. Hystero-epilepsy consecrated a spectacular form of hysteria, a space of expression of interest to the medical field.

## **2) A contested clinic**

Depending on the authors of the time, hystero-epilepsy is recognized either as a specific entity (Landouzy, 1846) or as a dimensional continuity of the great hysterical attack of which it constitutes a variety (de la Tourette, 1895). However, the two conditions can interact with each other to change the "symptomatology of epilepsy by hysteria, and hysteria by epilepsy" as a whole (Landouzy, 1846). Without being blamed for hystero-epilepsies, other manifestations of hysteria can be confused with non-motor epileptic seizures. Richer cites syncopal attacks, lethargy and catalepsy (Richer, 1885). The heirs of the Salpêtrière School's

knowledge protesters each boast of imposing a universal model of hysteria seen from the perspective of its causes. Bernheim seeks to move away from the agreed descriptions of the "culture hysteria" whose artificial and manufactured nature he castigates (Bernheim, 1913). The Nancy doctor refers to a symptomatic and etiological continuum between the small vulgar attack or hysteria and the large so-called hysterio-epileptic attack. Moreover, and unlike Charcot, Bernheim considers that any individual is potentially suggestible even if the hysterical subject is more so.

Babinski took up the work of his clinical professor to denounce several limits, including the false diagnosis of hysteria on a somatic pathology, and the error of confusing artificial disorder or pathomimy with hysteria (Babinski, 1909). Babinski scrupulously details the clinical examination to determine objective signs that will differentiate between hemiplegia of hysterical origin and hemiplegia of neurological origin. But the semiology of epileptoid seizures is more complex to authenticate and differentiate conversions involving the peripheral nervous system. To remedy this pitfall, the author defines Pithiatism by referring to the imitation of disorders that "the suggestion realizes, and that persuasion heals". If Babinski considers hysteria to be a purely psychogenic mechanism, it is of the subconscious mind or self-suggestion, the subject abandoning himself to his symptom.

### **3) The psychodynamic approach**

Fusegating on Charcot's lack of theorization and his non-recognition of the suggestion, Freud's work will be more retained by history. Freud treats hysterical patients with hypnotic

suggestion and thus explores the psychological processes of seizures. Hystero-epilepsy is considered here as a hysterical attack and apprehended as a result of the cleavage of conscious content. Freud and Breuer consider the crisis as a dissociation of the content of consciousness, reflecting the return of an unconscious traumatic memory. This repressed reminiscence is heard through the study of the passionate attitudes of the great crisis, whose hallucinations often evoke those of dreams. The exploration of thoughts under hypnosis also makes it possible to return to the previous and causal memory of the crises (Freud & Breuer, 1892). If Freud did not write a Treatise on Hysteria, the Vienna Master theorize the paradigm: hysteria inscribed in the body a language to be deciphered. The attack translates the subject's fantasies through a motor expression "represented in the form of pantomime" (Freud, 1909). Thus, thanks to this model, the unconscious dimension of the psyche is addressed by the interpretation of somatic symptoms and dreamed thoughts, coupled with the analysis of free associations.

As a result of these psychopathological conceptions, decades passed before modern authors recovered from this strange phenomenon of "psychogenic convulsions".

## **II. NON-EPILEPTIC PSYCHOGENIC SEIZURES: RENEWED EDITORIAL INTEREST OVER THE PAST DECADE**

### **1) Definitions and clinical description**

Non-epileptic psychogenic seizures can be defined as paroxysmal manifestations clinically suggestive of epileptic seizures but related to unconscious psychogenic processes. However, this definition is far from being consensual. Indeed, some authors question the psychogenic nature of PNESs both in reference to a cognitive unconscious of information processing and to the unconscious referenced to psychoanalysis. Other research rejects the purely psychogenic aspect of seizures. The different conceptual definitions of "non-epileptic seizures" are expressed through the multiple names of what appears to be the same clinical phenomenon. Scull identifies about twenty signifiers that can correspond to the framework of the PNES (Scull, 1997). ICD-11 hoarded them as dissociative disorders (WHO, 2018), while DSM V categorized them as conversion disorders (APA, 2013). The term "pseudo-seizures" is used particularly frequently in the literature, whereas dummy disorder or epileptoid pathomagnetism as well as epileptic simulation are only very rarely diagnosed. However, the dimensions of facticity and simulation may be evoked, taking extraordinarily varied clinical expressions that can be associated with an authentic organic disease.

The current renewed interest in the international press on the topic of PNESs is the result of an awareness of the importance of the phenomenon both with epidemiological point of view and, in terms of additional costs for the healthcare system (Auxéméry, 2012). The average diagnostic time is seven years, during which an anticonvulsant is regularly prescribed as a barrier to diagnostic wandering and medical nomadism (Markus Reuber & Elger, 2003). The PNESs would concern 10 to 50% of adults and 5 to 20% of children consulting a specialized epilepsy centre (Auxéméry, 2011; S. R. Benbadis & Allen Hauser, 2000; Uldall, Alving, Hansen, Kibaek, & Buchholt, 2006). This phenomenon is thought to be the cause of 20 to 30% of "crises" resistant to anticonvulsant therapy (Ettinger & Kanner, 2001). Epileptic (EC) and psychogenic seizures are clinically close. They are also co-morbid in 20 to 40% of cases.

By studying 43 patients whose PNESs began before the age of 16, Lancman et al. found a family history of epilepsy in one-third of cases (Lancman, Asconapé, Graves, & Gibson, 1994). A female predominance is noted by the majority of studies with a sex ratio of one fifth. In prepubescent children, PNES occurs more frequently in boys. But beyond the age of 12, PNESs are more frequently the prerogative of adolescent girls, gradually approaching the sex ratio found in the adult population (Kotagal, Costa, Wyllie, & Wolgamuth, 2002).

In modern medical practice, the diagnosis of PNES is a diagnosis after the elimination of somatic and psychiatric pathologies. Paraclinical examinations are performed based on clinical call points. Cardiological and neurological investigations are the most frequent. Semiological elements such as eye opening, lateral tongue bite, urinary loss and fall injury are nondiscriminatory. Clinical observation of abnormal movements also does not establish a reliable differential, even if their fluctuating nature suggests a psychogenic origin. It is traditionally considered that resistance to eye opening does not plead in favour of organicity, whereas nocturnal seizures are essentially epileptic. However, all clinical elements can be found to be deficient. The majority of PNESs are excito-motor and give rise to a generalized tonic-clonic convulsive seizure. However, other PNESs may wrongly refer to partial epileptic seizures or even absences or contact breakdowns. Reuber and Elger define three main clinical frameworks: the rigid catatonic crisis, the absence and finally the excessive tremor of the limbs, trunk and head (Markus Reuber & Elger, 2003).

## **2) The key place of video-EEG**

The clinical diagnosis of PNES is particularly difficult. Most patients with the disorder misuse anticonvulsant therapy with no efficacy and frequent side effects (McBride, Shih, & Hirsch,

2002). The question of differential diagnosis is just as significant in acute pathology given the frequency of "psychogenic epileptic conditions" that are regularly hospitalized in intensive care settings before being corrected (Holtkamp, Othman, Buchheim, & Meierkord, 2006).

Unanimously in the literature, an electroencephalographic recording coupled with a video recording is the gold standard (Benbadis, 2006). Epilepsy is established on electroencephalographic criteria, but the absence of these criteria is not sufficient to confirm or disprove the presence of PNES. On the other hand, there are non-specific paroxysmal electroencephalographic activities in the healthy subjects outside any clinical EC or PNES. A non-seizure subject, an epileptic patient and a hystero-epileptic patient may have intercritical abnormalities and only the critical EEG will allow differentiation. Epilepsy sensitization techniques such as hyperpnea or intermittent light stimulation may suggest PNES, even in an epileptic patient. Some authors have proposed seizure induction techniques by injecting a placebo that may induce stress induced EC or PNES in a suggestible patient (Baker, Brooks, Goodfellow, Bodde, & Aldenkamp, 2007). Unlike electrophysiology, no discriminant biological marker could be identified to establish a sensitive and specific differential (Willert, Spitzer, Kusserow, & Runge, 2004).

### **3) Etiopathogenic approach and therapeutic orientation**

Two main etiopathogenic orientations are mentioned in the literature. A psychological trauma as favouring dissociative disorders and a neurobiological origin that is far from the psychogen one.

PNESs are co-occurring with post-traumatic stress disorder and dissociative disorders

(Bowman & Markand, 1996; Dworetzky et al., 2005; Goldstein, Drew, Mellers, MitchellO'Malley, & Oakley, 2000). Seen as a dissociative disorder, seizures can be understood as reactions to psychological trauma. The latter takes the form of child abuse and particularly sexual violence (Akyuz, Kugu, Akyuz, & Dogan, 2004). PNEs are understood by some authors as an equivalent of traumatic repetition syndrome or recurrent dissociative disorder.

At the same time, other research teams are nuancing the psychogenic nature of seizures to reintegrate them into brain function. Thus, even minor head trauma is a risk factor for PNEs (Westbrook, Devinsky, & Geocadin, 1998). A neurobiological predisposition is suggested in the presence of electroencephalographic functional signs associated with structural abnormalities in neuroimaging. Devinski et al. evoke the role of the frontal cortex of the minor hemisphere (Devinsky, Mesad, & Alper, 2001) while Reuber et al. do not precisely determine a specific location (Reuber, Fernández, Helmstaedter, Qurishi, & Elger, 2002). Head-on epileptic seizures are not always detectable with EEG, they are in fact a very difficult differential paraclinical diagnosis. Further research is needed to refine the potential neurobiological determinants of PNEs.

The treatment of PNEs is complex. Some teams advocate diagnostic announcement by viewing the video recording to the patient, which is not necessarily of great interest in the psychodynamic sense as argued by other teams. Despite the diagnosis made on the subject and joint follow-up by a specialized team of neurologists and psychiatrists, the prognosis remains uncertain. Crises persist in many cases and even if they disappear, this element is not considered a factor for medical remission or better social rehabilitation.

### **III...FROM THE GREAT ATTACK TO THE PNES: CONTINUOUS OR DISCONTINUOUS?**

#### **1) A reference to "hysteria" is found over time**

From the École de la Salpêtrière to the present day, epileptic but not epileptic phenomena question the clinic and our theoretical references. There are many paradoxes whatever the time. The elders unanimously referred to hysteria. Hysteria was then both clinical evidence and an approximate, extensive and little theorized conceptual denomination. Epilepsy and Hysteria were considered according to the terminology of the time as "two neuroses" that differed from each other but whose etiological framework could follow a similar path (Charcot, 1892). Charcot sometimes considered hysteria from a neurological perspective and evoked some dynamic and temporary lesions of the nervous system, similar to epilepsy. The Treatises of Charcot's students are essentially descriptive and confine themselves to showing signs and symptoms observed by the doctor through the verb or sketch. This trend towards description is still palpable today through the desire to subdivide the PNESs into different subtypes. Betts differentiates three major syndromic executives. The pamoison with sudden fall, lack of response to external stimuli and rapid regaining of consciousness; the tantrum with fall and violent movements of the feet and arms contemporary with cries; the abreactive attack combining initial hyperventilation, fall and disordered movements or rigidity of the limbs and pelvis (Betts & Boden, 1992). In their famous literature review, Reuber and Elger distinguished excessive tremor of limbs, trunk and head; rigid catatonic seizure; absence with contact rupture (Markus Reuber & Elger, 2003). Prepubescent subjects are mostly "absent"

compared to postpubescent patients whose seizures are essentially motor (Verrotti et al., 2009). In a remarkable neurology thesis defended in Nancy, Hubsch found 7 PNES profiles by studying 145 videoEEG episodes (Hubsch, 2009). The main clinical forms are seizures with automatism and dystonia, sensitive paucikinetic seizures with preserved contact, convulsive seizures-like syncope seizures, prolonged fluctuating seizures with hyperventilation preceded by prodromes, and prolonged "large seizures" with axial tonic manifestations. However, clinical and paraclinical descriptions are rarely linked to etiopathogenic hypotheses.

Long after Babinski and his pithiatism, the École de Saint-Louis opposes a new dismemberment of hysteria to promote the genesis of quantitative psychiatric studies with a high degree of scientificity (Perley & Guze, 1962). To characterize the crises, however, a reference to hysterical terminology persisted until the early 1980s (Stewart, Lovitt, & Stewart, 1982; Williams, Spiegel, & Mostofsky, 1978). Psychic manifestations are now grouped within dissociative disorders that dissociate from somatic manifestations collected in somatoform disorders. This rupture of the classical unit is reflected in the phenomenon of PNESs by a paradox: the DSM-IV-TR collected them within somatoform disorders (APA, 2003), while the ICD-10 hoarded them in the category of dissociative disorders (WHO, 1993). Marchetti et al. propose the term "dissociative-conversion" to characterize PNESs, thus returning to unity (Marchetti et al., 2008).

2) From one time to another, the diagnosis of epileptic seizures is a diagnosis of clinical and paraclinical elimination

Babinski developed the sense of the neurological clinic in order not to wrongly attribute hysteria to well-somatic diseases. A subtle clinical examination must make the difference at a time when paraclinical exploration was rare. Babinski particularly detailed the semiological differences between hemiplegia of somatic origin and hysterical or pithiatic hemiplegia (Babinski, 1909).

His medical approach of challenging for hysteria, somatic pathologies such as simulations, is still relevant.

But unlike in the days of the Salpêtrière, it is a paraclinical examination that will in many cases guide the difference between PNES and epilepsy. The development of the technique used for the benefit of science makes it possible to refine diagnoses and reduce errors. However, the use of additional examinations sometimes returns to the uncertainty of the past because not all hyperexcitable neuronal discharges are necessarily visible at the EEG. The acquisition of the electrical signal is difficult in certain brain areas, including the frontal lobe. Neurophysiological exploration has limitations that will be pushed back by functional brain imaging explorations.

The logic of the diagnosis of elimination is quite understandable in the face of the need to prevent the known complications of a serious pathology. However, there are also limitations to this design. If it is legitimate to carry out certain additional examinations, it is inappropriate to establish a pseudo exhaustive logic resulting in medical nomadism.

#### **IV.... DISCUSSION: A PSYCHOTRAUMATIC DIMENSION FOUND FROM THE**

## **PAST TO THE PRESENT**

Without theorizing it, Landouzy held sexual and emotional relationships as the determining causes of hystero-epilepsy. Freud understood the hysterical crisis as understandable in reference not to the brain but to the psychic system: the critical symptom takes shape in the body as a metaphor for a hidden meaning. The crisis is the temporary resolution of an unconscious fantasy. The study of dreams echoed in the fourth phase of the great attack may help to give meaning to the symptom. Psychic trauma is by its nature an unspeakable experience that is frequently masked by other grievances. The reluctance of patients to abandon neurological consultation for that of the psychiatrist limits the psychopathological approach.

A multidisciplinary centre jointly associating neurologists and psychiatrists allows new research to be carried out. Fleischer et al. compare 31 PNEs (some of which are mixed) to 32 refractory CEs using psychometric scales. This study observes a post-traumatic profile in subjects with NSCLC who have more dissociative symptoms, post-traumatic stress disorder (PTSD) and psychotraumatic history compared to epileptic patients (Fleisher et al., 2002).

Similarly, there is more sexual and physical abuse (Alper, Devinsky, Perrine, Vazquez, & Luciano, 1993; Kuyk, Swinkels, & Spinhoven, 2003). Traumatic lifetime exposure of nonepileptic convulsive subjects is 80% to 100% (Bowman & Markand, 1996; Rosenberg, Rosenberg, Rosenberg, Williamson, & Wolford, 2000). Clinical translation is a lifetime PTSD rate of 50-100% in these same subjects (Bowman & Markand, 1996; Rosenberg et al., 2000).

Incriminated trauma is frequently child abuse and sexual violence (Bowman & Markand, 1996; Shen, Bowman, & Markand, 1990). Cross-referencing these epidemiological data, Dikel et al. argue for a chronology between psychological trauma and PNEs without,

however, retaining any direct causal evidence (Dikel, Fennell, & Gilmore, 2003). The onset of PNES may coincide with acute stress disorder (Bowman & Markand, 1996; Drake, Pakalnis, & Phillips, 1992). At the same time, other studies find that childhood trauma history increases dissociative disorders and PTSD (C. R. Brewin, Andrews, & Valentine, 2000; Keane, Kaufman, & Kimble, 2001) and thus, the development of PNES (Hingray et al., 2017). The reference to "dissociation" is understood here as the alteration and disjunction of normally integrated functions of consciousness (memory, identity, perception of the environment). Peritraumatic dissociation and acute stress disorder are predictive of subsequent post-traumatic stress in prospective studies (Ehlers, Mayou, & Bryant, 1998; Ozer, Best, Lipsey, & Weiss, 2003). It would be necessary to study with a dimensional perspective the evolution of the symptoms presented in the immediacy of psychotrauma in relation to their evolution towards traumatic repetitive stress disorder or possible dissociative resurgences. Some research has begun an attempt to read a subtype of PNES (motor versus absence) based on previous traumatic dimensions (dissociations versus repetitions) and their intertwined psychiatric comorbidities (Abubakr, Kablinger, & Caldito, 2003). Other authors consider dissociative elements as a dimension of PTSD, or even as a sign of its severity (Spiegel, Classen, & Cardena, 2000). Two subtypes of PTSD then emerge, one of which is characterized by a pre-eminence of repetitions and the other by its dissociative dimension. The latter would be more severe (Bryant, Marosszeky, Crooks, & Gurka, 2000; Marshall, Spitzer, & Liebowitz, 1999). Fiszman et al. consider PNESs as the atypical expression of PTSD illustrating a dissociative dimension of the condition (Fiszman, Alves-Leon, Nunes, D'Andrea, & Figueira, 2004). A continuity solution can thus develop between peritraumatic and post-traumatic dissociation. PTSD then manifests itself as recurrent dissociative episodes that take the form of seizures (Briere, Scott, & Weathers, 2005). The term "posttraumatic dissociative reactions" reflects this dimension (C. R. Brewin et al., 2000; Chris R. Brewin,

Andrews, & Rose, 2003). The main hypothesis is that during a trauma, the subject disappears from reality at the time of the events. Later, rather than repeating the traumatic scene through his sensations, he will be able to relive these "eclipses" or moments of dissociation. The psychogenic crisis may look like a mime to what was experienced during the initial trauma when the subject may have been a "shaken being", literally and figuratively. Repetition then takes the voice of dissociation (Auxéméry & Fidelle, 2011).

#### **V. Clinical example: Tom's psychogenic crises embody an ancient trauma.**

Tom, a young man of 16 years old, is referred for psychotherapy in the face of the emergence of PNES, a diagnosis validated by the video-EEG. "I have two illnesses: a SturgeWeber and something else that I won't be able to tell you," he confides at the first consultation. Tom has a history of Sturge-Weber-Krabbe disease, which was diagnosed at the age of six when he had his first coma, with progressively regressive right hemiplegia (a congenital condition manifested by capillary malformations that can lead to various skin and neurological symptoms). In Tom's case, several epileptic seizures are objectified from the early stages of disease activity and reported to bilateral angiodysplasia by neuroimaging investigations. An anticonvulsant treatment was prescribed at the time in order to suppress secondarily generalised partial comital seizures. This treatment is quickly effective to the point of stopping the attacks. Since then Tom has been receiving an annual MRI scan injected in a reference centre as part of a systematic follow-up that does not reveal any particular concern. The prognosis appeared to be good in the long term as both the intracranial vascular malformation and the epilepsy seemed to have stabilised . However, Tom was readmitted to hospital in early adolescence for "partial convulsive manifestations" taking multiple clinical forms, sometimes productive, sometimes deficient. The resurgence of partial epilepsy is of course first mentioned. However, despite the increase in the dosage of anticonvulsant

medication, the evolution is still marked by a continuation and even an intensification of the 'seizures'. Intercritical EEGs will remain noncontributory: only the video-EEG recording concludes to the non-comitial character of these "new" crises which persist. These paroxysmal motor - we could also say behavioural - events seem to be made even more anxious by the reaction of those around them, which perhaps contributes to their perpetuation. A mode of communication seems to be gradually taking hold between Tom and his family and friends, which passes through motor crises, excluding the possibility of verbalisations and mentalisations.

The psychotherapist conscientiously resumes the anamnesis far removed from somatic considerations. The new seizures initially took the mask of atypical partial epilepsy combining pain in the left leg and paraparesis in the right arm, without any neurological systematisation. Tom remembered his symptoms well, often at night during sleepwalking episodes or "waking nightmares", as he says: "it's my leg that gives out [...] I fall in front of my parents' bedroom [...] my left arm or my left leg, always at night, I get up to go to the toilet, I always fall in front of my parents' door". During the course of his discomfort, Tom reports a feeling of 'déjà vu or déjà vécu', a term borrowed from the classic vocabulary of epileptic semeiology, which may have led him to think of temporal epilepsy. A victim of sexual assault at a young age, Tom actually suffers from nightmares of traumatic repetition followed by acute manifestations of dissociation and anxiety. Daytime flashbacks are also present, as well as hypervigilance coupled with startle reactions. Posttraumatic stress disorder was probably triggered by Tom's relationship with his girlfriend, but until then it had been "masked" by the PNES'. The psychotherapeutic support allowed the traumatic event to be reintegrated into Tom's life trajectory, the "crises" gave way to the construction of a quality relationship with his girlfriend.

## **VI. Conclusion**

We have seen here the links we can make between hystero-epilepsy and non-epileptic psychogenic crisis through the work of Charcot, Freud or Breuer and current psychiatry. The traumatic history and dissociative disorders of subjects with PNES seem to play an important role in the genesis of this pathology. However, there is very strong inter-individual variability in the psychological and psychiatric consequences following a potentially traumatic event (Houllé, Silès, & Tarquinio, 2017). What if the PNES' were not just a pathology in their own right but a specific expression of the psychotrauma(s) of the subject and its dissociative disorders? The example of Tom is revealing: the interviews conducted led the psychotherapist to look for a traumatic origin to his disorders. The sexual violences that Tom experienced when he was younger probably led him to develop a complex psychotrauma, and the PNES' then became a somatoform dissociative symptom. After treatment, these crises disappeared, leaving him to develop in a healthier relationship. It would appear that, for patients with a history of repetitive traumas, PNES' are only the most visible part of a much more complex symptomatology (Hingray et al., 2017). This confirms once again that psychotrauma does not have a single clinical picture as PTSD might suggest, but rather a wide range of expressions, from hypervigilance to dissociative symptoms, including eating or addictive behavioural disorders. It is important to take into consideration these multitude of possibilities in order to improve the management of PNES' (and of all psychopathologies and somatoform disorders) by doctors and psychotherapists. This is a major theoretical opening in the treatment of these pathologies. In the light of these remarks, what we thought was incurable or difficult to treat a few years ago (schizophrenia, fibromyalgia...) could finally find a solution. Moreover n event is traumatic for an individual depending on the

meaning he gives to this unspeakable and the possible limits (often cultural such as the confrontation with death) at his disposal. What if the development of PNES' was also linked to an intercultural dimension? These elements may seem pleasant, however, as Dr. Hingray (2017) pointed out, not all people with PNES' necessarily had a traumatic history, it does mean there are still factors to be studied such as intrauterine development or simply the child's brain development. While tempting, psychotrauma cannot and will not be able to explain everything.

**CONFLICT OF INTEREST:** none. The ideas and positions expressed in this work are those of the author and should not be considered as the official point of view of the French Army Health Service.

## REFERENCES

- Abubakr, A., Kablinger, A., & Caldito, G. (2003). Psychogenic seizures: clinical features and psychological analysis. *Epilepsy & Behavior: E&B*, 4(3), 241-245.
- Akyuz, G., Kugu, N., Akyuz, A., & Dogan, O. (2004). Dissociation and childhood abuse history in epileptic and pseudoseizure patients. *Epileptic Disorders: International Epilepsy Journal with Videotape*, 6(3), 187-192.
- Alper, K., Devinsky, O., Perrine, K., Vazquez, B., & Luciano, D. (1993). Nonepileptic seizures and childhood sexual and physical abuse. *Neurology*, 43(10), 1950-1953.
- APA. (2003). *DSM-IV TR - Manuel diagnostique et statistique des troubles mentaux* (2e éd.; A. P. Publishing, Éd.). Issy-les-Moulineaux: Editions Masson.
- Association, A. P. (2013). *Diagnostic and Statistical Manual of Mental Disorders: Dsm-5* (5th Revised edition). American Psychiatric Publishing.
- Auxéméry, Y. (2011). Crises psychogènes non épileptiques chez l'enfant et l'adolescent. *Neuropsychiatrie de l'Enfance et de l'Adolescence*, 59(1), 61-67. <https://doi.org/10.1016/j.neurenf.2010.07.015>
- Auxéméry, Y. (2012). De l'hystéro-épilepsie à la crise psychogène non épileptique : continuité ou discontinuité ? [/data/revues/00034487/v170i9/S000344871100062X/](https://www.em-consulte.com/en/article/767611). Consulté à l'adresse <https://www.em-consulte.com/en/article/767611>

Auxéméry, Y., & Fidelle, G. (2011). Les crises psychogènes non épileptiques : une dimension psychotraumatique ? *REVUE FRANCOPHONE DU STRESS ET DU TRAUMA*, 1 vol 11, 5-11.

Babinski, J. (1909). *Démembrement de l'hystérie traditionnelle : pithiatisme*. Paris: Hachette Livre BNF.

Baker, G. A., Brooks, J. L., Goodfellow, L., Bodde, N., & Aldenkamp, A. (2007). Treatments for non-epileptic attack disorder. *The Cochrane Database of Systematic Reviews*, (1), CD006370. <https://doi.org/10.1002/14651858.CD006370>

Benbadis, S. (2006). The EEG in nonepileptic seizures. *Journal of Clinical Neurophysiology: Official Publication of the American Electroencephalographic Society*, 23(4), 340-352. <https://doi.org/10.1097/01.wnp.0000228863.92618.cf>

Benbadis, S. R., & Allen Hauser, W. (2000). An estimate of the prevalence of psychogenic nonepileptic seizures. *Seizure*, 9(4), 280-281. <https://doi.org/10.1053/seiz.2000.0409>

Bernheim, H. (1913). *L'hystérie : définition et conception, pathogénie, traitement*. Consulté à l'adresse <https://gallica.bnf.fr/ark:/12148/bpt6k65523f>

Betts, T., & Boden, S. (1992). Diagnosis, management and prognosis of a group of 128 patients with non-epileptic attack disorder. Part I. *Seizure*, 1(1), 19-26.

Bowman, E. S., & Markand, O. N. (1996). Psychodynamics and psychiatric diagnoses of pseudoseizure subjects. *The American Journal of Psychiatry*, 153(1), 57-63. <https://doi.org/10.1176/ajp.153.1.57>

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, 68(5), 748-766.

Brewin, Chris R., Andrews, B., & Rose, S. (2003). Diagnostic overlap between acute stress disorder and PTSD in victims of violent crime. *The American Journal of Psychiatry*, 160(4), 783-785. <https://doi.org/10.1176/appi.ajp.160.4.783>

Briere, J., Scott, C., & Weathers, F. (2005). Peritraumatic and persistent dissociation in the presumed etiology of PTSD. *The American Journal of Psychiatry*, 162(12), 2295-2301. <https://doi.org/10.1176/appi.ajp.162.12.2295>

Briquet, P. (1859). *Traité clinique et thérapeutique de l'hystérie*. Consulté à l'adresse <https://gallica.bnf.fr/ark:/12148/bpt6k10251330>

Bryant, R. A., Marosszeky, J. E., Crooks, J., & Gurka, J. A. (2000). Posttraumatic stress disorder after severe traumatic brain injury. *The American Journal of Psychiatry*, 157(4), 629-631. <https://doi.org/10.1176/appi.ajp.157.4.629>

Charcot, J.-M. (1892). *Leçons sur les maladies du système nerveux recueillies et publiées par Bourneville*. Consulté à l'adresse <https://gallica.bnf.fr/ark:/12148/bpt6k98763k> de la Tourette,

G. (1895). *Traité clinique et thérapeutique de l'hystérie. Seconde partie : hystérie paroxystique*. Consulté à l'adresse <https://gallica.bnf.fr/ark:/12148/bpt6k76944z>

- Devinsky, O., Mesad, S., & Alper, K. (2001). Nondominant hemisphere lesions and conversion nonepileptic seizures. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 13(3), 367-373. <https://doi.org/10.1176/jnp.13.3.367>
- Dikel, T. N., Fennell, E. B., & Gilmore, R. L. (2003). Posttraumatic stress disorder, dissociation, and sexual abuse history in epileptic and nonepileptic seizure patients. *Epilepsy & Behavior: E&B*, 4(6), 644-650.
- Drake, M. E., Pakalnis, A., & Phillips, B. B. (1992). Neuropsychological and psychiatric correlates of intractable pseudoseizures. *Seizure*, 1(1), 11-13.
- Dworetzky, B. A., Strahonja-Packard, A., Shanahan, C. W., Paz, J., Schauble, B., & Bromfield, E. B. (2005). Characteristics of male veterans with psychogenic nonepileptic seizures. *Epilepsia*, 46(9), 1418-1422. <https://doi.org/10.1111/j.1528-1167.2005.13004.x>
- Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology*, 107(3), 508-519.
- Ettinger, A., & Kanner, A. (2001). *Psychiatric Issues in Epilepsy: A Practical Guide to Diagnosis and Treatment*. Philadelphia, PA: Lippincott W and Wilkins Publishers.
- Fizman, A., Alves-Leon, S. V., Nunes, R. G., D'Andrea, I., & Figueira, I. (2004). Traumatic events and posttraumatic stress disorder in patients with psychogenic nonepileptic seizures: a critical review. *Epilepsy & Behavior: E&B*, 5(6), 818-825. <https://doi.org/10.1016/j.yebeh.2004.09.002>
- Fleisher, W., Staley, D., Krawetz, P., Pillay, N., Arnett, J. L., & Maher, J. (2002). Comparative study of trauma-related phenomena in subjects with pseudoseizures and subjects with epilepsy. *The American Journal of Psychiatry*, 159(4), 660-663. <https://doi.org/10.1176/appi.ajp.159.4.660>
- Freud, S. (1909). Considérations générales sur l'attaque hystérique. In *Névrose, Psychose et Perversion* (p. 161-165). Paris: PUF.
- Freud, S., & Breuer, J. (1892). Pour une théorie de l'attaque hystérique. In *Résultats, Idées, Problèmes. Tome I: 1890-1920* (p. 25-28). Paris: PUF.
- Goldstein, L. H., Drew, C., Mellers, J., Mitchell-O'Malley, S., & Oakley, D. A. (2000). Dissociation, hypnotizability, coping styles and health locus of control: characteristics of pseudoseizure patients. *Seizure*, 9(5), 314-322. <https://doi.org/10.1053/seiz.2000.0421>
- Hingray, C., Donne, C., Cohn, A., Maillard, L., Schwan, R., Montel, S., & El-Hage, W. (2017). Link between psychogenic nonepileptic seizures and complex PTSD: A pilot study. </data/revues/24687499/v1i2/S2468749917300066/>. Consulté à l'adresse <https://www.emconsulte.com/en/article/1123851>

Holtkamp, M., Othman, J., Buchheim, K., & Meierkord, H. (2006). Diagnosis of psychogenic nonepileptic status epilepticus in the emergency setting. *Neurology*, *66*(11), 1727-1729. <https://doi.org/10.1212/01.wnl.0000218299.15988.9d>

Houllé, W. A., Silès, J., Tarquinio, P., & Tarquinio, C. (2017). Trauma et culture : Influence des facteurs culturels dans la rencontre traumatique et perspectives psychothérapeutiques. *European Journal of Trauma & Dissociation*, *1*(2), 121-129. <https://doi.org/10.1016/j.ejtd.2017.03.008>

Hubsch, C. (2009). *Crises psychogènes non épileptiques. Analyse de 145 crises à partir d'une cohorte de 52 patients* (Thèse de Médecine). Université de Nancy, Nancy.

Keane, T., Kaufman, M., & Kimble, M. (2001). Peritraumatic dissociative symptoms, acute stress disorder, and the development of posttraumatic stress disorder: causation, correlation or epiphenomena. In L. Sanchez-Planell & C. Diez-Quevedo, *Dissociative states* (p. 21-43). Barcelone: Springer-verlag.

Kotagal, P., Costa, M., Wyllie, E., & Wolgamuth, B. (2002). Paroxysmal nonepileptic events in children and adolescents. *Pediatrics*, *110*(4), e46.

Kuyk, J., Swinkels, W. a. M., & Spinhoven, P. (2003). Psychopathologies in patients with nonepileptic seizures with and without comorbid epilepsy: how different are they? *Epilepsy & Behavior: E&B*, *4*(1), 13-18.

Lancman, M. E., Asconapé, J. J., Graves, S., & Gibson, P. A. (1994). Psychogenic seizures in children: long-term analysis of 43 cases. *Journal of Child Neurology*, *9*(4), 404-407. <https://doi.org/10.1177/088307389400900413>

Landouzy, H. (1846). *Traité complet de l'hystérie*. Consulté à l'adresse <https://gallica.bnf.fr/ark:/12148/bpt6k76936b>

Louyer-Villermay, J. (1818). Article « Hystérie ». In *Dictionnaire des sciences médicales* (Vol. 23, p. 226-272). Paris: Panckoucke.

Marchetti, R. L., Kurcgant, D., Neto, J. G., von Bismark, M. A., Marchetti, L. B., & Fiore, L. A. (2008). Psychiatric diagnoses of patients with psychogenic non-epileptic seizures. *Seizure*, *17*(3), 247-253. <https://doi.org/10.1016/j.seizure.2007.07.006>

Marshall, R. D., Spitzer, R., & Liebowitz, M. R. (1999). Review and critique of the new DSM-IV diagnosis of acute stress disorder. *The American Journal of Psychiatry*, *156*(11), 1677-1685. <https://doi.org/10.1176/ajp.156.11.1677>

McBride, A. E., Shih, T. T., & Hirsch, L. J. (2002). Video-EEG monitoring in the elderly: a review of 94 patients. *Epilepsia*, *43*(2), 165-169.

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychological Bulletin*, *129*(1), 52-73.

- Perley, M. J., & Guze, S. B. (1962). Hysteria--the stability and usefulness of clinical criteria. A quantitative study based on a follow-up period of six to eight years in 39 patients. *The New England Journal of Medicine*, 266, 421-426. <https://doi.org/10.1056/NEJM196203012660901>
- Reuber, M., Fernández, G., Helmstaedter, C., Qurishi, A., & Elger, C. E. (2002). Evidence of brain abnormality in patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior: E&B*, 3(3), 249-254.
- Reuber, Markus, & Elger, C. E. (2003). Psychogenic nonepileptic seizures: review and update. *Epilepsy & Behavior: E&B*, 4(3), 205-216.
- Richer, P. (1885). *Etudes cliniques sur la grande hystérie ou hystéro-épilepsie*. Consulté à l'adresse <https://gallica.bnf.fr/ark:/12148/bpt6k76939c>
- Rosenberg, H. J., Rosenberg, S. D., Williamson, P. D., & Wolford, G. L. (2000). A comparative study of trauma and posttraumatic stress disorder prevalence in epilepsy patients and psychogenic nonepileptic seizure patients. *Epilepsia*, 41(4), 447-452.
- Scull, D. A. (1997). Pseudoseizures or non-epileptic seizures (NES); 15 synonyms. *Journal of Neurology, Neurosurgery, and Psychiatry*, 62(2), 200.
- Shen, W., Bowman, E. S., & Markand, O. N. (1990). Presenting the diagnosis of pseudoseizure. *Neurology*, 40(5), 756-759.
- Spiegel, D., Classen, C., & Cardena, E. (2000). New DSM-IV diagnosis of acute stress disorder. *The American Journal of Psychiatry*, 157(11), 1890-1891. <https://doi.org/10.1176/appi.ajp.157.11.1890>
- Stewart, R. S., Lovitt, R., & Stewart, M. (1982). Are hysterical seizures more than hysteria? A research diagnostic criteria, DSM-III, and psychometric analysis. *The American Journal of Psychiatry*, 139(7), 926-929. <https://doi.org/10.1176/ajp.139.7.926>
- Uldall, P., Alving, J., Hansen, L. K., Kibaek, M., & Buchholt, J. (2006). The misdiagnosis of epilepsy in children admitted to a tertiary epilepsy centre with paroxysmal events. *Archives of Disease in Childhood*, 91(3), 219-221. <https://doi.org/10.1136/adc.2004.064477>
- Verrotti, A., Agostinelli, S., Mohn, A., Manco, R., Coppola, G., Franzoni, E., ... Curatolo, P. (2009). Clinical features of psychogenic non-epileptic seizures in prepubertal and pubertal patients with idiopathic epilepsy. *Neurological Sciences: Official Journal of the Italian Neurological Society and of the Italian Society of Clinical Neurophysiology*, 30(4), 319-323. <https://doi.org/10.1007/s10072-009-0107-x>
- Westbrook, L. E., Devinsky, O., & Geocadin, R. (1998). Nonepileptic seizures after head injury. *Epilepsia*, 39(9), 978-982.
- WHO. (1993). *CIM-10/ICD-10 : Descriptions cliniques et directives pour le diagnostic*. Paris: Editions Masson.
- WHO. (2018). ICD-11 - Mortality and Morbidity Statistics. Consulté 2 avril 2019, à l'adresse <https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/1620688958>

Willert, C., Spitzer, C., Kusserow, S., & Runge, U. (2004). Serum neuron-specific enolase, prolactin, and creatine kinase after epileptic and psychogenic non-epileptic seizures. *Acta Neurologica Scandinavica*, *109*(5), 318-323. <https://doi.org/10.1046/j.16000404.2003.00232.x>

Williams, D. T., Spiegel, H., & Mostofsky, D. I. (1978). Neurogenic and hysterical seizures in children and adolescents: differential diagnostic and therapeutic considerations. *The American Journal of Psychiatry*, *135*(1), 82-86. <https://doi.org/10.1176/ajp.135.1.82>